

ORIGINAL RESEARCH

Exploring the need to update lung age equations

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Abstract**Aims:** A renewed interest in lung age is evidenced by recent smoking cessation publications. This research compares the original Morris lung age equations (1985) with contemporary Australian lung age equations.**Methods:** Both lung age equations were applied to the spirometry results of two sub-groups (never-smokers n=340, and current smokers n=50) from an independent dataset. Means of both lung age estimates were compared to the mean of the chronological age of each group by paired Student's t-test.**Results:** The Morris lung age estimates were paradoxically lower (younger) than chronological age in both groups. The new Australian equation produced lung age estimates that were equivalent to chronological age in the never-smoker group and significantly higher (older) than chronological age in the current smoker group.**Conclusions:** These results strongly suggest that the Morris lung age equations are in need of review. The use of contemporary lung age equations may translate into greater success for smoking cessation programs. The new Australian equations seem to possess internal validity.

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See linked editorial by Quanjer and Enright on page 197, and letter from Hansen on page 286

Introduction

Smoking has been shown to be detrimental to health for many years.¹⁻⁷ Smoking has an adverse effect on forced expiratory volume in one second (FEV₁) throughout a lifetime, reducing the maximal FEV₁ achieved, bringing forward the age of onset of decline in FEV₁, and hastening the rate of decline.⁸ The single most useful intervention to improve lung function in smokers with or without chronic obstructive pulmonary disease (COPD) is smoking cessation. One way to increase the quit rate in smokers could be to communicate lung function results in a manner that is easily understood and stimulates the desire to quit.

Lung age is an estimate that uses the observed FEV₁ of a smoker to calculate the approximate age of a healthy non-smoker with the same FEV₁ based on predicted values, and can be a "potent motivator" in smoking cessation counselling.⁹ Equations for estimating lung age were first developed by Morris and Temple in 1985¹⁰ using earlier American predictive equations for spirometry published in 1971.¹¹ The most relevant of the forced expiratory measures to determine lung age values was deemed to be FEV₁.¹⁰

Lung age equations were developed as an aid for smoking cessation counselling¹⁰ and the concept has been explored in several recent publications. One qualitative study explored acceptance of a graphical means to communicate lung function decline caused by smoking using a bar graph that compared

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chronological age and estimated lung age, and found acceptance and better understanding of the concept of the greater rate of decline of FEV₁ in smokers in this simple depiction.¹²

Three publications describe randomised controlled trials (RCTs) of which one¹³ used a visual aid based on the Fletcher and Peto graph of age-related decline of lung function and the cumulative effect of smoking.³ In this study, Parkes *et al.* detected a significant improvement in the quit rate in their intervention group, using a graphical explanation of lung age compared to chronological age to give feedback of spirometry results. At 12 months follow-up the quit rate was 14% in the intervention group (lung age group) and only 6% in the control group.¹³ The recent Cochrane Review, "Biomedical risk assessment as an aid for smoking cessation"¹⁴ that conducted a meta-analysis of eleven RCTs, has identified Parkes' lung age study as the only intervention using spirometry which detected a significant effect.

The second RCT investigated perceived smoking-related health risks, worries, and desire to quit in college smokers with feedback on respiratory symptoms and a verbal explanation of lung age.¹⁵ Smoking cessation was not a primary outcome of this study. Perceptions of risk and worry, together with the desire to quit smoking, were not directly affected by lung age or respiratory symptoms feedback, possibly because of the young ages of the subjects. The third RCT used another adaptation of the same Fletcher and Peto graph,³ but only calculated lung age if FEV₁ percent predicted was below 80%, indicating possible impairment.^{16,17} This RCT failed to show differences in the quit rate between their intervention and control groups.^{16,17} In Japan, lung age has also been investigated in relation to smoking status and body mass index¹⁸ and in detecting pulmonary function abnormality in pulmonary diseases.¹⁹

Despite mixed results, this flurry of recent publications indicates a renewed interest in the use of lung age to educate smokers about the deterioration of their lung function caused by smoking. International guidelines for smoking cessation state that primary care encounters provide good opportunities for smoking cessation advice and that tailored advice may be more effective,²⁰ and also that motivational counselling is of most use when it is relevant to the subject's risk status.²¹ The guidelines for spirometry by the American Thoracic Society (ATS) recommend that predictive equations should be derived from a 'relevant' population²² and should be updated at least every 10 years.²³ These recommendations should equally apply to equations that estimate lung age. However, the lung age equations in use today are those that were published in 1985.

This paper describes the comparison of new lung age equations derived from contemporary Australian data²⁴ with the Morris lung age equations.¹⁰ The null hypothesis is that there will be no difference between lung age mean values.

Methods

The research methodology has previously been described in the literature.²⁴ Briefly, the inclusion criteria included Caucasian ethnicity, and ages between 25 and 74 years. Volunteers were recruited from the broad semi-rural community in South Australia (SA) by advertising in the local media and the snowball technique. Exclusion criteria were a current or previous history of asthma or other chronic lung disease, current acute respiratory infection, current smokers, and past smokers if they had smoked more than 10 cigarettes per day for more than five years.²⁴ The resulting Australian predictive normal equations for spirometric parameters were mathematically manipulated using the methods described by Morris and Temple¹⁰ with the equations being solved for age.

The age distributions of the samples used to generate both sets of predictive equations were examined. Lung age estimates obtained from both the SA and the Morris equations were compared using an independent workplace dataset, the Metropolitan Fire Service (MFS) of SA collected in 2007-8 (unpublished data). As this dataset contained few females (n=13), the analyses reported here are restricted to males only.

Lung age estimates of two subgroups of the MFS dataset (male never-smokers (NS), n=340 and male current smokers (CS), n=50) were calculated by applying both lung age equations to the observed FEV₁ values for these subjects. Smoking status was self-reported. Paired t-tests were performed to compare the mean of the chronological age of the NS with the mean of the lung age estimates produced by each lung age equation. Comparisons of chronological age and lung age were repeated for the CS subgroup. Statistical significance was set at p = 0.05. SPSS (Version 17) was used for statistical analysis.

Ethics approval for both the Port Lincoln study (H-086-2006) and the MFS study (H-057-2007) was given by the University of Adelaide Human Research Ethics Committee.

Results

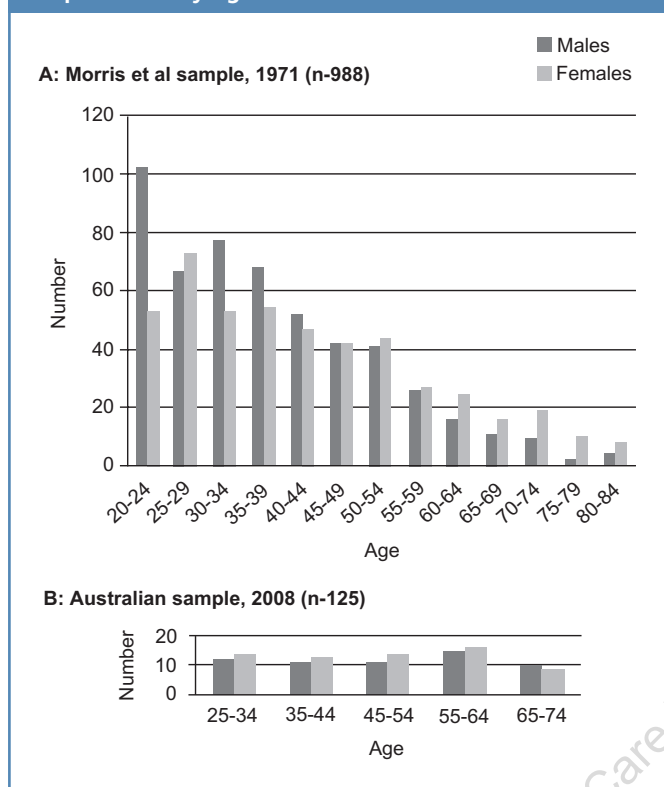
The preliminary Australian lung age equations derived from the Port Lincoln predictive normal equations for spirometry are given in Table 1. The age distribution of Morris' original sample¹¹ was strongly skewed to the right, while the Port Lincoln SA sample was evenly age-stratified.²⁴ The age distributions of both samples are shown in Figure 1.

Table 1. Preliminary Australian lung age equations.

Male
Lung Age (years) = 1.56*height(cm) - 33.69*Observed FEV ₁ - 85.62
Female
Lung Age (years) = 1.33*height(cm) - 31.98*Observed FEV ₁ - 74.65

Derived from contemporary Australian predictive equations for adults of Caucasian origin, aged between 25 and 74 years.¹³

Figure 1. Age distributions for the Morris sample (1971) and the Australian sample (2008). A: The Morris sample is strongly skewed to the right. B: The Australian sample is evenly age-stratified.



Demographic and clinical characteristics of the CS and the NS groups of the MFS sample are presented in Table 2.

The ages (mean and range) and the results of the t-tests for the NS and CS sub-groups are presented in Table 3. Diagrammatic representation of results is presented in Figure 2.

Never-smokers

The mean of the chronological age of the NS group was 42.4 years. The difference of +1.6 years between this and the mean of the lung age estimates (44 years) calculated using the Australian equation was not statistically significant ($p=0.08$).

Table 2. Demographic and clinical characteristics per smoking group.

	Current smokers	Never-smokers
n	50	340
Age (range)	42.9 (28-60)	43.1 (20-61)
Height*	180.2 (6.3)	180.3 (6.4)
Weight [†]	90.8 (11.1)	88.8 (11.6)
FEV ₁ [‡]	4.3 (0.7)	4.4 (0.7)
FVC [‡]	5.8 (0.8)	5.9 (0.8)
FEV ₁ /FVC [§]	73.3 (7.3)	75.3 (5.9)
Doctor diagnosis of Asthma, n (%)	8 (15.7)	40 (11.8)
Productive cough, n (%)		
- Mornings for more than 3 months	12 (23.5)	16 (4.7)
- Rest of day or night for more than 3 months	6 (11.8)	7 (2.1)

Mean and standard deviation (SD) unless otherwise stated.

*Height (cm), [†]Weight (kg); [‡]FEV₁, FVC - Litres, [§]FEV₁/FVC - %

The difference of -18.1 years in the means of chronological age and the lung age estimates using the Morris equation (24.3 years) was statistically significant ($p<0.001$).

Current smokers

The mean of the chronological age in the CS group was 42.9 years. The difference of +7.5 years between this and the mean of the lung age estimates (50.5 years) calculated using the Australian equation was statistically significant ($p=0.007$). In the comparisons using the Morris lung age equation the difference (-12.4 years) in the means of chronological age and lung age estimates (30.5 years) was also statistically significant ($p<0.001$).

Discussion

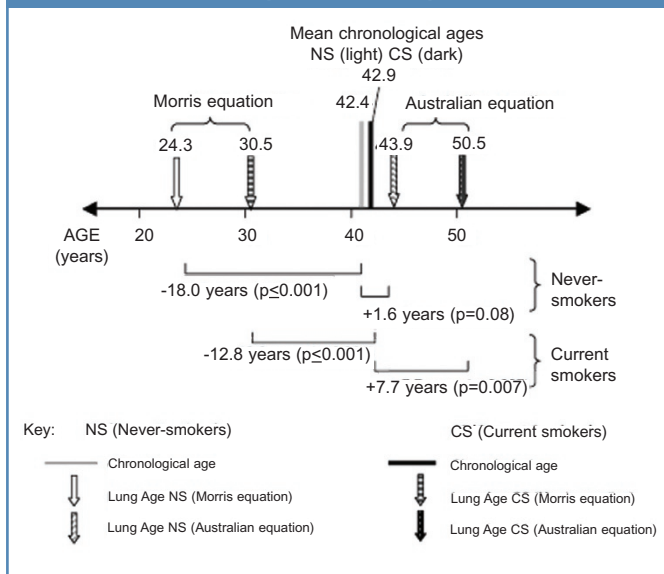
Preliminary lung age equations for adult Australian male Caucasians have been validated using the NS sub-group (males) of the MFS independent dataset. In this never-

Table 3. Paired t-test results comparing lung age estimates with chronological age, using the MFS dataset.

		Mean	SD	t(DF)	p value
Never-smokers	Chronological age	42.37	Range: 20-61		
	Australian lung age	43.95	18.66	-1.8(339)	0.08
	Morris lung age	24.3	17.89	21.2(339)	< 0.001*
Current smokers	Chronological age	42.88	Range: 28-60		
	Australian lung age	50.54	22.52	-2.8(49)	0.007*
	Morris lung age	30.52	20.87	4.9(49)	< 0.001*

Mean (Range) of chronological age of each sub-group; Mean and SD of lung age estimates using the Australian lung age equation and the Morris lung age equation. SD = Standard Deviation; t = t value; DF = degrees of freedom; * = statistically significant < 0.05.

Figure 2. Differences in means of Chronological Age and Lung Ages, using Morris' equation and the Australian equation for the NS (light arrows) and the CS (dark arrows) from the independent workplace dataset.



smoking sub-group, the contemporary Australian mean lung age closely agrees with the mean chronological age. However, the Morris equation calculates a lung age that is lower than the chronological age by approximately 18 years when applied to this never-smoking sub-group. This is a strong indication that the Morris lung age equations are not relevant for a current day Australian population.

In the CS sub-group the Australian lung age equation produced a lung age greater than chronological age suggesting that smoking causes lungs to deteriorate more quickly than the expected age-related decline, as predicted by Fletcher and Peto.³ In contrast, the Morris equation again produced lung age values less than chronological age (approximately 12 years lower) when applied to the CS sub-group. This result seems paradoxical in suggesting that smokers' lungs are 'younger' and presumably healthier than expected. There was indeed a difference of approximately 20 years in the lung age estimates produced by the Morris equations and the new equations in this current smoker sub-group.

These results mean that we can reject the null hypothesis that we would see no difference in the means of the lung age estimates produced by both lung age equations.

There are several possible reasons for this significant result and it is possible that all play a role. Firstly, the predictive equations that are the basis of the Morris lung age equations are approximately 40 years old. The cohort effect suggests that a 40 year-old today will not be the same as someone of the same age 40 years ago due to demographic and environmental

differences.²² Respiratory testing equipment and procedures have been progressively refined over the last 40 years in line with recommendations that have been regularly updated by the ATS.^{22,25-28} In particular, the Morris, Koski and Johnson results¹¹ were calculated using equipment and methods that may well give lower results than those currently recommended by the ATS.²⁹ Indeed this study predates even the first ATS guidelines on spirometry.²⁵

Secondly, 79% of Morris' original sample was from two church groups in rural USA.¹¹ As the doctrines of these two churches forbid tobacco smoking and the intake of alcohol or caffeine as well as advocating a vegetarian diet, this sample can't be described as representative of a 'normal' current day population. The SA sample was drawn from the broad rural community, targeting non-smokers with no history of lung disease.²⁴

A third reason could be that the age distribution of the Morris sample was strongly skewed to the right with over 30% of subjects in the youngest 10 year age bracket (Figure 1A).¹¹ This may mean that results are biased towards younger ages. One of the strengths of our SA sample was that it was evenly age-stratified, resulting in predictive equations that are equally relevant across the whole age range (Figure 1B).

A limitation of this study is that lung age is an estimated value based on the population mean and as such may have potential difficulties when predicting values for individuals. This is demonstrated by the large standard deviations of both lung age equations in Table 3. The variability of spirometry results of normal healthy subjects is itself quite wide, being approximately 80-120% predicted, and consequently wide variation in lung age estimates exists. There continues to be considerable debate about use of Lower Limits of Normal (LLN) or percent predicted, with the definitions of stages of disease easily described by percent predicted. Instead of a single lung age value it may be possible to communicate lung age as being "between x and y", based on 95% confidence intervals. Lung age's relationship to smoking may also be controversial as there is also a decline in lung function with increasing age as well as with diseases such as COPD. However, there is continued support in the literature for the more rapid decline in FEV₁ in smokers than in non-smokers.^{3,30-32} Other factors that also influence lung function throughout life include gestational age at birth, genetics, childhood infections, and environmental factors such as air quality and workplace exposures.

A further limitation of this study is that we have used predictive equations developed from a pilot study of 125 adults to produce the preliminary Australian lung age equations. However, this contemporary sample was comprised of healthy non-smokers and was evenly age- and sex-stratified. The predicted values for each of the spirometric parameters derived in the Port Lincoln study are remarkably similar to those

Summary box

What is already known:

- The Morris lung age equations in current use were produced in 1985, but are based on data published in 1971.
- Lung age has been shown to be an acceptable way to communicate spirometry results for smokers and patients with COPD.
- Lung age has had a positive impact on the quit rate in an RCT.

What this study adds:

- This study shows that Morris' equations significantly underestimate lung age in both Australian never-smokers and smokers.
- New lung age equations produce lung age estimates that are approximately 20 years greater than does the Morris equation, for Australian never-smoking and current smoking males.

Implications:

- Using the new equations may have a greater impact on smokers, and may increase quit rates further.
- These results highlight the importance of having current predictive equations available.

reported by Gore *et al.* in a previous study on a larger random sample (n=1302) of the South Australian population.^{24,33} Also, the subjects in the workplace dataset may not be representative of a normal population; they maintain a high level of fitness but contrarily have occasional occupational exposures to smoke when attending fires. The accuracy of the estimates of lung age for the NS subgroup using the Australian equation suggests that they are representative of the population.

Summary

Lung age can be calculated by modern spirometers and is a concept that can be easily communicated. In primary care, where much of the smoking cessation counselling takes place, lung age can be used as a simple non-pharmacological intervention to motivate smokers to quit. We have used contemporary data to produce preliminary lung age equations for adult Australians of Caucasian ethnicity (aged 25-74 years). International guidelines suggest that lung function equations should be derived from the same population to which they will be applied.²³ Our results strongly suggest that existing lung age equations are in need of review. Contemporary lung age equations can give a more powerful quit smoking message. Further research is planned to validate these equations in females as well as males, using a larger random population sample.

Conflict of interest declaration

There are no conflicts of interest for any of the authors.

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Authors' roles

Wendy Newbury: literature search, data collection, analysis, writing of article
Jonathan Newbury: analysis, writing of article
Nancy Briggs: statistical analysis, writing of article
Alan Crockett: analysis, writing of article

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