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PHOTOBIOMODULATION MAY REVERSE CELLULAR SENESCENCE BY INDUCING CELL PROLIFERATION AND PRESERVING NUCLEAR SIZE

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Background: Cellular senescence is an irreversible state of cell cycle arrest, thus being characterized by decreased cell proliferation and increased nucleus area, often acting as a tumor suppressor program. Photobiomodulation (PBM) has been used in several conditions to increase the mitochondrial response, promoting nuclear changes and cell proliferation. However, the effects of PBM on cells are still unclear.
Objectives: To verify the efficacy of photobiomodulation on cell senescence processes.
Methods: We utilized A172 glioblastoma cells transduced with H2B-mCherry by lentivirus to nuclear tagging. Treatment was done with GaAlAs Laser (850nm). Cells were divided by intensity into the following groups: C= Control, L1= 1J/cm^2, L2= 2.2J/cm^2, L3= 3J/cm^2, L9= 9J/cm^2, L15= 15J/cm^2, L21= 21J/cm^2, nuclear evaluation was performed at experimental times (0h, 24h, 48h and 72h). For data analysis, two-way ANOVA with the Tukey post hoc test was used. Differences were significant when $p<0.05$.
Results: PBM on intensities of 1J/cm^2, 2.2J/cm^2, 3J/cm^2, 9J/cm^2 e 15J/cm^2 showed a lower increase at the nuclear size when compared with time 0h and 72h in the control group. All intensities (1, 2.2, 3, 9, 15, and 21 J/cm^2) promoted cellular proliferation after 72 hours, while 15J/cm^2 presented an accentuated increase compared to groups L1, L2.2, and L3.
Conclusion: PBM enhanced cellular proliferation while causing a reduced nuclear increase in glioblastoma cells.
Implications: In this study, we found that the laser decreased the cellular senescence state from the evaluation of the morphological parameters, thus increasing cell proliferation and decreasing the nuclear area; therefore, it is an important therapeutic tool against the cellular aging process.
Keywords: Photobiomodulation, Glioblastoma, Cellular senescence

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PHYSICAL EXERCISE AND PHOTOBIOMODULATION INCREASE NRF2 EXPRESSION IN THE SKELETAL MUSCLE OF RATS WITH HEART FAILURE AND DIABETES MELLITUS

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Background: Heart failure (HF) and type 2 diabetes mellitus (DM2) are prevalent diseases worldwide, and both can cause muscle atrophy. Both disorders are related to increased autophagy and apoptosis in muscle cells, consequently reducing muscle volume. Physical exercise associated with photobiomodulation seems promising to attenuate the skeletal muscle changes caused by HF and DM2.
Objectives: To verify the influence of physical exercise and the association with photobiomodulation on autophagy, apoptosis, and cell survival signaling pathways in myocytes from rats with HF and DM2.
Methods: 18 male rats were divided into four groups: CT (not included in protocols), CT- (HF + DM2), EX+HF+D (HF + DM2 + aerobic exercise), and EX+HF+D+P (HF + DM2 + aerobic exercise + photobiomodulation). To induce DM2, streptozotocin (0.25 ml/kg, i.p.) was injected. To induce HF, coronary ligation was performed. After one week of disease induction, aerobic exercise, and photobiomodulation protocol were started for eight weeks. The protein expressions

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