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CORRESPONDENCE

What is the economic impact of preschool viral upper respiratory infection?

To the Editor:

In a previous issue of the *European Respiratory Journal* STEVENS *et al.* [1] suggested that preschool children with wheeze account for a significant proportion of the UK healthcare budget. Most preschool wheezing is caused by viral infection and, therefore, we wondered how much preschool children with upper respiratory tract infection (URTI) without wheeze would cost the National Health Service?

On average, a preschool child will catch 6–8 URTIs per year [2], and 11% of children <14 yrs will be taken to their family doctor when unwell. These figures may be higher in younger children [3]. If a child has six colds per year and is seen by a doctor for 11% of these infections, then, based on the 2.94 million UK preschool population estimate and a £16 consultation cost estimation [1], the primary care costs of viral URTI would be £31 million. This compares to the £34 million estimated by STEVENS *et al.* [1] as the cost of preschool wheezing. Therefore, the additional cost burden of wheeze seems small.

We believe the villains are not wheeze and asthma but the respiratory viruses!

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Exhaled breath condensate pH

To the Editor:

VAUGHAN *et al.* [1] are to be congratulated for the most extensive study of the pH of exhaled breath condensates (EBC) yet reported. However, there is reason to doubt that the pH of the condensate provides a reliable measure of the pH of fluid lining the airways. As noted in our recent study [2], the average concentration of NH_4^+ is 20 times greater than that of any other EBC cation in normal condensates, and NH_4^+ accounts for most of the ions and buffer in the condensate (as judged by total conductivity).

Several previous studies have shown that most of this NH_4^+ is derived from NH_3 generated in the mouth, in part from the bacterial degradation of urea [2–4]. In the study by VAUGHAN *et al.* [1], collection of condensates through endotracheal tubes did not seem to have an effect upon average pH. Since both intubation and tracheostomy significantly reduce NH_4^+ concentrations of the EBC [2, 4], they should be associated with more acidic condensates. This paradox could only be explained if intubation also reduced concentrations of some atmospheric or oral acid (*e.g.* residual CO_2 or acetic acid) in the EBC. It must be concluded that the pH of normal condensates reflects buffering by these volatile constituents from the mouth, rather than buffers in the airway fluid, and therefore EBC pH cannot provide reliable estimates of airway pH.

Although the normal condensate pH is set by oral and atmospheric buffers, HUNT and coworkers [5, 6] observed "acidopnoea" in asthmatics, which they attributed to airway acidification. We have suggested that reductions in exchange of NH_3 in the mouth and condenser are responsible in part for the reduction in EBC NH_4^+ seen in asthmatics [7], and consequently contribute to acidification. It is also possible that reflux of gastric fluid, which is extraordinarily common in asthmatics [8], is responsible for EBC acidification. Aerosolisation of tiny quantities of gastric acid (pH often 1–2) from the stomach or pharynx would be difficult to avoid or detect in these patients but could have a profound effect on EBC pH.

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