RED LIGHT PHOTOBIOMODULATION AND COLLAGEN REGULATION IN DERMAL FIBROBLASTS UNDER NICOTINE-INDUCED OXIDATIVE STRESS

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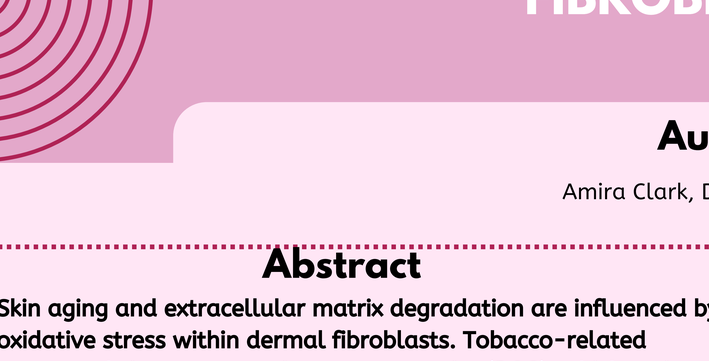
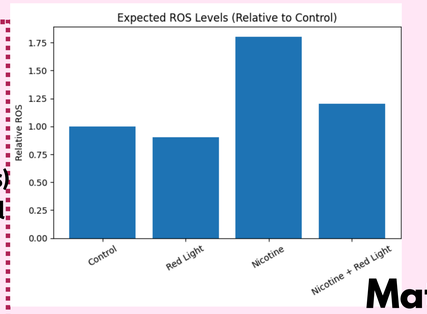
Abstract

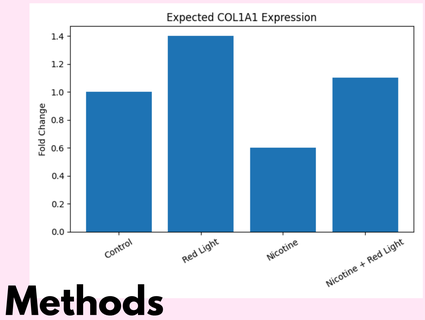
Skin aging and extracellular matrix degradation are influenced by oxidative stress within dermal fibroblasts. Tobacco-related compounds increase reactive oxygen species (ROS), which activate signaling pathways that elevate matrix metalloproteinases (MMPs) and promote collagen degradation. Photobiomodulation using red light (~630–660 nm) may influence mitochondrial activity and cellular signaling associated with tissue repair. This project examines how red light photobiomodulation influences oxidative stress and collagen regulation in human dermal fibroblasts exposed to nicotine-induced cellular stress. Fibroblasts will be cultured under controlled conditions and exposed to nicotine and/or red light treatment. Oxidative stress will be measured using mitochondrial ROS assays, and gene expression of collagen type I (COL1A1) and MMP-1 will be quantified using qPCR. Understanding these mechanisms may help identify non-invasive strategies for improving tissue repair and mitigating environmentally induced dermal damage.



Background

Skin aging and extracellular matrix degradation are influenced by oxidative stress in dermal fibroblasts. Tobacco-related compounds increase reactive oxygen species (ROS), which activate signaling pathways that elevate matrix metalloproteinases (MMPs) and promote collagen degradation. Red light photobiomodulation (~630–660 nm) has been shown to influence mitochondrial activity and cellular signaling involved in tissue repair and collagen regulation. Understanding these mechanisms may help identify non-invasive strategies for improving tissue repair and mitigating environmentally induced skin damage.





Materials and Methods

human dermal fibroblasts are cultured under standard laboratory conditions (37˚C, 5% CO_2). Cells will be divided into four experimental groups:
Control – untreated fibroblasts
Red light only – fibroblasts exposed to photobiomodulation (~630–660 nm)
Nicotine only – fibroblasts exposed to nicotine
Nicotine + red light – fibroblasts exposed to both treatments
Reactive oxygen species will be measured using mitochondrial ROS detection (MitoSOX Red fluorescence). Gene expression will be analyzed using quantitative PCR to evaluate collagen type I (COL1A1) and matrix metalloproteinase-1 (MMP-1).

Expected Results

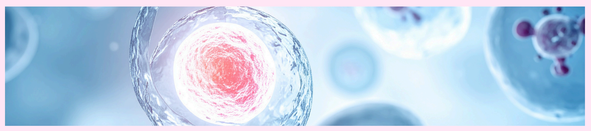
Nicotine exposure is expected to increase mitochondrial reactive oxygen species (ROS) and matrix metalloproteinase (MMP) expression while reducing collagen expression in dermal fibroblasts. Red light photobiomodulation (~630–660 nm) may reduce oxidative stress and increase collagen gene expression compared to nicotine-treated cells.

Expected Analysis

Fluorescence measurements will be used to quantify mitochondrial ROS levels across treatment groups. Gene expression data from qPCR will be analyzed to compare collagen type I (COL1A1) and MMP-1 expression between control, nicotine-treated, red light–treated, and combined treatment groups.

Conclusion

Based on prior research, irradiation at 660nm is expected to enhance fibroblast activity by increasing mitochondrial function and upregulating genes invloved in collagen synthesis and cellular repair. In contrast, exposure to nicotine and tobacco-derived compounds has been shown to induce oxadative stress and degrade extracellular matrix components, contributing to premature skin aging.



Key Sources & Acknowledgements

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List the primary sources used during the research, such as academic articles, books, and websites.

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