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Assessment of *PPAR-α* Gene Expression in Hens' Liver After Administration of Q10 and Endosulfan

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ABSTRACT

Endosulfan is a long-lasting organochlorine pesticide, whereas coenzyme Q10 (CoQ10) is a widespread antioxidant mainly found in mitochondria. *Peroxisome proliferator-activated receptor alpha* (*PPAR-α*), a regulatory transcription factor, plays a crucial role in hepatic regulation. The current study aimed to evaluate the effect of CoQ10 and endosulfan on *PPAR-α* gene expression in broiler chicken livers. A total of 40 one-day-old broiler chickens weighing 42 ± 2 grams were randomly divided into four groups. The first group served as the control and received distilled water by gavage (T1). The second group received CoQ10 orally at 40 mg/kg body weight (T2), the third group received endosulfan orally at 30 ppm (T3), and the fourth group received the combination of CoQ10 at 40 mg/kg body weight and 30 ppm of endosulfan (T4). On days 27 and 42 of the study, 20 chickens were euthanized, and liver samples were collected for analysis of *PPAR-α* gene expression by quantitative polymerase chain reaction (qPCR). On days 27 and 42, all treatments exhibited significant downregulation of *PPAR-α* gene expression compared to the control group. The most significant suppression was observed in the endosulfan with CoQ10 (T4) group, followed by the endosulfan group (T3), whereas CoQ10 (T2) exhibited the lowest level of downregulation among the treatment groups. On day 42, all treatment groups exhibited elevated *PPAR-α* gene expression compared to the control group. The highest *PPAR-α* expression level was observed in Group T2, followed by Group T3, while Group T4 demonstrated the lowest degree of upregulation among the treatment groups. The current findings indicated an age-dependent reversal pattern in *PPAR-α* expression. On day 27, chickens demonstrated a consistent downregulation of *PPAR-α*, whereas on day 42, an upregulation was observed. The current results indicated that CoQ10 administration produced differential stimulatory effects on *PPAR-α* gene expression.

Keywords: Coenzyme Q10, Endosulfan, Liver, *PPAR-α*

INTRODUCTION

Endosulfan is a prominent chlorinated hydrocarbon insecticide, chemically similar to heptachlor, chlordane, and aldrin, used to suppress insect pests in farms and houses (Berdowska and Bandurska, 2025). Due to its high persistence and capacity to accumulate in food chains, endosulfan negatively impacts wildlife populations, leading to regulatory restrictions and bans on its use (Berdowska and Bandurska, 2025). Numerous studies have indicated that pesticide residues and their metabolites have contaminated water, ground surfaces, and soil worldwide (Moore et al., 2014; Suliman et al., 2020). Pesticide compounds exert toxic effects on cells, including immunotoxicity, and inhibit the metabolic activity of splenocytes and blood lymphocytes in poultry (Tiwari et al., 2016; Cestonaro et al., 2022). Coenzyme Q10 (Q10) is a cofactor essential for numerous enzymatic activities within the body. Ubiquinone, commonly known as Q10, is essential for the respiratory chain and oxidative phosphorylation (Gopi et al., 2015).

Structurally, CoQ10 consists of a benzoquinone ring attached to an isoprenoid side chain. The letter Q indicates the quinone structure, while the length of the isoprenoid side chain ranges from 7 to 10 units. In mammals and humans, CoQ10specifically contains 10 isoprene units (Mantle et al., 2025). The reduced form of CoQ10, ubiquinol, is a powerful lipophilic antioxidant that can regenerate both itself and other antioxidants, including ascorbate and tocopherol (Gopi et al., 2015). In recent years, Q10 has gained popularity, and its pharmacodynamics and pharmacokinetics, including dosage, safety, and drug interactions, have been examined (Bayril et al., 2020). The *peroxisome proliferator-activated receptor alpha* (*PPAR-α*) gene has been proven to be highly expressed in laying hens (König et al., 2008). The *PPAR-α* gene plays a substantial role in lipid metabolism and energy homeostasis during fasting (Fuior et al., 2023). The *PPAR-α* gene encodes a nuclear receptor transcription factor that is essential for hepatic metabolic adaptation during fasting. The *PPAR-α* gene promotes the expression of key enzymes involved in peroxisomal and mitochondrial fatty acid oxidation, including acyl CoA oxidase (ACO) and carnitine palmitoyl transferase 1 alpha (CPT1α; Pawlak et al., 2015). Fatty acid oxidation involves genes for acyl-CoA dehydrogenases with different chain lengths, including medium-chain (MCAD) and long-chain (LCAD; Fuior et al., 2023). Therefore, *PPAR-α* is essential for fatty acid oxidation in mammals

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(Strömqvist et al., 2012; Du et al., 2022). The level of liver-type fatty acid-binding protein is regulated by *PPAR-α* activity in poultry, and the *liver fatty acid-binding protein* (*L-FABP*) gene may be linked to fat deposition in chickens (Zhang et al., 2023). The present study aimed to assess the effects of endosulfan and CoQ10 on *PPAR-α* gene expression in the hepatic tissue of broiler chickens.

MATERIALS and METHODS

Ethical approval

The current study was conducted within the animal laboratory of the College of Veterinary Medicine at the University of Mosul, Mosul, Iraq. The present study adhered to the ethical approval granted by the Animal Welfare Committee on July 20, 2025, with the approval number UM.VET.2025.061.

Experimental animals

A total of 40 one-day-old Ross 308 broiler chickens, weighing 40-45 g, were purchased from the Al-Walid Hatchery in the Al-Hamdaniya area of Mosul, Iraq. The broiler chickens were raised in controlled enclosures and randomly assigned within these areas, and fed a commercially mashed diet. The temperature started at 32°C and gradually decreased by 2-3°C each week. The cage environment was well controlled, with ventilation, humidity maintained at 50%-70%, and a 24-hour lighting system.

Study design

Forty chickens were randomly allocated into four groups. The first group served as the control (T1), the second group received CoQ10 at 40 mg/kg body weight (T2), the third group received endosulfan at 30 ppm (T3), and the fourth group received a combination of CoQ10 and endosulfan (T4). All groups were treated orally from day 1 to day 42. On days 27 and 42, the broiler chickens were euthanized via jugular vein incision, and liver tissue samples were collected and promptly frozen at -20°C for subsequent analysis.

RNA extraction

Approximately 50-100 mg of frozen liver tissue was placed in a microcentrifuge tube. Subsequently, 400 µL of lysis buffer, 2 µL of β-mercaptoethanol, and 20 µL of proteinase K solution were added to a 1 mL microcentrifuge tube containing the samples. The mixture was then vortexed and sonicated to homogenize the sample. After 10 minutes of incubation at 26°C, the mixture was centrifuged for three minutes at 13,000 rpm. A 2.0 mL collection tube was used to carefully transfer the supernatant to the upper reservoir (spin column 1). The mixture was then subjected to pulse-vortexing for 30 seconds and subsequently centrifuged at 13,000 rpm. The flux-through was kept constant. Subsequently, 400 µL of binding buffer was added to a collection tube and mixed thoroughly for 10 seconds using pulse-vortexing. The mixture was then centrifuged at 13,000 rpm for one minute. Afterward, 500-600 µL of the supernatant was transferred to a new micro-centrifuge tube (1.5 µL). Then, 200 µl of absolute ethanol (99.99%) was added and mixed thoroughly, followed by the addition of 600 µl of lysate buffer to the upper reservoir of spin column 2 (green ring) using a 2.0 ml collection tube. The mixture was centrifuged at 13,000 rpm for 10 seconds. The spin column was assembled and subsequently transferred into the 2 mL tube. The spin column was then filled with 500 µl of the washing solution and centrifuged for 10 seconds at 13,000 rpm. The spin column was assembled with the 2.0 mL collecting tube after the flow-through was poured out, and incubated for 15 minutes at room temperature (25°C). To eliminate any residual ethanol, the spin column was further centrifuged and dried for one minute at 13,000 rpm after the addition of 700 µl of washing solution. The spin column was moved to a 1.5 mL tube, then the total RNA was centrifuged at 13,000 rpm for one minute, and at -20°C, the extracted RNA was stored (Sharideh et al., 2020).

Quantitative polymerase chain reaction

The master kit was supplied by Addbio (Korea) for the real-time PCR. The kit was used for all procedures involving reverse transcription of RNA into cDNA, followed by real-time PCR amplification. Only primers and RNA samples were introduced into the single RT-PCR SYBR Master mix. The reference sequence used was NM_001001464.1, with the primer pair originally designed and validated by Sharideh et al. (2020). The forward and reverse primer sequences for *PPAR-α* were 5′-GGATGCTGGTAGCCTATGGA-3′ and 5′-GGACGATCTCCACAGCAAAT-3′, respectively (Wang et al., 2020). To design chicken *GAPDH* primers, the reference sequence K01458.1 was employed. The forward and reverse primer sequences were 5′-TGGTGACAGCCATTCTTCCA-3′ and 5′-TCCAACAAAGGGTCCTGCTT-3′, respectively (Wang et al., 2020). The total volume was 20 µl, which included 10 µl of One RT-PCR SYBR Master Mix, 0.5 µl of each primer, 3 µl of RNA sample, and 6 µl of nuclease-free water. According to the manufacturer's instructions,

optimal reaction conditions included reverse transcription for 20 minutes at 50°C, RNA hybrid denaturation and reverse transcriptase inactivation at 95°C for 10 minutes, followed by 40 cycles of qPCR. Each cycle consisted of denaturation for 15 seconds at 95°C and annealing for 60 seconds at 60°C. For real-time PCR, the Step One Plus Real Time PCR kit (USA) was employed.

Statistical analysis

The *PPAR-α* gene expression was quantified by qRT-PCR and expressed as fold change relative to the control group. Differences among groups were assessed by one-way ANOVA, followed by Tukey's post hoc test for comparison between groups. A p-value less than 5% ($p < 0.05$) was considered statistically significant.

RESULTS

On day 27, the control group exhibited ΔCt values ranging from 0.5 to 1.1, with a mean ΔCt of 0.9. All treatment groups demonstrated a significant downregulation of *PPAR-α* expression compared to the control group ($p \leq 0.05$; Figure 1). Group T3 exhibited ΔCt values ranging from 5.0 to 6.4, yielding ΔΔCt values between 4.1 and 5.5, which related to the extremely low fold changes (0.02-0.06). The present results indicated a strong suppressive effect of endosulfan on *PPAR-α* transcription. Group T3 resulted in a mean fold change of 0.04, indicating an approximate 96% reduction in *PPAR-α* expression compared to the control group. CoQ10 treatment (T2) decreased *PPAR-α* levels, but the effect was less pronounced compared to endosulfan treatment (T3). The ΔCt values ranged from 4.0 to 5.2, resulting in ΔΔCt values of 3.1-4.3 and fold changes of 0.05-0.12 in Group T2. Group T2 demonstrated a significant suppression of *PPAR-α* expression, with a mean fold change of 0.083, although this effect was milder than that observed in Group T3 ($p \leq 0.05$). Group T4 demonstrated the most significant downregulation of *PPAR-α*, with ΔCt values of 5.6-7.4, ΔΔCt values of 4.7-6.5, and fold changes ranging from 0.01 to 0.04. The mean fold change of 0.027 in Group T4 confirmed that the combination of endosulfan and CoQ10 exerted a synergistic downregulatory effect. The co-administration of CoQ10 with endosulfan did not mitigate the toxicity on day 27. The present result demonstrated that all groups significantly downregulated *PPARα* gene expression, with the greatest suppression observed in Group T4, followed by T3 and T2 on day 27 of age ($p \leq 0.05$).

On day 42, the ΔCt values of control samples ranged from 3.20 to 4.40, resulting in a mean ΔCt of 3.93. In contrast to the observations at day 27, all treatment groups exhibited negative ΔΔCt values on day 42, indicating the upregulation of *PPAR-α* in older broiler chickens (Figure 2). Endosulfan induced a moderate upregulation, with ΔΔCt values ranging from –3.43 to –0.93 and fold changes ranging from 1.9 to 10.8 (mean 5.3). The highest *PPAR-α* upregulation was observed in Group T2, which recorded ΔΔCt values of –4.53 to –2.53, corresponding to fold changes of 5.7–23.1 and a mean of 14. Group T4 exhibited mild to moderate upregulation, with a fold change ranging from 1.3 to 6.6 (mean 3.2), indicating that the antagonistic effect of endosulfan partially mitigated the stimulatory action of CoQ10 at 42 days. Notably, age differences resulted in an inverted expression pattern, in which 27-day-old chickens demonstrated downregulation of *PPAR-α* expression, whereas 42-day-old chickens exhibited significant upregulation of *PPAR-α* expression ($p < 0.05$).

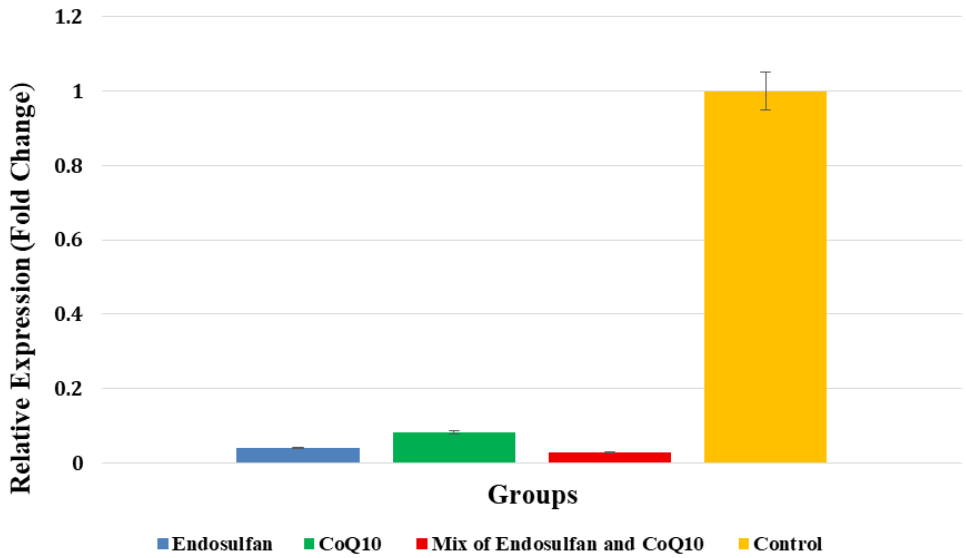

Figure 1. The *PPAR-α* gene expression across the groups at day 27

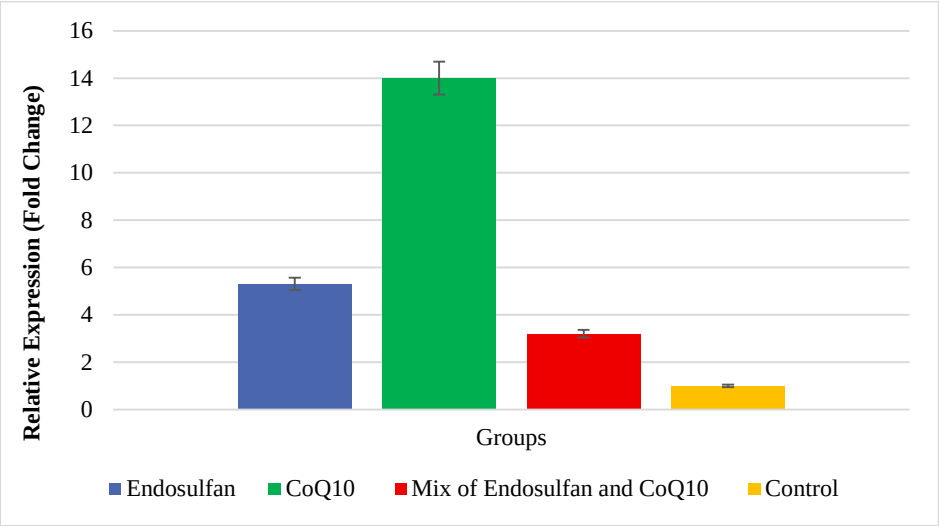

Figure 2. *PPAR-α* gene expression across the groups at day 42

DISCUSSION

The *PPAR* genes are nuclear transcription-modulating receptors that play vital roles in hepatic and overall body energy balance (Wang et al., 2020). In addition to their functions in glucose and lipid metabolism, multiple studies indicated that *PPAR* genes modulate inflammatory responses (Tan et al., 2017; Vázquez-Carrera and Wahli, 2022). New technologies, such as single-cell RNA sequencing, have helped reveal the complexity of *PPAR* expression, regulation, and signaling (Berthier et al., 2021). CoQ10 consistently enhanced *PPAR-α* expression and elicited the strongest induction in hepatic tissue, thereby indicating dose-dependent efficacy and age-related metabolic response in broiler chicken liver. The Q10 has been shown to reduce reactive oxygen species (ROS) production, thereby enhancing mitochondrial function and influencing the sensitivity of specific genes to oxidative stress, including adiponectin and *PPAR-α* (Vázquez-Carrera and Wahli, 2022). Adiponectin and *PPAR-α* are known as crucial factors in cholesterol and lipid metabolism (Zhang et al., 2023). As with other nuclear receptor ligands such as metabolic sensors, Q10 may be transported by proteins that facilitate its entry into the nucleus (Mantle, 2024). Two proteins known to bind Q10 are Q10p and Saposin B (SapB; Jin et al., 2008). The Saposin B protein facilitates the control and transport of Q10 within human cells and acts as a crucial Q10-binding protein (Jin et al., 2008). Additionally, previous studies have shown that *PPAR coactivator-1α* (*PGC-1α*) and *PPARα* bind to the D-loop promoter within mitochondria and are linked to enhanced *PPARα* gene expression (Puigserver 2005; Dominy and Puigserver 2013). The distinct responses observed between day 27 and day 42 suggested a developmental shift in hepatic sensitivity. Broiler chickens exhibited strong susceptibility to endosulfan, affecting nervous system receptors and suppressing metabolic genes, such as *PPARs*. In contrast, older broiler chickens exhibited adaptive metabolic activation, likely facilitated by an enhanced hepatic detoxification capacity and *PPAR-α*–mediated lipid mobilization. It has been indicated that *PPAR-α* regulates multiple genes involved in lipid metabolism (Pawlak et al., 2015).

Although gene regulation of liver lipid metabolism has been extensively investigated, the regulation of *PPAR* genes in the gastrointestinal tract has been poorly explored (Grabacka et al., 2024). The *PPAR-α* is one of the key receptors involved in regulating the metabolism of carbohydrates and lipids (Hernández-Valdez et al., 2023). Besides the similarity in expression patterns, RT-PCR analysis revealed that genes associated with transport and metabolism were downregulated in the layer exposed to quinalpos (Arjun et al., 2026). While endosulfan suppressed lipid metabolism genes in younger chickens by day 27, older chickens exhibited partial compensatory upregulation of the *PPAR-α* gene by day 42. The current findings might be attributed to the pesticides' mechanism of action and their effects on carbohydrate and lipid metabolism. The co-administration of CoQ10 and endosulfan demonstrated mild to moderate upregulation, suggesting that antagonistic effects of endosulfan partially mitigate the stimulatory action of CoQ10 at 42 days of age. Mild to moderate upregulation of gene expression might be attributed to oxidative stress induced by endosulfan and the protective and metabolic-enhancing effects of CoQ10.

CONCLUSION

The different responses of the *PPAR-α* gene expression at days 27 and 42 indicated an age-related transition in hepatic sensitivity, marked by suppression at day 27 and upregulation at day 42. Co-administration of CoQ10 and endosulfan in

broiler chickens affected *PPAR-α* gene expression, potentially due to mitochondrial metabolism and energy production modifications observed on day 42. The *PPAR-α* gene expression in broiler chickens was influenced by environmental variability, tissue-specific regulation, metabolic differences, and the inability of mRNA expression to fully reflect functional receptor activity. Further studies are necessary to examine the effects of CoQ10 and endosulfan on laying and breeder hens throughout the production phase.

DECLARATIONS

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Authors' contributions

Hamid and Al-Sabaawy contributed to the study design and conception, data analysis, and interpretation of results, prepared the initial draft of the manuscript, and critically reviewed it to ensure its scientific integrity. All authors have read and approved the final edition of the manuscript prior to publication in the current journal.

Availability of data and materials

The data are available upon reasonable request from the corresponding author.

Conflict of interests

The authors have declared that there is no conflict of interest related to this study.

Ethical considerations

All ethical aspects of the present study, including plagiarism, data integrity, consent to publish, and avoidance of misconduct or duplication, were carefully considered and approved by the authors. No AI tools were used in the preparation and writing the present study.

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