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Inhibition of the renin–angiotensin system in severe COPD

To the Editors:

In a recent issue of the *European Respiratory Journal*, ALBERT and CALVERLEY [1] analysed the treatment approaches in severe chronic obstructive pulmonary disease (COPD). However, they did not mention the effect of the inhibition of the renin–angiotensin system (RAS) in this setting.

Angiotensin converting enzyme (ACE) is present in very high concentrations in the lungs, and its activity is further increased by chronic hypoxia [2, 3]. Indeed, in patients with COPD, lower ACE activity may improve the efficiency of the peripheral use of oxygen and respiratory muscle function [3, 4].

Furthermore, mounting evidence suggests that COPD is characterised by systemic inflammation that might have an adverse impact on various extrapulmonary organs [5]. Interestingly, RAS blockade exerts an anti-inflammatory action in many systems [6].

Finally, even some side-effects related to therapy with inhibitors of the RAS, in certain instances, can prove beneficial in some patients with COPD. An intact and activated RAS has been shown to be an important determinant of erythropoiesis [7], and it has been found that the inhibition of the RAS may be a useful treatment for secondary erythrocytosis [8, 9]. Consequently, RAS blockade might have profound benefits in the long-term treatment of COPD-associated polycythaemia. Moreover, even cough, a well-characterised side-effect of ACE inhibitors, could be of some benefit in that it can decrease the risk of aspiration pneumonia in certain patient settings [10].

Therefore, we suggest that future studies should assess the overall health impact of renin–angiotensin system blockade in patients with chronic obstructive pulmonary disease.

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STATEMENT OF INTEREST

None declared.

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