

Table 1 Gender differences in health status in patients with chronic cough assessed with the Leicester Cough Questionnaire (LCQ)

	Females	Males	p Value (t test)
Number (%)	112 (63)	66 (27)	
Age, years (SEM)	56 (1)	54 (2)	0.5
Cough duration, years (SEM)	5.5 (0.9)	3.3 (0.6)	0.04
LCQ total (range 3–21) (SEM)	13.5 (0.4)	14.9 (0.5)	0.01

Health status questionnaires are designed to quantify quality of life numerically using the least number of questions. They are not a substitute for taking a good history. During the validation of the LCQ, health-related issues particularly pertinent to females were evaluated.¹ Several items, including stress incontinence, were excluded because only a minority of patients reported them. There was also evidence that alterations in health status due to symptoms such as stress incontinence were adequately captured by items that relate particularly to the psychosocial impact. It is important to note that health status questionnaires should be designed for use in the wider population rather than targeted to a specific subset of patients.

In conclusion, the LCQ is a brief, well-validated and widely used health status questionnaire for patients with cough and can be used to detect gender differences.

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Airway epithelial cells as guardians of immune homeostasis?

We read with interest the paper by Wang *et al* and accompanying editorial by Smyth showing that healthy murine airway epithelial cells (AECs) are potent inhibitors of dendritic cell (DC)-induced T cell activation.^{1,2} AECs infected with respiratory syncytial virus (RSV) lost this regulatory function, allowing activation of T cell responses and airway inflammation.¹ These in vitro observations match with the high concentrations of pro-inflammatory mediators and cells found clinically in the bronchoalveolar lavage fluid of infants with RSV bronchiolitis.²

The paper by Wang *et al* adds to a growing body of evidence that AECs are involved in maintaining airway immune homeostasis.³ Mayer *et al* previously showed that primary murine and immortalised human AECs induce an anti-inflammatory microenvironment inhibiting DC maturation and reducing T cell proliferation through constitutive secretion of transforming growth factor- β .³ Wang *et al* comment that further studies in human primary AECs are required to validate the findings in a clinical setting. Smyth also highlights the importance of research to investigate the function of AECs in health.²

Primary AECs cultured from protocol bronchoscopic brushings taken from clinically stable lung allograft recipients free from chronic allograft dysfunction represent a useful model to study AEC function in a healthy, steady state, albeit alloimmune environment. In a recent paper we have shown that epithelial cell-conditioned medium from stable lung allografts drives the production of macrophage-like cells from monocytes rather than DCs.⁴ It is unclear whether this effect only occurs in the airway of lung transplant recipients or if it reflects a general role for AECs in the homeostasis of DC populations in the lung. Nonetheless, our findings provide complementary human evidence to the murine observations of Wang *et al* and indicate that, in a steady state, AECs may be important in local immune homeostasis and promote an anti-inflammatory and pro-phagocytic airway milieu.

An emerging hypothesis that encompasses these observations is therefore that, in the healthy state, AECs regulate local immune homeostasis in the epithelium and promote anti-inflammatory conditions in the airway. In response to epithelial damage such as RSV infection, danger signals are released into the microenvironment by AECs which drive the production and maturation of professional antigen-presenting DCs, promoting T cell activation and airway inflammation.⁵

To explore this hypothesis more fully we suggest that future studies should include primary human tissue in both health and disease, and that this strategy can complement and extend animal studies.

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Are patients on treatment for pulmonary TB who stop expectorating sputum genuinely culture negative?

In patients receiving treatment for pulmonary tuberculosis (TB), change in sputum culture from positive to negative is the principal outcome measure of a therapeutic response in both clinical practice^{1,2} and drug trials.³ Patients will often stop producing sputum early in the course of treatment.⁴ We have tested the assumption that "no sputum" means that the patient is "culture negative", as this has never been confirmed experimentally.

We prospectively followed 16 patients with newly diagnosed pulmonary TB. Sputum samples were collected at diagnosis, during weeks 1 and 2, at months 1, 2 and 4, and on completing treatment. Those patients who were not producing sputum spontaneously had specimens collected by induction. This was performed in a purpose-built negative pressure isolation chamber (Elwyn E Roberts Isolators, Shropshire, UK) where patients inhaled 3% hypertonic saline via an ultrasonic nebuliser for 20 min. Samples were homogenised with Sputasol (Oxoid, Basingstoke, UK). A dilution series