

minimum of 4 to 6 weeks lag to a disease response as reported by Dr Summers et al, *all* subjects received 2 to 5 doses at least 4 weeks before June 9 (90%, ≥ 6 weeks). This compliance was identical by treatment group despite that subjects on *T suis* had an unexpected 3 to 19-fold higher rate of episodes with moderate to severe diarrhoea, abdominal pain, and flatulence (median duration, 2 days) compared with placebo.

Hepworth et al also write that a dose of 2,500 *T suis* ova every 21 days might be insufficient to effectively alter systemic responses considering the lack of colonization and rapid clearance of *T suis*. However, we observed an altered systemic *T suis* IgG response in more than 92% of subjects. In addition, Table 1 demonstrates that subjects who did not clear *T suis* by means of diarrhea or subjects who responded in the upper quartile of *T suis* IgG titers (>29.9 milligram antibody per liter serum) showed no reduction in their skin reactions to allergens or grass IgE titers after 6 months. A few insignificant changes in the size of skin reactions were likely an after effect of higher pollen and more variable indoor allergen exposure during the summer months. Finally, our demonstration of an absence of an effect of *T suis* on measures of allergic reactivity in skin, blood, and airways even after 6 months is not compatible with the insignificant effect on self-reported medication usage noted by Dr Summers et al.

In conclusion, an even closer look at our data suggests that *T suis* will not show sufficient efficacy in treatment of subjects who have already had an allergic disease.

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Obesity and asthma control in an urban population

To the Editor:

We read with interest the article by Clerisme-Beatty et al¹ in the July issue of the Journal. The authors highlight an important and increasingly common problem in the clinical management of asthma, particularly in urban and disadvantaged populations, an area in which the authors have considerable expertise. The impact that obesity has on asthma control and asthma severity has been researched by several groups and continues to be debated. The authors of this study, unlike others,²⁻⁴ did not find an association between asthma control and obesity.

We suggest that this discrepancy in their findings may be explained by the population enrolled in their study. The study had a large sample of 292 people with asthma attending primary care in an urban setting. Patients were recruited into the study if they had evidence of active asthma defined as recent asthma symptoms or reliever medication use. However, the very high group mean value for the Asthma Control Questionnaire (2.1), and correspondingly low mean value for the Asthma Control Test (15.4) indicate that the population as a whole had very poorly controlled asthma, particularly compared with previous studies addressing this issue.² This population bias may have made it more difficult to detect an association between body mass index and asthma control.

This limitation of the present cross-sectional study casts some doubt on the authors' conclusion that the National Asthma Education and Prevention Program guidelines advocating weight loss as a measure to improve asthma control may be premature. Weight loss has been associated with improvement in asthma control.^{5,6} A previous systematic review found an improvement in various asthma outcomes associated with weight loss.⁷ There is also evidence of improvement in respiratory health status in patients completing a weight loss program.⁸ Although further work is needed to understand the phenotype of the obese person with asthma, there appears to be adequate evidence to justify national guidelines recommending weight loss as part of strategies to improve asthma control.

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Reply

To the Editor:

We are writing in reply to the comments of Farah et al¹ regarding our findings published in the July issue of the Journal about the effects of obesity on asthma control in an urban population.²

As the authors report, our findings are contradictory to previous reports showing a positive association between obesity and asthma control.³⁻⁵ We acknowledge the authors' argument that compared to previous studies, our study population was, on average, more poorly controlled. However, our study population included a full spectrum of disease status ranging from intermittent to severe persistent asthma. We believe that a population bias would only have a major impact on our conclusions if it was necessary to include patients without current evidence for asthma (ie, patients with no symptoms or medication use in the previous 6 months). Our results generalize most readily to those with active symptoms, and thus, to those most likely to benefit from lifestyle changes, if they are eventually proven effective.

We, too, are enthusiastic about the emerging published literature on the effect of weight loss on asthma control, but we interpret the studies as supportive of a testable hypothesis, rather than definitive enough evidence on which to base guideline recommendations. The limitations of the existing reports, as articulated by Eneli et al,⁶ include small sample sizes (median, $N = 28$) as well as heterogeneity of asthma definitions, study interventions, and asthma outcomes. According to this systematic review, in only 5 of the 15 relevant studies was asthma the main outcome, and the only report relevant to pediatrics describes a

single adolescent with asthma in 1 study of bariatric surgery in a series of morbidly obese children.⁷ In some cases, it is unclear whether reports of improved asthma control after weight loss are a result of improvement in asthma physiology or improvements in respiratory mechanics attributable to weight loss. For example, in the trial of 38 obese patients with asthma,⁸ dyspnea improved more in the group assigned to a low calorie diet than those on standard diet, but there was no difference in the symptom cough. Likewise, the authors report greater improvements in FEV₁ and forced vital capacity (FVC) in the intervention group but do not report whether or not there were changes in the ratio FEV₁/FVC, leaving it uncertain whether or not any degree of obstruction was relieved. As we reported in our study, FEV₁ and forced vital capacity were both inversely related to the degree of obesity, but FEV₁/FVC was not.

We agree with the authors that more definitive trials are needed to elucidate better the effect of obesity on asthma risk and phenotype. Although weight loss for people who are overweight or obese should be recommended generally to improve overall health, as well as to improve exertional dyspnea, we do not believe there is yet sufficient evidence to tell patients that weight loss will effectively treat their asthma. Given the complexity of managing asthma with proven therapies, we feel that clinicians should focus their attention on proven therapies for improvement of asthma control.

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