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Mitochondrial dysfunction in COPD patients with low body mass index

To the Editors:

The potential importance of mitochondrial (dys)function in common chronic diseases is increasingly recognised [1]. Accordingly, we read with great interest the article by RABINOVICH *et al.* [2], which was recently published in the *European Respiratory Journal*. RABINOVICH *et al.* [2] showed that chronic obstructive pulmonary disease (COPD) patients with a low body mass index (BMI; $19.2 \pm 0.6 \text{ kg} \cdot \text{m}^{-2}$) have a dysfunctional mitochondrial electron transport chain in comparison with COPD patients with a “normal” BMI ($29.0 \pm 1.7 \text{ kg} \cdot \text{m}^{-2}$) and healthy controls ($27.9 \pm 1.9 \text{ kg} \cdot \text{m}^{-2}$). The function of the mitochondrial electron transport chain was assessed as acceptor control ratio (*i.e.* the efficiency of oxidative phosphorylation) in freshly obtained muscle biopsies.

We recently showed that cigarette smoke, which is the major cause of COPD in the Western world, can induce significant mitochondrial dysfunction [3]. This dysfunction was the consequence of a cigarette smoke-induced blockage of the mitochondrial electron transport chain, resulting in a decreased mitochondrial respiration rate and a corresponding decrease in adenosine triphosphate production.

In the study performed by RABINOVICH *et al.* [2], the prevalence of current and past smoking was not reported. As a consequence, the potential influence of smoking on the outcome of the study was neither analysed nor discussed. Due to our recent observations, and reports from other groups about the induction of mitochondrial dysfunction by cigarette smoke even in circulating cells that do not reside in pulmonary tissues [4], we thought it would be very interesting to know how the results of the clinical study performed by RABINOVICH *et al.* [2] would appear if current (and past) smoking were taken into account. Furthermore, it would be interesting to know how the significantly lower acceptor control ratio results reported in their study affect the principal function of mitochondria: adenosine triphosphate production.

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STATEMENT OF INTEREST

None declared.

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From the authors:

We would like to thank D-J. Slebos and co-workers for their interesting and assertive contribution to the present issue of the *European Respiratory Journal*. Following their arguments, one can reasonably state that because tobacco smoking is the main cause of chronic obstructive pulmonary disease (COPD), it might also play a role in a mechanism leading to the