Original Article

Antioxidant Effects of *Taraxacum officinale L.* (Dandelion) Root Extract on Acute Liver and Kidney Injuries Induced by D-Galactosamine in Male Rats

Entissar Mansour Abdul Rasool[1]*

[1]Department of Basic Science, College of Dentistry, Al_Iraqia University, Baghdad- Iraq
ORCID iD: https://orcid.org/my-orcidorcid=0009-0000-2230-6623
Email: entissar.m.abdulrasool@aliraqia.edu.iq

Abstract

Background: Liver and kidney injuries are major health concerns worldwide, often resulting from oxidative stress and inflammation. *Taraxacum officinale* (dandelion) root extract(TOE) is traditionally recognized for its antioxidant and hepatoprotective properties. However, its potential role in mitigating acute liver and kidney injuries induced by D-galactosamine (D-GaIN) remains underexplored. **Objective:** This study aims to investigate the antioxidant effects of *Taraxacum officinale* root extract on acute liver and kidney injuries induced by D-GaIN in male rats, evaluating its efficacy in both curative and prophylactic treatment strategies. **Method:** Fifty Albino adult male rats were divided into five groups: Group 1 (Control), Group 2(D-GaIN): Rats were injected with single dose of D-Galactosamine(D-GaIN) (300mg / kg.i.p.) on 1st day and followed the vehicle (0.5 ml.distilled water /kg / day) by gavage needle for consecutive 21 days.Group 3 (Curative treatment): Rats were injected with single dose of D-GaIN (300mg/kg.i.p.) on 1st day and followed administered by TOE (600mg/kg/ day) by gavage needle for consecutive 21 days Group 4(D-GaIN on 21nd): Rats were received the vehicle (0.5 ml.distilled water /kg / day) by gavage needle for period 21 days, and followed injected of D-GaIN (300mg/kg.i.p.) single dose on 21nd day and Group 5(Prophylactic treatment): Rats were treated by TOE (600mg/kg/ day by gavage needle for period 21 days, and followed injected of D-GaIN (300mg/kg.i.p.) single dose on 21nd day. Liver and kidney function markers, inflammatory cytokines (TNF-α, IL-6, IL-1β), oxidative stress biomarkers (MDA, GSH, SOD, CAT) were analyzed. **Results:** The administration of *Taraxacum officinale* root extract significantly reduced elevated liver enzymes (ALT, AST, ALP) and kidney function markers (urea, creatinine) induced by D-GaIN. Additionally, the extract lowered inflammatory cytokines and enhanced antioxidant enzyme activity, as demonstrated by decreased MDA levels and increased GSH, SOD, and CAT concentrations. analysis revealed notable improvements in liver and kidney enzyme levels, supporting the extract's protective effects. **Conclusion:** The findings suggest that *Taraxacum officinale* root extract exhibits strong antioxidants, anti-inflammatory, and organ-protective properties, mitigating D-GaIN-induced hepatic and renal injuries. These results support its potential curative and prophylactic applications in oxidative stress-related organ damage, warranting further investigation for clinical use.

Keywords: *Taraxacum Officinale*, D-galactosamine (D-GaIN), Liver Injury, Kidney Injury, Antioxidant Activity.

Introduction

Background

Liver and kidney injuries rank among the world's major health challenges[1], contributing significantly to morbidity and mortality. These organs play an essential role in maintaining physiological homeostasis, including metabolic regulation, detoxification of harmful substances, biosynthesis, water-electrolyte balance, and waste excretion. [2] However, their high metabolic activity and exposure to endogenous and exogenous toxins make them highly vulnerable to damage from toxic substances, drugs, infections, and environmental pollutants.[3,4] A major underlying mechanism in acute liver and kidney injuries is oxidative stress, defined as an imbalance between reactive oxygen species (ROS) production and the body's antioxidant defense systems. This imbalance triggers inflammatory cascades, cellular dysfunction, and apoptosis, resulting in progressive tissue damage. [5] D-galactosamine (D-GalN) is a well-established hepatotoxin widely used in experimental models to induce

Access This Article Online

Quick Response Code:

Website: www.jnsbm.org

DOI: https://doi.org/10.4103/jnsbm.JNSBM_16_1_11

Address for Correspondence: Department of Basic Science, College of Dentistry, Al_Iraqia University , Baghdad- Iraq
Email: entissar.m.abdulrasool@aliraqia.edu.iq

Submitted: 28th December, 2024 **Received:** 15th January, 2024
Accepted: 10th February, 2025 **Published:** 18th February, 2025

How to Cite This Article: Rasool E M A. Antioxidant Effects of *Taraxacum officinale L.* (Dandelion) Root Extract on Acute Liver and Kidney Injuries Induced by D-Galactosamine in Male Rats. J Nat Sc Biol Med 2025;16:98-107

acute liver and kidney injuries.[6] Its toxic effects arise from uracil nucleotide depletion, which disrupts RNA and protein synthesis, leading to hepatocellular injury. Furthermore, D-GalN toxicity stimulates excessive ROS production, mitochondrial dysfunction, and lipid peroxidation, exacerbating inflammatory cytokine release (TNF-α, IL-6, IL-1β) and activating NF-κB and caspase-dependent apoptotic pathways.[7] One of the most promising avenues for treatment is the use of natural antioxidants from medicinal plants.[8] These plant-based therapies offer several advantages, including low toxicity, affordability, and multi-target bioactive properties. Among such plants, *Taraxacum officinale L.* (dandelion) has been widely used in traditional medicine across various cultures.[9] It is recognized for its diuretic, anti-inflammatory, and hepatoprotective properties. *Taraxacum officinale* contains a rich array of phytochemicals, including phenolic acids, flavonoids, terpenoids, vitamins (A, C, E), and minerals, which exhibit strong antioxidant and free radical scavenging effects.[10] Recent studies suggest that *Taraxacum officinale* enhances antioxidant enzyme activities (SOD, CAT, GSH), reduces lipid peroxidation (MDA), and suppresses pro-inflammatory cytokine expression, which are crucial mechanisms in preventing oxidative stress-related organ damage.[11,12] Despite these promising findings, limited studies have investigated the therapeutic versus prophylactic efficacy of *Taraxacum officinale* in acute toxin-induced liver and kidney injuries, particularly those caused by D-GalN toxicity. Understanding its potential to prevent and reverse oxidative stress-induced organ damage is essential for advancing affordable and accessible natural therapies. Moreover, its ability to modulate multiple pathological pathways makes it a compelling candidate for further investigation in experimental hepatotoxicity and nephrotoxicity models. This review of the pharmacological and biological activities of *Taraxacum officinale* aims to provide a scientific rationale for its therapeutic applications in toxin-induced organ injuries, contributing to the growing body of research on natural hepatoprotective agents. Expanding knowledge in this area may help develop cost-effective, plant-based treatment options, particularly in resource-limited settings where access to conventional therapies is restricted.

Problem Statement

Despite the interest in natural antioxidants for therapeutic purposes in the current trend, data on both efficacy and mechanisms of *Taraxacum officinale* treatment in acute liver and kidney injuries induced by toxins such as D-GaIN is scarce. Specifically, the detailed mechanism of how this extract mediates its influence on inflammatory cytokines, oxidative stress biomarkers, and markers of organ function has not been defined. The comparative effectiveness given the curative and prophylactive use of such extracts from a Taraxaxcum official has not obviously been defined. This is a gap in knowledge that limits man's ability with respect to maximum therapeutic application/efficacy of this plant given the management option for organistic injuries.

Objective

The current study is specifically designed to investigate the antioxidant effects of *Taraxacum officinale* root extract on acute injuries of the liver and kidneys caused by D-GaIN in male rats. Specific objectives of the study will be to:

1. Assessing the influence of the extract on liver enzymes, kidney function markers, and inflammatory cytokines.
2. The evaluation of the effects of the extract on biomarkers of oxidative stress in liver and kidney tissues.
3. Comparison of the therapeutic and prophylactic efficacy of the extract in preventing D-GaIN-induced organ damages.

Significance of the Study

This study reveals therapeutic value in *Taraxacum officinale* for oxidative stress-related kidney and liver injuries in a cost-effective, organic form. By comparing curative and prophylactic activity in D-GalN toxicity, it fills a critical information vacuum in conventional herbal medicine studies. Observations can lay a basis for creating cheap, plant-originated alternatives for synthetic drugs for hepatic protection, particularly in developing nations with poor financial and infrastructure capacities. Besides, through its demonstration of *Taraxacum officinale's* function in modulating oxidative stress, inflammation, and apoptosis, this work opens a platform for future therapeutic application in kidney and liver protection.

Literature Review

Nowadays, it's common knowledge that oxidative stress plays a part in how acute liver and kidney damage occurs; thus, studies on therapeutic interventions that can offer protection against the process are ongoing.[13] Among the natural antioxidants, especially those of plant origin, it has become important due to its easy availability, low toxicity, and ability to exhibit multifunctionality.[14,15] Among the plants studied is *Taraxacum officinale L.* This has attracted the broad interest of many to this plant for its wide range of pharmacological activities, reaching thus far unparalleled heights, including a variety of activities: antioxidants, anti-inflammatory, and hepatoprotective.[16] In this vein, this review has been focused on the update of knowledge onthe antioxidant properties of *Taraxacum officinale* relevant for toxin-induced organ injuries with special emphasis on the efficacy in experimental models of oxidative stress. Research into natural antioxidants has demonstrated their significant potential in mitigating oxidative stress and its associated health conditions. Among medicinal plants, *Taraxacum officinale L.* (dandelion) Its antioxidant, anti-inflammatory, and hepatoprotective effects have been the subject of much research, with a growing interest in its potential to combat liver and kidney injuries.

Pfingstgraf *et al.*[17] investigated if *Taraxacum officinale* root extract may mitigate the consequences of experimental acute-on-chronic liver failure. In a study using a D-galactosamine and lipopolysaccharide paradigm, researchers found that *Taraxacum officinale* reduced indicators of liver damage and improved histopathological scores in ACLF rats. It is feasible to utilize the extract in the therapy of chronic liver disease because of the strong positive link between its antioxidant activity and its hepatoprotective and nephroprotective effects. Saoudi and El Feki[18] examined the efficacy of an extract from the stems of the Ficus carica tree in protecting male Wistar rats' livers from oxidative stress caused by methanol. Their results showed that Ficus carica exerted an antioxidant effect by restoring hepatic enzymes' activities and lowering lipid peroxidation in methanol-exposed rats, which in turn protected against its hepatotoxicity. This would be indicative of the wider pharmacological role played by plant antioxidants in preventing liver damage. Xing *et al.*[19] assessed the antioxidant and hepatoprotective effects of Reynoutria ciliinervis root extract. Characterisation of 12 bioactive compounds in the work, which showed a significant amount of antioxidant capacity from methanol extract, effectively protected liver injury in rats. Such results evidence that in plant extracts, there are bioactive compounds that form a very imperative field for application. García-Carrasco *et al.*[20] Explored the hypolipidemic and antioxidant activities of *Taraxacum officinale* extracts on mature adipocytes. Based on the obtained results, extracts from dandelion leaves and roots showed induction of inhibition in triglyceride accumulation and high free radical scavenging activity. These findings give a clue to the potential of *Taraxacum officinale* not only in antioxidant therapy but also in conditions related to obesity. Ishfaq *et al.*[21] examined the effects of Cichorium intybus, *Taraxacum officinale*, and Lectuca sativa on lipid peroxidation in mouse livers as antioxidants. Since the plants investigated, *Taraxacum officinale* showed the highest antioxidant activity and was able to inhibit lipid peroxidation, one can conclude its role as a vital natural antioxidant.

In the light of immense evidence for the antioxidant and hepatoprotective roles of *Taraxacum officinale*, not much emphasis has been found on studies about acute injuries in the liver and kidney that have been induced by certain toxins, like D-galactosamine. Most studies have emphasized either the chronic action of this plant or its role in isolated liver injury, hence leaving behind the unaddressed question about the dual potential for therapy and prophylaxis of the drug in models with acute injuries. Besides, the exact mechanism of how *Taraxacum officinale* can modulate biomarkers of oxidative stress, inflammatory cytokines, and markers of organ function in such models is still not well characterized. This research examines the therapeutic and preventive benefits of *Taraxacum officinale* root extract on D-GaIN-induced liver and kidney damage. The comprehensive analysis of serum markers, tissue oxidative stress, and histopathological changes provides new insights into its therapeutic efficacy and mechanisms of action.

Materials and Methods

Experimental Workflow

This study aims to examine the therapeutic and prophylactic effects of *Taraxacum officinale* root extract (TOE) on liver and kidney injuries caused by D-galactosamine (D-GaIN) in rats. A systematic experimental design was employed, as depicted in Figure 1, which outlines the methodology, including animal grouping, treatment protocols, sample collection, and analysis.

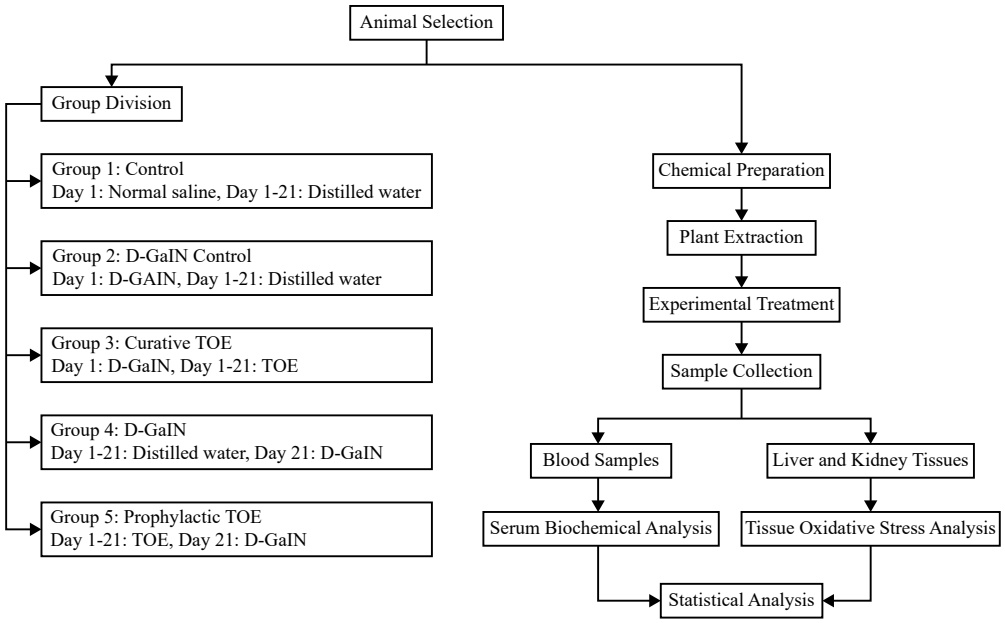

Figure 1: Flowchart of Experimental Design and Methodology.

Animal Model

Albino adult male rats , aged 3 to 4 months and weighing between 190 and 215 grams, were procured from the animal house facility of the Biotechnology Research Center at Al-Nahrain University. The rats were maintained in polypropylene cages under regulated environmental conditions (12-hour light/dark cycle; 25 ± 3°C) and were given a normal meal and water ad libitum to guarantee optimum health.

Inclusion and Exclusion Criteria in this Study

A- Inclusion Criteria

1. All experimental animals are *Albino* adult male rats
2. Rat aging between 3 to 4 months and weighing between 190-215 grams.
3. Healthy animals.
4. Acute liver and kidney failure in experimental animals latrogenically produced by administration of D-galactosamine.
5. All animals were euthanasia to prevent harmful effects.

Exclusion Criteria

In this study excluded all characteristics did not match the criteria for study protocol

1. Lab. animals under the maturity age or above 4 months of age.
2. Rats did not lie in the range of body weight considered in this study where excluded.
3. Exclusion had also been considered in terms of nonlogical data, which where excluded to avoid negative or positive impact on the significance of the results.

Chemical Reagents

D-Galactosamine (D-GaIN) was obtained from Sigma Aldrich Chemical Co., St. Louis, MO, USA, and used to produce hepatic and renal damage. All compounds were of analytical quality.

Plant Extraction Process

The roots of *Taraxacum officinale* were sourced from a local market. The roots were washed thoroughly, air-dried, and ground into fine powder. The extraction process was conducted as follows:

1. **Extraction:** A total of 100 g of powdered root was added to 500 mL of methanol and stirred on a magnetic stirrer at room temperature for 3 days.
2. **Filtration:** The mixture was filtered using Whatman No. 1 filter paper.
3. **Concentration and Storage:** The filtrates were concentrated with a rotary evaporator, dried in an incubator at 40°C, and stored at 4°C until use.

Experimental Design

Fifty *Albino* adult male rats were randomly distributed into five groups of ten. The treatment methods were as shown in Table 1.

Table1: Experimental Group Treatments and Timelines.

Group	Treatment	Timeline
Group 1 (Control)	Single dose of normal saline (0.5 mL, intraperitoneally) followed by distilled water (0.5 mL/kg).	Day 1: Normal saline Day 1–21: Distilled water
Group 2 (D-GaIN Control)	Single dose of D-GaIN (300 mg/kg, intraperitoneally) followed by distilled water (0.5 mL/kg).	Day 1: D-GaIN Day 1–21: Distilled water
Group 3 (Curative TOE)	Single dose of D-GaIN (300 mg/kg, intraperitoneally) followed by TOE (600 mg/kg/day).	Day 1: D-GaIN Day 1–21: TOE
Group 4 (D-GaIN)	Daily distilled water (0.5 mL/kg/day) for 21 days followed by a single dose of D-GaIN (300 mg/kg).	Day 1–21: Distilled water Day 21: D-GaIN
Group 5 (Prophylactic TOE)	Daily TOE (600 mg/kg/day) for 21 days followed by a single dose of D-GaIN (300 mg/kg).	Day 1–21: TOE Day 21: D-GaIN

Sample Collection

Twenty-four hours after the last treatment:

1. Blood samples were collected via retro-orbital puncture using heparinized capillary tubes.
2. Serum was separated by centrifugation (3000 rpm, 10 minutes) and stored at −20°C for analysis.
3. Rats were euthanized under diethyl ether anesthesia.
4. Liver and kidney tissues were harvested, washed with cold saline, and homogenized. Tissue homogenates were centrifuged, the supernatants were preserved at −20°C for biomarker analysis.

Biochemical Analysis of Liver and Kidney Functions

The following parameters were measured using commercial kits (Randox, Northern Ireland) according to the manufacturer's instructions:

- **Liver Function:** The following enzymes are involved in protein metabolism: albumin (ALB), total bilirubin (TB), alkaline phosphatase (ALP), and alanine transaminase (ALT).
- **Kidney Function:** Urea (U), creatinine (Cr), sodium (Na^+), potassium (K^+), and erythropoietin (EPO).
- **Inflammatory Markers:** The ELISA kits from Sunlong Biotech Co. Ltd, China, were used to measure tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), and interleukin-1β (IL-1β).

Tissue Oxidative Stress Biomarkers1

Oxidative stress was assessed in liver and kidney homogenates. The following biomarkers were measured:

1. **Lipid Peroxidation (MDA):** Assessed using the approach outlined by Gilbert *et al.*[22].

2. **Glutathione (GSH):** Evaluate using the procedure of Moron *et al.*[23].
3. **Superoxide Dismutase (SOD):** Examination performed using the procedure by Marklund and Marklund[24].
4. **Catalase (CAT):** Ascertained by the Aebi technique[25]

Statistical Analysis

Mean ± standard error (SE) was used to represent all findings. We used one-way analysis of variance (ANOVA) for our statistical study. Duncan's multiple range test was used to assess group differences, the significance level was established at ($p \leq 0.05$). We used IBM SPSS Statistics, version 23, to do the analyses.

Results

This research aimed to determine if adult male rats exposed to D-galactosamine (D-GaIN) liver and kidney damage would benefit from taking TO root extract. Two different treatment approaches were employed to assess the highest potential of TO extract in preventing organ injury:

1. **Curative Strategy**: D-GaIN was administered at the start of the experiment, followed by daily treatment with TO extract.
2. **Prophylactic Strategy**: TO extract was administered daily for 21 days, followed by a single dose of D-GaIN on the last day of the experiment.

A complete analysis of serum biomarkers and oxidative stress indicators was done to find the protective effects of TO extract.

Effect of TO Extract on Liver Enzymes in Rats Exposed to D-GaIN

The presence of liver injury was tracked by measuring ALT, AST, and ALP levels. Table 2 below displays the results. In contrast to control rats, Animals that were given D-GaIN had noticeably elevated ALT, AST, and ALP levels throughout the experiment ($p < 0.05$), suggesting severe liver injury. The increased enzyme levels were markedly decreased by the TO extract curative therapy, reaching a level like the control group (Group 3, $p < 0.05$). Although it was not as successful as the therapeutic approach, the preventative therapy (Group 5) did lower liver enzyme levels. Enzyme levels in Group 5 were still noticeably reduced ($p \leq 0.05$) compared to Group 4, which was treated with D-GaIN alone.

Table 2: Impact of TO Root Extract on Hepatic Enzymes in Rats Subjected to D-GaIN Exposure.

Treatment	ALT (U/L)	AST (U/L)	ALP (U/L)
Control	41.12 ± 2.47 c	73.75 ± 4.11 c	91.27 ± c
D-GaIN (300 mg/kg, Day 1) *	271 ± 6 a	258.8 ± 8.6 a	262.20 ± a
TO + D-GaIN (600mg/kg+300mg/kg) *	54.30 ± 2.8 c	78.88 ± 3.6 c	127.98 ± bc
D-GaIN (300 mg/kg, Day 21) **	288 ± 4.9 a	270.2 ± 11 a	278.20 ± a
TO + D-GaIN (600mg/kg+300mg/kg) **	173.8 ± 6.1 b	102.8 ± 5.41 b	163.54 ± b

Values are presented as Mean± SEM, n = 10 in each group,
Different letters in the same column refer to significance values ($p \leq 0.05$)
*D-GaIN is injected at the beginning of the experiment
** D-GaIN is injected at the end of the experiment

Effect of TO Extract on Relevant Cytokines in Rats Exposed to D-GaIN

To delve more into the subject of TO root extract's protective benefits, researchers examined the levels of pro-inflammatory cytokines—TNF-α, IL-6, and IL-1β—in the blood of rats. Oxidative stress and tissue damage are known to increase levels of these cytokines, which are important indicators of inflammation. Table 3 summarizes the results showing that there was a substantial ($p < 0.05$) rise in levels of TNF-α, IL-6, and IL-1β after D-GaIN injection compared to the control group, indicating an exacerbated inflammatory response. The levels of all three cytokines were significantly lowered after treatment with the curative dosage of TO extract (Group 3), reaching values that were statistically like the control group ($p < 0.05$). Although it was not as successful as the curative therapy, the preventative dosage of TO extract (Group 5) did considerably reduce cytokine levels. Both groups that received TO treatments had considerably smaller decreases ($p \leq 0.05$) compared to the groups who received just D-GaIN (Groups 2 and 4).

Table 3: Effect of *Taraxacum officinale* Root Extract on Relevant Cytokines in Rats Exposed to D-GaIN.

Treatment	TNF-α (pg/mL)	IL-6 (pg/mL)	IL-1β (pg/mL)
Control	52.40 ± 2.56 c	57.80 ± 2.63 c	37.90 ± 3.12 d
D-GaIN (300 mg/kg, Day 1) *	132.60 ± 4.3 a	93.40 ± 3.04 a	72.07 ± 3.42 a
TO + D-GaIN (600mg/kg+300mg/kg) *	66.10 ± 2.9 bc	66.40 ± 4.15 c	46.41 ± 2.54 c
D-GaIN (300 mg/kg, Day 21) **	130.65 ± 2.82 a	99.00 ± 4.85 a	75.57 ± 2.93 a
TO + D-GaIN (600mg/kg+300mg/kg) **	73.80 ± 1.85 b	79.20 ± 3.86 b	57.62 ± 2.37 b

Values are presented as Mean± SEM, n = 10 in each group,
Different letters in the same column refer to significance values ($p \leq 0.05$)
*D-GaIN is injected at the beginning of the experiment
** D-GaIN is injected at the end of the experiment

Effect of TO Extract on Kidney Function Tests in Rats Exposed to D-GaIN

The effects of TO root extract on kidney function were evaluated as an essential part of the study, given that the kidneys are a critical target organ for D-GaIN-induced toxicity. Key parameters, including U, Cr, and EPO, were analyzed to determine the extent of kidney function impairment and the protective effects of TO extract. The results are summarized in Table 4. D-GaIN administration caused a significant ($p \leq 0.05$) increase in both U and Cr levels compared to the control group, indicating kidney dysfunction. The curative treatment with TO extract (Group 3) effectively reduced U levels to those comparable to the control group ($p \leq 0.05$). Also, prophylactic treatment of Group 5 significantly reduced the level of U, although it was less intense compared with the curative group. Nevertheless, U concentration in Group 5 was considerably lower than in Group 4 treated with D-GaIN alone at $p \leq 0.05$. Regarding the Cr levels, both the curative and prophylactic doses of TO extract managed to reduce the Cr levels to a value like the control group. These results show the potential of TO extract for the mitigation of D-GaIN-induced kidney injury. EPO concentration was significantly reduced, $p \leq 0.05$, after treatment with D-GaIN. Both curative and prophylactic treatments with TO extract significantly increased EPO levels compared to D-GaIN-only groups, although the levels remained lower than those of the control group.

Table 4: Effect of *Taraxacum officinale* Root Extract on Kidney Function Tests in Rats Exposed to D-GaIN.

Treatment	U (mg/dL)	Cr (mg/dL)	EPO (pg/mL)
Control	4.45 ± 0.66 d	1.01 ± 0.48 b	112.12 ± 4.6 a
D-GaIN (300 mg/kg, Day 1) *	15.61 ± 0.49 a	1.75 ± 0.82 a	59.24 ± 7.1 c
TO + D-GaIN (600mg/kg+300mg/kg) *	5.02 ± 0.43 d	1.04 ± 0.68 b	104.40 ± 3.7 ab
D-GaIN (300 mg/kg, Day 21) **	13.37 ± 0.77 b	1.67 ± 0.11 a	76.10 ± 5.7 c
TO + D-GaIN (600mg/kg+300mg/kg) **	8.10 ± 0.40 c	0.95 ± 0.02 b	92.90 ± 3.2 b

Values are presented as Mean± SEM, n = 10 in each group,
Different letters in the same column refer to significance values ($p \leq 0.05$)
*D-GaIN is injected at the beginning of the experiment
** D-GaIN is injected at the end of the experiment

Effect of TO Extract on Blood Electrolytes in Rats Exposed to D-GaIN

To further validate the protective effects of TO root extract on kidney function, the concentrations of key blood electrolytes, sodium (Na^+) and potassium (K^+), were measured. These electrolytes are critical markers of kidney health and homeostasis. The results are summarized in Table 5. Administration of D-GaIN led to a significant ($p \leq 0.05$) elevation in both Na^+ and K^+ concentrations compared to the control group. This indicates an imbalance in electrolyte regulation and impaired kidney function due to D-GaIN-induced toxicity. Treatment with the curative dose of TO extract (Group 3) significantly ($p \leq 0.05$) reduced Na^+ levels, restoring them to values comparable to the control group. In contrast, the prophylactic treatment (Group 5) resulted in a smaller reduction in Na^+ levels, which were not markedly different from those recorded in the D-GaIN-only group (Group 4). These findings suggest that the curative administration of TO extract is more effective in normalizing sodium levels. Regarding K^+ levels, only the prophylactic dose of TO extract (Group 5) significantly ($p \leq 0.05$) reduced K^+ concentrations to levels comparable to the control group. The curative dose (Group 3), while effective, was less impactful in normalizing K^+ levels compared to Na^+.

Table 5: Effect of *Taraxacum officinale* Root Extract on Blood Electrolytes in Rats Exposed to D-GaIN.

Treatment	Na^+ (mmol/L)	K^+ (mmol/L)
Control	138.4 ± 2.85 c	5.18 ± 0.12 bc
D-GaIN (300 mg/kg, Day 1) *	150.0 ± 2.1 a	6.57 ± 0.2 a
TO + D-GaIN (600mg/kg+300mg/kg) *	142.1 ± 2.2 bc	5.89 ± 0.3 ab
D-GaIN (300 mg/kg, Day 21) **	147.2 ± 2.33 ab	6.45 ± 0.18 a
TO + D-GaIN (600mg/kg+300mg/kg) **	145.3 ± 2.94 abc	5.08 ± 0.37 c

Values are presented as Mean± SEM, n = 10 in each group,
Different letters in the same column refer to significance values ($p \leq 0.05$)
*D-GaIN is injected at the beginning of the experiment
** D-GaIN is injected at the end of the experiment

Effect of TO Extract on Some Metabolites in Rats Exposed to D-GaIN

To finalize the blood analysis in this study, metabolic indicators, including TB, TP, and ALB, were evaluated to assess the systemic effects of TO root extract. The results, presented in Table 6, provide insights into the metabolic disruptions caused by D-GaIN and the restorative potential of TO extract. D-GaIN administration significantly ($p \leq 0.05$) increased TB concentrations in both injection strategies compared to the control group, indicating impaired liver function. Treatment with TO extract, in both curative (Group 3) and prophylactic (Group 5) regimens, effectively reduced TB levels. The curative treatment restored TB concentrations to levels comparable to the control group, while the prophylactic treatment achieved a significant reduction, albeit slightly higher than control values. Similarly, ALB concentrations followed a parallel trend to TB. D-GaIN injection resulted in a significant ($p \leq 0.05$) increase in

ALB levels. Both curative and prophylactic TO treatments significantly reduced ALB levels, normalizing them to values comparable to the control group. In contrast, TP concentrations were significantly ($p \leq 0.05$) decreased by D-GaIN in all groups compared to the control. While the curative treatment with TO extract (Group 3) improved TP levels, they remained below control levels. Interestingly, the prophylactic TO treatment (Group 5) restored TP concentrations close to those observed in the control group, indicating a superior effect in maintaining protein synthesis.

Table 6: Effect of *Taraxacum officinale* Root Extract on Some Metabolites in Rats Exposed to D-GaIN.

Treatment	TB (mg/dL)	TP (g/dL)	ALB (g/dL)
Control	0.89 ± 0.48 b	6.46 ± 0.22 a	4.92 ± 0.36 b
D-GaIN (300 mg/kg, Day 1) *	2.34 ± 0.17 a	4.10 ± 0.31 c	9.64 ± 0.54 a
TO + D-GaIN (600mg/kg+300mg/kg) *	0.93 ± 0.11 b	4.92 ± 0.38 bc	5.50 ± 0.33 b
D-GaIN (300 mg/kg, Day 21) **	1.99 ± 0.21 a	4.95 ± 0.46 bc	10.68 ± 0.71 a
TO + D-GaIN (600mg/kg+300mg/kg) **	1.15 ± 0.15 b	5.54 ± 0.21 ab	6.50 ± 0.48 b

Values are presented as Mean± SEM, n = 10 in each group,
Different letters in the same column refer to significance values ($p \leq 0.05$)
*D-GaIN is injected at the beginning of the experiment
** D-GaIN is injected at the end of the experiment

Effect of TO Extract on Oxidative Stress Indices in the Liver of Rats Exposed to D-GaIN

To evaluate the effect of TO root extract on oxidative damage in liver tissues caused by D-GaIN, the following oxidative stress markers were employed: MDA, reduced GSH, SOD, and CAT. The findings are summarized in Table 7. Treatment with D-GaIN resulted in a notable rise ($p \leq 0.05$) in MDA levels, which indicate lipid peroxidation, and a notable fall ($p \leq 0.05$) in GSH, SOD, and CAT levels, which indicate heightened oxidative stress and compromised antioxidant defense mechanisms. Both curative (Group 3) and prophylactic (Group 5) treatments with TO extract significantly improved oxidative stress indices compared to their respective D-GaIN-only groups (Groups 2 and 4). MDA levels were reduced in both treatment groups, while GSH, SOD, and CAT levels were elevated. However, despite these improvements, the values for oxidative stress indices in TO-treated groups remained significantly different from the control group, indicating partial recovery.

Table 7: Effect of *Taraxacum officinale* Root Extract on Oxidative Stress Indices in the Liver of Rats Exposed to D-GaIN.

Treatment	MDA (nmol/mg tissue)	GSH (μmol/g tissue)	SOD (U/mg tissue)	CAT (U/mg tissue)
Control	3.75 ± 0.31 c	3.14 ± 0.35 a	10.64 ± 0.48 a	13.04 ± 0.35 a
D-GaIN (300 mg/kg, Day 1) *	7.78 ± 0.21 a	1.72 ± 0.10 c	6.75 ± 0.36 c	7.74 ± 0.25 c
TO + D-GaIN (600mg/kg+300mg/kg) *	6.26 ± 0.36 b	2.28 ± 0.32 b	9.43 ± 0.50 b	9.18 ± 0.16 b
D-GaIN (300 mg/kg, Day 21) **	7.56 ± 0.23 a	1.56 ± 0.14 c	7.85 ± 0.36 bc	7.64 ± 0.25 c
TO + D-GaIN (600mg/kg+300mg/kg) **	5.94 ± 0.29 b	1.91 ± 0.35 bc	8.86 ± 0.49 b	9.34 ± 0.43 b

Values are presented as Mean± SEM, n = 10 in each group,
Different letters in the same column refer to significance values ($p \leq 0.05$)
*D-GaIN is injected at the beginning of the experiment
** D-GaIN is injected at the end of the experiment

Effect of TO Extract on some Oxidative Stress Indices in the Kidney of Rats Exposed to D-GaIN

To determine if the root extract of TO protected kidney tissues from oxidative damage caused by D-GaIN, the oxidative stress indicators MDA, reduced GSH, SOD, and CAT were quantified in these tissues. The results, as shown in Table 8, show that there are clear variations when compared to the liver's oxidative stress profile. The delivery of D-GaIN resulted in a notable rise ($p < 0.05$) in MDA levels, which represents increased lipid peroxidation, and a notable decrease ($p \leq 0.05$) in GSH, SOD, and CAT levels, which signifies oxidative stress and compromised antioxidant defenses within kidney tissues. Both curative (Group 3) and prophylactic (Group 5) treatments with TO extract significantly reduced MDA levels compared to their respective D-GaIN-only groups (Groups 2 and 4). However, for GSH, SOD, and CAT, only the curative treatment (Group 3) restored these antioxidant indices to near-normal levels ($p \leq 0.05$). The prophylactic treatment (Group 5) had a minimal impact on these parameters, with values remaining significantly different from the control group.

Table 8: Effect of *Taraxacum officinale* Root Extract on Oxidative Stress Indices in the Kidney of Rats Exposed to D-GaIN.

Treatment	MDA (nmol/mg tissue)	GSH (μmol/g tissue)	SOD (U/mg tissue)	CAT (U/mg tissue)
Control	5.69 ± 0.23 c	8.14 ± 0.46 a	15.40 ± 0.44 a	19.66 ± 0.63 a
D-GaIN (300 mg/kg, Day 1) *	7.58 ± 0.30 a	5.54 ± 0.23 c	8.53 ± 0.23 c	12.50 ± 0.52 c
TO + D-GaIN (600mg/kg+300mg/kg) *	6.53 ± 0.32 bc	7.01 ± 0.37 b	13.06 ± 0.36 b	14.58 ± 0.42 b
D-GaIN (300 mg/kg, Day 21) **	7.37 ± 0.55 ab	5.34 ± 0.11 c	7.68 ± 0.41 c	12.88 ± 0.38 c
TO + D-GaIN (600mg/kg+300mg/kg) **	6.84 ± 0.21 b	5.72 ± 0.30 c	7.69 ± 0.14 c	13.89 ± 0.44 bc

Values are presented as Mean± SEM, n = 10 in each group,
Different letters in the same column refer to significance values ($p \leq 0.05$)
*D-GaIN is injected at the beginning of the experiment
** D-GaIN is injected at the end of the experiment

Discussion

This research presents substantial evidence for the hepatoprotective and nephroprotective advantages of TO root extract, as shown by numerical enhancements in biochemical, inflammatory, and oxidative stress markers. The notable decrease in liver enzymes, including ALT, AST, and ALP, highlights TO's effectiveness in alleviating D-galactosamine (D-GaIN)-induced hepatic injury. For example, ALT levels surged significantly from 41.12 ± 2.47 U/L in the control group to 271 ± 6 U/L in the D-GaIN-treated group, but therapeutic TO therapy decreased ALT levels to 54.30 ± 2.8 U/L, almost returning them to baseline. In Group 3 (curative TO), AST levels were diminished to 78.88 ± 3.6 U/L, while ALP levels were lowered to 127.98 ± 12.3 U/L, in contrast to the markedly higher levels seen in the untreated group. These results align with findings by Hamza *et al.*[26], where TO was shown to decrease hepatic injury markers and collagen deposition in fibrosis models. Comparable reductions in ALT and AST were also observed by Al-Malki *et al.*[27] in their investigation of liver damage brought on by carbon tetrachloride. The research also showed that TO decreased inflammatory cytokines such as IL-6, IL-1β, and TNF-α. In the group treated with D-GaIN, for example, TNF-α levels increased from 52.40 ± 2.56 pg/mL to 132.60 ± 4.3 pg/mL, but they were considerably lowered to 66.10 ± 2.9 pg/mL following curative TO therapy. IL-6 levels, similarly, were reduced to 66.40 ± 4.15 pg/mL in Group 3, highlighting TO's anti-inflammatory potential. This aligns with studies that emphasize TO's ability to suppress pro-inflammatory cytokine production through its phenolic and flavonoid components. This aligns with studies that emphasize TO's ability to suppress pro-inflammatory cytokine production through its phenolic and flavonoid components.[28,29] In terms of kidney function, TO effectively reduced U and Cr levels, which were significantly elevated by D-GaIN. U levels increased from 4.45 ± 0.66 mg/dL in the control group to 15.61 ± 0.49 mg/dL in the untreated group, but curative treatment normalized them to 5.02 ± 0.43 mg/dL. Cr levels followed a similar pattern, decreasing from 1.75 ± 0.82 mg/dL in the untreated group to 1.04 ± 0.68 mg/dL after TO treatment. These results are consistent with Esra and Betul's[29] findings, where TO significantly reduced nephrotoxicity markers in paracetamol-induced renal damage models. Lipid peroxidation markers, such as malondialdehyde (MDA), showed marked improvement with TO treatment. MDA levels in liver tissues were reduced from 7.78 ± 0.21 nmol/mg in Group 2 to 6.26 ± 0.36 nmol/mg in Group 3. Kidney MDA levels also decreased from 7.58 ± 0.3 nmol/mg to 6.53 ± 0.32 nmol/mg with TO treatment. Concurrently, antioxidant markers like GSH, SOD, and CAT were partially restored. For instance, GSH levels in the liver increased from 1.72 ± 0.1 µmol/g in the untreated group to 2.28 ± 0.32 µmol/g in Group 3, while CAT activity improved from 7.74 ± 0.25 U/mg to 9.18 ± 0.16 U/mg. These results are consistent with studies showing TO's capacity to enhance antioxidant defenses in oxidative stress models.[17] The normalization of metabolic parameters, such as bilirubin and total protein (TP), further highlights TO's systemic protective effects. Bilirubin levels, which rose to 2.34 ± 0.17 mg/dL in Group 2, were normalized to 0.93 ± 0.11 mg/dL with curative treatment. Similarly, TP levels increased from 4.1 ± 0.31 g/dL to 5.54 ± 0.21 g/dL in the prophylactic group, indicating partial improvement. TO's curative effects on D-GaIN-induced damage outperform many other natural and synthetic interventions. For example, butin, a flavonoid, demonstrated comparable reductions in ALT, AST, and inflammatory cytokines in similar models.[28] However, TO's broader benefits on kidney function and electrolyte balance make it a more comprehensive therapeutic agent. Sodium levels, for instance, were reduced from 150 ± 2.1 mmol/L to 142.1 ± 2.2 mmol/L, while potassium levels normalized only in the prophylactic group (5.08 ± 0.37 mmol/L vs. 6.57 ± 0.2 mmol/L in untreated rats), highlighting its specific regulatory effects.

Conclusion

This study confirms that *Taraxacum officinale* root extract effectively protects against D-GalN-induced liver and kidney injuries through oxidative stress inhibition, inflammation, and derangement of metabolism. The extract lowered pro-inflammatory cytokines (TNF-α, IL-6, IL-1β) and normalized markers of liver function (ALT, AST, ALP) and kidney function (urea, creatinine) in a significant manner. It boosted antioxidant defenses (GSH, SOD, CAT) and reduced lipid peroxidation (MDA), providing additional supporting evidence for its protective role. All these observations confirm that *Taraxacum officinale* can act as a naturally derived hepatoprotector and nephroprotector, offering a cheap alternative to conventional drugs. Future studies must include clinical evaluation, molecular mechanism, and pharmacokinetics to assess its therapeutic and safe use in humans in medical practice.

Limitations

Despite the encouraging results, several limitations in this study must be considered. The relatively small sample could restrict generalizability of the findings. Differences in dosing and duration of administration could impact efficacy of the extract, and additional studies in terms of dose-response will have to be conducted. In addition, strong preclinical efficacy in a model system cannot assure its efficacy in humans, and therefore, its translation to humans must rely on clinical trials for confirming its safety, its pharmacokinetics, and long-term consequences in healing liver and kidney injuries.

References

1. Asrani SK, Devarbhavi H, Eaton J, Kamath PS. Burden of liver diseases in the world. J Hepatol. 2019; 70(1): 151-71. doi: https://doi.org/10.1016/j.jhep.2018.09.014.
2. Wang FS, Fan JG, Zhang Z, Gao B, Wang HY. The global burden of liver disease: the major impact of China. Hepatology. 2014; 60(6): 2099-108. doi: https://doi.org/10.1002/hep.27406.

3. Petejova N, Martinek A, Zadrazil J, Teplan V. Acute toxic kidney injury. Ren Fail. 2019; 41(1): 576-94. doi: https://doi.org/10.1080/0886022x.2019.1628780.
4. Kehrer JP, Klotz LO. Free radicals and related reactive species as mediators of tissue injury and disease: implications for Health. Crit Rev Toxicol. 2015; 45(9): 765-98. doi: https://doi.org/10.3109/10408444.2015.1074159.
5. Chatterjee S. Chapter Two - Oxidative Stress, Inflammation, and Disease. In: Dziubla T, Butterfield DA, Eds. Oxidative Stress and Biomaterials. Academic Press; 2016:35-58. doi: https://doi.org/10.1016/B978-0-12-803269-5.00002-4.
6. Farghali H, Kgalalelo Kemelo M, Wojnarová L, Kutinová Canová N. In vitro and in vivo experimental hepatotoxic models in liver research: applications to the assessment of potential hepatoprotective drugs. Physiol Res. 2016; 65(Suppl 4): S417-s25. doi: https://doi.org/10.33549/physiolres.933506.
7. Muhamad Fadzil NS, Sekar M, Gan SH, et al. Chemistry, Pharmacology and Therapeutic Potential of Swertiamarin - A Promising Natural Lead for New Drug Discovery and Development. Drug Des Devel Ther. 2021; 15: 2721-46. doi: https://doi.org/10.2147/dddt.s299753.
8. Gurib-Fakim A. Medicinal plants: traditions of yesterday and drugs of tomorrow. Mol Aspects Med. 2006; 27(1): 1-93. doi: https://doi.org/10.1016/j.mam.2005.07.008.
9. Alamgir ANM. Biotechnology, In Vitro Production of Natural Bioactive Compounds, Herbal Preparation, and Disease Management (Treatment and Prevention). In: Alamgir ANM, Ed. Therapeutic Use of Medicinal Plants and their Extracts: Volume 2: Phytochemistry and Bioactive Compounds. Springer International Publishing; 2018:585-664. doi: https://doi.org/10.1007/978-3-319-92387-1_7.
10. Abou Seif HS. Physiological changes due to hepatotoxicity and the protective role of some medicinal plants. Beni Suef Univ J Basic Appl Sci. 2016; 5(2): 134-46. doi: https://doi.org/10.1016/j.bjbas.2016.03.004.
11. Azat Aziz M, Shehab Diab A, Mohammed AA. Antioxidant Categories and Mode of Action. In: Shalaby E, Ed. Antioxidants. IntechOpen; 2019. doi: https://doi.org/10.5772/intechopen.83544.
12. Kafi DK, Ayyash AN. Molecular structure, molecular docking and vibrational spectra of 2,2′-[1,3,5,7-tetraoxo-5,7-dihydropyrrolo[3,4-f-[isoindole 2,6(1H,3H) diyl]]] diacetohydrazide molecule by density functional theory approach as human immunodeficiency virus inhibitory. Spectrosc Lett. 2022; 55(10): 636-47. doi: https://doi.org/10.1080/00387010.2022.2139726.
13. Pavlakou P, Liakopoulos V, Eleftheriadis T, Mitsis M, Dounousi E. Oxidative Stress and Acute Kidney Injury in Critical Illness: Pathophysiologic Mechanisms-Biomarkers-Interventions, and Future Perspectives. Oxid Med Cell Longev. 2017; 2017: 6193694. doi: https://doi.org/10.1155/2017/6193694.
14. Mohammed RAK, fadhil Mohammed S, Hasan MS. Isolation of Tooth caries bacterial pathogens. Revista Iberoamericana de Psicologia del Ejercicio y el Deporte. 2022; 17(5): 272-74. Available from: https://psykebase.es/descarga/articulo/8679683.pdf.
15. Xu DP, Li Y, Meng X, et al. Natural Antioxidants in Foods and Medicinal Plants: Extraction, Assessment and Resources. Int J Mol Sci. 2017; 18(1): 96. doi: https://doi.org/10.3390/ijms18010096.
16. Akbar S. Handbook of 200 Medicinal Plants: A Comprehensive Review of Their Traditional Medical Uses and Scientific Justifications. Springer Cham; 2020. doi: https://doi.org/10.1007/978-3-030-16807-0.
17. Pfingstgraf IO, Taulescu M, Pop RM, et al. Protective Effects of Taraxacum officinale L. (Dandelion) Root Extract in Experimental Acute on Chronic Liver Failure. Antioxidants (Basel). 2021; 10(4): 504. doi: https://doi.org/10.3390/antiox10040504.
18. Saoudi M, El Feki A. Protective Role of Ficus carica Stem Extract against Hepatic Oxidative Damage Induced by Methanol in Male Wistar Rats. Evid Based Complement Alternat Med. 2012; 2012: 150458. doi: https://doi.org/10.1155/2012/150458.
19. Xing Z, Han Y, Pang H, et al. Evaluating the Root Extract of Reynoutria ciliinervis (Nakai) Moldenke: An Analysis of Active Constituents, Antioxidant Potential, and Investigation of Hepatoprotective Effects in Rats. Molecules. 2024; 29(19): 4701. doi: https://doi.org/10.3390/molecules29194701.
20. García-Carrasco B, Fernandez-Dacosta R, Dávalos A, Ordovás JM, Rodriguez-Casado A. In vitro Hypolipidemic and Antioxidant Effects of Leaf and Root Extracts of Taraxacum Officinale. Med Sci (Basel). 2015; 3(2): 38-54. doi: https://doi.org/10.3390/medsci3020038.
21. Ishfaq S, Sabir SM, Khurshid H, Zaman T, Ahmad Z. Antioxidant activities and inhibitory effect of Taraxacum officinale, Cichorium intybus and Lectuca sativa on prooxidant induced lipid peroxidation in mice liver. Croatian Journal of Food Science and Technology. 2018; 10(1): 16-22. doi: https://doi.org/10.17508/CJFST.2018.10.1.03.
22. Gilbert HS, Stump DD, Roth EF, Jr. A method to correct for errors caused by generation of interfering compounds during erythrocyte lipid peroxidation. Anal Biochem. 1984; 137(2): 282-6. doi: https://doi.org/10.1016/0003-2697(84)90086-1.
23. Moron MS, Depierre JW, Mannervik B. Levels of glutathione, glutathione reductase and glutathione S-transferase activities in rat lung and liver. Biochim Biophys Acta. 1979; 582(1): 67-78. doi: https://doi.org/10.1016/0304-4165(79)90289-7.
24. Marklund S, Marklund G. Involvement of the superoxide anion radical in the autoxidation of pyrogallol and a convenient assay for superoxide dismutase. Eur J Biochem. 1974; 47(3): 469-74. doi: https://doi.org/10.1111/j.1432-1033.1974.tb03714.x.

25. Aebi HE. Catalase: UV-method. Methods of enzymatic analysis. 1983; 111: 273-86.
26. Hamza AA, Mohamed MG, Lashin FM, Amin A. Dandelion prevents liver fibrosis, inflammatory response, and oxidative stress in rats. JoBAZ. 2020; 81: 1-13. doi: https://doi.org/10.1186/s41936-020-00177-9.
27. Al-Malki AL, Abo-Golayel MK, Abo-Elnaga G, Al-Beshri H. Hepatoprotective effect of dandelion (Taraxacum officinale) against induced chronic liver cirrhosis. Med Plants Res. 2013; 7(20): 1494-505. doi: https://doi.org/10.5897/JMPR12.1116.
28. Althurwi HN, Altharawi A, Alharthy KM, et al. Butin prevent liver damage triggered by D-galactosamine via regulating lipid peroxidation and proinflammatory cytokines in rodents. J King Saud Univ Sci. 2023; 35(9): 102934. doi: https://doi.org/10.1016/j.jksus.2023.102934.
29. Esra AS, Betul AY. Effect of Taraxacum officinale L. ethanol extract against kidney injuries induced by paracetamol in rats. GSC Biol Pharm Sci. 2022; 21(1): 144-51. doi: https://doi.org/10.30574/gscbps.2022.21.1.0385