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Oral care and pneumonia

Sir—D Simons and colleagues (May 22, p 1761)[1] report poor oral status of the institutionalised elderly in the UK, which may contribute to the eating disorders and low nutrient and vitamin C concentration in this group. Since aspiration of bacteria in oropharyngeal secretions is an important risk factor for nonsocomial pneumonia in the elderly,[2] poor oral health may also contribute to the development of pneumonia. We investigated whether oral care lowers the frequency of pneumonia in the institutionalised elderly.

We prospectively assessed the rate of pneumonia in elderly people receiving oral care and in those who did not receive oral care. Participants were selected from 11 nursing homes. Nurses or care-givers cleaned their teeth by toothbrush after each meal, and scrubbed the pharynx with an applicator with povidone iodine (1%) every day. Dentists assessed oral status once a week for the oral-care group.

Before the study, all participants underwent physical examination and chest radiography. Participants were randomly (by random numbers table) assigned oral care or no active treatment in September, 1996, and were followed up for 2 years. Criteria for diagnosis of pneumonia were new pulmonary infiltrate seen on chest radiograph, and cough, temperature higher than 37·8°C, or subjective dyspnoea. Two radiologists not involved in the study diagnosed pneumonia. 51 people were excluded from analysis because they died from causes other than pneumonia during follow-up. Of the remaining 366 people, 184 (mean age 82 years [SD 7]) received oral care at study entry and 182 (mean age 82 years [7]) received no active oral care.

During follow-up, pneumonia was diagnosed in 34 (19%) participants who did not receive oral care and 21 (11%) of those who received oral care. Relative risk of developing pneumonia on no active oral care compared with oral care was 1·67 (95% CI 1·01–2·75, p=0·04).

Oral care lowered the risk of pneumonia in institutionalised elderly. This finding underscores the necessity for the monitoring of specific oral hygiene courses for nurses and care-givers.

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Asthma and diabetes

Sir—Isabelle Douek and colleagues (May 29, p 1850)[1] and the EURODIAB ACE substudy group[2] suggest that low incidence of asthma among patients with type-1 diabetes mellitus is more than coincidental. The T-helper (Th)1/Th2 model suggests that the two diseases are unlikely to co-exist in the same individual because they are dominant immune response is driven by interleukin 2, interferon gamma, and cell-mediated immunity. By contrast, allergic asthma is a Th2 disease in which interleukin 4, interleukin 5, and IgE dominate the immune response. The distinct polarisation of type-1 diabetes and allergic asthma, however, provide a unique opportunity to re-examine the underlying pathogenesis of both diseases and to offer new approaches in the prevention of these diseases.

Since the progression of the Th1 or Th2 immune response is closely linked to HLA markers of the host, the susceptible gene for one disease might be a resistant gene for the other disease. Thus, molecular engineering may be possible to alter the susceptibility of individuals who are prone to autoimmune disease such as type 1 diabetes.

In an independent metabolic study,[3] Szczeklik and colleagues showed that glycaemic responses to tolbutamide or insulin were significantly different between children with atopic asthma and controls. They conclude that more economic use of insulin and reduction in hypoglycaemic effects of cate-cholamines in bronchial asthma might explain why asthmatics rarely develop diabetes. It is tempting to speculate that there is a hidden link between a genetic marker for neurohorminal control and immunological markers that together drive the host to develop either Th1 or Th2 disease.

In the Th1/Th2 model, cross-regulation of the Th1 immune response to suppress Th2 disease, or vice versa, is now possible. For instance, bacille Calmette-Guèrin infection can suppress allergic sensitisation.[4] Likewise, treatment with interleukin 4 suppresses the onset of type-1 diabetes.[5] The question now is, how long we need to maintain such immunotherapy in patients and what are its long-term consequences?

Whatever the answers turn out to be, any immunological intervention has to start early, at the onset or even before the onset of the disease. This early intervention is particularly important for type-1 diabetes, because the effect of therapy will not be felt once the β-cell mass in the diseased pancreas is substantially diminished.

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Sir—A positive association of haptoglobin *(Hp*1/*1)* genotype has been reported with human fertility[1] and atopic asthma in children.[2] Since atopic asthma is characterised by a preponderant Th2 response, and the maintainance of normal pregnancy seems to be dependent on Th2 immune response,[3] our data suggest that *Hp*1/*1* genotype may favour the development of a Th2 immune response.

Isabelle Douek and colleagues[4] suggest that the same genetic factors predisposing to a Th1-mediated