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CORRESPONDENCE

Women's respiratory vulnerability to tobacco smoking

To the Editor:

PRESCOTT *et al.* [1] report, in two independent population samples of adults, that smoking had a greater impact on the lung function of females than of males and the adjusted risk of being admitted to hospital for chronic obstructive pulmonary disease (COPD) was higher for females than for males. Although the authors state that some earlier reports do not confirm the higher risk of respiratory diseases related to tobacco smoking in women, the evidence of a greater vulnerability of women has now become unquestionable. In two French cross-sectional studies in the late 1970s we found that the prevalence of respiratory symptoms increased more sharply with increasing numbers of cigarettes smoked in female than in male subjects and that there was a dose-response relationship between smoking and lower levels of height-adjusted forced expiratory volume in one second (FEV_1), both in adults [2] and in teenagers [3]. Since then several cross-sectional and follow-up studies have reported similar results. A recent longitudinal study suggests that adolescent girls may be more vulnerable than boys to the effects of smoking on the growth of lung function [4]. It seems unlikely that results observed in different countries at different times, measuring different health outcomes are due to epidemiological biases.

We have also investigated whether females are more likely than men to have nonspecific bronchial hyperresponsiveness (BHR), which can be a risk factor for the accelerated decline of lung function. Using the data from 407 males and 392 females aged 20–44 yrs, from the Paris and Montpellier centres of the European Community Respiratory Health Survey (ECRHS) [5] we studied the sex ratio for BHR, defined as a 20% fall in FEV_1 when a maximal cumulative dose of 4 mg methacholine had been administered. The proportion of reactors was 37% in females and 19% in males (odds ratio (OR)=2.6 (1.9–3.6)). After excluding subjects with asthma, and adjusting for baseline FEV_1, BHR was still significantly more frequent in females (OR=1.9 (1.1–3.4)). The excess prevalence of BHR in women was related to smoking. The OR for BHR in heavy *versus* nonsmokers was 2.0 (0.99–4.1) and in heavy *versus* moderate smokers was 2.8 (1.3–6.4) in females, whereas the corresponding ORs were 1.1 (0.4–2.7) and 0.8 (0.3–2.3) in males.

The paper by PRESCOTT *et al.* [1] shows gender differences in severe respiratory morbidity. Thus, although the reasons for such sex-specific differences are not known, the greater respiratory vulnerability of females to smoking is consistently evidenced whatever the health outcomes measured, from asymptomatic bronchial hyperresponsiveness to hospitalizations for COPD.

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REPLY

From the authors:

We thank Liard, Leynaert, Neukirch and Bousquet for their comments regarding our recent paper [1] and for supplying additional data. In addition to our results regarding admission to hospital for chronic obstructive pulmonary disease (COPD), we have found that female smokers have a higher relative mortality risk from respiratory disease than male smokers, compared to never-smokers of the corresponding gender [2].

Regarding Liard and associates' interesting results on bronchial hyperresponsiveness (BHR), a higher prevalence of BHR in female smokers with early COPD was also found in the Lung Health Study [3], although this was suggested to be due to their smaller airway caliber [4]. In the same study BHR was shown to be a predictor of progression of airway obstruction in smokers [5]. Furthermore, it has repeatedly been reported that females in the Lung Health Study responded both more favourably to smoking cessation and with a further forced expiratory volume in one second (FEV_1) decline when restarting smoking. Although this information is still unpublished, it was most

recently presented by S. Buist during the discussion at the European Respiratory Society study on chronic obstructive pulmonary disease (EUROSCOP) session at the recent European Respiratory Society meeting in Berlin.

If we accept that female lungs are more susceptible to the deleterious effects of smoking, which Liard and associates find is unquestionable, can a possible mechanism be that smoking is more prone to trigger nonspecific bronchial hyperresponsiveness in women, causing airway obstruction to progress more rapidly?

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