If Smoking Results in COPD, Why Hasn't the Incidence Gone Down?

By Chuck Dinerstein, MD, MBA — June 22, 2020

Chronic Obstructive Pulmonary Disease, or COPD, is a limitation of how much air a person can inhale and exhale. Smoking has long been considered a major risk factor, but some smokers avoid the problem while some non-smokers develop COPD. A new study identifies a risk factor we haven't really considered that seems to play as large a role as smoking.

Chronic Obstructive Pulmonary Disease, COPD, is a limitation of how much air we can inhale and exhale, it is not fully reversible, is a significant cause of poor healthcare outcomes (it is the third most common cause of death in the US), and is frequently associated with smoking. But the rates of COPD have not declined in lockstep with the decline in smoking; more importantly, up to 30% of patients with COPD have never smoked. What is going on?

While smoking tobacco is a common COPD risk factor, other factors like occupational exposure or pollutants have been implicated. Interestingly, a 30-year longitudinal study of lung function has found that 50% of patients with COPD have an initial "low baseline lung function." A quick word about lung function skipping the abbreviations. COPD is associated with a decline in the ability to exhale forcefully. It is measured using a device called a spirometer, as the amount exhaled in one
second (FEV$_1$) divided by the amount totally exhaled (FVC).

The air we inhale and exhale travels through passages or airways. As the airways split off, their diameter diminishes until they reach the alveoli, the air sacs where oxygen and carbon dioxide are exchanged. The laws of physics appear to govern their changing diameters to maximize flow [1]. Still, in a subgroup of adults, there is a mismatch called dysanapsis – where airways are smaller than predicted for the accompanying lung volume. These changes are present early in life, at least as inferred by spirometry data, and these patients were the sparks that ignited the researchers’ interest.

The researchers used CT scans at full inspiration to measure 19 standard locations in the airways as well as to calculate total lung volume. The measurements which are computer calculated are very repeatable and show little variation over time. "To simplify interpretation," the researchers expressed their findings as a percentage of what was to be expected normally. Of course, while that provided a simplified number, it also introduced a variable related to how accurately they could predict normal. So as always, ignored the numbers and look at the trends.

The researchers made use of several different data sets, two community-based surveys in the US and Canada as well as a longitudinal case-controlled study for COPD. They collected the usual variables of interest. Roughly speaking, the participants were mid-60’s, slightly more male. All of the participants in the longitudinal study were current or former smokers. In the two community-based studies, half had never smoked, and only 8-11% were current smokers. Racially, the participants were primarily non-Hispanic white, followed by varying degrees of non-Hispanic black, Hispanic, and Chinese.

- The difference in airway size was about 9% of predicted between the smallest to largest airways. But that 9% reduction in size was associated with a 2-4 fold increase in COPD.
- In the multiple-factor regression, demographics like age, gender, height, and weight, as well as ethnicity accounted for 14% of that variation in COPD, primary smoking 5%, and secondary smoke 3%. Airway size was responsible for more than any of them at 17% [2]
- Using the data from the COPD study, the one with only current or former smokers, COPD was more prevalent in those with smaller airways. Again, smaller airways "explained" more of the COPD than smoking or other risk factors. Heavy smokers, with the largest airways, seemed to be less vulnerable to COPD.

"Our study shows that having an undersized airway tree compromises breathing and leaves you vulnerable to COPD later in life." Benjamin M. Smith, MD, assistant professor of medicine at Columbia University

It seems that airway size makes a difference and helps explain why patients who have never smoked develop COPD. Larger airway size in some smokers may provide a "physiologic reserve" and explain why not all smokers develop COPD. The study suggests that there are at least two pathways to COPD's altered lung function – smaller airways, and an accelerated decline in lung function associated with smoking.

There are several limitations associated with the study, most importantly that the airway measurements were not made early in life or that the measures were not wholly reflective of lung
function – form does not always follow function biologically. But we are left with several significant findings. COPD has a multifactorial basis; smoking accelerates the diminishment of airways, and smaller, initial, airway size is a considerable risk factor, posing a higher risk than smoking alone.


[2] One need also note that height, weight, gender, and ethnicity all impact genetics that may underlie airway size, meaning the role of airway size may be even more significant.

Source: Association of dysanapsis with Chronic Obstructive Pulmonary Disease Among Older Adults JAMA DOI: 10.1001/jama.2020.6918

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