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Making the case for causality: what role do lung microbiota play in idiopathic pulmonary fibrosis?

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Though the notion of a "lung microbiome", *i.e.* a complex community of microbes inhabiting and influencing the lower respiratory tract, is barely a decade old [1], there is nothing new about the idea that the lungs represent a crucial interface between us and the microbial universe we inhabit. Louis Pasteur demonstrated the presence of viable microbes in air [2], and one of his contemporaries calculated that healthy adults inhale between 1500 and 14 000 organisms each hour [3]. Numerous radiographic studies have repeatedly demonstrated the ubiquity of subclinical microaspiration [4–6], even in healthy, asymptomatic subjects. The surface area of the lungs is twice that of the gastrointestinal tract [7] and 30 times that of the skin [8], representing the body's largest interface with the outside environment. Thoughtful investigators of lung biology have long suspected that the mechanisms our lungs evolved to cope with this continuous microbial exposure (innate and adaptive immune defences, the establishment and resolution of fibrosis) play central roles in the pathogenesis of lung disease. As pulmonary biologist Galen Toews commonly said, long before the lung microbiome era: "All lung disease is infectious disease" (figure 1).