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Small airways and early origins of COPD: pathobiological and epidemiological considerations

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Until recently, the conceptual model of the natural history of COPD has been greatly focused on an accelerated decline of lung function, which would occur in adult life in susceptible individuals when chronically exposed to environmental noxious agents, such as cigarette smoke and other inhaled gases. However, 25%–45% of all COPD patients worldwide have never smoked [1]. In addition, pre- and peri-natal factors associated with diverse biological mechanisms can determine low lung function in young adults [2–4]. Among these factors, asthma and active and/or passive smoking act synergistically to affect early lung function deficits in young adulthood [5, 6]. The impact of smoke exposure in susceptible smokers is detectable with exposures to as little as 8–10 pack-years, in their late 30 s to early 40 s [7]. Similarly, early onset asthma is observed in 26% of those diagnosed with COPD at a mean age of 37 years, which represents a 20-fold increased risk of adult airway obstruction compared with those without asthma diagnosis [8]. However, what the combined effects of current active smoking and asthma are in a population of young adults is unknown.