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Research Category

Basic Science

Abstract Title

Antioxidant Modulation of Wood Smoke–Induced Injury in Murine and Human Lung Fibroblasts

Authors

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Background

Climate change is increasing wildfire activity and wood-smoke (WS) exposure, which can induce oxidative lung injury. Lung fibroblasts are essential for tissue repair following injury.

Objective

We hypothesized that wood smoke extract (WSE) would impair fibroblast wound closure and that vitamin C (VC), vitamin E (VE), and N-acetylcysteine (NAC) would mitigate these effects.

Methods

Murine CCL-206 and human MRC5 lung fibroblasts were exposed to WSE antioxidants, or their combinations. Wound-scratch assays measured closure at 24 and/or 48 h, and RT-qPCR assessed stress-, repair-, and fibroblast activation-associated genes.

Results

WSE impaired wound closure in a concentration-, time-, and cell-dependent manner. At 1% WSE, closure was significantly reduced at both 24 and 48 h in CCL-206 cells ($p=0.0100$ and $p=0.0024$) and MRC5 cells (both $p<0.0001$). At 0.5% WSE, closure was reduced at 48 h in CCL-206 cells ($p=0.0004$) and at both 24 and 48 h in MRC5 cells ($p=0.0279$ and $p=0.0224$). WSE altered stress- and repair-associated gene

expression, increasing Cyp1b1, Mgst1, and Hmox1 in CCL-206 cells and CYP1A1, WNT5B, and HMOX1 in MRC5 cells (all $p < 0.05$). Antioxidant effects on wound closure varied by compound and condition. VE (100 μM) increased closure in untreated CCL-206 cells at 24 h ($p = 0.0159$) and MRC5 cells at 48 h ($p = 0.0452$). In CCL-206 cells, co-treatment with 0.5% WSE and 100 μM VE increased closure above control levels at 48 h ($p = 0.0071$). However, VE did not restore closure following 1% WSE exposure, remaining impaired in CCL-206 cells at 48 h ($p = 0.0238$) and MRC5 cells at both timepoints ($p < 0.0001$). Other antioxidant treatments had no significant influence on wound closure.

Conclusion

WSE impairs lung fibroblast wound closure and alters oxidative stress-associated pathways. Antioxidant effects vary by compound, concentration, exposure severity, and cell type. VE improved wound closure under selected conditions. Further studies will assess VE protection and epithelial-fibroblast interactions following WSE exposure using lung organoid models.