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Effect of honey on blood and biochemical parameters of rats with induced anemia

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Abstract

Objective

Anemia is a multifactorial biological, nutritional, and socio-environmental health problem characterized by a deficiency of red blood cells or hemoglobin, which are responsible for oxygen transport throughout the body's tissues. This deficiency results in symptoms such as fatigue and general weakness. Honey, a natural sweetener produced by bees from flower nectar, has been used since ancient times for its medicinal properties due to its rich content of vitamins and minerals. The present study aimed to evaluate the role of honey in mitigating anemia-induced damage in male rats.

Materials and Methods

Anemia was induced in rats using phenylhydrazine, obtained from a pharmacy in Baghdad, at a dose of 40 mg/kg body weight. The drug was administered via intraperitoneal injection at a volume of 1 mL. A total of 30 adult male rats were randomly assigned to three groups (n=10 per group). Erythrocyte count, hemoglobin concentration, and packed cell volume were measured. Additionally, malondialdehyde (MDA), serum glutathione (GSH), and catalase (CAT) levels were assessed. Serum ferritin and hepcidin concentrations were estimated using an ELISA kit. Statistical analyses were conducted using SPSS software.

Results

The results indicated a significant decrease in red blood cell count (RBC), hemoglobin (Hb), and packed cell volume (PCV) in the anemia-induced group (T1), whereas these parameters increased in the honey-treated group (T2). Furthermore, serum ferritin, hepcidin, and iron levels were

significantly reduced in the T1 group, measuring 45.3 ng/mL, 18.7 pg/mL, and 32.6 mg/L, respectively. In contrast, these values significantly increased in the T2 group, reaching 85.7 ng/mL, 35.4 pg/mL, and 72.3 mg/L, respectively. Thyroid-stimulating hormone (TSH) levels were significantly elevated in the T1 group (1.43 ng/mL), while triiodothyronine (T3) and thyroxine (T4) levels were reduced (0.68 ng/mL and 3.42 ng/mL, respectively). In the T2 group, TSH decreased to 0.85 ng/mL, while T3 and T4 increased to 1.06 ng/mL and 5.49 ng/mL, respectively. Moreover, MDA levels increased in the T1 group (8.76 μmol/L) and decreased in the T2 group (4.62 μmol/L), while GSH and CAT levels decreased in the T1 group (8.3 μmol/L and 18.3 μmol/L, respectively) but increased in the T2 group (18.5 μmol/L and 34.7 μmol/L, respectively). Statistically, significant differences were observed between the T1 and T2 groups compared to the control group ($p \leq 0.05$).

Conclusions

This study demonstrates that honey supplementation plays a beneficial role in mitigating anemia-induced damage in male rats. Rats treated with honey exhibited significant improvements in red blood cell count, hemoglobin concentration, and packed cell volume compared to the anemia-induced group. Additionally, honey supplementation increased ferritin, hepcidin, and iron levels, modulated thyroid hormone levels, reduced oxidative stress markers (MDA), and enhanced antioxidant defense mechanisms (GSH and catalase). These findings suggest that honey may have potential therapeutic effects in alleviating anemia and its associated biochemical alterations.

Keywords: anemia, blood and biochemical parameters, honey, rats

Paper Type: Research Paper.

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Introduction

Anemia is a significant public health concern, influenced by a complex interplay of nutritional, biological, and mechanical factors, as well as various physical, economic, social, behavioral, demographic, and environmental determinants of health (Raiten et al. 2023). Clinically, anemia is defined as a hemoglobin concentration lower than the normal range for a given age, sex, physiological status, and altitude or as an absolute reduction in the number of red blood cells, leading to inadequate oxygen delivery to tissues and impaired physiological function (WHO 2011).

Anemia can be classified based on either the underlying physiological process responsible for red blood cell deficiency or the physical characteristics of red blood cells, including size, color, and shape. From a physiological perspective, anemia arises from three primary mechanisms: blood loss, increased red blood cell destruction, and impaired red blood cell production. Blood loss may be associated with either acute or chronic diseases, whereas increased red blood cell destruction (hemolytic anemia) can result from internal or external factors. Internal factors include inherited genetic disorders such as sickle cell anemia and thalassemia, while external factors encompass immune-mediated reactions, such as hemolysis due to blood transfusion incompatibility. Additionally, mechanical forces can contribute to red blood cell destruction (York 2017). Honey is a natural product widely recognized for its medicinal properties. It contains approximately 200 bioactive substances, with fructose and glucose as its primary components. Honey also contains fructooligosaccharides, amino acids, vitamins, minerals, and enzymes, with its composition varying depending on the floral source. Most natural honeys contain flavonoids (e.g., apigenin, pinocembrin, kaempferol, quercetin, galangin, chrysin, and hesperetin), phenolic acids (e.g., ellagic acid, caffeic acid, p-coumaric acid, and ferulic acid), ascorbic acid, tocopherols, catalase (CAT), superoxide dismutase (SOD), reduced glutathione (GSH), Maillard reaction products, and peptides (Bahador et al. 2016). These compounds interact synergistically to enhance honey's antioxidant properties. Phytobiotics and medicinal plants, which are used as natural antimicrobial growth promoters in place of antibiotics, have been shown to offer numerous benefits, including improved efficiency parameters, suppression of specific diseases (Amirteymoori et al. 2021; Mohammadabadi et al. 2023), antimicrobial and antioxidant activities (Hajalizadeh et al. 2019; Jafari Ahmadabadi et al. 2023), hypocholesterolemic effects, enhanced digestive enzyme activity, and improved liver function (Safaei et al. 2022; Shokri et al. 2023; Mohammadabadi et al. 2024). Studies have demonstrated that dietary supplementation with these plants enhances feed efficiency (Vahabzadeh et al. 2020; Shokri et al. 2023). Since honey is derived from nectar collected from various medicinal plants, it retains many of their beneficial properties. Nutritional deficiencies are a major cause of anemia; therefore, adequate intake of

essential vitamins and minerals, along with proper supplementation, plays a crucial role in prevention and treatment. Nutrients essential for anemia management include iron, folic acid, vitamin B12, vitamin B6, vitamin C, and protein. Honey has been identified as a natural source of these nutrients (Kiasari et al. 2020; Widowati 2023). It contains vitamins A, C, E, B12, beta-carotene, flavonoids, and iron, all of which contribute to hemoglobin synthesis and reduce oxidative stress. Additionally, honey enhances iron absorption due to its vitamin content. Other essential components of honey, including calcium, phosphorus, potassium, sodium, and enzymes, support various physiological functions. As a readily absorbable and nutrient-rich substance, honey has been associated with numerous health benefits, such as blood pressure regulation, enhanced energy levels, cardiovascular and muscular health, and disease prevention. Traditionally, honey has also been used to alleviate cough, abdominal pain, and digestive issues and to strengthen bones, teeth, and the nervous system (Asrida et al. 2022).

Emerging research suggests that honey may play a role in improving anemia. One study investigated the effects of daily honey consumption (1.2 g/kg body weight) in humans and found that honey supplementation increased serum iron levels, enhanced antioxidant activity, and improved hematological indices. Additionally, the study observed reductions in certain liver and muscle enzymes as well as fasting blood sugar levels (Al-Waili et al. 2006). Animal studies further support the potential role of honey in anemia management. In an experiment involving albino rats, those fed raw whole milk supplemented with 20% honey exhibited elevated hemoglobin levels compared to controls receiving milk with 16% sucrose. Notably, dark honey was more effective in maintaining hemoglobin levels than light honey, which resulted in a significant decline of approximately 30% from baseline. The hemoglobin levels in sucrose-fed controls also progressively decreased. These findings suggest that dark honey may be beneficial for the prevention and treatment of nutritional anemia, whereas light honey appears to be less effective as a hematopoietic agent (Haydak et al. 1942).

Anemia is a prevalent health issue during pregnancy, often characterized by low hemoglobin levels. Research has examined the potential of honey to improve maternal hemoglobin status. In one study, honey administration during the second trimester resulted in increased hemoglobin levels, suggesting that honey consumption may support maternal hematological health (Sianturi et al. 2022). Further supporting this, Hotima et al. (2022) identified anemia as a global health concern, particularly among pregnant women. Their study examined the effects of *Acacia crassicarpa* honey, which is rich in iron, on pregnant women diagnosed with anemia in the third trimester. Participants consumed honey for 15 days, leading to an increase in average hemoglobin levels from 9.4 g/dL to 12.6 g/dL. Based on these findings, the authors concluded that honey, due

to its rich vitamin and mineral composition, particularly its iron content, may effectively elevate hemoglobin levels in pregnant women. Given the growing body of evidence supporting honey's nutritional and medicinal properties, this study aimed to evaluate the effects of honey administration on hematological and biochemical parameters in an experimentally induced anemia model in rats.

Materials And methods

Chemicals: Phenylhydrazine (PHZ), used to induce anemia in rats, was obtained from a pharmacy in Baghdad. A dose of 40 mg/kg body weight was administered via intraperitoneal (I.P.) injection of 1 mL per animal (Diallo et al. 2008). Buckthorn honey was sourced from an apiary in Samawah, with a dosage of 500 mg/kg body weight (Abd Ali & Ismail 2012). The honey was dissolved in distilled water, and each animal received 1 mL orally per day using a specialized syringe with a hooked end.

Experimental Animals: This study was conducted in the animal house of the Faculty of Science, Al-Qadisiyah University, Iraq, from October 1, 2024, to December 1, 2024. A total of 30 male albino rats (200-270 g, 3-4 months old) were housed in plastic cages equipped with 500 mL water bottles. The cages contained sawdust bedding, which was replaced and disinfected every three days. The animals were maintained at a temperature of 23-27°C under a 12-hour light/dark cycle. Before the experiment, they were acclimatized for one week.

Experimental Design: The rats were randomly assigned to three groups (n = 10 per group):

- Group I (Control, C): Received 0.9% saline solution and served as the control group.
- Group II (T1): Anemia was induced by PHZ injection at 40 mg/kg body weight for two consecutive days.
- Group III (T2): Anemic rats (PHZ-induced) received honey treatment for 30 days. The scientific dose per kg body weight was calculated as follows: Scientific dose/kg body weight = required dose/animal weight.

Chemical Composition of Honey: Key components of the honey used in this study were: Iron (26.15 ppm), Magnesium (19.04 ppm), Calcium (169.02 ppm), Potassium (247.14 ppm), Zinc (1.27 ppm), Vitamin C (251 mg/100 g), and Gallic acid (21.45 mg/g).

Sample Collection: After 30 days, the animals were sacrificed. Anesthesia was induced using a combination of ketamine (0.3 mL/kg) and xylazine (1.0 mL/kg) via I.P. injection. Blood samples were collected via cardiac puncture and divided into two portions:

1. One portion was placed in tubes containing an anticoagulant for hematological analysis.

2. The other portion was placed in anticoagulant-free tubes, inclined, and centrifuged at 3000 rpm for 15 minutes to obtain serum. The serum samples were stored at -20°C until biochemical, antioxidant, and hormonal analyses were performed.

Measured Parameters-Hematological Parameters: Erythrocyte count, hemoglobin concentration, and packed cell volume were assessed using an automated hematology analyzer (GENEX), following the manufacturer's instructions.

Iron Parameters: Serum ferritin and hepcidin concentrations were measured using an ELISA kit (BioSystem, USA). Serum iron concentration was determined spectrophotometrically using BioSystem's kit, based on the reaction of ferrous ions with bipyridine, forming pink complexes, with absorbance measured at 520 nm.

Hormonal Parameters: Thyroid hormones (T3, T4, and TSH) were quantified using an Enzyme-Linked Immunosorbent Assay (ELISA) based on the method of Wisdom (1976), with absorbance recorded at 450 nm. Kits were obtained from Biocheck, Inc.

Antioxidant Parameters:

- **Malondialdehyde (MDA):** Serum MDA levels were determined following the method of Guidet & Shah (1989). Lipid peroxidation was assessed by measuring the reaction of MDA with thiobarbituric acid (TBA) in an acidic medium, producing a colored product with absorbance measured at 532 nm.
- **Glutathione (GSH):** Serum GSH levels were estimated using Ellman's reagent, following the modified method of James et al. (1982). The reaction of Ellman's reagent with GSH forms a colored product, measured at 412 nm.
- **Catalase (CAT):** Serum catalase activity was determined using the method described by Mueller et al. (1997), based on the decomposition of hydrogen peroxide (H_2O_2) into water and oxygen, with absorbance reduction used to quantify activity.

Statistical Analysis: Data were analyzed using SPSS software (Version 25.0, IBM 2017). One-way ANOVA and the Least Significant Difference (LSD) test were used to compare groups, with statistical significance set at $p < 0.05$.

Results

Blood Parameters: The results of the present study (Table 1) indicate a significant decrease in red blood cell (RBC) count, hemoglobin (Hb) levels, and packed cell volume (PCV%) in the anemia-induced T1 group compared to the control group. However, these parameters showed a significant increase in the T2 group compared to the T1 group. Moreover, a statistically significant

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difference was observed between the T1 and T2 groups when compared to the control group ($p \leq 0.05$).

Table 1. Effect of honey on blood parameters in male albino rats with PHZ-induced anemia

Groups	RBC (10^{12}/L)	Hemoglobin (g/L)	PCV (%)
Group C	7.24±0.05 [a]	13.7±0.23 [a]	41.82±0.09 [a]
Group T1	4.10±0.08 [c]	7.7±0.07 [c]	27.4±0.13 [c]
Group T2	5.76±0.05 [b]	11.38±0.06 [b]	36.42±0.11 [b]
LSD	0.512	0.352	0.895

Iron Parameters: The results presented in Table 2 indicate a significant decrease in ferritin, hepcidin, and iron levels in the T1 group, where anemia was induced. The observed values were 45.3 ng/mL, 18.7 pg/mL, and 32.6 mg/L, respectively, compared to the control group. However, in the T2 group, these parameters increased to 85.7 ng/mL, 35.4 pg/mL, and 72.3 mg/L, respectively, showing a significant improvement relative to the T1 group. Furthermore, significant differences were observed between the T1 and T2 groups when compared to the control group ($p \leq 0.05$).

Table 2. Effect of honey on ferritin, hepcidin, and iron levels in male albino rats with PHZ-induced anemia

Groups	Ferritin ng/mL Means± SE	Hepcidin pg/mL Means± SE	Iron mg/L Means± SE
Group C	125.6±10.2 [a]	42.6±0.45 [a]	95.4±8.2 [a]
Group T1	45.3±7.5 [c]	18.7±0.12 [c]	32.6±5.7 [c]
Group T2	85.7±8.9 [b]	35.4±0.17 [b]	72.3±6.5 [b]
LSD	4.251	2.014	3.562

Hormonal Parameters: The results presented in Table 3 indicate a significant increase in TSH levels and a decrease in T3 and T4 levels in the T1 group, where anemia was induced. Specifically, TSH, T3, and T4 levels were recorded as 1.43 ng/mL, 0.68 ng/mL, and 3.42 ng/mL, respectively, compared to the control group. In contrast, the T2 group exhibited a decrease in TSH levels (0.85 ng/mL) and an increase in T3 (1.06 ng/mL) and T4 (5.49 ng/mL) levels relative to the T1 group. Furthermore, the results demonstrated significant differences between the T1 and T2 groups compared to the control group ($p \leq 0.05$).

Table 3. Effect of honey on T3, T4, and TSH levels in male albino rats with PHZ-induced anemia

Groups	T3 ng/mL Means± SE	T4 ng/mL Means± SE	TSH ng/mL Means± SE
Group C	1.45±0.12 [a]	6.87±0.45 [a]	0.65±0.08 [c]
Group T1	0.68±0.09 [c]	3.42±0.32 [c]	1.43±0.15 [a]
Group T2	1.06±0.11 [b]	5.49±0.38 [b]	0.85±0.11 [b]
LSD	0.0551	0.324	0.0145

Antioxidant Parameters: The results presented in Table 4 indicate a significant increase in malondialdehyde (MDA) levels and a decrease in glutathione (GSH) and catalase (CAT) activity in the T1 group, where anemia was induced. Specifically, MDA, GSH, and CAT levels in the T1 group were 8.76 µmol/L, 8.3 µmol/L, and 18.3 µmol/L, respectively, compared to the control group. However, in the T2 group, MDA levels significantly decreased to 4.62 µmol/L, while GSH and CAT levels increased to 18.5 µmol/L and 34.7 µmol/L, respectively, when compared to the T1 group. Additionally, significant differences ($p \leq 0.05$) were observed between the T1 and T2 groups in comparison to the control group.

Table 4. Effect of honey on MDA, GSH, and catalase levels in male albino rats with PHZ-induced anemia

Groups	MDA µmol/L Means± SE	GSH (µmol/L) Means± SE	Catalase µmol/L Means± SE
Group C	2.45±0.22 [c]	24.6±2.1 [a]	42.6±3.5 [a]
Group T1	8.76±0.67 [a]	8.3±0.7 [c]	18.3±1.6 [c]
Group T2	4.62±0.41 [b]	18.5±1.6 [b]	34.7±2.9 [b]
LSD	0.754	1.478	1.044

Discussion

The results indicate that rats in the T1 group, treated with phenylhydrazine (PHZ) to induce anemia, exhibited decreased blood parameters, antioxidant levels, and thyroid hormones (Tables 1, 2, 3, and 4). PHZ is commonly used to induce anemia and oxidative stress in experimental animal models (Lee et al. 2014; Onyeabo et al. 2017). It causes hemolysis by oxidizing hemoglobin, leading to the formation of unstable intermediates such as methemoglobin, a form of hemoglobin incapable of binding oxygen. Consequently, this reduces the oxygen-carrying capacity of red blood cells (Shukla et al. 2012). Furthermore, the degradation of these unstable

intermediates generates reactive oxygen species (ROS), which cause oxidative damage to red blood cells and their membranes, ultimately leading to hemolysis. These free radicals initiate an oxidation-reduction cycle, reacting with oxygen to form superoxide anions, hydrogen peroxide, and other ROS (Adwas et al. 2019). Such reactive oxygen species can damage cellular macromolecules, including lipids, proteins, and DNA, leading to cellular dysfunction and destruction (Chinko et al. 2023). PHZ-induced oxidative stress also depletes endogenous antioxidant enzymes responsible for ROS neutralization (Banerjee et al. 2020).

A study by Unami et al. (1996) demonstrated that subchronic PHZ exposure (10 mg/kg per day for 8 days) induces severe hemolytic anemia, characterized by a significant decrease in erythrocyte count, hemoglobin levels, and packed cell volume (PCV). Phenylhydrazine-induced hemolysis leads to iron deficiency, thereby increasing the demand for erythropoietic iron. This process is expected to suppress hepcidin expression in the liver and enhance ferroportin expression, promoting intestinal iron absorption and iron recycling within the reticuloendothelial (RE) system (Latunde-Dada et al. 2004). Another study suggested that PHZ-induced anemia results from oxidative lipid damage in erythrocyte membranes, possibly due to the self-oxidation of the drug and the interaction of oxygen free radicals with membrane lipids (Jain & Subrahmanyam 1978).

PHZ has been shown to generate superoxide anions and hydrogen peroxide, leading to lipid oxidation and Heinz body formation (Shukla et al. 2012). Its toxicity is primarily linked to oxidative stress within erythrocytes (Kinuta et al. 1995). Since PHZ elevates hydrogen peroxide levels beyond the detoxification capacity of glutathione and catalase, it exacerbates oxidative stress, particularly in individuals with genetic deficiencies or aging-related reductions in glucose-6-phosphate dehydrogenase activity (Hochstein 1971). PHZ-induced oxidative stress may also impair thyroid function by inhibiting deiodinase secretion, suppressing T3 receptor activity, and degrading thyroxine-binding globulin (TBG) (Simpson 2009). Bhimte et al. (2012) suggested that reduced thyroid activity correlates with increased oxidative stress and decreased antioxidant levels, which negatively impacts mitochondrial function and cellular metabolism, thereby reducing thyroid hormone levels. The results further showed that anemia-induced reductions in blood indices and iron levels in the T1 group were reversed upon honey supplementation (T2 group) (Tables 1 and 2). This effect may be attributed to honey's iron, copper, zinc, and magnesium content. Iron is essential for hemoglobin synthesis, as it binds to protoporphyrin to form heme, a crucial component of erythrocytes (Al-Waili et al. 2006; Singh & Singh 2018). Additionally, zinc, a component of honey, enhances vitamin activity involved in red blood cell formation. Zinc also promotes hemoglobin and erythropoietin synthesis, thereby improving erythropoiesis and protecting erythrocytes from oxidative degradation (Chen et al. 2018).

The improved hematological parameters and increased antioxidant levels in the T2 group may also be attributed to magnesium, another essential mineral found in honey. Magnesium enhances erythropoiesis, increases reticulocyte formation, reduces oxidative stress, and strengthens immune function. Furthermore, magnesium activates vitamin D, which is essential for erythropoietic progenitor cell proliferation and stabilizes cellular function by maintaining antioxidant defenses and erythrocyte membrane integrity (Son et al. 2007). Honey's ability to donate electrons to ROS stabilizes red blood cell membranes, preventing oxidative damage. The observed increase in white blood cell (WBC) count in the T2 group suggests that honey promotes immune function, as it is a rich source of iron, zinc, and vitamin C, all of which are essential for WBC production and immune cell function (Chinko & Umeh 2023). The present findings align with studies demonstrating that oral honey administration significantly enhances PCV, hemoglobin, RBC, WBC, and neutrophil counts in experimental groups compared to untreated controls. These effects appear to be dose-dependent, as honey ingestion before PHZ exposure likely enhances hematopoiesis due to its mineral and vitamin content (James et al. 2009).

The results also indicated increased thyroid hormone levels (T3, T4, and TSH) in the T2 group (Table 3). This observation aligns with Al-Samarraie and Hommadi (2021), who reported that honey supplementation increased thyroid hormone levels by up to 75%, particularly after three weeks of administration. Since hypothyroidism is associated with iron deficiency anemia, iron supplementation can restore hormonal balance (Gökdeniz et al. 2010). Iron plays a crucial role in thyroid function by supporting the activity of thyroid peroxidase (TPO), an iron-containing enzyme that catalyzes the initial steps of thyroid hormone synthesis (Zimmermann 2006). Iron deficiency reduces TPO efficacy, impairing thyroid hormone synthesis (Gökdeniz et al. 2010). Studies in humans confirm that individuals with iron deficiency anemia have lower T3 and T4 levels, which normalize upon iron supplementation (Eftekhari et al. 2006). Therefore, honey's iron and mineral content may contribute to the observed improvement in thyroid function. The mechanisms underlying honey's effects on thyroid function include stimulation of the hypothalamic-pituitary-thyroid (HPT) axis, antioxidant and anti-inflammatory properties, enhancement of deiodinase activity, modulation of gut microbiota, and reduction of stress-induced cortisol levels, all of which collectively support thyroid hormone production and metabolism. The study further demonstrated that honey supplementation enhanced antioxidant capacity (Table 4), consistent with findings that honey reduces malondialdehyde (MDA) levels while increasing superoxide dismutase (SOD), catalase (CAT), and glutathione (GSH) levels in experimental groups (Peláez-Acero et al. 2022). Oxidative stress occurs when ROS production surpasses antioxidant defense capacity, leading to lipid peroxidation and cellular damage. PHZ

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stimulates excessive ROS generation, disrupting oxidative balance and causing tissue injury. Elevated MDA levels in PHZ-exposed animals indicate oxidative damage, whereas honey's antioxidant properties mitigate this effect. Increased antioxidant enzyme activity in the T2 group supports honey's protective role against PHZ-induced oxidative stress, likely due to its flavonoid content, which stabilizes red blood cell membranes and neutralizes extracellular oxidants (Fiorani et al. 2006). Several studies have emphasized honey's effectiveness in counteracting chemical-induced oxidative stress, including that caused by cadmium, gentamycin, and acetaminophen (Laaroussi et al. 2021; Mahesh et al. 2009; Ruslee et al. 2020).

Conclusion: This study concludes that honey supplementation in the T2 group improved hematological parameters (RBC, Hb, MCV, MCH, MCHC, PCV, and platelet count), iron status (ferritin, hepcidin, and iron levels), thyroid hormone levels (T3, T4, and TSH), and antioxidant capacity (MDA, GSH, and catalase) compared to the anemic T1 group. These findings suggest that honey has potential therapeutic benefits in mitigating anemia-induced damage, enhancing hematopoiesis, modulating thyroid function, and reducing oxidative stress, making it a promising natural intervention against anemia and associated metabolic disturbances.

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مجله بیوتکنولوژی کشاورزی
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تأثیر عسل بر پارامترهای خونی و بیوشیمیایی موش‌های صحرایی مبتلا به کم خونی ناشی از آن

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چکیده

هدف: کم خونی یک مشکل بیولوژیکی، تغذیه ای و سلامتی اجتماعی–محیطی چند عاملی است که با کمبود گلبول‌های قرمز خون یا هموگلوبین، که مسئول انتقال اکسیژن در سراسر بافت های بدن هستند مشخص می‌شود. این کمبود منجر به علائمی مانند خستگی و ضعف عمومی می‌شود. عسل یک شیرین کننده طبیعی است که توسط زنبورهای عسل از شهد گل تولید می‌شود و به دلیل داشتن ویتامین‌ها و مواد معدنی غنی از زمان‌های قدیم برای خواص دارویی آن استفاده می‌شده است. مطالعه حاضر با هدف بررسی نقش عسل در کاهش آسیب‌های ناشی از کم خونی در موش‌های صحرایی نر انجام شد.

مواد و روش‌ها: کم خونی با استفاده از فنیل هیدرازین که از داروخانه‌ای در بغداد با دوز ۴۰ میلی گرم بر کیلوگرم وزن بدن تهیه شده بود، در موش‌ها ایجاد شد. این دارو از طریق تزریق داخل صفاقی در حجم ۱ میلی لیتر تجویز شد. در مجموع ۳۰ موش صحرایی نر بالغ به طور تصادفی در سه گروه (۱۰ موش در هر گروه) قرار گرفتند. تعداد گلبول‌های قرمز، غلظت هموگلوبین و حجم سلول‌های متراکم اندازه گیری شد. علاوه بر این، سطوح مالون دی آلدئید (MDA)، گلوتاتیون سرم (GSH) و کاتالاز (CAT) مورد ارزیابی قرار گرفت. غلظت فریتین و هپسیدین سرم با استفاده از کیت الایزا برآورد شد. تجزیه و تحلیل‌های آماری با استفاده از نرم افزار SPSS انجام شد.

نتایج: نتایج نشان‌دهنده کاهش معنی‌دار تعداد گلبول‌های قرمز (RBC)، هموگلوبین (Hb) و حجم سلول‌های متراکم (PCV) در گروه ناشی از کم‌خونی (T1) بود، در حالی که این پارامترها در گروه تحت درمان با عسل (T2) افزایش یافت. علاوه بر این، سطوح فریتین، هپسیدین و آهن سرم به ترتیب در گروه T1 برابر ۴۵/۳ نانوگرم در میلی لیتر، ۱۸/۷ پیکوگرم در میلی لیتر و ۳۲/۶

میلی گرم در لیتر به دست آمد که به طور قابل توجهی کاهش یافت. در مقابل، این مقادیر به طور قابل توجهی در گروه T2 افزایش یافت و به ترتیب به ۸۵/۷ نانوگرم در میلی لیتر، ۳۵/۴ پیکوگرم در میلی لیتر و ۷۲/۳ میلی گرم در لیتر رسید. سطوح هورمون محرک تیروئید (TSH) به طور قابل توجهی در گروه T1 افزایش یافت (۱/۴۳ نانوگرم در میلی لیتر)، در حالی که سطوح تری یدوتیرونین (T3) و تیروکسین (T4) (به ترتیب ۰/۶۸ نانوگرم در میلی لیتر و ۳/۴۲ نانوگرم در میلی لیتر) کاهش یافت. در گروه T2، TSH به ۰/۸۵ نانوگرم در میلی لیتر کاهش یافت، در حالی که T3 و T4 به ترتیب به ۱/۰۶ نانوگرم در میلی لیتر و ۵/۴۹ نانوگرم در میلی لیتر افزایش یافت. علاوه بر این، سطوح MDA در گروه T1 افزایش یافت (۸/۷۶ میکرومول در لیتر) و در گروه T2 کاهش یافت (۴/۶۲ میکرومول در لیتر)، در حالی که سطوح GSH و CAT در گروه T1 کاهش یافت (به ترتیب ۸/۳ میکرومول در لیتر و ۱۸/۳ میکرومول در لیتر) اما در گروه T2 (به ترتیب ۱۸/۵ و ۳۴/۷ میکرومول در لیتر) افزایش یافت. از نظر آماری تفاوت معنی داری بین گروه های T1 و T2 نسبت به گروه کنترل مشاهده شد ($p\leq0.05$).

نتیجه‌گیری: این مطالعه نشان می‌دهد که مکمل عسل نقش مفیدی در کاهش آسیب ناشی از کم خونی در موش‌های صحرایی نر دارد. موش‌های تحت درمان با عسل در مقایسه با گروه ناشی از کم خونی، پیشرفت‌های قابل توجهی در تعداد گلبول‌های قرمز، غلظت هموگلوبین و حجم سلول‌های متراکم نشان دادند. علاوه بر این، مکمل عسل باعث افزایش سطح فریتین، هپسیدین و آهن، تعدیل سطح هورمون تیروئید، کاهش نشانگرهای استرس اکسیداتیو (MDA) و افزایش مکانیسم‌های دفاعی آنتی اکسیدانی (GSH و کاتالاز) شد. این یافته‌ها نشان می‌دهد که عسل ممکن است اثرات درمانی بالقوه‌ای در کاهش کم خونی و تغییرات بیوشیمیایی مرتبط با آن داشته باشد.

واژه‌های کلیدی: پارامترهای خونی و بیوشیمیایی، عسل، کم خونی، موش صحرایی

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