Smoking and Lung Function: Taxing the Lungs

According to a World Health Organization report, cigarettes kill an estimated 5 million people annually worldwide. Tobacco smoking killed 100 million people worldwide in the 20th century and it could kill one billion people around the world in the 21st century and by early 2030, tobacco related deaths would increase to about 10 million a year. Tobacco smoking rates have decreased in industrialized countries since 1975, but there has been a corresponding 50% increase in smoking rates in low-income countries.

In India, smoking is a common habit prevalent in both urban and rural areas in various forms that include cigarettes, bidis, pipes, cigar, hookah etc; commonest being bidis (54%), followed by smokeless tobacco (27%) and cigarettes (9%).

The cigarette/bidi smoke is a heterogeneous aerosol produced by the incomplete combustion of the tobacco leaf. Smoking is the primary causal factor for at least 30% of all cancer deaths, nearly 80% of deaths from chronic obstructive pulmonary disease (COPD), and for early cardiovascular disease and deaths. COPD is an important cause of morbidity and mortality being listed currently as the fourth leading cause of death worldwide and estimated to register an even further increase by 2020. India is one of the countries identified to have a significant increase in the burden of tobacco related mortality.

A theoretical model which plots measured FEV1 as a percentage of the maximal attainable FEV1 proposes that maximal lung development is attained at the age of 20 years. This is followed by an age range of 25-35 years called as the plateau phase during which the FEV1 remains relatively stable. Lung function starts to decline after 40 yrs of age, and slightly earlier in females than in males. Thus the predicted lung functions attained during adult life depends upon childhood growth, age of onset of the decline, accelerated decline or any combination of these three possible mechanisms.

Cigarette smoking is the most well known risk factor for accelerating lung function decline in adults. In the landmark study by Fletcher and colleagues, the rate of decline of FEV1 was up to 18mL/year more in smokers as compared to non-smokers. Almost similar observations have been made in other studies as well. In the current issue of the journal, Nadeem et al have reported on the lung function of smokers in Maharashtra and reported a decline in various parameters demonstrated on spirometric measurements.

The rate of decline in FEV1 has been largely dose related and nearly approaches that of the non-smokers upon cessation of smoking. The lung function recovers faster in women quitters than in men, as shown in the SPALDIA 2 study. However, current women smokers with airway obstruction experience a greater smoking related decline in lung function than men. Why women have more decline in their lung functions on exposure to smoking is largely unknown. One possible explanation could be a concentration of the smoke in the smaller size of the female lungs. The second possibility is because of the small calibre of airways and consequently to the smaller baseline FEV1. In the Swiss Study on Air Pollution and Lung Diseases in Adults in which current smokers between the age of 18 – 60 yrs were assessed, women current smokers with airway obstruction experienced a greater smoking related decline in lung function than men.

The effects of smoking on the development and functions starts from the womb. There are enough studies to quote that have shown detrimental long-term pulmonary outcomes in the offspring of mothers who smoke during pregnancy. Significant suppression of alveolarization, functional residual capacity, and tidal flow volume has been demonstrated in the offspring of women who smoked during pregnancy. The underlying mechanisms and effector molecules involved in this process are not completely understood. However, it has been shown convincingly that in utero nicotine exposure disrupts specific molecular paracrine communications.
between epithelium and interstitium that are driven by PTHrP and PPARα, resulting in transdifferentiation of lung lipofibroblasts to myofibroblasts, i.e., the conversion of the lipofibroblast phenotype to a cell type that is not conducive to alveolar homeostasis and is the cellular hallmark of chronic lung disease including asthma. Not only does maternal smoking affects the lung functions in the offsprings but paternal smoking has been also shown to effect the lung development. A cross-sectional study to investigate paternal smoking and children’s pulmonary function in rural communities of China showed that children whose fathers smoked ≥30 cigarettes/day had the largest deficits in both FEV₁ and FVC. They concluded that there is a monotonic exposure-response relationship between paternal smoking and decline of pulmonary function in children. This effect could be because of side smoke exposure of the smoke.²⁴

In utero exposure to cigarette smoke has been shown to have a negative effect on lung function and the effect persists for a long time. Bozen and colleagues showed that FEV₁ values at age 6 years were lower in children who had been exposed in utero to cigarette smoke. Large, persistent deficits in lung function have been documented in children who developed asthma.²⁴ Not only is there an accelerated decline in lung functions, but exposure to smoking in utero or during first few months of life leads to an increased incidence of asthma and its instability.²⁵-²⁸

Finally, parental smoking had clinically significant effects on the FEV₁/FVC ratio among adolescents with wheeze and asthma in a longitudinal study, and a subsequent cross-sectional population-based study in China.²⁹

In a study in adolescent males and females, Diane et al found a dose–response relation between smoking and lower levels of both FEV₁/FVC ratio as well as the FEF 25–75, suggesting that smoking not only affects the large airways (demonstrated in the ratio of FEV₁/FVC) but also the smaller airways. Each pack per day of smoking was associated with a 3.2 percent reduction in FEF 25–75 for girls (p = 0.01) and a 3.5 percent reduction in FEF25–75 for boys (p = 0.007). Whereas the FVC level was elevated in smokers, the rate of growth of FVC and FEV₁ was reduced. There was a positive correlation with the amount of smoking and absolute level of attainment of FEV₁. They also found that adolescent girls were more vulnerable to smoking in comparison to boys. This gender difference was also shown in another landmark trial called the Lung Health Study (LIIS). The LIIS study followed the enrolled participants for 5 years and demonstrated changes in lung function by gender, treatment group, and smoking history categories. Among participants who quit smoking in the first year, mean FEV₁ expressed as a percentage of the predicted value of FEV₁, given the person’s age, height, gender, and race (FEV₁,%) increased more in women (3.7% of predicted) than in men (1.6% of predicted). Across the 5-year follow-up period, among sustained quitters, women gained more in FEV₁, % of predicted than did men.

Cigarette smoking is by far the single most clearly established environmental risk factor for COPD.²³ So far, longitudinal studies are lacking to show whether in utero smoke exposure is indeed a risk factor for COPD. The observation that active smoking during adolescence is associated with a shortening of the plateau phase of FEV₁ that generally occurs between 20 and 35 years of age suggests that there is an overall negative effect of smoking in adolescence. Smoking cessation during adolescence has a positive impact on lung growth.²⁶

The study by Nadeem et al is just another admonition of the effects of smoking on lung function in the Indian context and only re-emphasizes that the efforts for smoking cessation must be propelled with greater force than ever.

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References


