INVESTIGACIÓN - MEDICINA

# PHOTOBIOMODULATION EFFECTS ON THE VIABILITY OF C2C12 MUSCLE CELLS EXPOSED TO DEXAMETHASONE

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INTRODUCTION: Skeletal muscle homeostasis relies on the balance between proliferation, differentiation, and regeneration, which can be impaired by pathological conditions or prolonged glucocorticoid exposure. Dexamethasone (DEXA), a synthetic glucocorticoid, is widely used for its anti-inflammatory and

immunosuppressive properties, but chronic or high-dose administration induces muscle atrophy. Photobiomodulation (PBM) has demonstrated anti-inflammatory and regenerative effects in muscle cells. However, its ability to counteract glucocorticoid-induced cytotoxicity remains underexplored. OBJECTIVE: This study aimed to evaluate the effects of PBM on the viability of C2C12 myoblasts exposed to different concentrations of DEXA during differentiation. METHODOS: C2C12 cells were cultured in DMEM with 10% fetal bovine serum for proliferation and differentiated in DMEM with 2% horse serum. Cells were incubated with DEXA at 5, 10, or 200 μM for 24, 48, or 72 h. PBM was applied using a 780 nm laser (70 mW, 15 s, 26.25 J/cm²). Cell viability was assessed by MTT assay after 48 and 72h. Experimental groups included: control, DEXA-only, and DEXA+PBM at each concentration and time point. Data were analyzed by ANOVA followed by Tukey's post-hoc test ($p<0.05$). RESULTS: DEXA induced a decrease in cell viability in a time- and dose-dependent manner, with the greatest reduction at the higher concentration (200 μM). PBM significantly increased viability in cells treated with 5 or 10 μM DEXA and partially restored viability at 200 μM. At 48 h, PBM mitigated the pronounced viability loss observed with 10 and 200 μM DEXA. At 72 h, although viability remained below control levels, PBM-treated groups consistently showed higher values than DEXA-only groups. PBM alone had no detrimental effect. CONCLUSION: PBM improved the viability of C2C12 myoblasts exposed to DEXA, particularly at intermediate concentrations, indicating its potential to mitigate glucocorticoid-associated muscle atrophy.

Palavras-chave: c2c12; dexamethasone; muscle cells; photobiomodulation.