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## Authors' response: hyperoxia in acute asthma

We appreciate the comments by Snelson and Tunnicliffe<sup>1</sup> regarding our study of the effects of high concentration oxygen therapy in acute exacerbations of asthma.<sup>2</sup> We concur with the view that the effect of high concentration oxygen therapy on arterial carbon dioxide pressure (PaCO<sub>2</sub>) is not clinically relevant in all patients presenting to the emergency department (ED) with acute severe asthma. However, we consider that the 3.9-fold greater risk of patients developing an increase in transcutaneous partial pressure of carbon dioxide (PtCO<sub>2</sub>) ≥8 mm Hg (22% vs 6% in the high concentration vs titrated oxygen groups, respectively) is likely to be of clinical relevance in life-threatening asthma. Even in our study, which excluded patients who were unable to speak or perform spirometry due to breathlessness, all 10 patients who had a final PtCO<sub>2</sub> ≥45 mm Hg had received high concentration oxygen therapy. These findings suggest that the routine administration of high concentration oxygen therapy in the ED setting is a determinant of respiratory failure, a recognised marker of near fatal asthma. This probably also applies to the routine use of high concentration oxygen therapy during ambulance transfer in patients with severe asthma, as has been noted in chronic obstructive pulmonary disease,<sup>3</sup> but this was not assessed in our study.

While permissive hypercapnia is an approach to the management of mechanical ventilation for severe asthma, this relates to intubated patients, in whom the purpose is to reduce the risk of complications associated with hyperinflation.<sup>4</sup> It certainly does not apply to prehospital or ED care.

We agree that there are many potential risks associated with hyperoxia, including but not limited to reductions in coronary and cerebral blood flow, decreased cardiac output, increased oxidative stress, delay in recognising a clinical deterioration and rebound hypoxaemia if oxygen therapy is abruptly stopped. However, in respiratory disorders such as severe asthma where there is significant ventilation/perfusion (V/Q) mismatch, hypercapnia represents another potential risk of high concentration oxygen therapy that needs to be recognised in clinical practice.

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## Increasing smokers' risk perception improves CT screening participation

We read with interest the article by Patel *et al*<sup>1</sup> and wish to comment on their findings with specific regard to smokers' risk perception, motivation and low participation rates in CT screening programmes.

Based on the studies to date, there is a consistent theme that smokers' participation in CT screening programmes for lung cancer is poor when their motivation is low and much greater when their perception of risk of lung cancer is high.<sup>1–2</sup> Despite overwhelming public health messaging, smokers continue to smoke, in large part, because they perceive their own risk from smoking to be low. This self-perception of low risk (termed optimistic bias) maintains a low level of motivational tension (the fear that smoking might indeed be harmful).<sup>3</sup> We propose that optimistic bias can be undermined, and motivational tension increased, when smokers are confronted with adverse 'personalised' risk data.<sup>3</sup> With advances in the understanding of the clinical and genetic factors underlying lung cancer susceptibility, we have developed a lung cancer susceptibility risk model.<sup>4</sup> This model assigns current and former smokers to moderate, high and very high risk. In a group of randomly

selected current smokers, 84% took up the offer of risk testing and, surprisingly, quit rates 6 months after testing were 20%, 36% and 40%, respectively (28% overall).<sup>5</sup> Just as with triggering a decision to quit smoking, we suggest uptake of (and possibly adherence to) CT screening might be improved by risk testing that enhances risk perception, undermines optimistic bias and increases motivational tension.<sup>5</sup>

We tested this proposition in a scenario-based telephone questionnaire involving 350 current and former smokers (mean age 67, age range 44–86 years, 59% male and mean pack years 45). When told of a survival benefit with CT screening versus no screening, we found 68% agreed to undertake CT screening while 95% agreed to gene-based risk testing. Likelihood of participation in CT screening for lung cancer was 25% higher (absolute increase) in those testing high and very high risk compared with those at moderate (average) risk. Collectively, the results of these studies support our suggestion that optimistic bias can be undermined, and motivational tension increased, in current and former smokers through the use of personalised risk testing. We suggest that personalised risk testing, incorporating genetic markers of susceptibility, may help identify and motivate 'high risk' smokers to engage in CT screening.

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