Correspondence

To the Editor:

Recently, you have published in this journal a paper by Leuenberger *et al.* SAPALDIA Study (1). The contents as well as its main conclusion lead us to ask a number of questions.

Our first question concerns study design. Cross-sectional surveys are unable to resolve the antecedent-consequence uncertainty. If a study cannot show that the exposure to a suspected risk factor occurred before the symptoms arose, then how can a statistical association be meaningful?

Our second question concerns the selection procedure for the study cohort. A random sample of 17,500 adults was drawn from the registries of the inhabitants of eight areas of Switzerland. Of these, only 9,651 (56%) were successfully recruited and completed the questionnaire. Does not a nonresponse rate of 44% suggest a self-selection bias, which must be fully understood before the study could be applied at large?

A third issue concerns the questionnaire. How was the questionnaire validated? No information is given in the paper. The reference to the ATS questionnaire is not helpful, because that questionnaire does not address ETS exposure in adults.

A fourth question concerns the validity of the questionnaire: How does it actually measure smoking status, ETS exposure, or disease status? Endexpiratory CO-measurements to control for smoking status are outdated for misclassification purposes. After an overnight sleep, smokers who declare themselves as nonsmokers, a common problem in ETS studies, simply cannot be identified. ETS exposure was assumed by questionnaire, an unreliable method for quantitative exposure measurements (2). In addition, besides the exposed and the not exposed group there was a third category of respondents, the "Don't know". How has this third group been accounted for? Disease status was assumed by self-diagnosis via questionnaire, even though the medical support for an objective diagnosis was available. None of these data were used to verify the self-reported symptoms.

A fifth question concerns statistical methods. Linear logistic regression depends on assumptions of the specific model. How good was the fit between the model and the data? Were interactions between the risk factors (e.g., hours of exposure per day) included? Which method was used for the trend test?

The SAPALDIA study was intended to be a "Swiss study on Air Pollution and Respiratory Diseases in Adults". How has it become an indoor air study on passive smoking?

Any epidemiologically unbiased risk estimate is one that seeks to represent as perfectly as possible (besides chance) the true value of the risk in the base population. The above questions suggest that this is not the case for the SAPALDIA study.

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1. Leuenberger, *et al.* 1994. Passive Smoking Exposure in Adults and Chronic Respiratory Symptoms (SAPALDIA Study). *Am. J. Respir. Crit. Care Med.* 150:1222–1228.
2. Phillips, *et al.* 1994. Assessment of Personal Exposures to Environmental Tobacco Smoke in British Nonsmokers. *Environment International* 20:693–712.

From the Authors:

Thank you for the opportunity to respond to the letter of Dr. Atteslander and Dr. Schneider. We have considered the points they raise about our study of the effects of environmental tobacco smoke, and find them unconvincing. For example, their first concern is that a cross sectional study cannot establish the direction of the association. We agree that there are two possible explanation for the associations that we have reported, and that the statistical analysis *per se* cannot distinguish them. The first is that passive smoking exposure produces chronic respiratory symptoms. The second is that persons who have chronic respiratory symptoms preferentially seek out smokey environments to an extent greater than persons without respiratory symptoms.

Their suggestion that only 60% of subjects being successfully recruited suggests a nonresponse bias seems equally peculiar. Nonresponse does not equal nonresponse bias. For selection to influence our results, nonresponse would have to be correlated *both* with exposure *and* with the presence of chronic respiratory symptoms. That is, never smokers without respiratory symptoms would be *less* likely to participate if they *were* exposed to ETS, but *more* likely to participate if they *were not* exposed. Moreover, we see a dose dependent increase in respiratory symptoms with, for example, hours of passive smoke exposure. For selection bias to have explained that, the asymptomatic persons would have had to have become progressively less likely to participate with increased hours of exposure, the opposite would be true for symptomatic persons. Dr. Atteslander and Dr. Schneider have offered no reason why this should have occurred, and we share their inability to come up with a plausible explanation for such an occurrence.

We are also puzzled by the suggestion that a study of air pollution should not be used to assess the impact of an indoor air pollutant, such as environmental tobacco smoke. Control for potential confounders is important in environmental epidemiology, and given the well established adverse effects of tobacco smoke, we would be remiss in contrasting the prevalence of chronic respiratory symptoms across regions of Switzerland with long term air pollution concentrations without control for this potential confounder. Having obtained data on passive smoking, we would be remiss in not examining its association with those outcomes.

The comment about testing the assumptions of linearity in the linear logistic model also seem puzzling. In our initial regression model exposure is a yes/no indicator. There is no issue of linearity; the logistic regression estimates the relative odds of respiratory symptoms in the exposed versus unexposed subjects. When we then turned to continuous exposure variables (e.g., hours of exposure per day) we used ordered categories to examine whether responses increased linearly with dose or not. For example, Figure 3 shows a roughly linear dose response relationship between wheezing and hours per day of ETS exposure in each of 4 strata defined by number of smokers and whether the exposure occurred at work.

We do not agree that end expiratory CO cannot distinguish between smokers and nonsmokers. In our data there was a clear bimodal distribution of end expiratory CO with the second peak occurring in self-reported smokers. CO was measured during the day, when an active smoker would have likely already had several cigarettes. Even if end expiratory CO were a poor maker for active smoking, exclusion of subjects based on an imperfect measure of active smoking would reduce the estimated effect of ETS exposure on respiratory symptoms if those effects resulted from misclassification of active smokers. In this analysis the exclusion of these subjects had little effect.