Reviewer's report

Title: Contribution of smoking and air pollution exposure in urban areas to social differences in respiratory health

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Contribution of smoking and air pollution exposure in urban areas to social differences in respiratory health

General

This is a potentially useful study in trying to understand what underlies the negative social class gradient for respiratory disease (i.e. why the poor usually have higher prevalences than the rich). The measurement of lung function using spirometry is a strength of the study

The findings are interesting, but there are some problems with the interpretation and in some respects the methods.

Background

Page 3: There are many more than the two studies cited by the authors on the association between SES and respiratory symptoms and lung function decline. They need to extend their literature search. E.g. there is a review by Hegewald and Crapo in Chest 2007; 32: 1608-14.

Page 3. It is not entirely clear what causal model the authors have in mind, and specifically whether they consider smoking and air pollution as mediators or confounders of the association between SES and respiratory health. In paragraph two, page 1, they imply mediation (â##â# strongly influenced by SES..â##) but in paragraph three, confounding is implied (â##....competing factorsâ#â##). This needs to be clarified.

Page 3, etc. It is common practice to use education as a proxy for SES, but it is this reviewerâ##s belief that this is an oversimplification and can lead to the lumping together of different causal phenomena. While it is true that in some societies (not all) education is predictive of income and wealth, education may have more direct effects on health, for example, through access to and interpretation of health information and ability to make better use of health care.

In this study, the husbandâ##s education level was used when it was higher than that of the participant. It would be useful to know in what proportion of cases this.
Interestingly, when the participant’s education level was used, the association
of SES with the respiratory outcomes was weaker with less attenuation by known
risk factors. This is interesting in its own right in respect of gender/sex influences
on respiratory health.

Methods

Page 5, etc. It is not clear what the reference level for current smoking is. Surely
cumulative smoking rather than current smoking, i.e. including ex-smokers, is
more important for COPD?

Results

Page 8: “Linear” trend. Some of the trends are not “linear”.

Page 8/ Table 1: While “physician diagnosed chronic bronchitis” showed no
trend, “frequent cough with phlegm production” which is a better
epidemiological measure of chronic bronchitis, did.

Page 8/ Table 2: The purpose of Table 2 is not clear and the columns seems to
be wrongly labelled (“...reporting no symptoms”).

Page 10 / Table 4: The arithmetical procedure used by the authors to estimate
fractional contribution to explained SES by other risk factors is \((OR_{unadjusted} \div OR_{adjusted}) / OR_{unadjusted}\). This understates the effect. \((OR_{u} - UR_{a}) / (OR_{u} - 1)\) is the correct way to do this. E.g. if \(OR_{u} = 2.0\) and \(OR_{a} = 1.0\), 100% of
the effect is attenuated, not 50%.

Discussion/General

A strong finding is that the SES effect is not greatly attenuated by known risk
factors. The authors need to start with methodological considerations as an
explanation, e.g. misclassification/ mismeasurement of the risk factors “this is
likely to be so for all the risk factors (including “current smoking” if it is
contrasted with “non current smoking”). The occupational exposure
variable, while widely used, is very crude. In Table 3, one is struck by the relative
weakness of the risk factor gradients by SES “if accurate, this may suggest
that it would be difficult to generalize to other populations (it is not clear what the
authors mean by “other industrialized regions” “would this include
Brazil, China, etc.?). Whatever the case, only once one is satisfied that the model
is properly specified and misclassification has been minimized, does the estimate
of the “residual” effect have meaning.

The authors treat FEV1 and FVC as if they have the same biological meaning.
However, one would expect the primary impact of inhaled toxicants to be on
airflow limitation and not on FVC. The similar findings in regard to FEV1 and FVC
suggest that SES has an effect on lung growth, which means early life influences
(including those of parental SES and childhood SES) on insults, growth and
development. (See e.g. Jackson “). This shows up the limitations of a
cross-sectional study attempting to unravel effects operating throughout the life
The authors might also want to consider that if one is looking at a total SES effect including SES in early life, then adjusting for height, as one does in using a reference equation for FEV1 and FVC, might be adjusting out an important intermediary process in the form of lung growth.