# Commentary

# Chronic obstructive pulmonary disease and tuberculosis

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Chronic obstructive pulmonary disease (COPD) and tuberculosis (TB) are two important causes of mortality and morbidity in our country and are among top 10 causes of death. <sup>[1]</sup> The interrelationship between TB and COPD is very complex. A substantial number of TB patients develop posttubercular airway disease or TB-associated COPD. <sup>[2-5]</sup> This is the most commonly reported relationship. However, many different associations have also been published.

- a. COPD patients are also at high risk of developing pulmonary  $TB^{\scriptscriptstyle [6]}$
- b. COPD is a common comorbidity in patients with TB, second only to diabetes<sup>[7-10]</sup>
- c. History of TB negatively impacts the long-term course of COPD with early mortality and increased frequency of exacerbations<sup>[11]</sup>
- d. COPD also alters the clinical presentation of TB and is a risk factor for increased morbidity and mortality from TB.<sup>[12,13]</sup>

In the article by Agarwal *et al.* published in this issue of Lung India, 32.4% of COPD patients had a history of TB.<sup>[14]</sup> In the PLATINO study which compared COPD patients with and without TB history, it was found that airway obstruction was observed in 30.7% of patients with a positive history of TB, while the incidence for the same was only 13%–9% in those without a prior history.<sup>[15]</sup> In our study on gender differences in COPD, history of TB was present in 28.4% of males and 29.7% of females.<sup>[3]</sup>

Even a study from Columbia has reported that the association between TB and airway obstruction was stronger than that observed between smoking and airway obstruction.[16] Yakar et al. evaluated the effect of TB history on natural course of COPD' according to them, patient with history of TB was diagnosed with COPD 5 years earlier, hospitalized more often due to COPD exacerbations, and had a life expectancy 5 years shorter[11] Further, these patients with COPD secondary to TB have been shown to have significantly low forced expiratory volume in 1 s, higher airway resistance, and poor positive bronchodilator response (27% vs. 82%) than only COPD patients.[17] The abnormalities in lung functions were significantly associated with extent of lung involvement with TB, duration of disease, episodes of TB and age over 40 years.[10,18,19]

Tobacco smoking is a major factor in the development of COPD. The association between cigarette smoking, accelerated loss of lung functions, and COPD is well established.<sup>[20,21]</sup>

Cigarette smoking also increases the risk of developing TB by 3–5 folds.<sup>[22,23]</sup> TB in smokers takes a more severe disseminated course, more extensive lung involvement, and less cavity closure.<sup>[12]</sup>

In a recent study from Taiwan on a large sample of 5567 TB patients, it was observed that smoking tobacco doubles the risk of recurrent TB.<sup>[24]</sup>

Smoking, TB, and COPD all damages the lungs. Smoking suppresses the innate and adaptive immune response with decreased levels of pro-inflammatory cytokines and circulating immunoglobulins and reduces the activity of alveolar macrophages, dendritic cells, and natural killer cells. [25,26]

Antigenic wall component of *Mycobacterium tuberculosis* lioparabinomannan stimulates the release of matrix metalloproteinases (MMPs). [27,28] The increased expression of MMPs, CD8 lymphocytes, neutrophils, interleukin 8 and vascular endothelial growth pathway results in structural changes seen in COPD. This complex interrelationship of smoking, COPD, and TB with increased expression of proteolytic enzymes, cytokines, and interleukins results in the structural damage seen in both COPD and TB.[29,30]

Smoking, however, is not the sole factor in the pathogenesis of COPD and TB and the structural lung damage seen as all the smokers do not develop COPD and COPD is known to occur in nonsmokers. [31] In a study of COPD in never smokers, only 5.3% of COPD patients had a history of TB. [32] It appears that susceptibility of an individual to develop active TB and COPD involves a complex interaction between genetic and environment, and the result depends on the net result of protease, anti-protease imbalance, and the extent of oxidative stress. [30,33-35]

The study by Agarwal *et al.* shows only one side of this association, there is strong need of further research on this complex interaction to determine the exact clinical presentation, further course of TB in COPD and COPD

in TB how adversely affects the disease progression, morbidity, and mortality.

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### **Conflicts of interest**

There are no conflicts of interest.

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