The Impact of Air Pollution Interventions on ILD

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Interstitial lung diseases (ILDs) comprise over 300 rare and heterogeneous disorders characterized by chronic inflammation and/or fibrosis of the lung parenchyma, leading to progressive functional decline and impaired gas exchange. Epidemiologic studies consistently associate ambient air pollution—particularly fine particulate matter ≤2.5 μm in diameter (PM□.□), nitrogen dioxide, and ozone—with increased ILD incidence, faster decline in forced vital capacity (FVC), higher risk of acute exacerbations (AEs), and increased mortality.

Mechanistic evidence demonstrates that air pollutants amplify pathological pathways already active in ILD lungs, including oxidative stress–mediated transforming growth factor-β (TGF-β) signaling, immune dysregulation, and cellular aging processes such as telomere shortening and epigenetic age acceleration. Gene–environment interactions, particularly in individuals with telomere-related gene mutations, further enhance susceptibility.

Despite strong biologic plausibility and robust observational data, there are no randomized controlled trials (RCTs) targeting air pollution exposure reduction specifically in ILD. Existing evidence, largely extrapolated from chronic obstructive pulmonary disease, asthma, and general populations, shows that personal-level measures—high-efficiency particulate air filtration (HEPA), appropriate respirator mask use, and structured behavioral modification—can lower PM□.□ exposure and modestly improve symptoms or quality of life, particularly with high adherence. Policy-level interventions, such as occupational dust regulation in pneumoconiosis, have reduced disease burden by over 40% in two decades, illustrating the value of sustained exposure control.

The absence of ILD-specific intervention trials limits guideline development and policy translation. Mechanistically vulnerable ILD patients may benefit most from integrated personal–policy approaches. Future priorities include multi-component RCTs combining HEPA filtration, personalized behavioral coaching, and policy integration, with outcomes spanning both clinical endpoints (FVC decline, AE incidence) and mechanistic biomarkers (oxidative stress, telomere biology, epigenetic modifications).

In conclusion, ambient air pollution acts as a potent modifier of ILD trajectory. ILD-focused intervention research is urgently needed to inform clinical practice and environmental policy.