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A Computational Model of Lung Fibroblast Migration with In Vitro Validation

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Chronic Obstructive Pulmonary Disease (COPD) is currently the 3rd leading age-adjusted cause of death in the United States. The primary cause of this disease is known to be tobacco smoke, yet it is unclear which cellular pathways produce these symptoms. Fibroblasts are known for their roles in tissue inflammation and remodeling, and these functions have been found to be inhibited in COPD patients. To evaluate how lung fibroblast populations from COPD patients differ from healthy ones, we developed an agent-based model of lung fibroblasts during wound healing using the NetLogo platform. This model separates the healing response in terms of the migration, proliferation, death and senescence rates of these cells, and accounts for the effects of serum deprivation and cigarette smoke condensate exposure. Simulations were performed in BehaviorSpace to select biologically suitable parameter sets for each cell type. Model results were validated using data gathered from *in vitro* experiments consisting of scratch-migration assays and MTT assays. This model is the first step in creating a computational tool that will allow us explore the role of these fibroblast functions on the overall disease progression and evaluate responses to therapeutics for COPD.