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optimised in a way that takes into account the known underlying physical mechanisms.

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#### References

- Paredi P, Caramori G, Cramer D, et al. Slower rise of exhaled breath temperature in chronic obstructive pulmonary disease. Eur Respir J 2003; 21: 439–443.
- Walker JE, Wells CRE, Merrill EW. Heat and water exchange in the respiratory tract. Am J Med 1961; 30: 259– 267.
- 3. Caldwell PRB, Gomez DM, Fritts HW Jr, *et al.* Respiratory heat exchange in normal subjects and in patients with pulmonary disease. *J Appl Physiol* 1969; 26: 82–88.
- 4. Primiano FP, Saidel GM, Montague FW Jr, Kruse KL, Green CG, Horowitz JG. Water vapour and temperature dynamics in the upper airways of normal and CF subjects. *Eur Respir J* 1988; 1: 407–414.
- 5. Gilbert IA, Fouke JM, McFadden ER Jr. Heat and water flux in the intrathoracic airways and exercise-induced asthma. *J Appl Physiol* 1987; 63: 1681–1691.
- 6. Baile EM, Dahlby RW, Wiggs BR, Pare PD. Role of tracheal and bronchial circulation in respiratory heat exchange. *J Appl Physiol* 1985; 58: 217–222.
- Solway J, Leff ID, Dresha I, et al. Circulatory heat sources for canine respiratory heat exchange. J Clin Invest 1986; 78: 1015–1019.
- 8. Serikov VB, Fleming NW. Pulmonary and bronchial circulations: contributions to heat and water exchange in isolated lungs. *J Appl Physiol* 2001; 91: 1977–1985.
- 9. McFadden ER Jr, Pichurko BM, Bowman KF, et al. Thermal mapping of the airways in humans. *J Appl Physiol* 1985; 58: 564–570.
- 10. Cole P. Further observations on conditioning of respiratory air. *J Laryngol Otol* 1953; 67: 669–681.

### From the authors:

We would like to thank J.B. McCafferty and J.A. Innes for their interest and constructive criticism relating to our paper which reported low exhaled breath temperature gradients in chronic obstructive pulmonary disease patients compared to normal subjects [1].

J.B. McCafferty and J.A. Innes suggest that it is controversial that bronchial blood flow has a role in airway heat exchange. We disagree and would like to draw their attention to numerous papers that demonstrated the opposite effect. First, it has been shown that changes in airway blood volume are reflected in fluctuations in intra-thoracic heat exchange, whereas even large increases in blood flow through the pulmonary circulation do not influence airstream temperature [2]. This proves that bronchial and not pulmonary blood flow plays a major role in airway heat exchange. Secondly, changes in bronchial blood flow can alter airway responsiveness and temperature [3], confirming that the bronchial circulation may control airstream temperature and contribute to airway narrowing. Thirdly, temperature changes induce bronchoconstriction, which can be prevented by reducing mucosal blood supply using inhaled vasoconstrictors [3], indicating that airway temperature and bronchial blood flow are correlated. Furthermore, preliminary data presented as an oral presentation at the American Thoracic Society meeting in Atlanta last year by our group showed a correlation between bronchial blood flow, measured noninvasively by an inhaled soluble gas technique, and exhaled breath temperature gradients [4]. Therefore, there is substantial evidence that airway blood flow is the major determinant of airway heat exchange.

In their letter, J.B. McCafferty and J.A. Innes also state that heat transfer occurs in the upper airways (above the glottis) when breathing normally at room temperature. They quote a manuscript published in 1953 by COLE [5], but they have not interpreted the manuscript correctly and have ignored more recent publications by the same author. Even though we agree that inspired air is mostly conditioned in the upper airways, in our study we concentrated on exhaled air. The work of COLE [6] has demonstrated that "during expiration heat and water are returned to the exhaled air along the whole of the respiratory tract", including the bronchial tree. We would also like to stress that the patients included in our study, as described in the method session, were not "respiring at low flow rates" as indicated by J.B. McCafferty and J.A. Innes. Rather, they were breathing normally at tidal volume before being asked to exhale at a constant exhalation flow rate (10 L·min<sup>-1</sup>) after a deep inhalation to total lung capacity.

Regarding the methodology for the measurement of exhaled breath temperature gradients, J.B. McCafferty and J.A. Innes point out that we did not report the method of flow targeting. However, after describing the exhalation manoeuvre as flow and pressure controlled, we refer the reader to a previous publication by our group in which all the details related to the pressure (4 cmH<sub>2</sub>O) and flow control (visual feedback) are fully described (see reference [14] in the text of [11).

J.B. McCafferty and J.A. Innes make further comments regarding our manuscript stating that because we did not control the exhalation flow rate our measurements actually reflect expiratory pattern rather than heat exchange. However, we did control exhalation flow rate (see above and see the Methods section in the manuscript [1]). Furthermore, a more thorough reading of the literature would have shown that expiratory air at the mouth is little influenced by ventilation rate [6] and pattern (tidal volume and respiratory rate changes with the same minute ventilation) [7].

We agree that the measurement of humidity of the exhaled breath would add important information related to the balance between airway temperature and evaporation of epithelial lining fluid in the airways. We are therefore currently developing a new method for the measurement of exhaled breath humidity. For the standardisation of the method (exhalation flow rate, mouth pressure, distance of the thermocouple from the mouthpiece, body core temperature, room temperature, etc.) we refer J.B. McCafferty and J.A. Innes to references [13] and [14] in our manuscript [1].

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### References

- 1. Paredi P, Caramori G, Cramer D, Ward S, Kharitonov SA, Barnes PJ. Slower rise of exhaled breath temperature in COPD. *Eur Respir J* 2003; 21: 439–443.
- Gilbert IA, Regnard J, Lenner KA, Nelson JA, McFadden ERJ. Intrathoracic airstream temperatures during acute

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- expansions of thoracic blood volume. *Clin Sci (Colch)* 1991; 81: 655–661.
- 3. Gilbert IA, McFadden ERJ. Airway cooling and rewarming. The second reaction sequence in exercise induced asthma. *J Clin Invest* 1992; 90: 699–704.
- 4. Paredi P, Kharitonov SA, Barnes PJ. Exhaled breath temperature gradients in asthma and the effect of beta 2 agonists. *Am J Respir Crit Care Med* 2002; 165: A237.
- 5. Cole P. Further observations on conditioning of respiratory air. *J Laryngol Otol* 1953; 67: 669–681.
- 6. Cole P. Recordings of respiratory temperature. *J Laryngol Otol* 1954; 68: 295–307.
- Ingenito E, Solway J, McFadden ERJ, Pichurko BM, Cravalho EG, Drazen J. Finite difference analysis of respiratory heat transfer. J Appl Physiol 1986; 61: 2252– 2259.