

and non-invasive way of obtaining samples to assess airway inflammation and diagnose pulmonary infections such as tuberculosis (TB).<sup>1,2</sup> Data published on the safety of SI are largely derived from subjects with obstructive lung disease. Yet on this basis a Working Group of the European Respiratory Society (ERS) recommended that all patients undergoing SI should receive pre-procedure bronchodilation with monitoring throughout by either serial forced expiratory volume in 1 s or peak expiratory flow rate (PEFR) measurements.<sup>3</sup> Subjects with other respiratory conditions may not be at the same risk of bronchoconstriction as the original reference group. If so, then the recommended steps could be removed from routine SI, leading to a simplified procedure with no compromise in patient safety. Here we report our findings from routine clinical practice.

We prospectively observed 100 consecutive and unselected adult subjects undergoing SI to investigate possible TB. SI was performed using 3% hypertonic saline delivered via an ultrasonic nebuliser (Sunrise Medical, Wollaston, UK). To ensure good infection control, bronchospasm was assessed with disposable Mini-Wright peak flow meters (Clement Clarke International, Harlow, UK). PEFR was measured at baseline and then at 5, 10, 15 and 20 min. The preselected criteria for discontinuing SI were: (1) a fall in PEFR of >15% at any time point, (2) patient choice, (3) if the patient were deemed by the clinician performing the test to be unable to continue. Bronchodilators and resuscitation equipment were available if required. Equipment costs for generic salbutamol metered dose inhaler devices and the PEFR meter were calculated from the *British National Formulary* (March 2007) and manufacturer's data.

Median age of the 100 subjects was 43 years (range 18–68) and 60 were male. Six were ex-smokers and 26 were current smokers. Median baseline PEFR was 450 l/min (range 220–710). No subject had to stop the procedure because of respiratory symptoms or a fall in PEFR of >15% from baseline. The median change in PEFR was –6.6% (range –14% to +29%) with 76 subjects having a fall in PEFR, eight no change and 16 an increase in PEFR at 20 min. None of the patients required a bronchodilator post-procedure or developed symptoms thereafter. Three of 100 patients stopped at 15 min: one with headache, one because of nausea and one as they had collected an adequate sample.

Our data argue against the current ERS guidelines. We found a low rate of bronchospasm or complications following SI in our patients being investigated for TB (including current and ex-smokers). This suggests that it is safe to omit routine bronchodilation and PEFR measurements during SI in these subjects. We estimate that removing these

steps produces a saving of approximately £10 in cost and 10 min in time per procedure (which usually takes 30 min without this step). We would be interested to know whether other centres have had a similar experience in their clinical practice.

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## Margarine: a supplement may be decisive

A recent study in *Thorax* described margarine intake as a risk factor for allergic diseases (*Thorax* 2007;**62**:677–83). This association has now been replicated in about 10 studies, but without any convincing explanation.<sup>1</sup> Margarine is the only food factor to date that is associated with allergic diseases without provoking any direct allergic reaction.

Margarine was invented by the French chemist Hippolyte Mège-Mouriès at the demand of emperor Napoleon III in 1869 as a cheap replacement food for his troops. The initial recipe contained water, skimmed milk and suet. Its use spread rapidly as a butter substitute, with the first factory in Oss/Netherlands exporting approximately 40 000 tons of margarine to Great Britain in 1883. As a raw material, lard was used at that time as well as copra, palm or train oil. As sales in the mid of the last century suffered from a somewhat poor quality image, modifications have been introduced, for example, to raise the content of polyunsaturated fats by using sunflower oil.

Since 1952, vitamin D<sub>3</sub> has also been added to margarine in West Germany. According to a new monograph on the history of vitamin D supplementation in Germany,<sup>2</sup> the company Merck delivered a highly concentrated oily vitamin solution to the Margarine Union who supplemented about the half of the 230 000 tons annually produced at that time. Since then margarine

has been one of the few if not the only continuously fortified food in Germany with a major brand containing ~1 IU/g D<sub>3</sub>.

Given more recent experimental and epidemiological findings on the immunological action of vitamin D and its metabolites,<sup>3</sup> it is possible that this vitamin D supplement may be responsible for the observed effect. Changing from butter to margarine indeed leads to ~30% higher 25-OH-calciferol serum level<sup>4</sup> which may be in the effective range of serum levels linked to allergic rhinitis in the NHANES study.<sup>5</sup>

Possibly a trial on the Crete island could answer the question of whether or not there is any causal relationship between a margarine ingredient and allergy. Vitamin D supplements could be banned from margarine for a limited time as on a sunny island there is no major fear of vitamin D deficiency in the population. Will the allergy risk remain?

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## Authors' reply

We read with great interest the letter by Wjst proposing an alternative hypothesis for margarine supplementation by vitamin D as the risk factor underlying its positive associations with asthma and allergies. In our recently published study (*Thorax* 2007;**62**:677–83), we found that margarine intake was a risk factor for asthma and rhinitis symptoms among children in Crete. We hypothesised that the high concentration of n-6 PUFAs in margarine could partially explain the observed associations through modulation of the synthesis of IgE and inflammatory mediators.<sup>1</sup>

The evidence regarding the effects of vitamin D on the development of asthma and allergies is controversial. Vitamin D has been shown to inhibit Th1 immune responses but its effects on Th2 responses are more complex and not fully elucidated. Moreover, genetic studies have provided early, although not clear, evidence of the