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Myricetin Modulates Redox Balance in MDBK Cells in a Dose-Dependent Manner

Cansu Celik
Gaziantep University

Gul Fatma Yarim
Ondokuz Mayis University

Murat Yarim
Ondokuz Mayis University

Abstract: Myricetin (MYR), a natural flavonoid, exerts antioxidant effects by scavenging free radicals and enhancing cellular defenses. However, its dose-dependent redox-regulatory effects in Madin-Darby bovine kidney (MDBK) cells remain unexplored. This study aimed to investigate the dose-dependent effects of myricetin on redox balance in MDBK cells by measuring total antioxidant capacity (TAC) and total oxidant capacity (TOC). MYR was dissolved in 0.1% DMSO. Cells were treated for 24 hours in quadruplicate, with untreated cells as the negative control (NC) and 0.1% DMSO as the vehicle control. Cell viability was assessed by MTT assay, confirming that MYR treatments maintained >95% cell survival. TAC and TOC levels were measured using a commercial spectrophotometric kit, and each measurement was carried out in duplicate. Oxidative stress index (OSI) was calculated as the ratio of TOC to TAC. Statistical analysis was performed using one-way ANOVA followed by Tukey's post hoc test, with $p<0.05$ considered significant. TAC levels (mmol Trolox equivalent/L) increased from 1.07±0.03 in the NC group to 1.29±0.07 with MYR 10 µM ($p=0.008$), 1.52±0.07 with MYR 50 µM ($p<0.01$), and 1.73±0.12 with MYR 100 µM ($p<0.01$). Conversely, TOC levels (µmol H_2O_2 equivalent/L) decreased from 10.19±0.28 in NC to 8.02±0.17 with MYR 10 µM ($p<0.01$), 7.33±0.17 with MYR 50 µM ($p<0.01$), and 6.09±0.11 with MYR 100 µM ($p<0.01$). The 0.1% DMSO control did not significantly alter TAC (1.16±0.06, $p>0.05$ vs NC) but caused a slight decrease in TOC (9.50±0.18, $p>0.05$ vs NC). These results indicate that myricetin modulates oxidative balance in a dose-dependent manner. In conclusion, MYR significantly increased antioxidant capacity and reduced oxidative stress in MDBK cells in a dose-dependent manner. The dose-dependent decrease in OSI values indicates a shift towards redox balance. These findings, which reveal the cellular redox regulatory potential of MYR, are expected to contribute to the understanding of flavonoid-mediated antioxidant mechanisms in veterinary and cell biology research, as well as in nutraceutical studies as a feed supplement.

Keywords: Myricetin, MDBK cells, Oxidative stress, TAC, TOC, OSI, Flavonoid.

Introduction

Reactive oxygen species (ROS), continuously produced as products of cellular metabolism, play a crucial role in cellular homeostasis. However, excessive ROS production disrupts the balance between antioxidants and oxidants, leading to oxidative stress. Oxidative stress can damage the structure of proteins, lipids, and DNA. This damage leads to cellular dysfunction, resulting in numerous pathologies, including inflammation, metabolic disorders, and degenerative diseases. Therefore, maintaining intracellular redox balance is essential for the organism's overall health (Kozlov et al., 2024; de la Lastra et al., 2026). Cells have a defense system that neutralizes ROS (reactive oxygen species) using enzymatic and non-enzymatic antioxidants such as superoxide

dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and glutathione, thereby protecting cellular survival (Ighodaro and Akinloye, 2018; Irato and Santovito, 2021). As the severity of oxidative stress increases, these defense systems become insufficient, necessitating the use of exogenous antioxidants. Biological damage associated with oxidative stress can lead to numerous degenerative health problems that have a significant impact on the overall performance and productivity of livestock (Yang et al., 2013). Natural flavonoids, due to their strong antioxidant and cytoprotective effects, are successfully used in preventing oxidative stress (Zahra et al., 2024; Trivedi, 2025).It is also considered a functional feed additive that improves redox balance and reduces oxidative stress in ruminants (Onu et al., 2024).

Myricetin (3,3′,4′,5,5′,7-hexahydroxyflavone; MYR) is a dietary flavonoid abundant in fruits, vegetables, and medicinal plants that exhibits significant biological effects, including antioxidant, anti-inflammatory, and anti-apoptotic properties. In addition to its ability to directly scavenge free radicals formed in cells, myricetin also has the potential to modulate cellular antioxidant systems. It has been reported that MYR reduces intracellular ROS levels and increases the activity of antioxidant enzymes during oxidative stress (Wang et al., 2010; El-Ghareeb et al., 2023; Almatroodi and Rahmani, 2025). Myricetin has also been shown to prevent oxidative stress-induced apoptosis by modulating signaling pathways involved in cell survival, including the PI3K/Akt and MAPK pathways (Taheri et al., 2020; Goyal et al., 2024; Sabir and Dyary, 2026). Myricetin has been observed to exhibit protective effects in different cell types exposed to oxidative stress (Pandey et al., 2009; Xie et al., 2023; Wang et al., 2023). It has been shown to reduce hydrogen peroxide-induced oxidative damage and apoptosis in bovine mammary epithelial cells, highlighting the potential importance of MYR in veterinary and agricultural research (Kan et al., 2021). The fact that MYR maintains cellular ultrastructure and improves cell viability in oxidative stress models supports its cytoprotective potential (Berker et al., 2025). Despite existing studies in literature, the dose-dependent effects of MYR on the redox balance of MDBK cells have not, to our knowledge, been evaluated. This study aimed to investigate the dose-dependent effects of MYR on redox balance in MDBK cells by measuring TAC and TOC. Investigating how different concentrations of MYR affect the redox balance in MDBK cells, an important in vitro model in veterinary biomedical research, as assessed by TAC and TOC measurements, not only provides valuable insights into potential biomedical effects, but it may also provide insight into the diet-based management of oxidative stress in ruminants and offers mechanistic data supporting its potential use as a nutraceutical agent.

Method

Cell Culture and Myricetin Treatment

This study was conducted in continuous cell culture of MDBK, and cells were cultured in Dulbecco's minimum essential medium (DMEM) containing 10% fetal bovine serum and 1% penicillin/streptomycin. The cells were cultured with fresh medium every 2 days. After the cells reached a concentration of 80-90% in a T-25 culture flask, they were washed with PBS and then trypsinized with 1.5 mL of 0.25% trypsin-EDTA solution for 2-3 minutes at room temperature. The cells were transferred to T-75 culture flasks and cultured until they reached sufficient confluence. Cells were treated for 24 hours in quadruplicate, with untreated cells as the negative control (NC) and 0.1% DMSO as the vehicle control. Myricetin (Cat. No. 476275, Sigma-Aldrich, Wicklow, Ireland) was dissolved in 0.1% dimethyl sulfoxide (DMSO) as a stock solution. MDBK cells were treated with 10, 50, or 100 µM MYR for 24 hours in quadruplicate. Untreated cells served as the negative control (NC), while cells treated with 0.1% DMSO were used as the vehicle control. Cell viability was assessed by MTT assay, confirming that MYR treatments maintained >95% cell survival.

Measurement of TAC and TOC

Total antioxidant capacity (TAC) levels in the cell culture media were determined using a colorimetric assay kit (Rel Assay Diagnostics, Gaziantep, Turkey). The assay was performed according to the manufacturer's instructions. This method quantified the antioxidant status of the samples based on the principles described by Erel (2004). Total oxidant capacity (TOC) levels in the cell culture media were determined using a colorimetric assay kit (Rel Assay Diagnostics, Gaziantep, Turkey). The assay was performed following the manufacturer's instructions. This method measures the oxidant status of the samples, as described by Erel (2005).

Calculation of OSI

The OSI was calculated to evaluate the balance between oxidants and antioxidants in the cells. OSI was defined as the ratio TOC (µmol H_2O_2 equivalent/L) to TAC (mmol Trolox equivalent/L) multiplied by 100. Because the units of TAC and TOC differed, TAC values were first converted from mmol/L to µmol/L by multiplying by 1000 (Alp et al., 2010; Karkucak et al., 2010).

Statistical Analysis

Statistical evaluation of the data was performed using SPSS 25.0 (IBM Corp., Armonk, NY, USA). Data are presented as mean ± standard deviation (SD). Normality of the data was assessed using the Shapiro–Wilk test. One-way analysis of variance (ANOVA) followed by Tukey's post hoc test was used for group comparisons. A p-value <0.05 was considered statistically significant.

Results

Effect of MYR on TAC Levels

TAC levels were significantly increased in a dose-dependent manner following MYR treatment. TAC levels in the negative control (NC) group were 1.07±0.03 mmol Trolox equivalent/L. Treatment with MYR at 10 µM increased TAC to 1.29±0.07 ($p=0.008$), 50 µM increased it to 1.52±0.07 ($p<0.01$), and 100 µM further increased TAC to 1.73± 0.12 (<0.01). The 0.1% DMSO control did not significantly alter TAC (1.16±0.06, $p>0.05$ vs NC). These results indicate that MYR effectively enhances antioxidant capacity in a concentration-dependent manner (Figure 1).

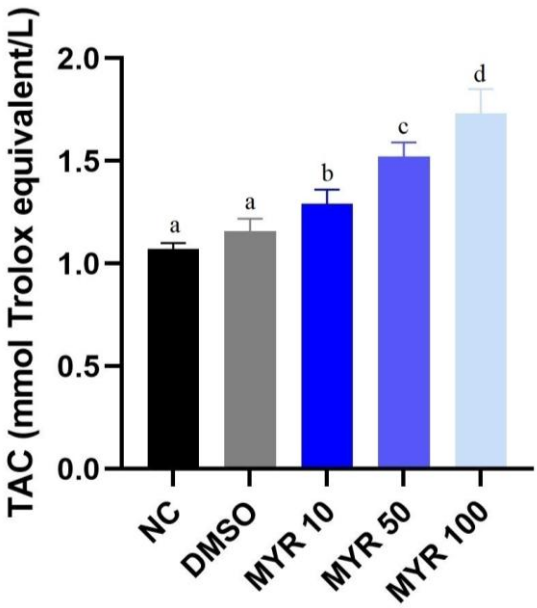

Figure 1. Effect of MYR on TAC in the cells. Data are presented as mean±SD (n=4). Different letters (a, b, c, d) indicate significant differences among groups according to one-way ANOVA followed by Tukey's post hoc test ($p<0.05$). TAC: Total antioxidant capacity; NC: Negative control; DMSO: Dimethyl sulfoxide; MYR: Myricetin.

Effect of MYR on TOC Levels

TOC levels were significantly decreased in a dose-dependent manner following MYR treatment. TOC levels in the negative control (NC) group were 10.19±0.28 µmol H_2O_2 equivalent/L. Treatment with MYR at 10 µM decreased TOC to 8.02±0.17 ($p<0.01$), 50 µM decreased it to 7.33±0.17 ($p<0.01$), and 100 µM further decreased TOC to 6.09±0.11 ($p<0.01$) compared to NC. The 0.1% DMSO control did not significantly alter TOC (9.50±0.18, $p>0.05$ vs NC). These results indicate that myricetin effectively reduces oxidative stress in a concentration-dependent manner (Figure 2).

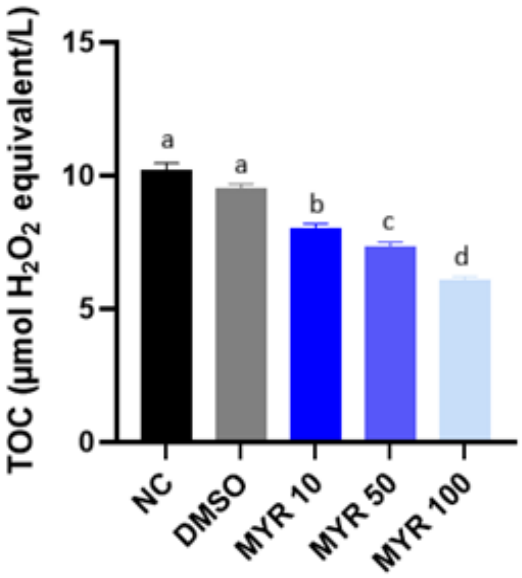

Figure 2. Effect of MYR on TOC in the cells. Data are presented as mean±SD (n=4). Different letters (a, b, c, d) indicate significant differences among groups according to one-way ANOVA followed by Tukey's post hoc test ($p<0.05$). TAC: Total antioxidant capacity; NC: Negative control; DMSO: Dimethyl sulfoxide; MYR: Myricetin.

OSI Values

OSI values were 0.95 in NC, 0.62 with MYR 10 µM, 0.48 with MYR 50 µM, and 0.35 with MYR 100 µM. The 0.1% DMSO control showed an OSI of 0.82. These results demonstrate that myricetin effectively enhances antioxidant capacity and reduces oxidative stress in a concentration-dependent manner.

Discussion

This study demonstrated that MYR increased TAC in MDBK cells in a dose-dependent manner while simultaneously decreasing TOC and OSI. These findings suggest that the cellular environment becomes more antioxidant as MYR concentrations increase. These findings are consistent with the well-documented cellular antioxidant effects of MYR, which occur both through direct ROS scavenging and upregulation of endogenous antioxidant defenses. Flavonoids such as MYR are known for their notable antioxidant effects due to multiple hydroxyl groups that facilitate free-radical scavenging and electron transfer (Agraharam et al., 2022; Devi et al., 2025). MYR's catechol and pyrogallol moieties, in particular, confer high reducing power that enables effective neutralization of ROS and stabilization of radical intermediates, consistent with observed increases in TAC with escalating MYR doses (Semwal et al., 2016; Imran et al., 2021; Chaabani et al., 2026).

It has been demonstrated that myricetin increases antioxidant capacity in bovine-derived cells and alleviates oxidative stress and lipotoxicity (Yang et al., 2022). In bovine hepatocytes, myricetin increased SOD activity, which is associated with antioxidants, whilst reducing the levels of GRP78 and PERK, which are associated with endoplasmic reticulum stress, and NF-κB and TNF-α, which are associated with inflammation, during high fatty acid loading. Investigators have suggested that myricetin intake may exert an antioxidant effect partly by promoting fatty acid mitochondrial oxidation, thereby reducing endoplasmic reticulum stress, inflammation and lipotoxicity (Yang et al., 2022). Our study's observation of dose-dependent redox modulation and antioxidant effect profile in bovine kidney cells suggests that myricetin may exhibit similar protective effects in different cell types responding to metabolic stress, as seen in hepatocytes.

MYR's ability to regulate redox balance is not limited to direct radical scavenging; it also protects cells from oxidative stress-induced damage by supporting cellular antioxidant enzymes. MYR treatment has been reported to restore superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) activities after oxidative damage such as hydrogen peroxide exposure, thereby reducing DNA and lipid oxidative damage (Wang et al., 2010; Yang et al., 2024). In addition to these direct enzyme-mediated mechanisms, MYR also modulates intracellular signaling pathways that affect redox homeostasis and cell survival. MYR has been

observed to reduce oxidative stress-induced apoptosis by regulating the PI3K/Akt and MAPK pathways, which are known to interact with redox-sensitive signaling networks and influence cellular defense responses (Song et al., 2021; Devi et al., 2025). The dose-dependent redox modulation we identified in this study is consistent with the findings of previous studies focusing on the effects of flavonoids on redox biology. These findings suggest that mirisetin could be considered a potential nutraceutical component for reducing oxidative stress-induced cellular damage. However, it appears that flavonoids reported in scientific studies exhibit more antioxidant effects at low concentrations and pro-oxidant effects at high concentrations (Chobot and Hadacek, 2011; Chobot et al., 2020; Imran et al., 2021). The TAC and TOC findings determined in this study clearly demonstrate that MYR increases antioxidant capacity and suppresses oxidative stress in healthy MDBK cells. It is well known that flavonoids such as MYR can regulate cellular redox balance in a dose-dependent manner, not only by directly neutralizing free radicals but also by influencing antioxidant defense systems and cellular signaling (Kerimi & Williamson, 2018). In our study, the decreases in OSI associated with increasing MYR concentrations indicate that MYR shifts intracellular redox balance towards a protective antioxidant state.

Conclusion

Our findings demonstrated that MYR regulates cellular redox balance in MDBK cells in a dose-dependent manner. By increasing TAC and decreasing TOC, MYR shifts the intracellular environment towards a favorable state for antioxidants and supports cellular defense mechanisms. It has been shown that redox balance in the kidneys, one of the target organs of oxidative stress in ruminants, can be modulated; therefore, the investigation of bioactive compounds such as myricetin at the cellular level contributes to our understanding of their potential effects on ruminant nutrition. This study provides novel insights into the dose-dependent redox regulatory effects of MYR in renal epithelial cells, contributing to the understanding of flavonoid-mediated antioxidant mechanisms in nutraceutical research and laying a foundation for future research on natural compounds in the prevention of oxidative stress.

Scientific Ethics Declaration

* The authors declare that the scientific, ethical, and legal responsibility of this article published in the EPHELS journal belongs to the authors.

Conflict of Interest

* The authors declare that they have no conflicts of interest.

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Author(s) Information

Cansu Celik
Department of Veterinary Medicine
Araban Vocational School
Gaziantep University, Gaziantep, Türkiye
ORCID iD: https://orcid.org/ 0000-0002-4682-4617
*Contact e-mail: *cansud@gantep.edu.tr*

Gul Fatma Yarim
Department of Biochemistry
Faculty of Veterinary Medicine
Ondokuz Mayis University, Samsun, Türkiye
ORCID iD: https://orcid.org/0000-0003-4050-429X

Murat Yarim
Department of Pathology
Faculty of Veterinary Medicine
Ondokuz Mayis University, Samsun, Türkiye
ORCID iD: https://orcid.org/0000-0002-0636-4214
*Corresponding author(s) contact e-mail

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