Red Light Photobiomodulation and Collagen Regulation in Dermal Fibroblasts Under Nicotine-Induced Oxidative Stress

Skin aging and extracellular matrix degradation are influenced by oxidative stress and environmental exposures. Tobacco-related compounds increase reactive oxygen species (ROS) within dermal fibroblasts. These compounds may activate signaling pathways that elevate matrix metalloproteinases (MMPs) and accelerate collagen degradation. This process contributes to structural changes in the dermal matrix and visible aging of the skin. Recent research suggests that red light photobiomodulation (PBM) may influence mitochondrial activity and cellular signaling involved in tissue repair and collagen synthesis. This project examines the effects of red light photobiomodulation on collagen expression and oxidative stress pathways in human dermal fibroblasts exposed to nicotine-induced cellular stress. Fibroblasts will be cultured under standard conditions and divided into four experimental groups: untreated control, red light exposure only, nicotine exposure only, and nicotine combined with red light treatment. Red light wavelengths in the ~630–660 nm range will be used to stimulate photobiomodulation pathways associated with mitochondrial signaling. Oxidative stress will be measured using mitochondrial ROS detection assays (MitoSOX Red), and gene expression analysis will be conducted using quantitative PCR to evaluate changes in collagen type I (COL1A1) and matrix metalloproteinase-1 (MMP-1). Results from this work may provide insight into mechanisms through which red light photobiomodulation influences oxidative stress signaling and collagen regulation in dermal fibroblasts. Understanding mechanisms may contribute to the development of non-invasive strategies for improving tissue repair, mitigating environmentally induced dermal damage, and advancing light-based biomedical therapies.