Authors’ reply

We thank Drs Young and Hopkins for their interest in our study and their interesting explanation for the results observed. It is indeed likely that the relationship between chronic obstructive pulmonary disease (COPD) and cancer (both pulmonary and extrapulmonary) is attributed to cytokine-induced inflammation mediated by guanosine triphosphatase (GTPase) signalling molecules. This is advocated by the results of Man et al who showed that the increased inflammatory state in patients with COPD is associated with future cancer mortality including extrapulmonary cancers.

Statins are associated with reduced cardiovascular morbidity and mortality in patients with cardiovascular disease. Besides the reduction in low-density lipoprotein cholesterol levels, statins also reduce inflammation through reduced expression of inflammatory cytokines which is known as one of the pleiotropic effects of statins. A recent double-blind placebo controlled trial in patients who had undergone vascular surgery showed that patients who were treated preoperatively with fluvastatin had significantly decreased levels of interleukin at the time of surgery compared with the placebo group (−35% and −4%, respectively; p<0.001). The same was observed for high-sensitivity C reactive protein, another marker of inflammation, which was decreased by 21% in the fluvastatin group and increased by 3% in the placebo group (p<0.001). Furthermore, patients with elevated inflammatory levels are more likely to benefit from statin therapy than those without elevated levels. This might explain the increased beneficial effects of statins in patients with COPD and cancer observed in our study. Although the results of our study are in line with those of previous studies which suggest that statins might have an important role in patients with COPD (with or without cancer), further studies are needed before statin treatment can be recommended for patients with COPD.

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