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From the authors:

In R.M. Effros′ frequent letters in response to data regarding exhaled breath condensate (EBC) pH, there is one constant: he states that his speculations must be correct, despite the accumulation of substantial data to the contrary. To his credit, R.M. Effros now accepts that indeed there are acids involved in the acidification of exhaled breath condensate. Additionally edifying is that, in this current letter, he has not repeated his previous strongly stated, yet incorrect, notions that: pH cannot be measured in EBC; that glass electrodes will provide irreproducible readings; that asthmatic hyperventilation causes EBC acidification; or that facilitated diffusion would cause increased ammonia to be exhaled when the airways are acidic [1]. Much data have emerged that have thoroughly negated these notions.

This time R.M. Effros has manufactured a false paradox compelling him to speculate an unnecessary solution. He makes the false assumption that removal of oral ammonia should be acidifying to a relevant degree. Indeed, R.M. Effros′ "paradox" disappears when we discard his underlying false assumption. Because EBC pH and ammonia experiments indeed solidly dispel his assumption, we are easily left with no paradox, and an internally consistent set of findings regarding EBC pH.

Although R.M. Effros references his suggested mechanism for artifactual EBC pH decline in asthmatics, we respectfully remind him that the data in our paper specifically and directly disprove his suggestion. Scientists uniformly agree that it is more reasonable to discard hypotheses proven wrong, than to discard data inconsistent with hypotheses.

We have to agree with R.M. Effros that oral ammonia, as a base, should indeed have some alkalinising effect. However, the data show clearly that this effect in EBC is too tiny to be noted without a huge enrollment of subjects, and such a quest is pointless. Neither oral ammonia, nor the lack of it, is responsible for the profound EBC acidification seen in various diseases. Indeed, that EBC acidification is characteristic of intubated patients with lung disease, but not of intubated patients without lung disease [2–4], helps further to dispel R.M. Effros′ argument that the mouth is the controller of EBC pH. As part of the airway, the mouth assuredly contributes to EBC. But the mouth is a small part of the airway, and its contribution to EBC may be likewise.

Gastro-oesophageal reflux certainly affects tracheal pH [5], with tracheal pH probe readings falling to 4.0, and therefore we believe aspirated acid to be one mechanism that acidifies EBC. We are convinced that reflux contributes to lung disease, but if reflux is the sole reason why EBC pH falls, then reflux must be a factor in most every lung disease patient, even while intubated, which is more than even the most vocal advocates suggest.

Over the years, we have appreciated R.M. Effros′ strongly and repeatedly asserted, albeit incorrect, speculations. Without these thoughtful speculations, we might not have so aggressively and rigorously tested every facet of the exhaled breath condensate pH assay. In the end, exhaled breath condensate pH has proven to be thoroughly robust and entirely useful. Patients with respiratory diseases, including asthma, chronic obstructive pulmonary disease, bronchiectasis [6], cystic fibrosis [7], acute lung injury and acute respiratory distress syndrome [2] exhale more acid. This is particularly true of patients who are undergoing disease exacerbations. To us, this is very interesting indeed both as a marker of disease activity, and as a clue to underlying pathologic mechanisms.

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