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Radiation risk of screening with low dose CT

I read with interest the article by MacRedmond *et al*¹ on screening for lung cancer using low dose CT scanning and the related editorial by Gleeson² which provides a comprehensive summary of the benefits and potential pitfalls of such a screening. However, I noticed that, in both articles, the important issue of the potential radiation risks associated with low dose CT screening for lung cancer has not been addressed.

Previously published reports have suggested radiation risks even with a low dose CT scan as part of a regular screening programme³ and also of a possible synergistic interaction between the risk from smoking and radiation exposure.^{4–7} Brenner³ estimates that, if 50% of all current and former smokers in the US population aged 50–75 years received annual CT screening, the estimated number of lung cancers associated with radiation from screening would be 1.8% (95% credibility interval 0.5% to 5.5%) more than the otherwise expected number. Considering an upper limit of a 5.5% increase in lung cancer risk attributable to annual CT related radiation exposure, he feels that a mortality benefit of considerably more than 5% would be necessary to outweigh the potential risks of radiation. This estimation was derived from cancer incidence data for atomic bomb survivors.⁷

Several other reports^{6–8} have suggested a link between radiation exposure and lung cancer. Potential radiation risks associated with multiple CT scans should therefore be

considered as one of the limiting factors for such screening.

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Dr MacRedmond was asked to comment but no reply who received by the time this issue of *Thorax* went to press.

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Effect of dichotomising age in multivariate model analysis

We read with interest the paper by Soler-Cataluña and colleagues¹ that examined—in an impressive prospective study with 5 years follow up—factors predicting poor prognosis and mortality in patients with severe acute exacerbations of chronic obstructive pulmonary disease (AECOPD). Their findings are complementary with the current available literature in identifying that older age, arterial carbon dioxide tension, and acute exacerbations were independent predictors of mortality in their cohort group.

We have concerns, however, regarding both their analyses and conclusions. Firstly, several studies^{2–4} have given advice on the limitations of dichotomising continuous predictors as they come at a cost “as explanatory variables could be seriously misleading, both in respect of which variables are significant in the model, and perhaps also with respect to the overall predictive ability”.² Soler-Cataluña and colleagues state that in their multivariate model “the frequency of acute exacerbations, age and Charlson index were analysed as categorical variables”.¹

Secondly, and perhaps more importantly, the authors have reported older age (clearly a non-modifiable factor) as a predictor of death. They do not state whether they believe this to be old age per se or an age related potentially modifiable variable. Have the authors collected data on social support, physical disability, depression, quality of life, and any palliative care their patients may have received during the follow up period? These variables may have some effect on

mortality in this exclusively male COPD patient cohort. Our own group has recently published data on 1 year mortality following hospitalisation for AECOPD in a slightly older group of subjects (mean age 73 years v 71 years in the patients studied by Soler-Cataluña and colleagues) with worse baseline spirometry (mean percentage predicted FEV₁ 39%). In our study age was not a mortality predictor on either univariate or multivariate analysis. Quality of life, level of disability, severity of depression, readmission, use of long term oxygen therapy, and duration of original admission (all of which are arguably related to age) were all univariate predictors of 12 month mortality, with only the quality of life score remaining a significant predictor on multivariate analysis.

We wonder whether the inclusion of age related variables in the study by Soler-Cataluña *et al.*, together with the use of age as a continuous variable, might have resulted in qualitatively or quantitatively different conclusions regarding the effect of age on prognosis. However, the inclusion of duration of original admission and of frequency of readmission in our own list of predictors³ would support their suggestion that severe AECOPD could have an adverse impact on longer term mortality.

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Authors' reply

We wish to thank Dr Yohannes for his interest and comments on our study¹ and have the following comments on the questions he raises.

Firstly, although it is true that in some cases the transformation of continuous variables into dichotomised variables may induce some changes in the results obtained, in other cases the use of continuous data may conceal some partial effect, particularly if the predictive relation is non-linear. In fact, in our study the only age group to show a poorer prognosis were those aged ≥75 years (odds ratio (OR) 5.26, 95% CI 2.70 to 10.24). In the same way, categorisation of the number of exacerbations allowed us to review the differential effect of repeated exacerbations. For these reasons, and in order to make interpretation of the results easier, we