6. **Xu X**, Li B, Wang L. Gender difference in smoking effects on adult pulmonary function. *Eur Respir J* 1994;**7**:477–83.
7. **Xu X**, Weiss ST, Rijcken B, *et al*. Smoking, changes in smoking habits, and rate of decline in FEV1: new insight into gender differences. *Eur Respir J* 1994;**7**:1056–61.
8. **Xu X**, Dockery DW, Ware JH, *et al*. Effects of cigarette smoking on rate of loss of pulmonary function in adults: a longitudinal assessment. *Am Rev Respir Dis* 1992;**146**:1345–8.
9. **DeMeo DL**. The Yin and Yang of COPD: sex/gender differences in the National Emphysema Treatment Trial. *Am J Respir Crit Care Med* 2007;**176**:222–3.
10. **Soerheim IC**, Johannessen A, Bakke P, *et al*. Gender differences in COPD—are women more susceptible to smoking effects? *Am J Respir Crit Care Med* 2009;**179**:A3750.
11. **Soerheim IC**, Johannessen A, Grydeland TB, *et al*. Case—control studies on risk factors for COPD: how does the sampling of the cases and controls affect the results? *Clin Respir J* 2009. doi:10.1111/j.1752-699X.2009.00154.x.
12. **Bakke P**, Gulsvik A, Lilleng P, *et al*. Postal survey on airborne occupational exposure and respiratory disorders in Norway: causes and consequences of non-response. *J Epidemiol Community Health* 1990;**44**:316–20.
13. **Brogger J**, Bakke P, Eide GE, *et al*. Contribution of follow-up of nonresponders to prevalence and risk estimates: a Norwegian respiratory health survey. *Am J Epidemiol* 2003;**157**:558–66.
14. **Anon**. Standardization of spirometry, 1994 update. American Thoracic Society. *Am J Respir Crit Care Med* 1995;**152**:1107–36.
15. **Gulsvik A**, Tosteson T, Bakke P, *et al*. Expiratory and inspiratory forced vital capacity and one-second forced volume in asymptomatic never-smokers in Norway. *Clin Physiol* 2001;**21**:648–60.
16. **Johannessen A**, Omenaas ER, Bakke PS, *et al*. Implications of reversibility testing on prevalence and risk factors for chronic obstructive pulmonary disease: a community study. *Thorax* 2005;**60**:842–7.
17. **Bakke PS**, Hanoa R, Gulsvik A. Educational level and obstructive lung disease given smoking habits and occupational airborne exposure: a Norwegian community study. *Am J Epidemiol* 1995;**141**:1080–8.
18. **Prescott E**, Godtfredsen N, Vestbo J, *et al*. Social position and mortality from respiratory diseases in males and females. *Eur Respir J* 2003;**21**:821–6.
19. **Silverman EK**, Weiss ST, Drazen JM, *et al*. Gender-related differences in severe, early-onset chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 2000;**162**:2152–8.
20. **Whittemore AS**, Perlin SA, DiCiccio Y. Chronic obstructive pulmonary disease in lifelong nonsmokers: results from NHANES. *Am J Public Health* 1995;**85**:702–6.
21. **Becklake MR**, Kauffmann F. Gender differences in airway behaviour over the human life span. *Thorax* 1999;**54**:1119–38.
22. **Ben-Zaken Cohen S**, Pare PD, Man SF, *et al*. The growing burden of chronic obstructive pulmonary disease and lung cancer in women: examining sex differences in cigarette smoke metabolism. *Am J Respir Crit Care Med* 2007;**176**:113–20.
23. **Han MK**, Postma D, Mannino DM, *et al*. Gender and chronic obstructive pulmonary disease: why it matters. *Am J Respir Crit Care Med* 2007;**176**:1179–84.
24. **Fabbri LM**, Romagnoli M, Corbetta L, *et al*. Differences in airway inflammation in patients with fixed airflow obstruction due to asthma or chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 2003;**167**:418–24.
25. **Chapman KR**, Tashkin DP, Pye DJ. Gender bias in the diagnosis of COPD. *Chest* 2001;**119**:1691–5.
26. **Patrick DL**, Cheadle A, Thompson DC, *et al*. The validity of self-reported smoking: a review and meta-analysis. *Am J Public Health* 1994;**84**:1086–93.
27. **Dransfield MT**, Washko GR, Foreman MG, *et al*. Gender differences in the severity of CT emphysema in COPD. *Chest* 2007;**132**:464–70.
28. **Martinez FJ**, Curtis JL, Sciurba F, *et al*. Sex differences in severe pulmonary emphysema. *Am J Respir Crit Care Med* 2007;**176**:243–52.
29. **Grydeland TB**, Dirksen A, Coxson HO, *et al*. Quantitative computed tomography: emphysema and airway wall thickness by sex, age and smoking. *Eur Respir J* 2009;**34**:858–65.
30. **Johannessen A**, Lehmann S, Omenaas ER, *et al*. Post-bronchodilator spirometry reference values in adults and implications for disease management. *Am J Respir Crit Care Med* 2006;**173**:1316–25.

Lung alert

IL-23 and allergic airway inflammation

This study examines the role of interleukin 23 (IL-23) in airway inflammation in asthma, focusing on the ability of IL-23 to promote inflammation independently of the known IL-17 pathway and via its effect on T helper 2 (Th2) polarisation. These interactions are studied using in vitro and in vivo techniques.

Mice exposed to allergen showed a significant increase in IL-23-specific p19 mRNA and IL-23 receptor mRNA. In mice induced to overexpress IL-23, an increased inflammatory cell infiltrate was observed after allergen challenge. In IL-23 knockout mice, exposure to the same allergen challenge resulted in a significant reduction in airway inflammation.

Having established a role for IL-23 in airway inflammation the authors next examined its signalling pathway. No significant alteration in IL-17 expression was seen between the IL-23 knockout and IL-23-expressing mice, thus suggesting a possible pathway independent of IL-17. In contrast, the levels of Th2 cytokines (IL-4, IL-5 and IL-13) were seen to vary significantly between the two groups. In the knockout mice, a dramatic reduction in Th2 cytokines was observed in lymphatic cultures.

Further examination of this pathway in vitro used CD4 T cells activated via an antigen-presenting cell-dependent or -independent mechanism. In both cases IL-23 affected Th2 differentiation. Partial Th2 inhibition was seen in the absence of IL-23 and increasing differentiation was observed in the IL-23-positive cell line.

This study confirms the importance of IL-23 and its receptor in regulating airway inflammation and identifies a possible signalling pathway between IL-23 and Th2 cells independent of IL-17.

► **Peng J**, Yang XO, Tesfaigzi Y, Chang SH *et al*. IL-23 signalling enhances Th2 polarization and regulates allergic airway inflammation, lung function, and COPD in high-risk populations. *Cell Res* 2010;**20**:62–71.

Charlotte Addy

Correspondence to Charlotte Addy, ST4, Bristol Royal Infirmary, UK; chill1981@doctors.org.uk

Thorax 2010;**65**:485. doi:10.1136/thx.2010.141101