Jurnal Pijar MIPA
https://jurnalfkip.unram.ac.id/index.php/JPM

ISSN 1907-1744 (Print)
ISSN 2460-1500 (Online)

Effect of Giving Ethanol Extract of *Keji Beling* (*Strobilanthes crispa L. Blume*) Leaves on Histology and Liver Physiology in White Rats (*Rattus norvegicus* L.) Induced by Sodium Benzoate

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Received: April 15, 2026. Accepted: August 8, 2026. Published: August 31, 2026

Abstract. Sodium benzoate is one of the most widely used food preservatives in the food and beverage industry due to its effectiveness in inhibiting microbial growth. However, excessive and prolonged consumption of sodium benzoate may induce adverse health effects, including gastrointestinal disturbances, neurological disorders, and liver damage resulting from oxidative stress. Natural medicinal plants with antioxidant properties have attracted considerable attention as potential therapeutic agents against chemical-induced organ toxicity. *Keji Beling* leaves (*Strobilanthes crispa* L. Blume) are traditionally utilized in Indonesia for various medicinal purposes and are known to contain bioactive compounds such as flavonoids, polyphenols, tannins, and saponins that exhibit hepatoprotective activity. This study aimed to evaluate the effect of ethanol extract of *keji beling* leaves on liver histology and liver physiological function, as indicated by serum glutamate pyruvate transaminase (SGPT) and serum glutamate oxaloacetate transaminase (SGOT) levels, in sodium benzoate-induced white rats (*Rattus norvegicus* L.). The experiment employed a Completely Randomised Design with five treatment groups at different extract dosages. Data were analyzed using one-way analysis of variance (ANOVA), followed by Duncan's Multiple Range Test at a 95% confidence level. The results demonstrated that sodium benzoate administration significantly induced liver histopathological alterations characterized by cellular degeneration, inflammation, and necrosis, accompanied by elevated SGPT and SGOT levels. Treatment with the ethanol extract of *keji beling* leaves significantly improved liver histology and reduced liver enzyme levels compared with the sodium benzoate-induced group. The hepatoprotective effect was dose-dependent, with the dose of 500 mg/kg body weight showing the most effective recovery of liver tissue and normalization of SGPT and SGOT levels. These findings suggest that the ethanol extract of *Strobilanthes crispa* leaves has promising potential as a natural hepatoprotective agent against sodium benzoate-induced liver injury.

Keywords: Liver; Sodium Benzoate; SGOT; SGPT; Vile Shard Leaves.

Introduction

Preservatives are food additives that are usually used to prevent or inhibit fermentation, acidification, or other microbial decomposition of food [1]. Preservatives are usually used to protect food that is easily damaged, maintain its nutritional value, and extend its shelf life [2]. Widely used preservatives on the market are benzoates, such as sodium benzoate, because they are more soluble than other forms. Sodium benzoate is a water-soluble salt derived from benzoic acid. It acts as a preservative and antimicrobial agent in environments with a pH level between 2 and 4. Commonly used as a preservative in food, pharmaceutical products, & cosmetics.

Cosmetic preparations may contain additional preservatives, but the maximum permitted is 0.5% [3]. In addition to its use in cosmetics, sodium benzoate is also used in the production of soft drinks, fruit foods, pickles, and sauces. Giving food preservatives such as sodium benzoate at inappropriate doses can cause organ damage, especially the liver [4].

The liver is a vital organ in the human body. The liver has a role in the intermediary metabolism of all food components. The liver is the primary site of synthesis, breakdown, and detoxification. inside the body. Apart from that, the liver also plays a role in removing blood pigment. The liver is very susceptible to the effects of food preservatives and dangerous chemicals, such as sodium benzoate. Liver damage is caused by several factors, including viral infections, alcohol consumption, and use of certain drugs (isoniazid, tetracycline, and aspirin) [5]-[6]. Apart from affecting liver histology, it will also affect the levels of Serum Glutamic Pyruvic Transaminase (SGPT) & Serum Glutamic Oxaloacetate Transaminase (SGOT) due to liver histology damage in mice, resulting in increased levels of liver enzymes [7].

When liver cells or enzymes are disrupted or overproduced, the liver's ability to function is impaired. Therefore, treatment is necessary to improve liver function, and one approach is traditional medicine [8]. Keji Shards are a natural ingredient commonly used in traditional herbal medicine. Keji shard leaves (Strobilanthes crispa L. Blume) contain various substances, such as potassium, calcium, sodium, silicic acid, alkaloids, saponins, polyphenols and flavonoids [9]. The aim of this research is to determine the effect of giving ethanol leaf extract *keji beling* (*Strobilanthes crispa* L. Blume) on the morphology & histology of the liver of white rats (Rattus norvegicus L.) induced by sodium

How to Cite:
R. A. W. Dari, H. Febriani, and S. Syukriah, "Effect of Giving Ethanol Extract of Keji Beling (Strobilanthes crispa L. Blume) Leaves on Histology and Liver Physiology in White Rats (Rattus norvegicus L.) Induced by Sodium Benzoate", *J. Pijar.MIPA*, vol. 21, no. 4, pp. 848–852, Aug. 2026. https://doi.org/10.29303/jpm.v21i4.10679

benzoate, to determine the effect of administering ethanol extract of *keji beling* leaves (Strobilanthes crispus L.) on the SGPT & SGOT levels of the liver of white rats (Rattus norvegicus L.) induced by sodium benzoate [10]. The author intends to determine the hepatoprotective potential of ethanol extract derived from *keji beling* leaves (Strobilanthes crispa L. Blume) [11]. Specifically, this study investigated whether the extract could reduce liver damage and lower SGPT & SGOT levels induced by free radicals generated by sodium benzoate exposure [12].

Research Methods

This study employed a laboratory experimental design with male Wistar rats (Rattus norvegicus L.) as subjects. The research aimed to evaluate the hepatoprotective effect of ethanol extract of *keji beling* leaves (*Strobilanthes crispa* L. Blume) on liver damage induced by sodium benzoate. The experimental animals were randomly divided into several groups, including a normal control group, a sodium benzoate group, and treatment groups receiving ethanol extract at different doses. Sodium benzoate and plant extract were administered orally via a gastric probe at the predetermined dosage.

The research procedure began with preparing *keji beling* leaf extract with ethanol via maceration, followed by evaporation on a rotary evaporator to obtain a concentrated extract. Experimental rats were acclimatized for several days prior to treatment and maintained under standard laboratory conditions with adequate food and water. Sodium benzoate was administered to induce liver damage, while treatment groups received different doses of ethanol extract to evaluate its therapeutic effect [13].

After the treatment period, the rats were sacrificed, and liver tissues were collected for histopathological examination. The liver samples were fixed in 10% buffered formalin, dehydrated in graded alcohol solutions, cleared in xylol, embedded in paraffin, and sectioned on a microtome. The tissue sections were then stained with hematoxylin-eosin (H&E) to observe histological changes, including parenchymal degeneration, hydropic degeneration, and necrosis, under a light microscope.

Blood samples were also collected to measure biochemical parameters, particularly SGOT and SGPT enzyme levels, using a haematology analyzer. The obtained data were analyzed quantitatively and presented as mean ± standard deviation. Statistical analysis was conducted to determine significant differences between treatment groups. This research procedure adopted and modified previously established experimental methods for evaluating hepatoprotective activity in laboratory animals, as described in earlier studies.

Results and Discussion

Liver damage caused by exposure to chemical compounds can be identified through histopathological observations and biochemical parameters in blood serum. Sodium benzoate, commonly used as a food preservative, can induce oxidative stress and hepatocellular injury at high doses. Therefore, evaluation of liver tissue structure and liver enzyme activity is essential for determining the extent of hepatotoxic effects and the potential protective role of natural compounds. In this study, histological changes in liver cells and the levels of liver enzymes were analyzed to assess the impact of sodium benzoate administration and the therapeutic potential of ethanol extract from *keji beling* leaves.

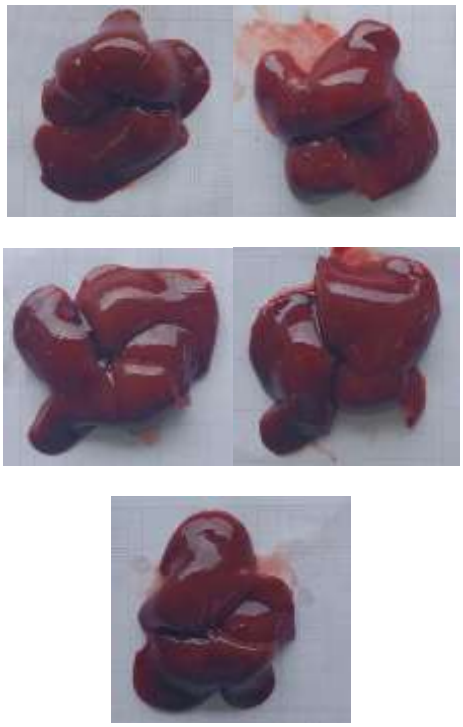

Figure 1. Histological picture of rat liver after administration of ethanol extract of *keji beling* leaves and sodium benzoate.

Table 1. Average value of the number of degenerations. hydropic, necrosis and degeneration. parenchymatous.

Normal	Degenerasi Parenkimatosa	Degenerasi Hidropik	Nekrosis
Normal group	4.50±1.00^{a}	7.50±1.73^{a}	11.00±2.00^{a}
Group Natrium Benzoat	9.50±1.00^{b}	14.25±1.50^{c}	20.00±0.00^{c}
Treat. 1	9.00±1.15^{b}	13.50±1.73^{bc}	18.00±4.00^{bc}
Treat. 2	7.50±2.51^{b}	10.50±3.00^{ab}	17.00±3.83^{bc}
Treat. 3	5.00±1.15^{a}	8.25±2.87^{a}	14.00±2.30^{ab}
p=value	0.001	0.002	0.004

Based on the data in Table 1, the extent of liver damage, particularly parenchymal degeneration, hydropic degeneration, and necrosis, differed markedly between the normal group and the group treated with sodium benzoate at doses P1 (300 mg per kg) and P2 (400 mg per kg). Treatment 3 (P3) dose (500 mg per kg) showed a large impact on the control group. These findings indicate that the ethanol extract from *keji beling* leaves has a therapeutic effect on liver cell injury induced by sodium benzoate. Increasing the dose resulted in more pronounced adverse effects on liver cells in the group. Treatments 1, 2, and 3 were compared with the sodium benzoate group.

The SGPT enzyme, which is synthesized in hepatocytes, shows greater specificity for liver diseases compared to other enzymes. SGPT is mostly localized in the liver, whereas its presence in the heart and skeletal muscle is

less common than that of SGOT. Elevated serum levels are mainly seen in cases of liver damage, in contrast to SGOT values. The SGPT enzyme catalyses the transfer of the amino group from alanine to α-ketoglutarate. The transaminase reaction produces two reversible products: pyruvate & glutamate. Serum SGPT levels are a very sensitive marker of liver damage due to the limited number of diseases that may affect these levels, other than liver-related problems [14]. SGOT is an enzyme that catalyses the reaction between aspartic acid & α-ketoglutaric acid. The heart contains a higher concentration of the SGOT enzyme than the liver. Enzymes are found in skeletal muscle, brain and kidneys. The range of SGOT levels in rat blood is generally 10-40 U/liter [13]. Increases rapidly when there are changes in myocardial infarction.

Table 2. Average value. SGPT & SGOT amount

Normal	SGPT (U/L)	SGOT (U/L)	p=value
Normal Group	44.75±4.64[a]	36.75±3.50[a]	
Natrium Benzoat Group	88.75±4.78[e]	120.50±7.04[d]	0.000
Treatment 1	76.50±1.91[d]	91.50±3.69[c]	
Treatment 2	63.25±1.70[c]	60.25±3.59[b]	
Treatment 3	53.00±2.16[b]	42.00±2.16[a]	

Table 2 showed that there were significant differences in SGPT and SGOT levels between the normal group (KN) and the sodium benzoate (KNB) group, as well as between treatment one (P1) dose (300 mg per kg BW) and treatment two (P2) dose (400 mg per kg). kg). Substantial effects were observed in the normal group (KN) when treated with 500 mg/kg (treatment 3; P3). Sodium benzoate can increase SGPT & SGOT levels in the bloodstream. Increased SGOT & SGPT levels may be due to hepatocellular injury caused by the administration of sodium benzoate. The increase in SGOT & SGPT levels is caused by excessive harmful substances in the body, which are then processed by cytochrome P450 enzymes in the liver, producing free radicals [15]. Furthermore, these unpaired electrons bind to hepatocytes in the liver, increasing liver membrane permeability. Liver damage can arise from oxidative stress mechanisms caused by increased formation of Reactive Oxygen Species (ROS) & decreased catalase antioxidant activity. This inflammatory process increases SGOT and SGPT levels [16].

Based on Figure 1, liver morphology analysis of mice in the KN group (Normal Group), KNB (Sodium Benzoate Group), and P1. (Treatment one), P2. (Treatment two), & P3 (Treatment three) showed no abnormalities. The liver does not have any hardening, the surface looks smooth & brownish in color. A healthy liver is characterized by an outer part that is flat and unblemished, and has a brownish-red hue [17]. On the other hand, an unhealthy liver has an uneven surface, is cyst-shaped, and experiences color variations. The distinctive heart exhibits a sleek exterior and tough texture. Visually, there were no visible variations in color or heart shape between the control group and the treatment group. The liver color seen in the control and treatment groups was brownish-red or dark red [18]. A healthy liver usually has a reddish-brown hue. This phenomenon is caused by the large volume of blood circulating through blood vessels [19]. A healthy liver usually has a reddish-brown hue due to increased blood flow to the organ. Based on research findings, no important changes were observed in the structure and appearance of rat liver after treatment with sodium benzoate and ethanol extract of *keji beling* leaves (Strobilanthes crispa L. Blume). These findings indicate that sodium benzoate and ethanol extracts derived from *keji beling* leaves (Strobilanthes crispa L. Blume) do not have a detrimental effect on the morphology, function or color of the liver of mice.

Table 1 shows that liver cell damage (necrosis) has the highest value among the damage types, followed by parenchymal degeneration and hydropic degeneration. A significant degree of necrosis was seen in the sodium benzoate group (20.00 ± 0.00c), indicating that sodium benzoate induced liver damage leading to cell death. Necrosis can arise from various factors, including inadequate blood flow, toxic substances, lack of nerve innervation, extreme temperatures, radiation exposure, and mechanical injury. Prolonged exposure to harmful agents will cause lobular necrosis. Necrosis begins with morphological changes in cell nuclei, particularly pyknosis. Next, the nucleus undergoes a rupture called karyohexis, which causes loss of the nucleus called karyolysis. Pyknosis can result from intracellular damage, such as damage to membranes, followed by damage to mitochondria and the Golgi apparatus, which disrupts the cell's ability to remove water and triglycerides, thereby causing their accumulation in the cytoplasm.

The amount of sodium benzoate that enters the liver is so large that it becomes toxic to the liver and causes degeneration of liver tissue. Next, necrosis occurs, potentially damaging liver tissue. It is important to continue taking precautions to reduce the dangers associated with excessive amounts of food preservatives. The presence of food preservatives in the bloodstream causes damage to many organs, such as the liver. This phenomenon is caused by the tendency of food preservatives to generate free radicals in the body, thereby reducing antioxidant capacity and leading to oxidative stress. Many studies have shown that food preservatives can directly disrupt normal biochemical processes in the hepatobiliary system, leading to liver cell necrosis. Post administration of food preservatives, important changes include hydropic degeneration, parenchymal degeneration, and necrosis. Parenchymal degeneration is a type of moderate degeneration characterized by swelling & turbidity in the cytoplasm. This degeneration is reversible because it occurs only in the mitochondria and endoplasmic reticulum and is caused by oxidative stress. Injured cells cannot excrete water, so water accumulates within the cells, causing swelling. Hydropic degeneration is a more severe form of destruction characterised by water-filled vacuoles in the cytoplasm that lack fat or glycogen. These changes occur from metabolic diseases, such as hypoxia/chemical poisoning. These setbacks can be overcome, but if the cause of the injury persists, then the damage may be irreversible. Furthermore, injured cells can sustain plasma membrane lacerations and nuclear changes.

Table 2 shows the normal group. (KN), Treatment three (P3) had significant results; there was no parenchymal degenerative damage. The normal control group (KN) showed marked differences from the sodium benzoate

(KNB), treatment one (P1), and treatment two (P2) groups, as evidenced by parenchymal degeneration, hydropic degeneration, and necrotic damage.

Parenchymal degeneration is a form of progressive hydropic degeneration that results in permanent damage. Parenchymal degeneration is a cellular reaction of hepatocytes to toxins that damage the fat metabolism system. Individual parenchymal constellation refers to a condition in which liver function is disrupted by the accumulation of fat in the cytoplasm of liver cells. Upon microscopic examination, small, translucent lipid droplets can be observed within the cells. This may result from ischemia, anemia, toxic chemicals, or excessive fat and protein intake. Hydropic degeneration refers to cell damage characterized by expansion of the cytoplasm. This phenomenon arises due to the disruption of active transport so that cells are unable to release Na^+ ions, causing an increase in the concentration of Na^+ ions in the cell. Cytoplasmic swelling increases significantly due to the cell's inability to maintain homeostasis [20].

Table 2 shows the significant impact of liver SGPT & SGOT levels in the normal group and in the 3-dose therapy (500 mg/kg). Increasing the extract dose decreases SGPT & SGOT levels. SGPT & SGOT levels in each treatment group gradually decreased and ultimately approached the values observed in the normal group. This is in line with the process of reducing histological damage obtained from the ethanol extract of *keji beling* leaves. Reducing SGPT & SGOT levels is achieved through the antioxidant activity of sodium benzoate, which effectively binds and neutralizes free radicals. Antioxidants inhibit the activity of free radicals, preventing them from causing damage to liver tissue and thereby disrupting the mechanism of liver damage. When cells remain undamaged, the concentrations of SGPT and SGOT in the bloodstream are lower because fewer cells are affected and their release into the blood is reduced.

Administration of *keji beling* leaf extract as an antioxidant resulted in significant changes compared to the control group, as evidenced by decreased SGPT and SGOT levels. The presence of flavonoids in the ethanol extract of *keji beling* leaves is responsible for its therapeutic effects. Flavonoid levels were determined at 40.2353 using the DPPH technique.

Flavonoids consist of aromatic chemicals that have antioxidant effects. Antioxidants can inhibit oxidation driven by free radicals, thereby forming unreactive molecules. Flavonoid molecules have the ability to ward off the harmful effects of free radicals [21]. Flavonoids function as antioxidants by providing hydrogen ions directly to ward off the negative effects of free radicals. Flavonoids function as antioxidants by indirectly stimulating the production of antioxidant genes. Endogenous in several ways. Activation of nuclear factor erythroid 2-related factor 2 (Nrf2) can increase antioxidant gene expression. This activation increases the expression of genes involved in the production of natural antioxidant enzymes, such as the SOD (Superoxide dismutase) gene [122]. The flavonoids found in *keji beling* leaves can mitigate increases in SGPT and SGOT levels [23]. These chemical components work together to effectively suppress the effects of free radicals [24-26].

The administration of sodium benzoate to white mice increases urea and creatinine levels and causes histological damage to the kidneys, including nuclear necrosis, tubular degeneration, and proximal tubular dilation [22]. However, administration of ethanol extract of *keji beling* leaves at a dose of 500 mg/kg BW can reduce urea and creatinine levels, and reduce kidney histological damage. The antioxidant activity of ethanol extracts of *keji beling* and sambiloto leaves, as well as their combination, using the DPPH method. The results showed that the combination of *keji beling* and sambiloto extracts exhibited higher antioxidant activity than either extract alone, with an IC_{50} of 6.36 ppm for the (2:1) combination [27].

Conclusion

Administration of ethanol extract of *keji beling* leaves did not cause any real changes or anomalies in liver morphology. However, this had a major impact on the mean liver histological damage score. Administration of ethanol extract. Vile leaves, glass: a dose of 500 mg per kg BW is safe and optimal for reducing liver tissue damage induced by sodium benzoate. Administration of ethanol extract. Keji leaves resulted in decreased SGPT and SGOT levels in sodium benzoate-induced rats. Administration of ethanol extract. A dose of 500 mg/kg BW of *keji beling* is safe and optimal for improving recovery of liver function after sodium benzoate induction.

Author Contributions

R.A.W. Dari: Conceptualization, methodology, investigation, data collection, formal analysis, and writing, original draft. H. Febriani: Conceptualization, methodology, supervision, validation, and writing, review and editing. Syukriah: Supervision, validation, data interpretation, and writing, review and editing. All authors have read and approved the final manuscript.

Acknowledgements

The authors gratefully acknowledge the Department of Biology, Faculty of Science and Technology, Universitas Islam Negeri Sumatera Utara, for providing facilities and support during this research. The authors also thank all individuals who contributed technical assistance during the experimental and laboratory work.

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