Published in final edited form as:

J Allergy Clin Immunol. 2010 March 1; 125(3): 770. doi:10.1016/j.jaci.2009.12.993.

Reply to Farah

Emmanuelle M Clerisme-Beaty, M.D., MHS, Cynthia Rand, PhD, and Gregory B. Diette, M.D., MHS

Department of Medicine, Johns Hopkins University, Baltimore MD

To the Editor,

We are writing in reply to Farah et al.'s comments (1) 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 asthmatics. 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 (i.e. patients with no symptoms or medication use in the prior 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, include small sample sizes (median N=28), as well as heterogeneity of asthma definitions, study interventions and asthma outcomes (6). 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 one study of bariatric surgery in a series of morbidly obese children (7). In some cases, it is unclear whether reports of improved "asthma control" following weight loss are due to improvement in asthma physiology or to improvements in respiratory mechanics attributable to weight loss. For example, in the trial of 38 obese asthmatics (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 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

Corresponding Author: Gregory B. Diette M.D., MHS., Division of Pulmonary Critical Care Medicine, 1830 E Monument St, Fifth Floor Baltimore, MD 21205, USA., Telephone: 410-955-3467, Fax: 955-0036, gdielte@jhmi.edu.
Clerisme-Beaty EM, M.D., MHS. Department of Medicine, Johns Hopkins University, Baltimore MD.
Cynthia S. Rand, PhD. Department of Medicine, Johns Hopkins University, Baltimore MD
Gregory B. Diette M.D., MHS. Department of Medicine, Johns Hopkins University, Baltimore MD

None of the authors have any financial conflicts or relationships to declare.

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not any degree of obstruction was relieved. As we reported in our study, FEV₁ and FVC 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 better elucidate the effect of obesity on asthma risk and phenotype. While 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.

Acknowledgments

Declaration of all sources of funding: This work is supported by National Heart, Lung, and Blood Institute grant 5U01HL072455, and NIH K12 RR017627.

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