

# Nitrated Fatty Acid Inhibition of Bleomycin-Mediated Pulmonary Fibrosis

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Interstitial lung disease (ILD) is highly prevalent in the United States, with limited treatment options leading to high morbidity and mortality. ILD is characterized by interstitial remodeling and fibrosis, which can be evaluated by collagen deposition. Nitrated fatty acids, such as nitrooleic acid (OA-NO<sub>2</sub>), are endogenously formed signaling mediators that have demonstrated anti-inflammatory and anti-fibrotic properties but have not been extensively studied in the lung. To assess their potential to improve fibrosis, an intraperitoneal bleomycin (IPB) model was employed. We hypothesize that OA-NO<sub>2</sub> will limit pulmonary fibrosis in a model of ILD based on its anti-inflammatory, pro-survival properties. C57BL6/J mice were injected with 0.1U IPB or PBS every 3d for 15d and were administered 50μg OA-NO<sub>2</sub> intratracheally on days 0, 4, 9, 16, 25, and 35. Mice were allowed to recover to 40d, after which lung tissue was collected for immunohistochemistry (IHC) and Mason's Trichrome staining to observe collagen deposition. In trichrome stained tissues, mice administered IPB had higher fibrosis scores than control mice (1±0.3 vs 4.5±1.3\*score). This was mitigated by the administration of OA-NO<sub>2</sub> (2.5±2.3<sup>#†</sup>). Increased collagen deposition in IPB mice compared to controls (5±2.7 vs 28±8.8\*count), which was mitigated by OA-NO<sub>2</sub> administration (6±1.6<sup>†</sup>count), supported these findings. In summary, bleomycin-mediated increases in collagen deposition were mitigated by the administration of OA-NO<sub>2</sub>, indicating its potential as an anti-fibrotic therapeutic in ILD. Supported by the ASPET SURF Intern Program and NIH R25ES020721.

