

## CORRESPONDENCE

## Azithromycin and severe asthma

Brusselle *et al*<sup>1</sup> performed an interesting academic-based randomised controlled trial Azithromycin for Prevention of Exacerbations in Severe Asthma (AZISAST) of azithromycin 250 mgm taken thrice weekly versus placebo as add-on therapy for 26 weeks in non-smoking patients with severe asthma, defined as Global Initiative for Asthma (GINA) steps 4–5, taking high dose inhaled corticosteroids/long-acting bronchodilators and with at least two severe exacerbations within 6 months of study entry. In the half of subjects with lower blood eosinophil counts ( $\leq 200/\mu\text{l}$ ) they detected a significant effect on the primary endpoint (severe exacerbations and/or lower respiratory tract illnesses requiring antibiotics) and also on asthma quality of life (AQL). These results lend additional empirical evidence to support my clinical experience that a significant subgroup of patients with uncontrolled severe asthma will experience important clinical benefits from azithromycin, and that this benefit appears to be ‘all-or-none’.<sup>2</sup> My most recent primary care-based trial confirmed this ‘all-or-none’ response pattern and also found that response persisted long after the azithromycin treatment was completed.<sup>3</sup> The primary care severe asthma subjects differed from the AZISAST subjects by including smokers and having worse AQL at randomisation (AQL 4.12 v 5.35 in AZISAST). Many of these primary care responders identified themselves correctly as good candidates for azithromycin.<sup>4</sup>

A pilot trial provided preliminary data that *Chlamydia pneumoniae* IgA, but not IgG, might be a predictor of treatment response and this needs to be studied further as another possible relevant biomarker.<sup>5</sup> I believe that AZISAST brings us closer to the time when we will understand the mechanisms underlying the azithromycin-asthma response and be able to categorise candidates for treatment, but I do not think that we are there yet.

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