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## *CORRESPONDENCE*

# Asthma: surfactant eliminates the early allergen-induced response

*To the Editor:*

In their recent article BABU *et al.* [1] have just published a remarkable finding in which they have clearly demonstrated how the inhalation of synthetic surfactant abolishes the early allergen-induced response in asthmatics. In interpreting these impressive results, however, they invoke theory based upon oedema at whose liquid-air interface surfactant could exert a "high surface pressure".

In pursuing this approach, the authors appear to have ignored a basic principle of surface physics. Forces derived from surface pressure/tension can only be translated into pressure differences for driving fluid ($\Delta P$) if there is appreciable curvature to the interface as expressed by the Laplace equation, viz:

$$\Delta P = \gamma\left\{\frac{1}{r_1}+\frac{1}{r_2}\right\} \quad (1)$$

where $\gamma$ is the surface tension (reciprocating surface pressure) and $r_1$ and $r_2$ are radii of curvature in mutually perpendicular planes. Thus, respirologists focus much attention upon surface tension at the alveolar level where curvature is high ($r_1=r_2\cong 175$ μm), $\Delta P$ amounting to as much as 8.0 cm water gauge (cm.w.g) if surfactant were not reducing the surface tension of water ($\gamma$=69.9 $mN\cdot m^{-1}$). However, at the bronchial level ($r_1$=1 cm; $r_2=\infty$), the fluid pressure ($\Delta P$) generated by pure water would be less than 0.07 cm.w.g. Hence, even at its highest surface pressure ($\gamma\cong 0$), surfactant could have no physiological effect whatsoever. Surfactant would simply cause the surface contribution to oedema formation ($\Delta P$) to fall from one insignificant level to another.

It would therefore seem more likely that surfactant is acting upon the asthmatic lung by the alternative "barrier" mechanism, referenced by the authors [2], whereby surface-active phospholipid (SAPL) binding by adsorption to bronchial epithelium is "masking" irritant receptors which elicit the bronchoconstrictor reflex. However, this alternative approach now invokes the mechanism of adsorption of SAPL to solid surfaces, which conflicts with a belief, culturally embedded in respirology, that surfactant acts only at liquid-air interfaces. It is interesting that this "belief" also conflicts with the roles of SAPL in nonpulmonary sites *in vivo* and with vast experience in the physical sciences of surfactants studied at solid surfaces where they often form barriers [3].

Any role of surface tension/pressure by surfactant acting at the liquid-air interface of oedema must surely be a red herring, but it should not detract from a most exciting clinical finding.

**B.A. Hills**
Golden Casket Paediatric Research Laboratory, Mater Medical Research Institute, Brisbane, Queensland, Australia.

### References

1. Babu KS, Woodcock DA, Smith SE, Staniforth JA, Holgate ST, Conway JH. Inhaled synthetic surfactant abolishes the early allergen-induced response in asthma. *Eur Respir J* 2003; 21: 1046–1049.
2. Hills BA. Asthma: is there an airway receptor barrier? *Thorax* 1996; 51: 773–776.
3. Hills BA. An alternative view of the roles of surfactant and the alveolar model. *J Appl Physiol* 1999; 87: 1567–1583.

# Role of interleukin-10 in idiopathic pulmonary fibrosis

*To the Editors:*

We have read with great interest the article of BERGERON *et al.* [1], concerning the cytokine profile in tissues of patients with idiopathic pulmonary fibrosis (IPF). We strongly agree that finding out the roles of cytokines in IPF might be the key in understanding the pathogenesis of the disease, as well as inventing new therapy strategies for this foetal and unresolved disorder. We think that outlining the cytokine profile in the tissues of those patients is very important, even though, as the authors also mention, the number of patients included in their study (five) was unfortunately small.

We conducted a study in the University Hospital in Thessaly (central Greece), involving 20 patients with IPF and 11 patients with pulmonary fibrosis of a known cause, as well as 40 healthy volunteers, in whom we measured the serum levels of several cytokines, such as interleukin (IL)-2, IL-4, IL-8, IL-10 and interferon gamma. Interestingly, we found several differences between the serum levels of the two different patient groups, as well as those in patients and healthy volunteers. One striking result was that IL-10 was detected in increased levels in sera of patients with IPF, in comparison to healthy volunteers ($p<0.05$), and was not detected at all in patients with pulmonary fibrosis of a secondary cause. We have suggested the use of IL-10 in the differential diagnosis of patients with IPF, while BERGERON *et al.* [1], suggest it might be a possible therapeutic target for IPF.

We agree with the observation of BERGERON *et al.* [1], and think that IL-10 might be an important cytokine in IPF. Although the above observations have been made in both studies in a "given moment", the elevation in the amounts of IL-10 in both the tissues and serum of patients with IP, is an observation, which, we think, should not be ignored. Nevertheless, we assume that those "given moments" happened to be the same in both studies, since the serum and tissue samples have been obviously collected during the onset of the disease, before any treatment was administered. However, this was not very clear in the paper of BERGERON *et al.* [1], and we would like to know if it really happened as we assume.

The existing literature of observations concerning the IL-10 profile in pulmonary fibrosis is scarce. Most papers suggest that IL-10 has an antifibrotic effect, and derives from the