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Leveraging early markers of cystic fibrosis structural lung disease to improve outcomes

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The natural history of cystic fibrosis (CF) lung disease has been described as a complex process of obstruction, infection, and inflammation, all contributing to airways injury and eventual structural disease (*i.e.* bronchiectasis). Consequences of the CF genetic abnormality begin *in utero* and are the basis for newborn screening for elevated levels of circulating immunoreactive trypsinogen (IRT) [1]. Thus, it should be no surprise that the process of airway obstruction begins immediately at birth. Ample cross-sectional evidence has been published of airway obstruction, inflammation, bacterial infection, bronchial wall thickening, air trapping and bronchiectasis in the first year of life for infants with CF [2].