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EDITORIAL

Should we use 'lung age'?

See linked article by Newbury *et al*. on page 242, and letter from Parkes and Greenhalgh on page 295

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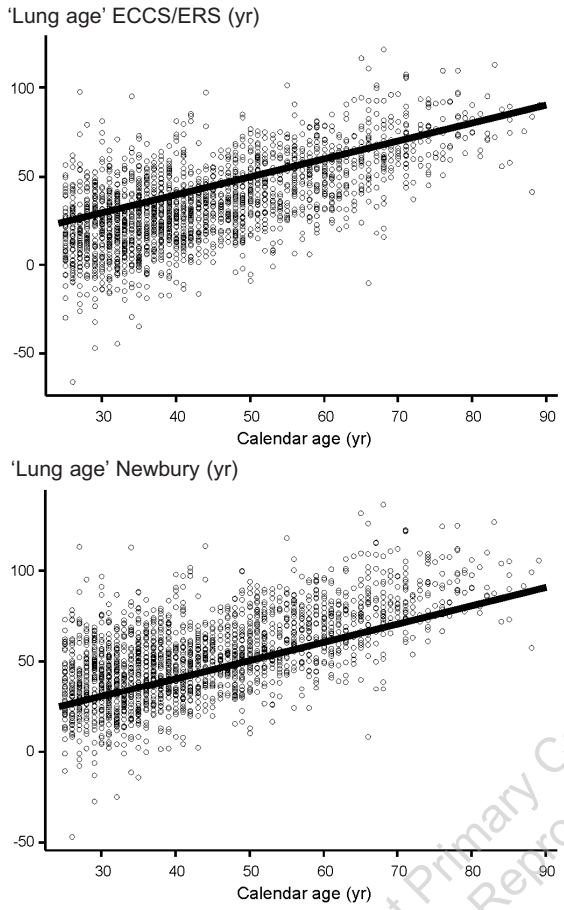
Chronic obstructive pulmonary disease (COPD) is an important cause of morbidity, and the fourth commonest cause of death worldwide.[1] Since it is largely caused by smoking, smokers are the natural target of efforts to prevent the development of clinically important COPD. The underlying assumption of preventive counselling is that confronting the smoker with evidence of potential lung damage will increase quit rates. However, the majority of smokers who are found to have normal lung function during COPD screening programs may subconsciously use that information as an excuse to continue smoking. The effects of smoking mimic premature lung ageing; hence 'lung age' as an index of potential lung damage was introduced by Morris and Temple in 1985 as a tool for motivating cessation of cigarette smoking.[2] Lung age is commonly estimated from regression equations for the forced expiratory volume in one second (FEV_1) in healthy never-smokers, and constitutes the age at which the FEV_1 measured in an individual equals the predicted value of FEV_1, taking into account age, height, sex and ethnicity.[3,4]

The original equations for 'lung age' were obtained in 1971 with techniques, and from a population sample, that does not comply with today's standards. In this issue of the *PCRJ*, Newbury and colleagues[5] present new equations based on a study of healthy lifelong nonsmokers in Australia. Their very careful study reveals that the Morris equation 'lung age' differs appreciably from chronological age in a representative sample of a healthy male non-smoking Australian population aged 20-61 years. After developing equations that fit the Australian population, they found that in current smokers, 'lung age' exceeded chronological age by 18 years. This illustrates the need to use equations that fit the local population, and confirms that prolonged smoking damages the lung, so that on average the FEV_1 in smokers is below the predicted value.

Many authors speculate that confronting smokers with their 'lung age' will persuade them to quit smoking.[2-4,6,7] Regrettably, in practice these expectations have not been fulfilled. In a retrospective analysis, Bednarek *et al*.[8] reported that the smoking cessation rate in those with airway obstruction was 16.3% compared with 12.0% in those with normal spirometry; however, they did not randomise their study participants to spirometry or no spirometry.[9] In the only positive study using lung age, Parkes *et al*.[10] reported that providing patients with their 'lung age' was associated with higher quit rates at 12 months in the intervention group (13.6%) when compared to the control group (6.4%). However, those with higher 'lung age' (i.e., worse lung function for their age) were no more (or less) likely to quit than those with normal 'lung age.' Lin[11] rejected Parkes' conclusions because "the study does not establish the independent motivational effectiveness of doing spirometry screening versus not doing it, which would require a randomised trial in which the control arm did not receive spirometry." Prior to publication of the Parkes study, three reviews[12-14] and one study targeted at college students[15] concluded that the evidence was very weak that confronting smokers with their pulmonary function improves quit rates.

Apart from the above, and as shown by Newbury *et al*. in this issue, calculated 'lung age' varies widely according to the prediction equations used. For example, for a 178 cm tall, 50 yr-old man, with an FEV_1 of 3.20 litres, the 'lung age' using prediction equations from ECCS/ERS,[16] Crapo,[17] Morris[12] and Newbury[5] is 68, 83, 62 and 89 years, respectively – so what is this person's 'lung age'? In addition, there is considerable scatter around the predicted value for FEV_1 in healthy nonsmokers, indicating that published prediction

Figure 1. Relationship between 'lung age' and calendar age in healthy male English lifelong nonsmokers[22] according to two prediction equations.[5,16] The bold line is the line of identity.



equations cannot satisfactorily take into account differences between individuals due to differences in body build, properties of lungs and airways, the level of physical training, and other factors. Pulmonary function tracks in healthy subjects,[18-20] so that people who start with a relatively high or low level of FEV_1 (i.e. lung age younger or older than calendar age, respectively) will usually retain that position as they grow older. Unless we have good quality spirometry results from several years previously for an individual (and can perform trend analysis), we should not attribute an FEV_1 in the low normal range to accelerated ageing.[21]

This can be demonstrated as follows. We calculated 'lung age' for a healthy non-smoking subset of English participants from the Health Survey for England,[22] which included 3107 men. Figure 1 shows 'lung age' as a function of calendar age using the ECCS/ERS equations[16] which are widely used in Europe, as well as using Newbury *et al.*'s equations.[5] The preponderance of values below the identity line with the ECCS/ERS equations, and conversely above the line of identity with Newbury's equation, means that the equations do not fit the English population. The ECCS/ERS equations identify too few subjects whose 'lung age' exceeds calendar age, Newbury's equations too many. Subjects whose FEV_1 is higher than average have a younger lung than calendar age. Should we tell these people that they can happily continue smoking? Definitely not. But should we tell people whose FEV_1 is below 100% predicted but above the lower limit of normal (LLN) that their 'lung age' indicates lung damage due to smoking? In fact, most of them will have had an older 'lung age' even before they started smoking…

Obesity reduces both FEV_1 and the forced vital capacity (FVC), but does not accelerate ageing of the lung, since subsequent weight loss increases lung function. There is often a rapid improvement in FEV_1 after the inhalation of salbutamol, both in patients with asthma and in some patients with COPD,[23] but it makes no sense that the age of the lung would improve within ten minutes.

So it is back to basics. Only if both the FEV_1/FVC ratio and the FEV_1 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 plausible to associate smoking with harm done to the lung.[24]

Conflict of interest declaration

None

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