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STATE OF REDOX FORMS OF HEPATIC UBIQUINONE UNDER DRUG-INDUCED LIVER INJURY AND PREVENTIVE ADMINISTRATION OF ETHANOLIC EXTRACT OF *HERICIUM FLAGELLUM*

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The study is devoted to determining the levels of ubiquinone redox forms and the activity of ubiquinol:cytochrome c oxidoreductase in liver mitochondria of animals administered an ethanolic extract of Hericium flagellum against the background of paracetamol-induced intoxication. Four experimental groups of animals were used in the study: C – intact rats; ATI – rats administered a toxic dose of acetaminophen (1250 mg/kg orally as a 2% starch gel) for two consecutive days; EEHF – animals that received the ethanolic extract of Hericium flagellum at a dose of 200 mg/kg body weight for 10 days; EEHF+ATI – rats that received the Hericium flagellum extract (200 mg/kg) for 10 days prior to acetaminophen administration. It was established that paracetamol administration leads to a significant decrease in the total coenzyme Q_{10} (CoQ_{10}) pool. Simultaneously, the pool of the reduced form of ubiquinone – ubiquinol – is virtually depleted, reflecting the development of severe oxidative stress. Concurrently, an elevated level of the oxidized form of ubiquinone is observed, which may result in dysfunction of the mitochondrial respiratory chain. It was shown that under conditions of toxic liver injury, ubiquinol:cytochrome c oxidoreductase activity decreases, potentially leading to a reduced efficiency of oxidative phosphorylation. The administration of Hericium flagellum extract promotes the restoration of total CoQ_{10} levels and an increase in ubiquinol content, indicating a pronounced antioxidant and membrane-stabilizing effect. Furthermore, the decrease in the level of oxidized ubiquinone upon administration of the fungal extract suggests an improvement in the functional state of the mitochondrial respiratory chain. The obtained results demonstrate the ability of Hericium flagellum extract to restore energy metabolism and the redox state of coenzyme Q_{10} under conditions of acetaminophen-induced toxic liver injury, substantiating the expediency of further research into the mechanisms of its biological activity.

Keywords: liver, mitochondria, acetaminophen, Hericium flagellum, CoQ_{10} redox forms, ubiquinol:cytochrome c oxidoreductase

Introduction. Acetaminophen (paracetamol, APAP)-induced liver injury represents one of the most prevalent causes of drug-induced toxic damage (McGill, Hinson, 2020). The acute toxic effect of this drug on hepatocytes directly depends on the administered dose and exposure duration. The development of paracetamol-induced toxicity typically stems from dosing errors, accidental ingestion, or intentional exceeding of the therapeutic threshold (Li et al., 2025). Acetaminophen overdose leads to hepatocyte damage and cell death mediated by the activation of a complex, multi-stage process that remains incompletely understood (Liao et al., 2023). This process is characterized by the metabolic bioactivation of APAP into reactive intermediates, depletion of endogenous antioxidant defenses, pronounced mitochondrial dysfunction, and impairment of cellular membrane integrity (Tsuchiya et al., 2018).

Coenzyme Q (ubiquinone) serves as a key lipophilic link in the mitochondrial redox system, functioning as a mobile electron carrier within the electron transport chain (Guerra, Pagliarini, 2023). Beyond its role in oxidative phosphorylation reactions, ubiquinone exhibits potent antioxidant properties by preventing lipid peroxidation of mitochondrial membranes. According to scientific literature, this compound is capable of counteracting the effects of oxidative stress and suppressing inflammatory cascades. Such properties underscore the therapeutic potential of ubiquinone for the preventive treatment of hepatotoxicity, particularly in acetaminophen poisoning (Fouad, Jresat, 2012). Concurrently, the ratio of reduced to oxidized forms of ubiquinone can be considered an informative indicator of the mitochondrial redox state (Voloshchuk et al., 2020).

A promising approach to addressing redox

imbalance involves the application of bioactive molecules of natural origin, particularly those isolated from fungi (Łysakowska et al., 2023). This unique kingdom of nature encompasses an estimated 2.2 to 3.8 million species, with more than 200 officially recognized as medicinal. Since fungi produce a rich spectrum of secondary metabolites possessing potent anti-inflammatory and antioxidant effects, mycological extracts are actively investigated as agents for liver tissue protection and restoration (Soares et al., 2013).

Among medically significant macromycetes, *Hericium flagellum* (a member of the Hericiaceae family) is of particular scientific interest. This species is characterized by a high content of bioactive molecules, among which polysaccharide complexes, phenolic compounds, and terpenoid derivatives play a key role (Gonkhom et al., 2021). The antioxidant and cytoprotective effects of its extracts are realized through the regulation of cellular redox status and control over signaling cascades. As this fungus is a rich source of β-glucans and other immunotropic metabolites, studying its therapeutic potential as a prospective protective agent against oxidative liver damage under conditions of acetaminophen intoxication is well substantiated (Qi et al., 2024).

Therefore, evaluating the balance between oxidized and reduced forms of ubiquinone in the liver of animals subjected to acetaminophen overdose and subsequent administration of *Hericium flagellum* extract represents a timely and scientifically grounded task. This will allow for a deeper understanding of mitochondrial antioxidant system function and expand opportunities for identifying effective natural compounds to alleviate redox imbalance.

The aim of this work was to evaluate the redox state of ubiquinone and the enzymatic activity of ubiquinol:cytochrome *c* oxidoreductase in hepatocyte mitochondria of animals under acute paracetamol intoxication and following treatment with an ethanolic extract of the macromycete *Hericium flagellum*.

Materials and methods. The study was conducted on outbred white male rats weighing 120–150 g (aged 2.5–3 months). Experiment design, animal care, and all handling procedures were performed in strict compliance with the provisions of the *European Convention for the Protection of Vertebrate Animals used for Experimental and other Scientific Purposes* (Strasbourg, 1986) and national guidelines for bioethical expertise in preclinical studies (Kyiv, 2006). Laboratory animals were housed in plastic cages with sand bedding and had free access to drinking water (*ad libitum*) (Voloshchuk, Schneider, 2025).

The ethanolic extract of the macrofungus was obtained according to a previously described protocol. Briefly, to prepare a 70% ethanolic extract, 5 g of powdered raw material was mixed with 50 mL of 70% ethanol. The mixture was vigorously stirred and incubated at room temperature on an orbital shaker (150 rpm) for 24 h. Subsequently, the solution was centrifuged at 12,000 rpm for 15 min; the supernatant was filtered through Whatman filter paper, and the remaining pellet was subjected to re-extraction under identical conditions. The combined extracts were pooled and concentrated under reduced pressure at 40 °C using a Labfreez RE-2000E rotary evaporator. The resulting dry/concentrated extract was stored in the dark at 4 °C (Boonsong et al., 2016). For oral administration, working solutions were prepared to deliver a dosage of 200 mg/kg body weight.

Animals were randomly assigned into four experimental groups: control group (C) – intact control; toxic injury group (ATI) – isolated acetaminophen-induced toxicity; *Hericium flagellum* extract group (EEHF) – animals received the extract alone (200 mg/kg, *per os*, for 10 consecutive days); preventive treatment group (EEHF+ATI) – animals received the extract (200 mg/kg, *per os*, for 10 days) prior to the induction of liver damage.

Hepatic toxicity was induced by intragastric administration of acetaminophen via a gavage tube (twice, orally, at a dose of 1250 mg/kg). A 2% starch gel suspension served as the vehicle (Gao et al., 2017).

The hepatic mitochondrial fraction was isolated by differential centrifugation at 0–4 °C. All preparation and isolation steps were carried out using pre-chilled reagents and instruments.

The levels of total and oxidized coenzyme Q in rat liver mitochondria were evaluated spectrophotometrically based on the absorbance of the oxidized form at λ=275 nm. Following lipid extraction from mitochondria using an organic solvent mixture and removal of protein components, the samples were analyzed in quartz cuvettes with a 1-cm optical path length. Calculations were performed using a molar extinction coefficient of 12.25 $mM^{-1}cm^{-1}$. The concentration of reduced ubiquinol was determined mathematically as the difference between total coenzyme Q and oxidized ubiquinone levels (Voloshchuk et al., 2020).

The enzymatic activity of mitochondrial respiratory chain Complex III (ubiquinol:cytochrome *c* oxidoreductase) was measured spectrophotometrically. The incubation mixture contained 50 mM Tris-HCl, 4 mM sodium azide (NaN_3), 0.05% Tween 20, and 0.1 mg/mL

bovine serum albumin (BSA). The reaction was initiated by sequential addition of 0.0625 mM cytochrome c, 0.25 mM decylubiquinol, and 100 µL of isolated mitochondrial suspension (normalized to 1 mg of protein). The rate of decylubiquinol-dependent cytochrome c reduction was monitored by tracking changes in absorbance at λ=550 nm (Claus et al., 2013).

Statistical processing of the experimental data was carried out using Microsoft Excel tools. Data from nine independent measurements (n=9) were expressed as mean ± standard error of the mean (M±SEM). Intergroup differences were analyzed using Student's t-test, with the threshold of statistical significance established at $p<0.05$.

Results and discussion. Modelling acetaminophen-induced hepatotoxicity is a widely established experimental approach for inducing oxidative imbalance and mitochondrial homeostasis disruption (Jaeschke, Ramachandran, 2025). Although the reactive acetaminophen metabolite, N-acetyl-p-benzoquinone imine (NAPQI), interacts with various cytosolic proteins, it is specifically the formation of mitochondrial protein adducts that correlates with the progression of hepatocyte damage (Jaeschke, Ramachandran, 2025; Xie et al., 2015). NAPQI has been shown to induce the generation of reactive oxygen species (ROS) and lipid peroxidation (LPO) within mitochondria; furthermore, scavenging mitochondrial ROS, peroxynitrite, and LPO products effectively prevents hepatocyte death (Ramachandran, Jaeschke, 2019). Additionally, nitrotyrosine protein adducts have been identified in mitochondria, and an elevated level of oxidized glutathione (GSSG) has been confirmed (Du et al., 2017). NAPQI selectively binds to proteins of the mitochondrial matrix and inner membrane, including Complexes I and II of the electron transport chain, as well as the α-subunit of ATP synthase. The mitochondrial enzymatic Complex II exhibits higher sensitivity to direct inhibition by NAPQI *in vitro*, whereas *in vivo* models demonstrate that APAP overdose significantly suppresses Complex I activity (Du K, Ramachandran, 2016). Moreover, APAP impairs the assembly of respiratory supercomplexes via the negative regulator methylation-controlled J-protein, thereby decreasing ATP production and increasing ROS accumulation (Barbier-Torres, 2017).

The coenzyme Q system occupies a distinct position in mitochondrial metabolism due to its unique properties. It functions as a mobile electron carrier transferring electrons from Complexes I and II to Complex III, thereby maintaining adequate levels of oxidative phosphorylation and ATP generation. Beyond its energy-transducing role, ubiquinone acts as a potent antioxidant that effectively scavenges reactive oxygen species and mitigates the destructive impact of oxidative stress on mitochondrial membranes. Impairment of ubiquinone functionality or a decrease in its pool size can lead to mitochondrial dysfunction, reduced ATP production, and exacerbated oxidative stress (Hidalgo-Gutiérrez et al., 2021).

Thus, coenzyme Q serves as an integrative biomarker linking cellular bioenergetics with redox homeostasis. Because cellular resilience to toxic injury directly depends on its functional state, determining the ratio of its redox forms is crucial for understanding the mechanisms underlying acetaminophen-induced hepatotoxicity.

The study results revealed that administration of a toxic dose of acetaminophen (ATI group) causes substantial alterations in the parameters of the ubiquinone system (Fig. 1A–1C). Specifically, a marked decrease in total ubiquinone content was observed compared to the control group, which under these conditions may result in both electron transport chain dysfunction and the depletion of antioxidant capacity, leading to intensified oxidative stress.

Concurrently, a decrease in the content of the reduced form of ubiquinone (Fig. 1B) accompanied by an increase in its oxidized form (Fig. 1C) was observed in the ATI group animals, indicating a disruption of the coenzyme Q redox balance, which may account for the suppression of ubiquinone-dependent mitochondrial enzyme complexes. We demonstrated a significant decrease in the activity of ubiquinol:cytochrome c oxidoreductase, which reflects the functional state of Complex III of the electron transport chain (Fig. 1D), suggesting the inhibition of bioenergetic processes and the development of an energy deficit.

Administration of the ethanolic extract of *Hericium flagellum* to healthy animals led to an increase in both total ubiquinone levels and its reduced form compared to the control group (Fig. 1A–1C), as well as an elevation in ubiquinol:cytochrome c oxidoreductase activity (Fig. 1D), indicating a stimulation of mitochondrial functional activity.

Preventive 10-day administration of the macrofungal ethanolic extract demonstrated a restoration of the analyzed parameters to values close to control levels, pointing to a pronounced corrective effect of the *Hericium flagellum* extract against acetaminophen-induced impairment.

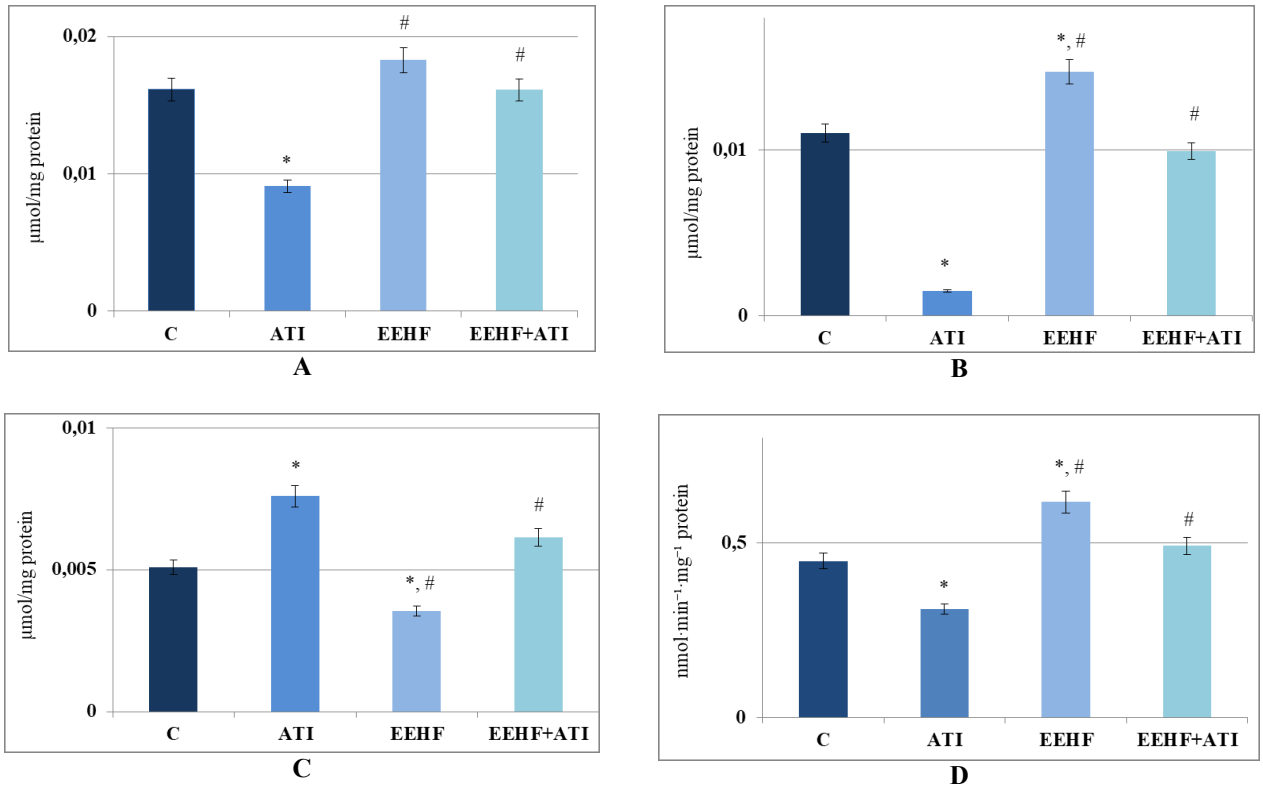


***Fig. 1*. Content of redox forms of ubiquinone and ubiquinol:cytochrome *c* oxidoreductase activity in rat liver mitochondria under the administration of *Hericium flagellum* ethanolic extract and paracetamol-induced intoxication (A – total ubiquinone, B – reduced ubiquinone, C – oxidized ubiquinone, D - ubiquinol:cytochrome *c* oxidoreductase activity)**

Note. C – control; ATI – acute toxic injury induced by paracetamol; EEHF – animals administered the ethanolic extract of Hericium flagellum (200 mg/kg body weight) for 10 days; EEHF+ATI – animals administered the Hericium flagellum extract prior to the modeling of paracetamol-induced intoxication.

** – differences are statistically significant relative to the control group ($p < 0.05$);*

 # – differences are statistically significant relative to the ATI group ($p < 0.05$).

In conclusion, the ethanolic extract of *Hericium flagellum* fruiting bodies exhibits a marked protective effect. This highlights its potential application for correcting mitochondrial dysfunction and restoring cellular bioenergetic homeostasis under conditions of acetaminophen intoxication.

Conclusions. It was established that acetaminophen-induced toxic liver injury is accompanied by a reduction in total CoQ_{10} content and its reduced form, ubiquinol, alongside an elevation in oxidized ubiquinone levels, indicating a shift in the redox balance toward pro-oxidant processes. Administration of *Hericium flagellum* extract prior to the induction of toxic damage facilitated the restoration of the total CoQ_{10} pool, increased ubiquinol levels, and restored the ratio of ubiquinone redox forms to values close to control levels, demonstrating the antioxidant properties of the investigated extract.

Under conditions of acetaminophen-induced intoxication, a decrease in the activity of ubiquinol:cytochrome c oxidoreductase (Complex III of the respiratory chain) was observed, which may indicate the suppression of mitochondrial respiration processes and the development of an energy deficit in liver cells. Pre-treatment with *Hericium flagellum* extract was accompanied by an increase in the activity of the analyzed enzyme and the restoration of its parameters to levels near control values, highlighting its potential application for correcting mitochondrial impairment in acetaminophen toxicity.

Conflict of Interest. *The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.*

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СТАН РЕДОКС-ФОРМ УБІХІНОНУ ПЕЧІНКИ ЗА УМОВ МЕДИКАМЕНТОЗНОГО УШКОДЖЕННЯ ТА ПРЕВЕНТИВНОГО ВВЕДЕННЯ ЕТАНОЛЬНОГО ЕКСТРАКТУ HERICIUM FLAGELLUM

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Роботу присвячено визначенню показників редокс-форм убіхінону та активності убіхінол:цитохром с оксидоредуктази в мітохондріях клітин печінки тварин, яким вводили етанольний екстракт Hericium flagellum на тлі інтоксикації парацетамолом. У дослідженні використані 4 групи тварин: К – інтактні щури; ТУ – щури, яким протягом двох діб вводили токсичну дозу ацетамінофену (1250 мг/кг у вигляді 2% крохмального гелю перорально); ЕГФ – тварини, які протягом 10 днів отримували етанольний екстракт Hericium flagellum у дозі 200 мг/кг маси тіла; ЕГФ+ТУ – щури, які перед введенням ацетамінофену протягом 10 днів отримували екстракт Hericium flagellum (200 мг/кг). Встановлено, що введення парацетамолу призводить до суттєвого зниження загального пулу коензиму Q_{10} (CoQ_{10}). При цьому пул відновленої форми убіхінону – убіхінолу – практично виснажується, що відображає розвиток вираженого окиснювального стресу. Одночасно спостерігається підвищення рівня окисненої форми убіхінону, наслідком чого може бути порушення функціонування дихального ланцюга. Показано, що за умов токсичного ураження печінки спостерігається зниження активності убіхінол:цитохром с оксидоредуктази, наслідком чого може бути зниження ефективності окисного фосфорилювання. Застосування екстракту Hericium flagellum сприяє відновленню загального рівня Q_{10} та підвищенню вмісту убіхінолу, що вказує на виражений антиоксидантний і мембраностабілізуючий ефект. Крім того, зниження рівня окисненої форми убіхінону при введенні екстракту досліджуваного гриба свідчить про покращення функціонального стану мітохондріального дихального ланцюга. Отримані результати вказують на здатність екстракту Hericium flagellum забезпечувати відновлення енергетичного метаболізму та редокс-стану коензиму Q_{10} за умов ацетамінофен-індукованого токсичного ураження печінки, що обґрунтовує доцільність подальших досліджень механізмів його біологічної дії.

Ключові слова: печінка, мітохондрії, ацетамінофен, Hericium flagellum, редокс-форми CoQ_{10}, убіхінол:цитохром с оксидоредуктаза

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