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The overarching objective of this proposal is to gain in-depth understanding of how macrophages orchestrate the development of a specific lung disease, idiopathic pulmonary fibrosis (IPF). Many of the salient features of IPF are similar to the disease of the geriatric lung, termed pulmonary fibrosis of aging (PFoA). This proposal specifically examines how the regulatory pathway known as the translational/signal transduction-1 (TS-1) pathway is regulated in the geriatric lung to promote a fibrogenic response (loss of E-cadherin and gain of fibronectin) in resident macrophages.

Recently, we made the novel observation that these fibrogenic pathways in macrophages are markedly suppressed in aged mice as compared to young mice. Further studies demonstrated that pharmacologic therapy with 4-phenylbutyric acid (4-PBA) maintained E-cadherin expression and thereby inhibited the fibrotic phenotype in aged mice. In contrast, inhibition of TS-1 pathways using dithiothreitol (DTT) suppressed E-cadherin expression and increased fibronectin in aged mice. This is a novel finding in geriatric lung disease. Further studies showed that aged macrophages also exhibit higher levels of

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