12 **Kuperman DA**, Huang X, Koth LL, *et al*. Direct effects of interleukin-13 on epithelial cells cause airway hyperreactivity and mucus overproduction in asthma. *Nat Med* 2002;**8**:885–9.
13 **Oldfield WL**, Kay AB, Larché M. Allergen-derived T cell peptide-induced late asthmatic reactions precede the induction of antigen-specific hyporesponsiveness in atopic allergic asthmatic subjects. *J Immunol* 2001;**167**:1734–9.
14 **Haselden BM**, Larché M, Meng Q, *et al*. Late asthmatic reactions provoked by intradermal injection of T-cell peptide epitopes are not associated with bronchial mucosal infiltration of eosinophils or T(H)2-type cells or with elevated concentrations of histamine or eicosanoids in bronchoalveolar fluid. *J Allergy Clin Immunol* 2001;**108**:394–401.
15 **Ying S**, Humbert M, Barkans J, *et al*. Expression of IL-4 and IL-5 mRNA and protein product by CD4+ and CD8+ T cells, eosinophils, and mast cells in bronchial biopsies obtained from atopic and nonatopic (intrinsic) asthmatics. *J Immunol* 1997;**158**:3539–44.
16 **Buckley MG**, VariendS, Walls AF. Elevated serum concentrations of beta-tryptase, but not alpha-tryptase, in sudden infant death syndrome (SIDS). An investigation of anaphylactic mechanisms. *Clin Exp Allergy* 2001;**31**:1696–704.
17 **Mochizuki A**, McEuen AR, Buckley MG, *et al*. The release of basogranulin in response to IgE-dependent and IgE-independent stimuli: validity of basogranulin measurement as an indicator of basophil activation. *J Allergy Clin Immunol* 2003;**112**:102–8.
18 **Gavett SH**, Chen X, Finkelman F, *et al*. Depletion of murine CD4+ T lymphocytes prevents antigen-induced airway hyperreactivity and pulmonary eosinophilia. *Am J Respir Cell Mol Biol* 1994;**10**:587–93.
19 **Hogan SP**, Matthaei KI, Young JM, *et al*. A novel T cell-regulated mechanism modulating allergen-induced airways hyperreactivity in BALB/c mice independently of IL-4 and IL-5. *J Immunol* 1998;**161**:1501–9.
20 **Diaz P**, Gonzalez MC, Galleguillos FR, *et al*. Leukocytes and mediators in bronchoalveolar lavage during allergen-induced late-phase asthmatic reactions. *Am Rev Respir Dis* 1989;**139**:1383–9.
21 **Aalbers R**, Kauffman HF, Vrugt B, *et al*. Bronchial lavage and bronchoalveolar lavage in allergen-induced single early and dual asthmatic responders. *Am Rev Respir Dis* 1993;**147**:76–81.
22 **Gratziou C**, Carroll M, Montefort S, *et al*. Inflammatory and T-cell profile of asthmatic airways 6 hours after local allergen provocation. *Am J Respir Crit Care Med* 1996;**153**:515–20.
23 **Leckie MJ**, ten Brinke A, Khan J, *et al*. Effects of an interleukin-5 blocking monoclonal antibody on eosinophils, airway hyper-responsiveness, and the late asthmatic response. *Lancet* 2000;**356**:2144–8.
24 **Phipps S**, Flood-Page P, Menzies-Gow A, *et al*. Intravenous anti-IL-5 monoclonal antibody reduces eosinophils and tenascin deposition in allergen-challenged human atopic skin. *J Invest Dermatol* 2004;**122**:1406–12.
25 **Bentley AM**, Meng Q, Robinson DS, *et al*. Increases in activated T lymphocytes, eosinophils, and cytokine mRNA expression for interleukin-5 and granulocyte/macrophage colony-stimulating factor in bronchial biopsies after allergen inhalation challenge in atopic asthmatics. *Am J Respir Cell Mol Biol* 1993;**8**:35–42.
26 **Kay AB**, Ali FR, Heaney LG, *et al*. Airway expression of calcitonin gene-related peptide in T cell peptide-induced late asthmatic reactions in atopics. *Allergy* 2007;**62**:495–503.

LUNG ALERT

What factors are predictive of survival in patients with non-small-cell lung cancer treated with gefitinib?

▲ Satouchi M, Negoro S, Funada Y, *et al*. Predictive factors associated with prolonged survival in patients with advanced non-small-cell lung cancer (NSCLC) treated with gefitinib. *Br J Cancer* 2007;**96**:1191–6.

This study identified factors associated with prognostic benefit from gefitinib chemotherapy. Japanese patients who had received gefitinib monotherapy between 2002 and 2005 at the Hyogo Medical Centre for Adults in Japan (n = 221) were included in the study. Their clinical parameters were retrospectively examined for potential predictive factors of survival.

Median survival time was better in females, 13.3 vs 6.8 months (= 0.036); patients with adenocarcinoma, 9.3 vs 3.6 months (p = 0.137); never smokers, 14.5 vs 6.5 months (p<0.001); those with favourable performance status, 11.1 vs 2.1 months (p<0.001); and patients with epidermal growth factor receptor (EGFR) mutation, 24.9 vs 7.4 months (p<0.001). The lower the smoking exposure (Brinkman Index: cigarettes per day × years smoked) the longer the mean survival time (p<0.001). Multivariate analysis showed that positive EGFR mutation status and performance status 0–1 were independent predictors of a favourable prognosis.

Prognosis was significantly different according to EGFR mutation status (with the same smoking status), but not according to smoking status (with the same EGFR mutation status). The authors suggest that although smoking is not a direct predictor of prognosis, it may be useful as a surrogate marker for EGFR mutation status. They concluded that EGFR mutation status is the most important independent predictor of survival benefit with gefitinib treatment.

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