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Antioxidant effects of quercetin on Chinese hamster ovary (CHO) cells and their electroporation

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Electroporation is a phenomenon when a brief application of a strong electric field affects biological membranes, temporarily increasing their permeability to ions and molecules. As a result, transient pores are created in the plasma membrane. During this process, electrochemical reactions occur both at the electrode-solution interface and within the medium, leading to the generation of reactive oxygen species (ROS) that can impair cell viability. In this study, we investigated the effects of ROS induced by hydrogen peroxide (H_2O_2) and electroporation on Chinese hamster ovary (CHO) cells, as well as the potential antioxidant protection provided by quercetin. CHO cell suspensions were exposed to various concentrations of H_2O_2 and quercetin and electroporated using a single 2 ms duration electric field pulse of varying intensities (0.5–2.0 kV/cm). Cell viability was evaluated using a colony formation assay, and ROS levels were assessed via Amplex Red fluorescence. Quercetin showed no cytotoxicity up to 2 μM, but reduced cell viability at higher concentrations. It significantly improved survival in cells exposed to H_2O_2 across all tested concentrations, with the most notable effect observed at 10 μM H_2O_2, increasing viability. Similarly, quercetin enhanced post-electroporation viability by 11% and 17% at 0.5 and 1.0 kV/cm, respectively. However, at 2.0 kV/cm, its protective effect was no longer observed. These results suggest that quercetin mitigates oxidative stress induced by both chemical and physical stimuli and may be a useful cytoprotective agent in electroporation-based biomedical applications.

Keywords: electroporation, quercetin, Chinese hamster ovary cells, CHO, oxidative stress

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INTRODUCTION

Electroporation technique is commonly used to transfer various molecules, nucleic acids, antibodies and antitumor drugs into cells (Rakozcy et al., 2022). The increase in the permeability of cell membranes is not the only consequence of the electroporation process. It is known that during a short strong electrical pulse, various electrochemical and chemical reactions occur when an electric current flows, which can noticeably change the parameters of the medium: pH, temperature or chemical composition (Kotnik et al., 2001). In this scenario, once the electric field intensity threshold is attained, an oxidative burst occurs (Gabriel, Teissié, 1994). Additionally, electric pulses create stress in cells, resulting in the generation of reactive oxygen species (ROS) (Fig. 1). Such ROS as superoxide anions ($O_2\bullet^-$), hydrogen peroxide (H_2O_2), hydroxyl radicals (•OH), peroxynitrite ($ONOO^-$), and singlet oxygen ($^1O_2$) are produced through mitochondrial dysfunction, NADPH oxidase activation, Fenton chemistry, and electrochemical processes during electroporation (Teissie, 2017). These species are capable of damaging lipids, proteins, and nucleic acids, ultimately reducing cell viability.

Generally, oxidative stress occurs when there is a disparity between the generation of reactive oxygen species and the efficiency of antioxidants. While reactive oxygen species are produced in the course of regular cellular metabolism, they have the potential to harm vital organic molecules (Jelic et al., 2021). Oxidative stress in cells is a detrimental process, since reactive oxygen species and other free radicals can harm important biomolecules, including membrane lipids, proteins, and DNA (Jomova et al., 2023). Oxidative stress caused by free radicals influences the development of various pathogenic diseases: diabetes, neurodegenerative and cardiovascular diseases, and cancer (Reddy, 2023).

It is known that antioxidants can neutralise free radicals. Quercetin is one of the most widely studied polyphenolic flavonoid compounds. It can be found in tea, fruits, berries, vegetables, and red wine (Xu et al., 2019). The name of quercetin originates from the Latin word *quercetum* 'oak forest', and it was first isolated in 1936 (Rusznyák, 1936). Numerous studies have demonstrated its strong antioxidant and cytoprotective properties, particularly its ability to prevent oxidative stress-induced cell death (Zhao et al., 2022; Sul, Ra, 2021). These effects are largely attributed to its hydroxyl-rich chemical structure, which allows quercetin to donate electrons and hydrogen atoms to stabilize free radicals. It effectively scavenges a broad spectrum of reactive oxygen species (ROS), including superoxide anions ($O_2\bullet^-$), hydrogen peroxide (H_2O_2), hydroxyl radicals (•OH), peroxynitrite ($ONOO^-$), and singlet oxygen ($^1O_2$) (Cyzmarova et al., 2023; Matías-Pérez et al., 2023; Xu et al., 2019).

Given that electroporation generates multiple ROS, quercetin's multifaceted antioxidant mechanisms make it a promising candidate

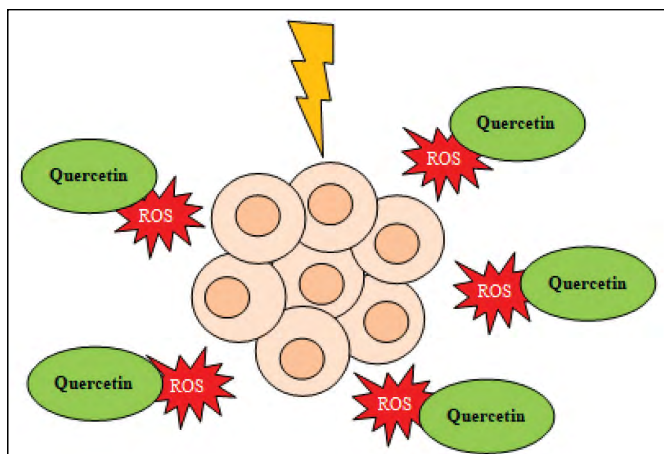

Fig. 1. Reactive oxygen species (ROS) generated by electroporation can be mitigated by antioxidant quercetin

for mitigating ROS-induced damage in electroporated cells. Nevertheless, the intracellular events and ROS-neutralising effects of quercetin under electroporation conditions remain poorly understood. To our knowledge, no prior studies have directly evaluated its protective potential in this context. Therefore, this study aimed to assess whether quercetin can attenuate oxidative damage and improve cell viability in Chinese hamster ovary (CHO) cells subjected to hydrogen peroxide or electroporation-induced oxidative stress.

MATERIALS AND METHODS

Cell culture

The Chinese hamster ovary (CHO) cells were used in this study. The cells were cultured in Dulbecco's Modified Eagle Medium (DMEM) (Sigma-Aldrich, Chemie GmbH, Steinheim, Germany), supplemented with 10% foetal bovine serum (FBS) (Sigma-Aldrich Chemie GmbH, Steinheim, Germany), 1% glutamin (Sigma-Aldrich Chemie GmbH, Steinheim, Germany), penicillin (100 U/mL), and streptomycin (100 µg/mL) (Sigma-Aldrich Chemie, GmbH, Steinheim, Germany). The cells were maintained at 37°C in a humid atmosphere with 5% CO_2.

Clonogenic Cell Survival

Cell viability was assessed using the standard colony formation assay (Franken et al., 2006). Serially diluted cell suspension was plated on antibiotic-supplemented DMEM. The plated cells were incubated for seven days. Colonies were treated with 1 mL of 96% ethanol (Stumbras, Kaunas, Lithuania), stained with Gram crystal violet solution (Fluka Chemie GmbH, Buchs, Germany), rinsed and air-dried and counted under bright-field microscope. Cell groupings consisting of at least 50 cells classified as a colony.

The effect of quercetin solvent on the viability of CHO cells

Approximately 300–400 CHO cells were seeded in Petri dishes with 2 mL of DMEM supplemented with antibiotics and 233 µl of different concentrations of quercetin dihydrate (Carl Roth GmbH, Karlsruhe) dissolved in 0.1% DMSO (Sigma-Aldrich, Chemie GmbH, France). The final concentrations of the quercetin dihydrate solutions in the medium ranged from 0.2 to 100.0 µM. Cell viability was assessed by their cloning efficiency.

Evaluation of the formation of ROS during a high-voltage pulse

Fluorescent Amplex Red (AR) dye was used for the detection of H_2O_2 (Audi et al., 2018). AR was prepared in PBS buffer (Sigma–Aldrich, Chemie GmbH, Steinheim, Germany) and electroporated between two stainless steel electrodes with a 0.2 cm gap between them. Electroporation was performed using 2 ms square electrical pulses with an intensity of 0.4–1.2 kV/cm (80–240 V). After treatment, 50 µl of electroporated AR was pipetted into the wells of the microplate twice to provide 100 µl of solution per well. Non-electroporated AR was used as a control. Fluorescence intensity was measured with a spectrofluorometer (Tecan Austria GmbH, Grödig, Austria) at a wavelength of 535–590 nm.

Assessment of quercetin ion effects on CHO cells following H_2O_2-induced oxidative stress

Approximately 300–400 CHO cells were plated in Petri dishes, each containing 2 mL of DMEM medium with antibiotics and 233 µl of 2 µM quercetin dihydrate solution. When studying the combined effect, H_2O_2 aqueous solutions were additionally added to the dishes to reach a final concentration of 0.1, 1, 10 or 100 µM. Cells exposed to H_2O_2 solutions only, with PBS added instead of quercetin, were used as controls. The cells were incubated for 8–10 days at 37°C in a 5% CO_2 environment. Cell viability was assessed by their cloning efficiency.

In parallel, CHO cells were cultured in Petri dishes for seven days to assess the effects of H_2O_2 on the cells over time. After seven days, cells were stained with 2 µM of calcein-AM dye (Sigma-Aldrich, Chemie GmbH,

Steinheim, Germany) for 20 min at 37°C. After staining, 1.5 mL of DMEM without phenol red and 0.5 mL of 3% of H_2O_2 were added to Petri dishes. The final concentration of H_2O_2 was 0.75%. Cell fluorescence was observed at 0, 5, 15, 30, and 40 minutes using a Motic BA400 microscope (Motic BA400, Germany). Viable cells emitted green fluorescence.

Evaluation of CHO cell viability after electroporation and exposure to quercetin ions

Cells were electroporated using a pulsed electroporator (ECM 2001, BTX, Holliston, USA), generating rectangular pulses of 2 ms duration and up to 1000 V amplitude. A 2 ms pulse duration was applied at voltages of 0.5 kV/cm, 1.0 kV/cm, and 2.0 kV/cm. Electroporation was performed using a reusable stainless steel electrode cuvette, into which 50 µl of cell suspension in Serum-like Modified Eagle's Medium (SMEM) (M8167, Sigma-Aldrich, Chemie GmbH, Steinheim, Germany) was added. After electroporation, cells were seeded into Petri dishes containing 2 mL of DMEM supplemented with antibiotics and 233 µl of a 2 µM quercetin dihydrate solution. Cell viability was assessed by their cloning efficiency.

RESULTS

Effects of quercetin dihydrate ions on cell viability

This stage of the study investigated the effect of quercetin dihydrate on CHO cell viability. As shown in Fig. 2, low concentrations of quercetin (1–2 µM) had no significant effect on cell viability ($p > 0.05$). However, starting from a concentration of 5 µM, cell viability began to decrease significantly to 83.3 ± 3.5% and progressively declined with increasing concentrations. The strongest cytotoxicity effect was observed at a concentration of 5 µM, when cell viability decreased significantly to 4.2 ± 0.5% indicating lethal concentration.

A concentration of 2 µM was chosen for subsequent experiments as it did not exhibit cytotoxicity in CHO cells ($p > 0.05$), while higher concentrations (≥5 µM) significantly reduced viability. This sub-cytotoxic dose ensured that the observed effects of quercetin in later experiments would not be confounded by toxicity.

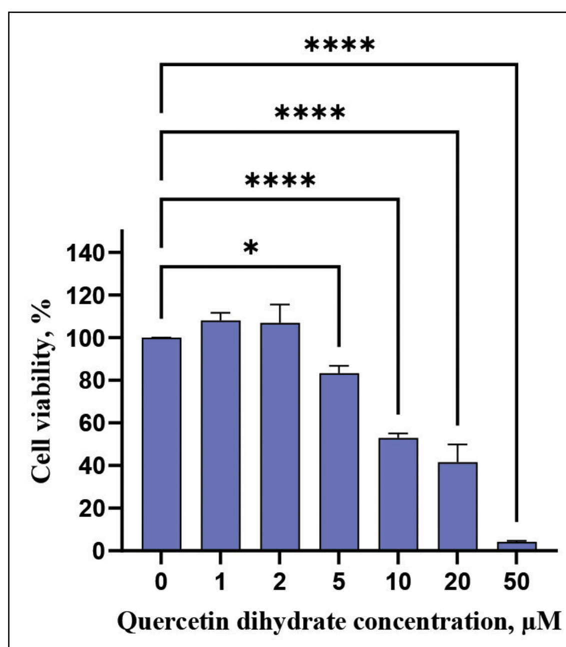

Fig. 2. Concentration-dependent effect of quercetin dihydrate on CHO cell viability. Whiskers represent standard deviation (SD). Significance was measured by using the one-way ANOVA test following Dunnet's post-hoc test. *, **** represent significance $p < 0.05$ and $p < 0.0001$, respectively

Dependence of Amplex Red fluorescence on high voltage intensity pulses

To assess the amount of ROS generated during electroporation, the fluorescent marker Amplex Red, which specifically reacts with H_2O_2, was used (Audi et al., 2018). The obtained results showed that as the electric field intensity increased from 0 to 1.2 kV/cm, the fluorescence intensity increased significantly from 355.2 ± 20.5 RLU to 1326.5 ± 35.5 RLU (Fig. 3).

These results confirm that oxidative stress induced by electrical pulses in cells is proportional to the intensity of the applied field.

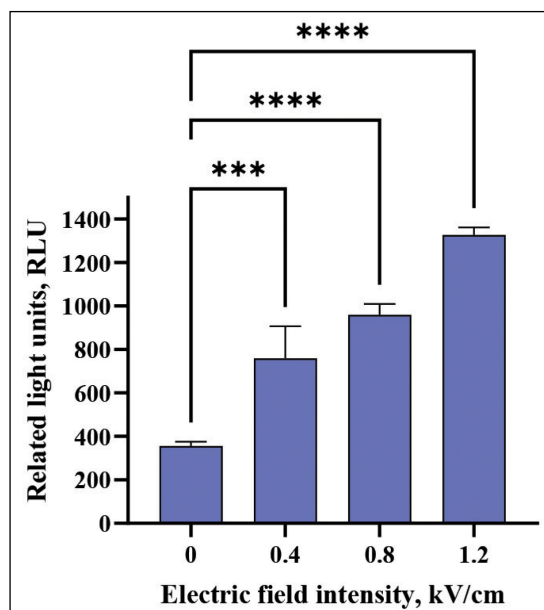

Fig. 3. Effect of high-voltage pulses on AR fluorescence intensity. Whiskers represent standard deviation (SD). Significance was measured by using the one-way ANOVA test following Dunnet's post-hoc test. ***, **** represent significance $p < 0.001$ and $p < 0.0001$, respectively

Effect of quercetin dihydrate on CHO cell viability after H_2O_2-induced mimic oxidative stress

Following H_2O_2 exposure, cell viability was also visualised using calcein-AM fluorescent staining. As shown in Fig. 4, the fluorescence signal remained stable for up to 15 min, indicating that during this period, the majority of cells maintained membrane integrity and esterase activity. However, after 30 and especially 40 min of exposure to 0.75% H_2O_2, a marked decrease in fluorescence intensity was observed, consistent with progressive loss of cell viability due to oxidative damage.

To assess protective effects of quercetin against oxidative stress, CHO cells were exposed to various concentrations of H_2O_2 (0.1–100 μM), both with and without quercetin (2 μM).

As shown in Fig. 5, at all concentrations of H_2O_2 tested, the presence of quercetin significantly increased cell viability compared to exposure to H_2O_2 alone. Despite increasing

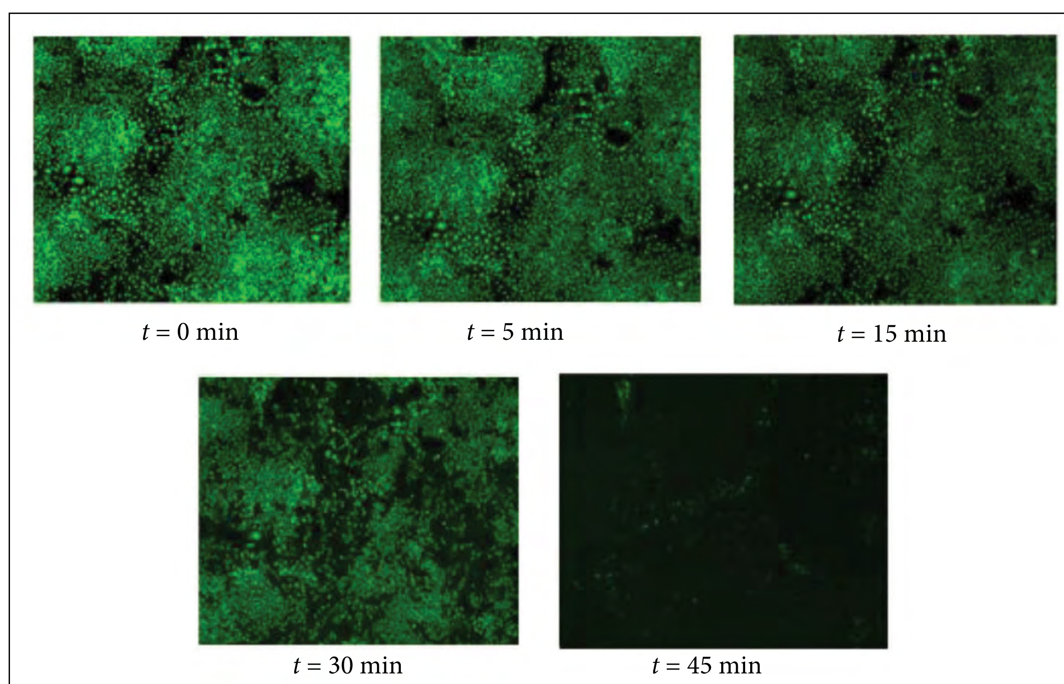

Fig. 4. Time-dependent effect of hydrogen peroxide on cell viability. Fluorescence microscopy images showing CHO cell viability at different time points (0, 5, 15, 30, and 40 minutes) after exposure to 0.75% H_2O_2. Cells were stained with calcein-AM, which labels live cells with green fluorescence

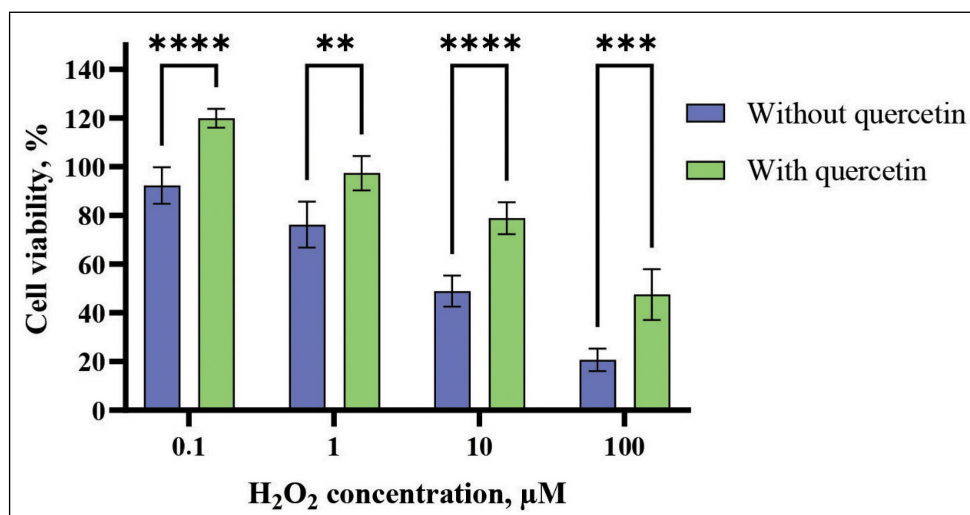

Fig. 5. CHO cell viability after H_2O_2 and quercetin treatment. Whiskers represent standard deviation (SD). Significance was measured by using the two-way ANOVA test following Tukey's post-hoc test. **, ***, **** represent significance $p < 0.01$, $p < 0.001$, and $p < 0.0001$, respectively. Control: 100% cell viability, 0 µM H_2O_2

oxidative stress with higher H_2O_2 concentrations, quercetin consistently demonstrated a statistically significant protective effect, keeping cell viability rates about 20–30% higher than when the antioxidant was not present. The most significant protective effect was noted at 10 µM H_2O_2, as quercetin raised cell viability from 48.9% to 78.9%, indicating a 30% enhancement.

Effects of electrical impulse and antioxidant quercetin ions on CHO cell viability

Having confirmed that quercetin dihydrate increases cell viability after H_2O_2-induced oxidative stress, the study further evaluated its effects after electroporation. The results are presented in Fig. 6.

At 0.5 and 1.0 kV/cm pulses, the presence of quercetin significantly increased cell viability around 11% and 17%, respectively, compared to electroporated cells without antioxidant. However, at a stronger pulse (2.0 kV/cm), quercetin no longer had a significant effect: in both cases, cell viability remained low (~20%), indicating that the strong electrical effect causes detrimental changes that quercetin is no longer able to compensate.

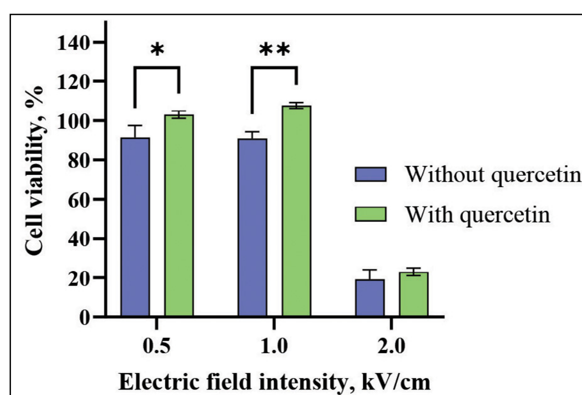

Fig. 6. CHO cell viability after electrical pulses and quercetin treatment. Whiskers represent standard deviation (SD). Significance was measured by using the two-way ANOVA test following Tukey's post-hoc test. *, ** represents significance $p < 0.05$ and $p < 0.01$ respectively. Control: non-electroporated cells with 100% viability at 0 µM quercetin

DISCUSSION

In this study, the antioxidant properties of quercetin were evaluated in CHO cells subjected to oxidative stress induced by hydrogen peroxide (H_2O_2) and electroporation. The initial experiments demonstrated that quercetin has

a concentration-dependent effect on cell viability: concentrations up to 2 μM were non-toxic, while concentrations from 5 μM and higher significantly reduced cell viability. This trend is consistent with previous studies. For example, Chen et al. (2019) reported that low doses of quercetin enhanced the viability of porcine intestinal cells, whereas higher concentrations (>20 μg/mL) were cytotoxic. These discrepancies suggest that the biological effects of quercetin are dose- and cell type-dependent.

A concentration of 2 μM was therefore selected for further experiments as it provided a balance between biological activity and safety, aligning with previous literature showing cytoprotective effects at comparable doses (Chen et al., 2019; Sul, Ra, 2021). Exposure of cells to H_2O_2 alone resulted in a concentration-dependent reduction in viability, while co-treatment with quercetin significantly increased survival at all tested H_2O_2 concentrations. The strongest protective effect was observed at 10 μM H_2O_2, where quercetin improved viability by 30%. This supports the idea that quercetin can attenuate chemically induced oxidative stress.

The protective effect of quercetin was also evident under physical stress conditions induced by electroporation. At 0.5 and 1.0 kV/cm, quercetin improved CHO cell viability by 11% and 17%, respectively. However, this benefit diminished at 2.0 kV/cm, suggesting that damage at higher intensities may exceed the antioxidant capacity of quercetin. These findings are consistent with prior reports indicating that electroporation generates ROS through both intracellular sources (mitochondrial dysfunction, NADPH oxidase activation) and extracellular mechanisms such as electrochemical reactions at the electrode–solution interface (Gabriel, Teissié, 1994; Pakhomova et al., 2012). The ROS involved include superoxide anions, H_2O_2, hydroxyl radicals, peroxynitrite, and singlet oxygen (Macarone et al., 1995), all of which are known to damage cellular macromolecules.

Compared to other antioxidants tested in the context of electroporation – such as Trolox, α-tocopherol, melatonin, sodium pyruvate, vitamin C, and tempol – quercetin stands out for its broad ROS-targeting profile and non-disruptive nature. While some antioxidants have been shown to impair electroporation efficiency depending on their timing and concentration (Tóthová et al., 2016), quercetin appeared to improve viability without interfering with the electroporation process under moderate field strengths (Kanduser et al., 2019; Nuccitelli et al., 2014; Markelc et al., 2012; Guo et al., 2021).

Taken together, these findings indicate that quercetin is a promising antioxidant for biomedical or biotechnological applications involving electroporation, particularly in the contexts in which oxidative stress compromises cell viability. Nevertheless, certain aspects were not addressed in this study. Specifically, intracellular levels of ROS and cell death mechanisms (e.g., apoptosis or necrosis) were not directly evaluated. To gain a deeper insight into the mechanistic role of quercetin and its long-term protective effects following electroporation, future studies should incorporate fluorescent ROS indicators (e.g., DCFDA), apoptosis markers (e.g., Annexin V/PI), and lipid peroxidation assays (e.g., TBARS).

CONCLUSIONS

This study demonstrated that quercetin exhibits protective antioxidant effects in CHO cells subjected to oxidative stress caused by both chemical agents and electroporation. The findings confirm that quercetin can mitigate damage induced by reactive oxygen species, enhancing cell survival under moderate stress conditions. While its protective effect diminishes at higher electric field intensities, quercetin remains a promising candidate for use in protocols in which maintaining cell viability is critical, such as in gene transfer or electrochemotherapy. These results provide further support for the potential application of naturally derived antioxidants in electroporation-based biomedical technologies.

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ANTIOKSIDACINIS KVERCETINO POVEIKIS KININIO ŽIURKĖNUKO KIAUŠIDŽIŲ (CHO) LĄSTELĖMS IR JŲ ELEKTROPORACIJAI

Santrauka

Elektroporacija yra toks reiškinys, kai trumpas stipraus elektrinio lauko impulsas laikinai padidina biologinių membranų pralaidumą jonams ir molekulėms. Šio proceso metu plazminėje membranoje susidaro laikinos poros, o elektrodų ir tirpalo sąveikos paviršiuje bei pačiame tirpale vyksta elektrocheminės reakcijos, dėl kurių formuojasi reaktyviosios deguonies formos (ROS) ir gali sumažėti ląstelių gyvybingumas. Šiame darbe buvo vertintas ROS, sukeltų vandenilio peroksido (H_2O_2) ir elektroporacijos, poveikis kininio žiurkėnuko kiaušidžių (CHO) ląstelėms bei galimas antioksidacinis kvercetino apsauginis poveikis. CHO ląstelių suspensijos buvo paveiktos įvairiomis H_2O_2 ir kvercetino koncentracijomis bei elektroporuotos vienetiniu 2 ms trukmės, skirtingo intensyvumo elektrinio lauko impulsu (0,5–2,0 kV/cm). Ląstelių gyvybingumas vertintas kolonijų formavimo metodu, o ROS kiekis nustatytas naudojant *Amplex Red* fluorescencinį testą. Mažesnės nei 2 μM koncentracijos kvercetinas nebuvo citotoksiškas, tačiau didesnės jo koncentracijos mažino ląstelių gyvybingumą. Visose tirtose H_2O_2 koncentracijose kvercetinas reikšmingai padidino ląstelių išgyvenamumą, ypač esant vidutiniam oksidaciniam stresui. Kvercetinas taip pat padidino gyvybingumą po elektroporacijos, kai elektrinio lauko intensyvumas buvo 0,5 ir 1,0 kV/cm, tačiau apsauginis poveikis išnyko esant 2,0 kV/cm. Gauti rezultatai leidžia teigti, kad kvercetinas veiksmingai mažina oksidacinį stresą, sukeltą tiek cheminiu, tiek fiziniu būdu, ir gali būti naudinga citoprotekcinė priemonė biomedicininiuose taikymuose, susijusiuose su elektroporacija.

Reikšminiai žodžiai: elektroporacija, kvercetinas, kininio žiurkėnuko kiaušidžių ląstelės, CHO, oksidacinis stresas