

LETTER TO THE EDITOR

Lung age is a useful concept and calculation

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Dear Sir,

I must disagree with the editorial by Professors Quanjer and Enright in the last issue of the *PCRJ* – two distinguished and respected pulmonary physicians who object to measuring and using “lung age” to help reduce smoking.¹

First, they pick two equations,^{2,3} both using age, height, and forced expiratory volume in one second (FEV₁), to plot major discrepancies in estimating “lung age”. This is no surprise, since, for any given age, height and gender, the mean absolute values of FEV₁ and forced vital capacity (FVC) of all reference series differ markedly more than the % FEV₁/FVC. In their editorial they cite another publication⁴ with a new equation, useful for both men and women of at least three ethnicities, but did not test it: $\text{age}(\text{lung}-\text{calendar}) = 3x(\text{predicted}-\text{actual}) \% \text{ FEV}_1/\text{FVC}$. Compared to other referenced equations¹, this equation⁴ neither over- nor under-estimates average lung age from age 20 to 80 years in 743 never-smoking healthy white men in the Third National Health and Nutrition Evaluation Survey (NHANES-3) (see Figure 1A). Figure 1B, using the same equation, shows the increasing age(lung-calendar) in 680 NHANES-3 current-smokers.

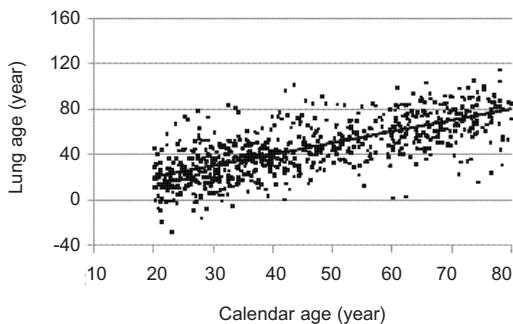
Second, for a 50-year-old man 178 cm tall with FEV₁=3.20L, Quanjer and Enright calculate lung ages ranging from 62 to 89 years from four reference equations. However, if they had used the Hansen lung age equation⁴ and recent Gutierrez, Quanjer, Hansen, and Hankinson reference equations for FEV₁=3.20L, FVC = 4.19L, and % FEV₁/FVC = 76.4%, his lung ages would be quite similar: 57, 56, 55, and 54 years, respectively.

Third, they end their editorial¹ by stating and agreeing with a Task Force⁵ opinion, “Only if both the FEV₁/FVC ratio and the FEV₁ value are below the LLN after effective therapy for airway disease - and only in patients who have smoked for more than 20 years – is it possible to associate smoking with the harm done to the lung.” By my chi-squared analysis, NHANES-3 white smokers had higher lung age than never-smokers by age 20-29 ($p<0.001$), with a rapid acceleration thereafter.

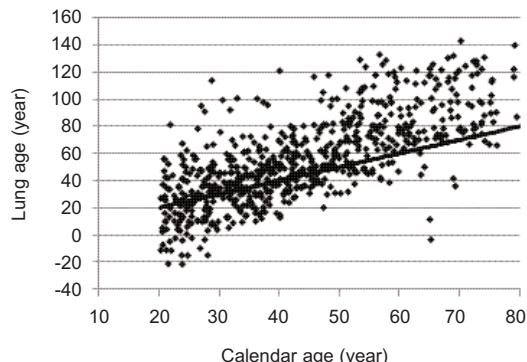
Since evidence presented here and elsewhere is so powerful that smoking damages the

Figure 1. Relationship between lung age and calendar age in healthy male never-smokers (A) and ambulatory current-smokers (B). The bold lines are the lines of identity. Note the shrinking percentage of smokers below the line of identity as age increases.

A. White male non-smokers



B. White male current-smokers



lungs, plus many other organs and systems, why should we physicians wait until a smoker is below the 95% confidence limits for both % FEV₁/FVC and FEV₁ before identifying him or her as harmed or very likely to have been harmed by smoking? Let us see if the use of new, valid, and easily-calculated lung age measurements using only % FEV₁/FVC can help us influence smoking behaviour.

Conflict of interest declaration

No known conflicts of interest.

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Authors' reply

We thank Dr Hansen for his interest¹ in our paper.²

Effectively prompting patients to change lifestyles which they have developed over decades, such as smoking and excessive carbohydrate or alcohol intake, is time-consuming but worthwhile. For smoking cessation, we recommend brief motivational interviewing,³ followed by pharmacological therapy which has proven efficacy.⁴ There are several reasons why spirometry testing of smokers to try to prompt smoking cessation for the subset with a high "lung age" is not a good substitute for evidence-based interventions, which take less time than does spirometry for busy health care providers:

1) Fewer than one in five current or former smokers over age 40 have or are developing COPD, the only smoking-related lung disease measured by spirometry. 2) There are many causes of a low FEV₁ other than COPD. 3) Even if FEV₁/FEV₆ is used to estimate lung age, asthma is just as common a cause of airway obstruction (a low ratio) in adult smokers as is COPD. 4) Only 1 of 3 studies found a higher rate of smoking cessation in patients told their lung age, but the study design was flawed (all patients had spirometry testing) and stating lung age was only one of three interventions. The other two were discussion of a graph showing rapid decline in lung function and follow-up with a personalised letter. 5) The "number needed to treat" (with spirometry testing and the triple intervention for the subset with a high estimated lung age) to prompt a single extra subject to

quit smoking was up to 46 subjects (95% CI). That's not very effective. 6) Lung age is any calendar age compatible with actual lung function; in healthy subjects it has a large scatter. Individual lung function tracks along centiles;⁵⁻⁷ hence 50% of people start with a higher than average lung age before becoming smokers. Values exceeding calendar age are routinely attributed to effects of smoking without taking the normal scatter into account. Hence the concept is basically flawed. 7) Hansen's approach⁸ is definitely preferable over the calculation of "lung age" since it takes into account normal variability, but this improvement has no bearing on the successfullness of intervention.

We should not wait until lung function indices are clearly abnormal before trying to persuade smokers to give up the smoking habit. Unfortunately, the use of spirometry results does not effectively improve their motivation to quit.

Conflict of interest declaration

No conflict of interest.

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