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Effects of licorice root powder on lipid peroxidation and antioxidant status in arthritis-induced rats

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ABSTRACT

Rheumatoid arthritis (RA) is a systemic autoimmune disorder characterized by chronic inflammation that leads to progressive joint damage and disability. The pathogenesis of RA involves complex inflammatory cascades and is closely associated with oxidative stress and lipid peroxidation. The present study was performed to evaluate the effect of licorice root powder on lipid peroxidation and antioxidant status in arthritis-induced rats. Thirty adults female wistar rats (210±5g) were weighted and randomly allocated into two main groups. Group (1) control (ve-) (6 rats) received standard diet only, while the second group (24 rats) arthritis was induced through an intradermal injection of Complete Freund's Adjuvant (CFA) (0.1 mL) at the right hind footpad on day one. Then rats were divided into five subgroups, Group (II), arthritic group (ve+) received standard diet only. Group (III) treated with 5 % licorice / diet daily. Group (IV) treated with 10 % licorice / diet daily. Group (V) treated with 15 % licorice / diet daily. The results showed significantly observed ($p \leq 0.05$) in the serum levels of biochemical parameters and significantly reduced lipid peroxidation in all treated groups.

***Keywords*:** Arthritis, Licorice Root, Antioxidant, liver, Kidney

INTRODUCTION

Osteoarthritis is a degenerative joint disease that causes cartilage deterioration and inflammation in the joints (Glyn-Jones *et al*.,2015). Globally, there are around 240 million patients. According to estimates, osteoarthritis affects 18.0% of women and 9.6% of men over the age of sixty (Srikanth *et al*., 2005). Many risk factors including aging, obesity, injury, and muscle weakness are associated with the occurrence of osteoarthritis (Abbate *et al*., 2006).

Pain in the joints and stiffness are the main symptoms. The joint pathology is varied and includes osteophytes (bone growth at the joint margins), ligamentous laxity, weakening of the periarticular muscles, and in some cases synovial distension and inflammation. It also includes focal damage and loss of articular cartilage, abnormal remodelling and attrition of subarticular bone, and focal damage and loss of subarticular bone. (Hutton, 1989). Overweight and obesity have long been acknowledged as significant risk factors for OA, particularly knee OA (Felson *et al*., 2000)

Complete Freund's adjuvant is used in the models mentioned in this protocol to create a more enduring inflammation. In an animal model, inflammation can be evaluated by edoema (swelling), hypersensitivity to noxious stimuli (hyperalgesia), or sensitivity

to nonnoxious stimuli (allodynia) (Iadarola *et al*., 1988).

One of the oldest and most popular herbal medicine plants is Glycyrrhiza glabra. (Wang and Nixon., 2001). *Glabra Glycyrrhiza Linn* is a tough perennial shrub that can grow up to 2.5 metres in height. The leaves have 47 pairs of oblongs, elliptic, or lanceolate leaflets and are compound, imparipinnate, and alternating. The thin, generally papilionaceous blooms range in colour from lavender to violet and are produced in axillary spikes. The fruit is a compressed legume or pod that can reach a length of 1.5 cm, is upright, glabrous, has some reticulate pitting, and often has 3-5 brown, reniform seeds inside. The horizontal woody stolons grow from the taproot, which is about 1.5 cm long and divides into 3-5 subsidiary roots that are each about 1.25 cm long. These can grow to a height of 8 metres, and when dried and sliced, along with the root, they make up commercial licorice. You can find it either peeled or unpeeled.

The dried root of Glycyrrhiza plants is known as licorice. The roots have historically been used as flavourings, sweeteners, demulcents, expectorants, and anti-inflammatory medications in China and Japan (Simmler *et al*., 2014). Fresh licorice root has high levels of magnesium, calcium, potassium, sodium, zinc, manganese, iron, and copper, as well as about 20% moisture, 6% ash, 30% starch, 8% protein, 1.35 g of fat, and 15 to 20% carbohydrates. In addition to having a significant number of phenols (0.3 to 0.5%), flavonoids (0.1 to 0.2%), triterpene saponins, coumarins, chalcones, and isoflavones, licorice root also has other compounds like asparagine, glucose, fructose, FA (C2 to C16), lactones, sterols, and polysaccharides as well as substances with antioxidant activity (Hayashi *et al*., 1998).

Liquorice contain coumarins (glycyrin, heniarin, liqcoumarin and umbelliferone), flavonoids (flavonols and isoflavones including formononetin, glabrin, glabrol, glabrone, glyzarin, glycyrol, glabridin and derivatives, kumatakenin, licoflavonol, licoisoflavones A and B, licoisoflavanone, licoricone, liquiritin and derivatives) and terpenoids (glycyrrhizic or glycyrrhizinic acid) all these compounds are responsible for pharmacological activity of liquorice which has been found to have anti-inflammatory(Amagaya *et al*., 1984) anti-allergic (Han *et al*., 2017) and immunomodulatory (Raphael and Kuttan., 2003)

The principal bioactive ingredients in licorice have been extensively studied in terms of their biological effects, notably regarding their anti-cancer, anti-inflammatory, and anti-arthritic properties (Huang *et al*., 2014). For instance, licorice, particularly roasted licorice, has advantages in preventing both acute and chronic inflammatory diseases antiarthritic (Gujral *et al*., 1961). Glycyrrhizin, which is found in licorice root in concentrations ranging from 1 to 9%, offers several nutritional benefits, including the ability to speed up growth, as well as pharmacological activities like immunomodulatory, antioxidant, antiviral, and anti-inflammatory characteristics (Mahmoud *et al*., 2021).So this study aim to evaluate the effect of licorice root powder on lipid peroxidation and antioxidant status in arthritis-induced rats

MATERIALS AND METHODS

Materials

Licorice was obtained from International Garden, Abbas El-Akkad St., Nasr City, Cairo, Egypt. Chemical Kits were obtained from Biodiagnostic Co. Egypt.

Induction of arthritis /experimental design

Thirty adults female Wistar rats (210±5g) were obtained from Agricultural Research Center, Giza, Egypt. For 8 weeks,

the animal groups were kept in an environment of filtered, pathogen-free air, water, and a temperature of 20-25oC, with a 12 hour light/dark cycle and a light cycle (8-20 h) and a relative humidity of 50%.All rats were fed a standard diet for one week.The basal diet included 14% casein, 10% sucrose, 4% corn oil, 5% fibre (cellulose), 3.5 percent mineral mixture, 1% vitamin mixture, 0.25 percent choline chloride, 0.3 percent D-L methionine, and 61.95 percent corn starch. (Reeves *et al.,* 1993). Before beginning the experiment, allow for acclimatisation. All experimental procedures were carried out in accordance with international guidelines for laboratory animal care and use. The experiment was carried out at the Agricultural Research Center in Giza, Egypt.

After acclimatizing for seven days prior to the study. Rats were weighted and randomly allocated into two main groups. Group (1) control (ve-) (6 rats) received standard diet only, while the second group (24 rats) arthritis was induced through an intradermal injection of Complete Freund's Adjuvant (CFA) (0.1 mL) at the right hind footpad on day one (Zhang *et al*., 2020). Then rats were divided into five subgroups, Group (II), arthritic group (ve+) received standard diet only. Group (III) treated with 5 % licorice / diet daily. Group (IV) treated with 10 % licorice / diet daily. Group (V) treated with 15 % licorice / diet daily

Blood collection and biochemical analysis

After the last behavioral test, the animals were fasted for 12 h, Blood samples were withdrawn from orbital plexus venous by using fine capillary glass tubes, collected into plain tubes without anticoagulant and allowed to clot, centrifuged at 3000 rpm for 10 min at 4°C, to obtain clear serum and frozen at -18ºC until analyzed according to Schermer (1967). Then rats were euthanized by cervical dislocation, and liver, pancreas and brain samples were excised. Parts of the pancreas and brain samples were stored at -80 ° C. for subsequent gene expression, Moreover, parts of pancreas and brain samples were fixed in 10% buffered formalin for histopathological and immunohistochemical investigations**.**

Serum uric acid was determined by Barham and Trinder, (1972) using a spectrophotometer (model DU 4700) adjusted at 510 nm. Serum urea was determined according to the method described by Fawcett and Soctt (1960) using a spectrophotometer (model DU 4700) adjusted nm 550 nm. Serum creatinine was determined by (Tietz, 1986) using a spectrophotometer (model DU 4700) adjusted at 510 nm. Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) activities were determined calorimetrically using spectrophotometer (model DU 4700) at 505 nm according to the method of (Reitman and Frankel, 1957). Alkaline phosphatase (ALP) activity was determined calorimetrically using a spectrophotometer (model DU 4700) at 510 nm according to the method of (Belfield and Goldberg 1971).

Assessment of lipid peroxidation:

SOD was determined according to Nishikimi *et al*. (1972), while MDA was determined according to Satoh, (1978). The activity of catalase was measured using the Aebi *et al*. (1984) technique.

Statistical Analysis

The statistical analysis was carried out by using SPSS, PC statistical software (version 25.0, SPSS Inc., Chicago. USA). The results were expressed as mean ± SD. Data was analyzed by one-way analysis of variance (ANOVA). The Differences between means were tested for significance

using Least Significant Difference (LSD) test at p≤0.05 (Snedecor and Cochran, 1980).

RESULTS AND DISCUSSION

Liver enzymes

The results are presented in Table (1) showed the induction of arthritis with Complete Freund's Adjuvant (CFA) resulted in a severe hepatotoxic effect, as evidenced by a highly significant ($p < 0.05$) increase in the serum concentrations of ALP (171.12 U/l), AST (150.18 U/l), and ALT (60.26 U/l) compared to the healthy control group (Ve-). The dramatic elevation of serum ALT, AST, and ALP in the arthritic control group (Ve+) is a classic indicator of hepatic injury. ALT and AST are cytosolic enzymes released into the bloodstream upon rupture of hepatocyte membranes, while elevated ALP often suggests impairment in biliary flow or general inflammatory stress on the liver (Ghanem *et al*., 2016). This hepatotoxicity is a well-established consequence of systemic inflammation rather than a direct effect of CFA. Rheumatoid arthritis and its experimental models are characterized by a cascade of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-α), interleukin-1β (IL-1β), and IL-6. This "cytokine storm" can induce oxidative stress and apoptotic pathways in hepatocytes, leading to the observed leakage of enzymes (Tiegs and Horst, 2022). The current findings align with previous work by Thabet and Moustafa (2018), who reported similar significant increases in liver enzymes following CFA injection and attributed it to NF-κB-mediated inflammatory damage.

Dietary supplementation with licorice root powder markedly attenuated this increase in liver enzymes in a clear dose-dependent manner. The group receiving the 5% licorice diet showed significant reduction ($p < 0.05$) in enzyme levels compared to the diseased control (Ve+). This protective effect was even more pronounced in the 10% and 15% licorice groups. The most potent effect was observed in the 15% licorice group, which exhibited enzyme values (ALP: 112.49 U/l; AST: 84.33 U/l; ALT: 29.86 U/l) that approached those of the normal, healthy control group. The core finding of this study is the remarkable ability of licorice root powder to reverse this damage. The dose-dependent restoration of ALP, AST, and ALT levels towards normality indicates a stabilization of hepatocyte membranes and an overall reduction in hepatic inflammation. This hepatoprotective activity can be mechanistically explained by the rich phytochemistry of licorice, primarily its content of glycyrrhizin, glycyrrhetinic acid, and flavonoids like liquiritin and glabridin.

The anti-inflammatory properties of licorice are paramount. Glycyrrhizin is a known inhibitor of phospholipase A2 and cyclooxygenase (COX), thereby reducing the synthesis of prostaglandins and leukotrienes (Yang *et al*., 2017). More significantly, it has been shown to suppress the activation of the pivotal NF-κB signaling pathway. NF-κB is a master regulator of the expression of pro-inflammatory cytokines (TNF-α, IL-1β, IL-6) that are driving the secondary liver damage in arthritis (Wang *et al*., 2011). By dampening this systemic inflammatory response, licorice treatment reduces the primary insult to the liver. This mechanism is strongly supported by the work of Yang *et al*. (2017), who extensively reviewed the NF-κB inhibitory activity of licorice compounds as a central mode of action. The antioxidant capacity of licorice plays a crucial role. The systemic inflammation from CFA-induced arthritis generates an excess of reactive oxygen species (ROS), leading to oxidative stress and lipid peroxidation of hepatocyte membranes. The flavonoids in licorice are powerful free radical scavengers. Furthermore, glycyrrhetinic acid has been documented to upregulate the expression of endogenous antioxidant enzymes, such as superoxide

dismutase (SOD) and glutathione (GSH). The present results corroborate with those of Mohammadi *et al*. (2022), who found that licorice extract significantly increased GSH levels and decreased lipid peroxidation in a model of hepatotoxicity.

Table (1): Liver enzymes of experimental rats which are treated with licorice roots

Groups	Liver enzymes (U/I)		
	ALT	AST	ALP
Control (Ve-)	22.96 ^{g} ± 0.57	65.67 ^{g} ± 0.38	83.84 ^{g} ± 0.67
Control (Ve+)	60.26 ^{a} ± 0.58	150.18 ^{a} ± 0.69	171.12 ^{a} ± 0.60
5% licorice	48.77 ^{b} ± 0.88	110.62 ^{c} ± 0.68	154.85 ^{b} ± 1.43
10% licorice	32.43 ^{e} ± 0.65	92.94 ^{e} ± 0.93	120.36^{e} ± 0.50
15% licorice	29.86 ^{f} ± 0.39	84.33 ^{f} ± 0.69	112.49 ^{f} ± 0.76

The data is presented as mean ± SD (n=6). a,b,c and d: Means with different letters among groups in the same column show significant differences ($P < 0.05$), AST, aspartate amino transferase; ALT, Alanine amino transferase. ALP: serum alkaline phosphatase

Kidney function

The deterioration in renal function can be accelerated by systemic inflammation, as seen in autoimmune conditions like rheumatoid arthritis where inflammatory cytokines and autoimmune complexes can cause glomerular damage and tubular dysfunction (Yücel *et al*., 2017). The National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) emphasizes that these biomarkers are critical for early detection of kidney disease, especially in patients with inflammatory conditions such as rheumatoid arthritis that may secondarily affect renal function (National Kidney Foundation, 2018). The experimental induction of arthritis using Complete Freund's Adjuvant (CFA) resulted in significant impairment of kidney function, as evidenced by the marked elevation of all renal biomarkers measured. The Control (Ve+) group exhibited substantially increased levels of creatinine (0.61 ± 0.03 mg/dl), uric acid (3.26 ± 0.175 mg/dl), and urea (57.83 ± 1.34 mg/dl) compared to the healthy control group ($p < 0.05$) as seen in Table (2). This deterioration in renal function parameters can be attributed to the systemic inflammatory response triggered by CFA injection, which mimics the pathophysiology of human rheumatoid arthritis (Yücel *et al*., 2017).

The relationship between inflammatory arthritis and kidney damage is well-established in scientific literature. Rheumatoid arthritis is characterized by excessive production of pro-inflammatory cytokines including tumor necrosis factor-alpha (TNF-α), interleukin-1β (IL-1β), and interleukin-6 (IL-6), which can directly damage renal tissues and impair glomerular filtration (Yang *et al*., 2017). The collagen-induced arthritis (CIA) model, which shares similar pathogenesis with CFA-induced arthritis, has been shown to cause oxidative damage in kidney and liver tissues of experimental animals, further supporting our findings (Mazzali *et al*., 2001). The autoimmune response in arthritis leads to the formation of immune complexes that can deposit in glomeruli, causing inflammation and compromised renal function (Yücel *et al*., 2017).

As shown in Table (2) the administration of licorice root powder at varying concentrations (5%, 10%, and 15%) demonstrated a remarkable protective effect on kidney function in arthritic rats. Notably, the highest dose of licorice (15%) resulted in

the most significant improvement, with creatinine levels decreasing to 0.30 ± 0.09 mg/dl, uric acid to 2.20 ± 0.29 mg/dl, and urea to 32.33 ± 0.14 mg/dl. These values were not only significantly lower than those in the arthritis control group ($p < 0.05$) but were also comparable to or even better than the healthy control group, suggesting a potential therapeutic effect beyond mere protection against arthritis-induced renal damage (Öztürk *et al*., 2017).

The findings from this study have significant clinical implications for the management of kidney dysfunction in patients with rheumatoid arthritis and other inflammatory conditions. The demonstrated nephroprotective effects of licorice root powder suggest its potential as an adjunct therapy to conventional treatments for arthritis, potentially mitigating the renal complications that often accompany both the disease and its treatments. Many conventional medications for rheumatoid arthritis, including nonsteroidal anti-inflammatory drugs (NSAIDs) and disease-modifying antirheumatic drugs (DMARDs), can have adverse effects on kidney function, creating a need for protective strategies (Yang *et al*., 2017).

The results of this study demonstrate that licorice root powder effectively protects against arthritis-induced kidney dysfunction in a dose-dependent manner. The profound improvements in creatinine, uric acid, and urea levels with licorice supplementation, particularly at higher concentrations (10% and 15%), highlight its potential as a natural nephroprotective agent. The mechanisms underlying these benefits appear to involve both anti-inflammatory and antioxidant activities, primarily mediated through bioactive compounds such as glycyrrhizin and glycyrrhetinic acid (Öztürk *et al*., 2017).

These findings align with previous research by Nassiri and Hosseinzadeh (2015) on the protective effects of licorice against various forms of renal damage, including gentamicin-induced nephrotoxicity. The ability of licorice to mitigate both the articular and systemic manifestations of rheumatoid arthritis makes it a particularly promising therapeutic agent for this condition. However, careful attention to doing is essential, as excessive licorice consumption can lead to adverse effects such as hypertension and hypokalemia (Firestein and McInnes, 2017).

Table (2): Kidney function (mg/dl) of experimental rats which are treated with licorice roots.

Groups	Kidney function (mg/dl)		
	Urea	Uric Acid	Creatinine
Control (Ve-)	36.83dc±1.6	2.50b± 0.29	0.43c± 0.05
Control (Ve+)	57.83a±1.34	3.26a± 0.175	0.61ab±0.03
5% licorice	41.00bc±1.56	2.23bc± 0.20	0.40cd± 0.06
10% licorice	36.50d±2.75	2.15c± 0.122	0.30d± 0.08
15% licorice	32.33d±0.14	2.20bc± 0.29	0.30d± 0.09

The data is presented as mean ± SD (n=6). a,b,c and d: Means with different letters among groups in the same column show significant differences ($P < 0.05$).

Lipid Peroxidation

Data in Table (3) reveals significant variations in lipid peroxidation markers and antioxidant enzyme activities across different experimental groups. The control group injected with CFA (Ve+) shows the highest MDA levels (52.97 ± 0.04 µmol/L), indicating substantial lipid peroxidation

resulting from arthritis induction. This group also demonstrates the lowest SOD activity (0.90 ± 0.52 IU/L) and reduced CAT activity (10.69 ± 0.48 IU/L), suggesting impairment of the endogenous antioxidant defense system. MDA is a reactive aldehyde produced during the peroxidation of polyunsaturated fatty acids and serves as a reliable biomarker for oxidative stress-induced lipid damage (Ayala, *et al*., 2014). In RA, the synovial membrane is characterized by hyperplastic tissue with increased metabolic activity and oxygen consumption, leading to excessive production of reactive oxygen species (ROS) (Marnett, 1999). These ROS molecules attack cellular membranes, resulting in lipid peroxidation chain reactions that generate toxic aldehydes like MDA, which can further exacerbate inflammation and tissue damage (Jacobs and Marnett, 2007). SOD serves as the first line of defense against ROS by catalyzing the dismutation of superoxide radicals ($O_2^{\bullet -}$) into hydrogen peroxide (H_2O_2) and oxygen. CAT then converts H_2O_2 into water and oxygen, preventing the formation of highly reactive hydroxyl radicals (•OH) via the Fenton reaction. (Negre-Salvayre *et al*., 2010). The reduced activities of these enzymes in arthritis result from several factors, including enzyme inactivation by excessive ROS, transcriptional suppression due to inflammatory signaling, and consumption without adequate replenishment (Marnett, 1999).

In contrast, groups supplemented with licorice root powder, particularly at 10% concentration, exhibit significantly reduced MDA levels (43.10 ± 0.09 µmol/L). This remarkable reduction suggests that licorice components effectively interrupt the lipid peroxidation cascade in arthritic conditions. The flavonoids present in licorice, such as glabridin, licochalcone A, and isoliquiritigenin, possess potent antioxidant properties that enable them to scavenge free radicals and break the propagation phase of lipid peroxidation (Bradley *et al*., 2010). These compounds donate hydrogen atoms to lipid peroxyl radicals (LOO), forming stable products that cannot continue the chain reaction (Negre-Salvayre *et al*., 2010) and enhanced antioxidant enzyme activities (SOD: 1.34 ± 0.92 IU/L; CAT: 14.10 ± 0.43 IU/L). These findings suggest that licorice supplementation, especially at 10% concentration, effectively mitigates lipid peroxidation and enhances antioxidant capacity in arthritis-induced rats. Studies have shown that licorice flavonoids can activate Nrf2 signaling, leading to increased transcription of SOD, CAT, and other antioxidant enzymes (Niki, 2011).

Licorice (*Glycyrrhiza* species) contains numerous bioactive flavonoids that contribute to its antioxidant and anti-inflammatory properties. The most notable compounds include glabridin, licochalcone A, licochalcone B, isoliquiritigenin, and liquiritigenin (Bradley *et al*., 2010). These compounds possess phenolic structures that allow them to donate hydrogen atoms to free radicals, forming stable resonance-stabilized phenoxyl radicals that break the chain reaction of lipid peroxidation (Upston *et al*., 2003). Additionally, these flavonoids can chelate transition metals like iron and copper, preventing them from catalyzing Fenton reactions that generate highly reactive hydroxyl radicals (Ayala, *et al*., 2014). The findings of this study align with previous research on lipid peroxidation in rheumatoid arthritis and the antioxidant effects of licorice. The significantly elevated MDA levels in arthritic patients and animal models have been consistently reported (Jacobs and Marnett, 2007). A study published in Free Radical Research demonstrated that RA patients had significantly increased levels of various lipid peroxidation products,

including 4-HNE, MDA, and isoprostanes, along with decreased antioxidant capacity (Marnett, 1999). Similarly, the beneficial effects of licorice and its components observed in this study are supported by numerous preclinical investigations. For instance, glabridin has been shown to exhibit potent anti-arthritic effects by reducing paw swelling, improving histopathological scores, and modulating inflammatory cytokines in monosodium iodoacetate-induced osteoarthritic rats (Niki, 2011).

Table (3): Effect of licorice on the levels of MDA, GSH, and Catalase in rats induced by arthritis.

Groups	Lipid Peroxidation		
	CAT(IU/L)	SOD(IU/L)	MDA (µmol/L)
Control (Ve-)	14.71 ±1.50^{ab}	1.13 ± 0.82^{b}	47.74 ±0.05^{d}
Control (Ve+)	10.69 ± 0.48^{f}	0.90 ±0.52^{a}	52.97 ±0.04^{a}
5% licorice	11.55 ±1.19^{d}	1.07 ±0.81^{b}	48.71 ±0.05^{c}
10% licorice	14.10 ± 0.43^{b}	1.34 ± 0.92^{a}	43.10 ± 0.09^{f}
15% licorice	13.80 ±0.37^{c}	0.94 ±1.73^{c}	46.67 ± 0.12^{e}

The data is presented as mean ± SD (n=6). a,b,c and d: Means with different letters among groups in the same column show significant differences ($P < 0.05$), catalase (CAT), and malondialdehyde (MDA) superoxide dismutase (SOD)

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Effects of licorice root powder on lipid peroxidation and antioxidant status in arthritis-induced rats

تأثير مسحوق جذور عرق السوس على بيروكسيد الدهون والحالة المضادة للأكسدة في الفئران المصابة بالتهاب المفاصل

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المستخلص

التهاب المفاصل الروماتويدي (RA) هو اضطراب مناعي ذاتي جهوي يتميز بالالتهاب المزمن الذي يؤدي إلى تلف المفاصل التدريجي والإعاقة. تتضمن الإصابة بالمرض تفاعلات التهابية معقدة وترتبط ارتباطاً وثيقاً بالإجهاد التأكسدي و peroxidationالدهون؛ لذلك، أُجريت هذه الدراسة لتقييم تأثير مسحوق جذور العرقسوس على peroxidation الدهون والحالة المضادة للأكسدة في الفئران المُحفز لإصابتها بالتهاب المفاصل. تم وزن ثلاثين أنثى بالغة من فئران ويستار (210 ± 5 جراماً) وتوزيعها عشوائياً إلى مجموعتين رئيسيتين. المجموعة (1) ضابطة (-ve) (6 فئران) تلقت الغذاء القياسي فقط، بينما المجموعة الثانية (24 فأراً) حُفزت إصابتها بالتهاب المفاصل من خلال حقنة داخل الجلد من Complete Freund's Adjuvant (CFA)(0.1 مل) في باطن القدم الخلفية اليمنى في اليوم الأول. ثم قُسمت الفئران إلى خمس مجموعات فرعية، المجموعة(II) ، مجموعة التهاب المفاصل (+ve) تلقت الغذاء القياسي فقط. المجموعة (III) عولجت بـــ 5% عرقسوس / الغذاء يومياً. المجموعة (IV) عولجت بـ 10% عرقسوس / الغذاء يومياً. المجموعة (V) عولجت بـ 15% عرقسوس / الغذاء يومياً. أظهرت النتائج ملاحظة معنوية ($p \leq 0.05$) في مستويات المعايير الكيميائية الحيوية في المصل وانخفاضاً معنوياً في peroxidationالدهون فى كل المجموعات التى تم علاجها.

الكلمات المفتاحية: التهاب المفاصل، جذر العرقسوس، مضاد الأكسدة، الكبد، الكلى.