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Research Article

Antrodia cinnamomea Reduces Oxidative Injury and Protects Cardiac Histology in Doxorubicin-Induced Cardiotoxicity

Antrodia cinnamomea, Doksorubisin Kaynaklı Kardiyotoksisitede Oksidatif Hasarı Azaltarak Kalp Histolojisini Koruyor

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Abstract

Doxorubicin (DOX) causes heart damage mainly through oxidative stress. In this study, we evaluated whether *Antrodia cinnamomea* can reduce DOX-induced cardiac injury in rats. Twenty-four male Sprague-Dawley rats were randomly assigned to one of four groups: Control, DOX (2 mg.kg-1 i.p., every other day for 10 days), *A. cinnamomea* (100 mg.kg-1 oral gavage, daily), or DOX + *A. cinnamomea* (2 mg.kg-1 i.p., every other day for 10 days and 100 mg/kg oral gavage, daily). H&E staining was used to evaluate the hearts and assign a semi-quantitative injury score. We investigated malondialdehyde (MDA), superoxide dismutase (SOD) enzyme activity, and reduced glutathione (GSH) in left-ventricular homogenates. DOX caused a lot of damage to the heart muscle, disorganized myofibrils, created vacuoles in the cytoplasm, changed the shape of the nucleus, and caused interstitial edema. The injury scores were much higher than in the Control group ($p < 0.05$). Biochemically, DOX elevated MDA levels while reducing SOD enzyme activity and GSH levels ($p < 0.05$). *A. cinnamomea* alone was similar to the Control. Co-treatment (DOX + *A. cinnamomea*) significantly reduced MDA and partially restored SOD activity and GSH levels compared with DOX (all $p < 0.05$), consistent with lower histopathology scores; however, the values did not fully revert to Control levels. *A. cinnamomea* reduces DOX-induced cardiotoxicity by improving redox balance and protecting heart muscle structure. These results support *A. cinnamomea* as a potential adjunct to alleviate anthracycline cardiotoxicity and affirm the need for mechanistic and translational studies incorporating apoptosis/inflammation markers, ultrastructural validation, and pharmacokinetic profiling.

Keywords: Antrodia cinnamomea, Cardiotoxicity, Doxorubicin, Myocardial injury, Oxidative stress.

Öz

Doksorubisin (DOX) başlıca oksidatif stres yoluyla kalp hasarına neden olur. Bu çalışmada, *Antrodia cinnamomea*'nın sıçanlarda DOX kaynaklı kardiyak hasarı azaltıp azaltamayacağını değerlendirdik. Yirmi dört erkek Sprague-Dawley sıçan rastgele dört gruba ayrıldı: Kontrol, DOX (2 mg/kg i.p., 10 gün boyunca gün aşırı), *A. cinnamomea* (100 mg/kg oral gavaj, günlük) ve DOX + *A. cinnamomea* (2 mg/kg i.p., 10 gün boyunca gün aşırı ve 100 mg/kg oral gavaj, günlük). Kalp dokuları H&E boyaması ile değerlendirildi ve yarı kantitatif hasar skoru verildi. Sol ventrikül homojenatlarında malondialdehit (MDA), süperoksit dismutaz (SOD) enzim aktivitesi ve indirgenmiş glutatyon (GSH) düzeyleri incelendi. DOX, miyokardda belirgin hasara, miyofibril düzensizliğine, sitoplazmik vakuol oluşumuna, nükleer şekil değişikliklerine ve interstisyel ödem gelişimine yol açtı; hasar skorları Kontrol grubuna göre anlamlı derecede yüksekti ($p < 0.05$). Biyokimyasal olarak DOX, MDA düzeylerini artırırken SOD aktivitesi ve GSH seviyelerini azalttı ($p < 0.05$). *A. cinnamomea* tek başına Kontrol grubuna benzer bulundu. Eş tedavi (DOX + *A. cinnamomea*), DOX grubuna kıyasla MDA düzeylerini anlamlı biçimde düşürdü ve SOD aktivitesi ile GSH seviyelerini kısmen düzeltti (tüm $p < 0.05$); bu bulgular daha düşük histopatoloji skorları ile uyumluydu, ancak değerler tamamen Kontrol düzeylerine dönmedi. *A. cinnamomea*, redoks dengesini iyileştirerek ve kalp kası yapısını koruyarak DOX kaynaklı kardiyotoksisiteyi azaltmaktadır. Bu sonuçlar, *A. cinnamomea*'nın antrasiklin kardiyotoksisitesini hafifletmede potansiyel bir yardımcı tedavi olabileceğini desteklemekte ve apoptoz/inflamasyon belirteçleri, ultrastrüktürel doğrulama ve farmakokinetik profil içeren mekanistik ve translasyonel çalışmalara gereksinimi vurgulamaktadır.

Anahtar Kelimeler: Antrodia cinnamomea, Doksorubisin, Kardiyotoksisite, Miyokard hasarı, Oksidatif stres.

Introduction

Doxorubicin (DOX) is a cornerstone anthracycline chemotherapeutic used across a broad spectrum of solid and hematologic malignancies (Kciuk et al. 2023). A common anthracycline chemotherapeutic, DOX, exhibits dose-dependent cardiotoxicity characterized by oxidative stress, mitochondrial dysfunction, inflammation, and apoptosis, limiting its clinical usefulness (M. Camilli et al. 2024; Huang et al. 2022).

Numerous studies have shown that the myocardium can be protected from DOX-induced damage by pharmacologic or natural interventions that reduce levels of reactive oxygen species (ROS) and malondialdehyde (MDA) or strengthen antioxidant defenses (Rawat et al. 2021). Lipid peroxidation and mitochondrial dysfunction caused by ROS subsequently result in the characteristic histopathology of myofibrillar disarray, cytoplasmic vacuolization, nuclear alterations, and interstitial edema (Carrasco et al. 2021; Mitry&Edwards, 2016). Natural substances with potent anti-inflammatory and antioxidant qualities have been studied extensively in this regard; for example, *Boesenbergia rotunda* extract protected against DOX-induced cardiac damage (Zhang et al. 2023), and Mokko Lactone mitigated cardiotoxic effects via combined antioxidant and anti-inflammatory mechanisms (Sirwi et al. 2022).

The medicinal mushroom *Antrodia cinnamomea*, also known as *Taiwanofungus camphoratus*, is native to Taiwan and has long been prized for its detoxifying qualities (Liu et al. 2017). *A. cinnamomea* is a fungus that is very famous because of its various medicinal properties and its considerable participation in the traditional Asian medical system. This fabulous healing mushroom is valued primarily as an adjuvant treatment alongside other therapies for many diseases, including cancer (Qiao et al. 2015). The full acknowledgment of its use is mainly due to the presence in its composition of highly bioactive compounds, such as triterpenoids, polysaccharides, and ubiquinones, which are responsible for the substance's strong antioxidant, anti-inflammatory, and immune-modulatory effects (Li et al. 2024). According to recent pharmacological research, *A. cinnamomea* contains bioactive metabolites with strong anti-inflammatory, anti-apoptotic, and antioxidant properties, including triterpenoids, polysaccharides, and benzenoids, which suppress lipid peroxidation, enhance antioxidant enzyme activity, and scavenge free radicals. These substances help maintain cellular redox homeostasis (Geethangili & Tzeng, 2011; Shaker et al. 2010).

Given the well-documented cardiotoxic potential of DOX and the emerging evidence that *A. cinnamomea* exerts potent antioxidant and anti-inflammatory effects, the present study was designed to evaluate whether *A. cinnamomea* supplementation can mitigate DOX-induced myocardial injury. Specifically, we sought to determine if *A. cinnamomea* attenuates oxidative stress and histopathological alterations in rat hearts subjected to cumulative DOX exposure. We hypothesized that *A. cinnamomea* administration would significantly reduce DOX-induced myocardial damage by lowering oxidative stress markers (MDA), enhancing antioxidant defenses (SOD, GSH), and alleviating histopathological injury in cardiac tissue.

Materials and Methods

Animals Procurement, Care, and Feeding

In this study, 24 male Sprague-Dawley rats, 12 weeks old and weighing 250–300 g, were used. The experimental animals were obtained from the Experimental Animals Production and Research Center (MAKÜ-DEHAM) at Burdur Mehmet Akif Ersoy University. All the experimental procedures received approval from the Burdur Mehmet Akif Ersoy University Animal Experiments Local Ethics Committee (approval no: 1536, date: July 9, 2025). During the experiment, the animals were kept in a ventilated space

maintained at 22 ± 2 °C, with a relative humidity of 50–60% and a light/dark cycle of 12 hours. Each rat was kept in appropriately sized polycarbonate cages in groups and was provided with free access (ad libitum) to sterile drinking water and standard laboratory chow.

Experimental Design and Groups

Animals were allocated into four groups (n = 6 per group), and the following treatments were administered:

- Control (n=6): intraperitoneal injections of 0.9% saline every other day and daily oral gavage of the *A. cinnamomea* vehicle (distilled water) for 10 days; dosing volumes were matched to those of the corresponding treatment groups to control for handling and vehicle effects.
- DOX (n=6): Doxorubicin 2 $mg.kg^{-1}$ (Eisvand et al. 2022), intraperitoneally (i.p.), every other day for a total of 10 days.
- *A. cinnamomea* (n=6): *A. cinnamomea* 100 $mg.kg^{-1}$ (Tain et al. 2023), by oral gavage once daily for 10 days.
- DOX + *A. cinnamomea* (n=6): Doxorubicin 2 $mg.kg^{-1}$ (i.p., every other day) (Eisvand et al., 2022) and *A. cinnamomea* 100 $mg.kg^{-1}$ (oral gavage) (Tain et al. 2023) for 10 days.

Final Study Procedures and Tissue Collection

At the end of the exposure period, all animals were anesthetized with isoflurane (3–5% in oxygen). After confirming the level of deep anesthesia, the animals were humanely euthanized by cervical dislocation in accordance with ethical guidelines. The hearts were then dissected and removed. Portions of the cardiac tissue were allocated for histological evaluation (fixed in 10% neutral buffered formalin for 48 h), and the remaining portions were stored at −80 °C for biochemical analyses.

Histological Processing and Light Microscopic Evaluation

After being fixed in 10% neutral buffered formalin (48 h), hearts were rinsed in running tap water (10 min) and trimmed to obtain consistent blocks from the left ventricular (LV) free wall and interventricular septum (mid-papillary level). Tissues were processed through graded ethanol (70% 1×60 min; 80% 1×60 min; 96% 2×60 min; 100% 2×60 min), cleared in xylene (3×30 min), and embedded in paraffin at 58–60 °C. Paraffin blocks were sectioned at 4–5 μm using a rotary microtome. Sections were mounted on poly-L-lysine–coated slides and dried at 60 °C for 45–60 min.

Light microscopic evaluation and injury scoring

Stained sections were examined under bright-field microscopy (10×, 20×, 40× objectives; oil 100× as needed). For each heart, ten non-overlapping fields from the LV free wall and five from the septum were captured at 20× using identical camera exposure, illumination, and white-balance settings. Observers were blinded to group allocation. H&E-stained cardiac sections were examined under a light microscope (10×, 20×, and 40× objectives). Observers were blinded to group allocation during evaluation. Histopathological alterations were semi-quantitatively graded on a 0–3 scale according to the system described by Alwaili et al. (2025):

- Score 0: Normal histology, no evident pathological changes.
- Score 1: Mild myofibrillar disorganization (<10% of the area), minimal cytoplasmic vacuolization, limited nuclear alterations.
- Score 2: Moderate myofibrillar disruption (≈10–30% of the area), evident cytoplasmic vacuolization, vascular congestion, and nuclear deformation.
- Score 3: Extensive myofibrillar damage (>30% of the area), marked loss of contractile components, pronounced nuclear deformation, interstitial edema, and inflammatory cell infiltration.

For each section, the mean score was calculated; the average of individual animal scores was used for statistical analyses (Alwaili et al. 2025).

Biochemical Analyses

Assessment of oxidative stress by ELISA (MDA, SOD, GSH)

Myocardial malondialdehyde (MDA), superoxide dismutase (SOD), and reduced glutathione (GSH) were quantified using BT Lab ELISA kits according to the manufacturer's instructions. Briefly, samples from the LV free wall were processed on ice to prepare 10% (w/v) homogenates in cold PBS (pH 7.4, 0.1 mM EDTA; tissue: buffer 1:9). Homogenates were centrifuged at 10,000–12,000 ×g for 15 min at 4 °C, and supernatants were used for assays. Total protein was determined by the Lowry method, and all outcomes were normalized to mg of protein. Analyte concentrations/activities were calculated from four-parameter logistic (4-PL) standard curves and reported as $\mu mol.mg^{-1}$ protein for MDA and GSH, and $U.mg^{-1}$ protein for SOD.

Statistical Analyses

All data were displayed as mean ± standard deviation (SD). We used one-way analysis of variance (ANOVA) to compare the groups. When notable differences were observed, Tukey's post hoc test was used to assess intergroup differences. We used the Shapiro–Wilk test to assess normality and Levene's test to assess homogeneity of variances. We used GraphPad Prism (version 9.0; GraphPad Software, San Diego, CA, USA) for statistical analyses. A p-value of less than 0.05 is considered statistically significant.

Results

Histological Outcomes Underscore DOX Cardiotoxicity and A. cinnamomea Benefit

Evaluation of H&E-stained cardiac sections demonstrated that the Control group exhibited normal myocardial architecture, with preserved myofibrillar organization and no evidence of cytoplasmic vacuolization, nuclear alterations, or inflammatory infiltration (Figure 1a). The *A. cinnamomea* group showed histological features similar to those of the Control, maintaining normal tissue morphology (Figure 1b). In contrast, the DOX group displayed pronounced myocardial injury, characterized by extensive myofibrillar disorganization, cytoplasmic vacuolization, nuclear deformation, interstitial edema, and prominent inflammatory cell infiltration (Figure 1c). Correspondingly, mean injury scores were significantly elevated in this group ($p < 0.05$) (Table 1). The DOX + *A. cinnamomea* group exhibited a marked reduction in the severity of histopathological lesions, with notably less myofibrillar disruption and edema compared with DOX alone (Figure 1d). However, scores did not return fully to baseline Control levels (Table 1).

Biochemical Outcomes Highlight DOX-Induced Oxidative Stress and A. cinnamomea–Mediated Improvement

Biochemical analyses showed that the Control and *A. cinnamomea* groups had comparable redox profiles, with low MDA (malondialdehyde), higher SOD (superoxide dismutase) activity, and preserved GSH (reduced glutathione). The DOX group showed significantly increased MDA levels, accompanied by reduced SOD activity and GSH content ($p < 0.05$ vs. Control), consistent with pronounced oxidative stress. Co-treatment (DOX + *A. cinnamomea*) partially reversed these changes, lowering MDA and restoring SOD and GSH levels relative to DOX alone ($p < 0.05$), although not to Control levels. All values are shown in Table 2.

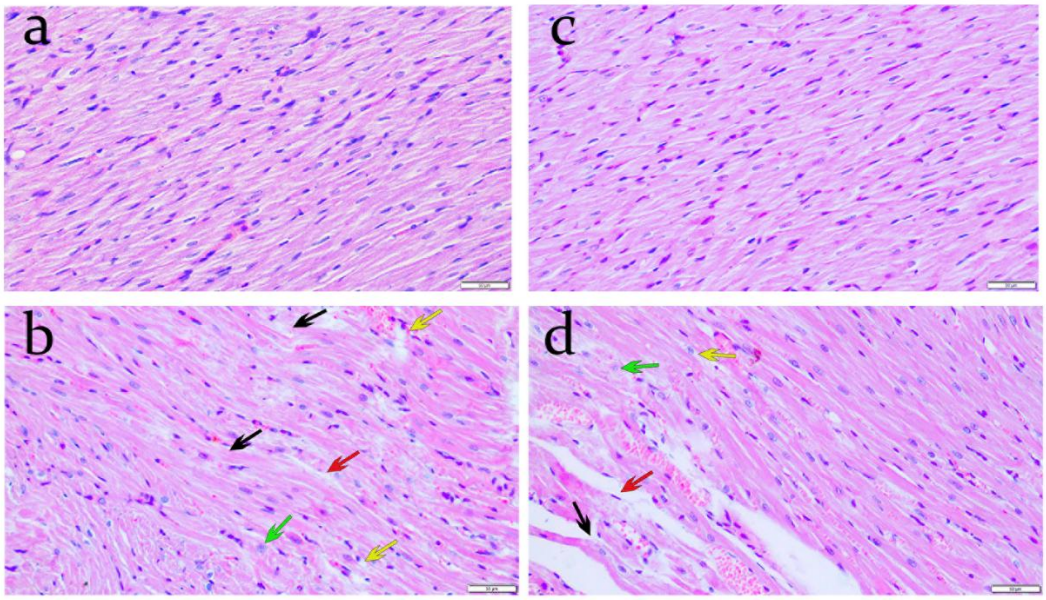

Figure 1. Representative photomicrographs of H&E-stained rat cardiac tissue (panels a-d). (a) Control group, (b) *A. cinnamomea* group, (c) DOX group exhibiting marked myocardial injury. Black arrows indicate myofibrillar disorganization, yellow arrows highlight cytoplasmic vacuolization, red arrows mark interstitial separation/edema, and green arrows point to nuclear deformation. (d) DOX + *A. cinnamomea* group showing attenuated histopathological alterations compared with DOX alone, with improved fiber organization, reduced vacuolization, and more preserved nuclear morphology.

Table 1. Semi-quantitative H&E injury scores by group.

Group	Mean ± SD
Control	0.00 ± 0.00^{a}
A. cinnamomea	0.00 ± 0.00^{a}
DOX	2.17 ± 0.41^{c}
DOX + *A. cinnamomea*	1.08 ± 0.20^{b}

Different superscript letters denote $p<0.05$ (one-way ANOVA, Tukey).

Table 2. Oxidative stress parameters in cardiac tissue across treatment groups.

Group	MDA (μmol.mg^{-1} protein)	SOD (U.mg^{-1} protein)	GSH (μmol.mg^{-1} protein)
Control	1.36 ± 0.30^{a}	6.39 ± 1.02^{c}	2.04 ± 0.21^{c}
A. *cinnamomea*	1.33 ± 0.10^{a}	6.27 ± 0.29^{c}	2.15 ± 0.31^{c}
Dox	3.01 ± 1.89^{c}	4.49 ± 0.04^{a}	1.36 ± 0.30^{a}
Dox + A. *cinnamomea*	1.89 ± 0.30^{b}	5.00 ± 0.61^{b}	1.88 ± 0.11^{b}

Different superscript letters denote $p<0.05$ (one-way ANOVA, Tukey).

Discussion

Cancer is one of the most significant causes of morbidity and mortality worldwide today. Chemotherapeutic agents used in its treatment can prolong life but may also cause serious side effects. Among these treatments, DOX is a highly effective anthracycline derivative widely used in many solid and hematological malignancies,

particularly breast cancer, leukemia, and lymphoma (Li et al. 2024). However, one of the most important limiting factors in clinical use is dose-dependent cardiotoxicity. The toxicity of DOX in cardiac tissue is mediated by multifactorial mechanisms, primarily increased oxidative stress, mitochondrial dysfunction, triggering of the inflammatory response, and apoptosis (Rawat et al. 2021; Wenningmann et al. 2019). In particular, increased mitochondrial ROS production initiates lipid peroxidation, leading to protein and DNA damage and resulting in typical histopathological changes such as myofibrillar disorganization, cytoplasmic vacuolization, and nuclear deformities (Massimiliano Camilli et al. 2024; Maria A. Mitry & John G. Edwards, 2016).

In recent years, numerous studies have examined the effects of pharmacological and natural antioxidant substances on reducing DOX-induced cardiotoxicity. The primary goal of antioxidant treatments is to suppress ROS production, support endogenous antioxidant enzymes (SOD, GSH, etc.), and thereby maintain intracellular redox homeostasis. Indeed, the literature has demonstrated that various natural products alleviate DOX-induced myofibrillar disruption, interstitial edema, and nuclear changes (Sirwi et al. 2022; Zhang et al. 2023). Similarly, natural products have been shown to reduce MDA levels and enhance defense systems, such as SOD and GSH, through their antioxidant capacity (Rawat et al. 2021). In this study, the selection of *A. cinnamomea* as a natural antioxidant agent is based not only on its potent antioxidant properties but also on its previously demonstrated cardiovascular benefits. Although most natural antioxidants in the literature have been reported to provide partial protection against DOX cardiotoxicity, *A. cinnamomea*'s triterpenoids distinguish it from other plant products by their ability to simultaneously suppress oxidative stress and inflammation in the heart muscle (Ma et al. 2024). Furthermore, its demonstrated protective effects against hypertrophy, ischemic damage, and metabolic disorders in experimental models beyond its traditional uses have made this agent a more suitable candidate against the complex pathophysiological processes caused by DOX (Geethangili & Tzeng, 2011). The findings obtained in our study are highly consistent with this literature. The increase in MDA levels, the decrease in SOD and GSH activities, and the histological observation of myofibrillar disorganization and cytoplasmic vacuolization in the DOX-treated group are consistent with the fundamental pathophysiological mechanisms of cardiotoxicity. In contrast, the significant improvement in oxidative stress markers and milder histopathological findings in the *A. cinnamomea*-treated group indicate that this substance plays a protective role in cardiac tissue, owing to its strong anti-inflammatory and antioxidant effects.

Conclusion

In conclusion, the histopathological healing pattern observed in our study (preservation of myofibrillar continuity, reduction in vacuolization, improvement in nuclear morphology) aligns with the improvement in oxidative stress markers, confirming the central role of oxidative stress in DOX cardiotoxicity while supporting *A. cinnamomea* as a meaningful protective candidate at the histo-morphological level. Future studies integrating light microscopy findings with ultrastructural (TEM) validation and apoptosis/inflammation markers (e.g., caspase-3, TNF-α/IL-1beta) will further strengthen the mechanistic inferences.

Conflict of interest: The authors have no conflicts of interest to report.

Authors' Contributions: S.Y.: Conceptualization, Methodology, Formal analysis, Investigation, Histopathology evaluation, Writing – Original Draft, Visualization. **S.Ö.:** Supervision, Formal analysis, Resources, Validation.

Ethical approval: This study was carried out at Burdur Mehmet Akif Ersoy University, Experimental Animals Production and Research Center (MAKÜ-DEHAM). The research protocol titled "Investigation of the Protective Effects of *Antrodia cinnamomea* in a Rat Model of Doxorubicin-Induced Cardiotoxicity" was reviewed and approved by the Local Ethics Committee for Animal Experiments of Burdur Mehmet Akif Ersoy University (Approval No: 1536, Date: 09/07/2025).

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Availability of data and materials: The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

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