MS-based analytical technique. The principal findings of this study are (i) PTR-MS evidence for the flow dependency of exhaled acetone; (ii) changing minute ventilation can both increase and decrease the concentrations of selected exhaled trace gases; and (iii) concentrations of certain volatiles were not significantly altered by respiratory manoeuvres in healthy volunteers.

These preliminary observations may have important implications regarding the standardisation requirement for measuring and reporting the concentrations of exhaled trace gases in the future. Further larger studies both in healthy and diseased subjects are necessary to expand on these observations and to provide mechanistic insights into exchange kinetics of affected volatiles. Such studies may help to further define the exact role of on-line MS technologies in non-invasive diagnosis and monitoring pulmonary and systemic diseases.

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► An additional table is published online only. To view this file please visit the journal online (<http://thorax.bmj.com>).

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Association of IgG4-related disease and sarcoidosis

Autoimmune pancreatitis (AIP) is a syndrome characterised by an enlarged pancreas with an irregular narrowing of the main pancreatic duct, a high serum IgG4 concentration and IgG4-positive plasma cell tissue infiltration.

A wide variety of lesions have been associated with AIP, including pulmonary lesions.¹ Consequently, the terms “IgG4-positive multi-organ lymphoproliferative syndrome (IgG4+ MOLPS)” seem more appropriate.²

To our knowledge, sarcoidosis has never been reported in association with AIP. We report the case of an association of AIP with sarcoidosis in an elderly woman.

An 80-year-old woman presented with a 1-year history of chronic diarrhoea and a weight loss of 12 kg during the previous 6 months. Physical examination was normal. Chest and abdominal CT scan disclosed an increased volume of the pancreas, coeliac and

Figure 1 Contrast-enhanced coronal reformatted CT view: hilar and mediastinal adenomegaly, diffuse homogeneous hypoattenuating enlargement of the pancreas with minimal peripancreatic stranding.

hilar lymphadenopathy and pulmonary lesions (figure 1). Pancreas biopsies revealed chronic pancreatitis. Accessory salivary gland and coeliac adenopathy biopsies showed non-caseating giant-cell epithelioid granuloma. The tuberculin purified protein derivative test and the Quantiferon assay were both negative. Laboratory analysis revealed a polyclonal hypergammaglobulinemia with IgG level at 35 g/l; serum IgG4 level was increased at 6.8 g/l (normal <0.8 g/l), white blood cell count revealed a lymphopenia (1000 mm³/l) and ACE was within the normal range.

Because of the histological picture of non-tuberculous granulomas, and mediastinal lymph nodes with pulmonary involvement on chest CT, sarcoidosis associated with an IgG4+ MOLPS was diagnosed. Corticosteroids (1 mg/kg/day) led to a dramatic improvement in the general and digestive manifestations within a 1-year follow-up.

AIP is a form of chronic pancreatitis characterised by a high serum IgG4 concentration and abundant IgG4-bearing plasma cell infiltration in the pancreatic lesion. This entity has been reported to be associated with a variety of extrapancreatic lesions. It is generally accepted that this form of pancreatitis is a part of a multi-systemic clinical syndrome, and this disease was redefined as 'IgG4-positive multi-organ lymphoproliferative syndrome'.²

Recently, Tsushima *et al*³ compared the clinicopathological features of pulmonary lesions in 19 patients with AIP and 8 patients with sarcoidosis; 17 of the 19 patients with AIP showed bilateral hilar lymphadenopathy, while 8 showed pulmonary nodules. IgG4-positive plasma cells were identified in the pulmonary lesions of patients with AIP. Our patient presented an authentic chronic pancreatitis with a significant increase in serum IgG4 level. She fulfilled the revised diagnostic criteria for AIP.⁴ Because of the presence of pulmonary lesions and hilar lymphadenopathies, salivary gland and coeliac adenopathies were biopsied, and they both revealed non-caseating epithelioid cell granulomas. Although sarcoidosis is uncommon in the elderly, the presence of disseminated granulomatous lesions led us to suspect sarcoidosis. However, it is difficult to determine whether our 80-year-old patient has an IgG4-related disease with systemic granulomatous lesions or an association of AIP with true sarcoidosis.

To our knowledge, such an association of AIP with granulomatous lesions mimicking sarcoidosis has never been reported previously in the literature, and this enlarges the spectrum of IgG4-related disease.

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CORRESPONDENCE

Gender differences in COPD: are women more susceptible to smoking effects than men?

We read the paper by Sørheim *et al*¹ with interest since possible clinical bias with regard to sex and disease in terms of diagnosis and treatment is clearly an important issue. The main problem with comparing the effect of a disease such as chronic obstructive pulmonary disease (COPD) between the two sexes is how one expresses the lung function deficit so that the data for the two sexes can be correctly analysed together.

We believe the method used by Sørheim *et al* introduces a sex bias that may be incorrectly influencing their result. The authors used percentage predicted to express the degree of abnormality and, depending on the equations used, this may bias the result with regard to sex and age.² Using the equations used by Sørheim *et al*,³ the scatter about the predicted value is the same for both sexes although the absolute predicted values for men are higher. This means that a man and a woman with forced expiratory volume in 1 s (FEV₁) values at equivalent deviation from predicted in population terms—for example, 1.645 standard deviations below predicted (equivalent to the 5th centile)—will have quite different percentage predicted values. Thus, for men of 1.80 m and women of 1.65 m (average height of the authors' prediction equations³) at the age of

25 years, the FEV₁ values at the 5th centile are 86.3% and 83.7% of predicted, respectively, and at the age of 70 they are 81.7% and 76.7% of predicted, respectively. In the paper by Sørheim *et al* the mean height of their subjects is not given but, assuming the above values and using the mean ages of the groups in table 1 in the paper, the 5th centile FEV₁ values would be at 82.5% and 78.6% predicted for the men and women, respectively, with COPD and 84.2% and 80.9% predicted for those without COPD.

This demonstrates how percentage predicted falsely suggests that subjects with equivalently low FEV₁ values in population terms appear to be different, with a bias towards women having apparently worse values than men. This bias is greater in older subjects and those with worse lung function. When using the ECCS prediction equations, this effect is still present but is much less than that seen with the equations used by Sørheim *et al*. Thus, using percentage predicted with the authors' prediction equations automatically makes low results for women appear worse than equivalently low results for men. We do not believe the paper by Sørheim *et al* has proved that women are more susceptible to smoking effects and their conclusion could well be an artefact based on the incorrect method used for expressing lung function abnormality. We suggest that the authors should rework their data with statistically valid methodology with their equations, such as using standardised residuals⁴ or centile values, and perhaps verify this with the generic equations of Stanojevic *et al*⁵ in order to determine if women are truly more susceptible than men to the effects of smoking.

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