Body Mass Index and Asthma Severity Among Adults Presenting to the Emergency Department*

Carey Conley Thomson, MD, MPH; Sunday Clark, MPH; Carlos A. Camargo, Jr, MD, DrPH, FCCP; on Behalf of the MARC Investigators

Study objectives: Among adults presenting to the emergency department (ED) with acute asthma, we sought to determine the prevalence of obesity, and the relation of body mass index (BMI) to asthma severity in this high-risk population.

Design: Multicenter, prospective cohort study.

Setting: Twenty-six North American EDs.

Participants: Five hundred seventy-two patients aged 18 to 54 years presenting with acute asthma.

Interventions: None.

Measurements and results: A standardized interview assessed demographic characteristics, asthma history, and details of the current asthma exacerbation. Data on ED medical management and disposition were obtained by chart review. Three of four asthmatic patients were either overweight (BMI, 25 to 29.9; 30%) or obese (BMI, ≥ 30; 44%). Normal weight/underweight, overweight, and obese patients did not differ on several markers of chronic asthma severity; obese subjects tended to rate symptoms more severely and to use more inhaled β-agonists in the 6 h hours prior to ED presentation despite a significantly higher initial percentage of predicted peak expiratory flow (PEF) [44%, 45%, and 51%, respectively; p < 0.05]. The three BMI groups responded similarly to acute therapy in the ED, with all groups demonstrating reversible airway obstruction. The sex distribution by BMI group differed markedly (p < 0.001), with women less often overweight (40% vs 24%) and more often obese (30% vs 52%). Since women were more likely have a higher initial PEF (45% vs 53%, p < 0.001), we stratified by sex to further examine the relation of BMI to asthma severity. The observed BMI-asthma associations were due largely, but not entirely, to confounding by sex.

Conclusions: Despite lingering concerns about the veracity of “asthma” among obese individuals, asthma exacerbations among obese and nonobese adults were remarkably similar. Potential differences (e.g., in symptom perception, use of inhaled β-agonists before ED presentation, initial PEF rate) were due, in large part, to confounding by sex. (CHEST 2003; 124:795–802)

Key words: asthma, adult; body mass index; obesity; sex

Abbreviations: BMI = body mass index; BRFSS = Behavioral Risk Factor Surveillance System; CI = confidence interval; ED = emergency department; IQR = interquartile range; MARC = Multicenter Airway Research Collaboration; OR = odds ratio; PEF = peak expiratory flow

Excess body fat is a health problem affecting millions of people worldwide.1,2 Overweight is defined by a body mass index (BMI) of 25 to 29.9, and obesity by a BMI ≥ 30.3,4 The National Health and Nutrition Examination Survey III (from 1988 to 1994)5 estimated the prevalence of adult obesity in the United States as 18 to 23%, while more recent national survey data (1999)6 estimate 27%; the Behavioral Risk Factor Surveillance System (BRFSS) [2000] indicates an obesity prevalence of 20%.6,7

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Increases in obesity are largest among women and minority groups. In the United States, obesity costs approximately $99 billion, of which 52% is due to medical costs.

Asthma is another common, chronic disorder, and its prevalence also is rising in the United States and worldwide. Between 1980 and 1994, the prevalence of self-reported asthma increased from 31 to 55 per 1,000 population. Like obesity, asthma increased more among women and minority groups. In the United States, asthma costs approximately $11 billion according to most recent estimates.

The concomitant rise in both obesity and asthma has led several groups to examine a possible causal relation between these conditions. Some groups have raised concerns that the “asthma” of obese individuals may not be real, or that it may differ, on a pathophysiologic basis, from that of nonobese people. Specifically, these groups propose that “obese asthma” may not involve bronchial hyperresponsiveness or reversible airway obstruction but instead reflect dyspnea related to excess weight and subsequent misdiagnosis. Along those lines, the relationship between BMI and acute asthma is not known. In this multicenter, prospective study, we determined the prevalence of obesity among 572 adults presenting to urban emergency departments (EDs) with acute asthma, and examined the relationship between BMI and acute and chronic asthma severity in this high-risk population.

Materials and Methods

This prospective cohort study was performed during November 2000 to May 2001 as part of the Multicenter Airway Research Collaboration (MARC). Using a standardized protocol, investigators at 26 EDs in 15 US states and 1 Canadian province provided 24-h per day coverage for a median of 2 weeks. All patients were managed at the discretion of the treating physician. Inclusion criteria were physician diagnosis of acute asthma, age 18 to 54 years, and ability to give informed consent. Repeat visits by individual subjects were excluded. Five hundred seventy-two of 590 enrolled adults (97%) reported their weight and height.

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BMI was used as a measure of excess body fat. Normal/underweight is defined as a BMI < 25, overweight is defined as a BMI of 25 to 29.9, and obesity is defined as a BMI ≥ 30. Median family income was estimated using home ZIP codes obtained from hospital administration. Primary care provider status was assigned on the basis of the following question: “Do you have a primary care provider (such as a family doctor, internist, or nurse practitioner)?” If yes, patients were asked to provide the name and address of their provider. Smoking status was coded as never smoker, former smoker, and current smoker. The severe symptoms classification was assigned to patients who reported severe symptoms during the 24 h preceding the ED visit on at least one of two questions (ie, asthma symptoms “most of the time” or “severe” discomfort and distress due to their asthma).

Peak expiratory flow (PEF) was used to assess acute asthma severity, and is expressed as percentage of the value, based on age, sex, race, and height. Changes in PEF are expressed as the relative change in percentage of predicted (eg, an improvement from 40% predicted to 70% predicted would be expressed as a change of 75%).

Statistical Analysis

All analyses were performed using STATA 7.0 (StataCorp; College Station, TX). Data are presented as proportion (95% confidence interval [CI]), mean ± SD, or median (interquartile range [IQR]). The prevalence of obesity in the study population was compared with the prevalence of obesity among adults from the general population, as reported in national studies. The association between BMI and asthma severity was examined using χ² test, analysis of variance, or Kruskal-Wallis test, as appropriate. Stratified analyses and multivariate linear and logistic regression models were performed. Factors associated with BMI (or with the outcome of interest) at p < 0.10 in univariate analysis were evaluated for inclusion in multivariate regression models. All odds ratios (ORs) are presented with 95% CIs. All p values are two sided, with p < 0.05 considered statistically significant.

Results

The 572 patients had a mean age of 37 ± 10 years, and 66% (95% CI, 62 to 70) were women. As expected in this urban population, most subjects were black (44%) or Hispanic (26%), with only 30% white. The smoking status of these ED patients was never smoker (38%), former smoker (26%), and current smoker (36%). Only 3% reported comorbid COPD and the exclusion of these patients from analyses did not materially change any results (data not shown).

Figure 1 shows the BMI classification of these patients, with three of every four patients either overweight (30%) or obese (44%). The high prevalence of BMI ≥ 25.0 did not materially differ by socioeconomic status. For example, the prevalence of overweight or obesity was comparable among those who had and had not graduated from high school (74% vs 73%, p = 0.83) and across quartiles of median household income (74%, 77%, 77%, and 75%, respectively; p = 0.83).

The obesity prevalence in this cohort of adults...
presenting to the ED with acute asthma was significantly greater than the prevalence among adults from the general population using either of the obesity prevalence estimates: 44% vs 27% (p < 0.001) in 1999 national survey,5 and 44% vs 20% (p < 0.001) in 2000 BRFSS.6,7 The national prevalence of obesity would have to rise to 41% for this difference to become nonsignificant. By contrast, the overweight prevalence in our sample of asthmatic patients did not differ from the National Health and Nutrition Examination Survey III estimate (30% vs 34%, p = 0.93),5 but did differ significantly from the BRFSS estimate (30% vs 37%, p < 0.001).6,7

Comparisons of the three BMI groups are shown in Table 1. As previously noted, obese patients were significantly more likely to be female. Although the total with BMI ≥ 25 did not differ between the 196 men and 376 women (70% vs 76%, p = 0.10), the distribution within BMI categories did (p < 0.001): women were less often overweight (40% vs 24%) and more often obese (30% vs 52%). Overall, men had a mean BMI of 28.4 ± 5.8 while women had a mean BMI of 32.1 ± 9.3 (p < 0.001). Obese participants were slightly older, but did not differ according to several other important sociodemographic factors.

Chronic asthma factors also are presented in Table 1. Obese individuals did not differ by several markers of chronic asthma severity, such as history of asthma medication use, health-care utilization, and smoking status. In all groups, these factors pointed toward a very “real” asthma with a likely classification of patients as having moderate-to-severe persistent asthma.33

Acute asthma presentation and ED course are shown in Table 2. Compared to normal/underweight and overweight individuals, the obese groups had a significantly higher initial PEF (48% vs 53%, p < 0.04), despite a trend toward more severe subjective symptoms (76% vs 81%, p = 0.15) and more inhaled β-agonist puffs within 6 h of presenting to the ED (four puffs vs six puffs, p = 0.03). Although nonobese subjects had a shorter duration of symptoms than their obese counterparts (duration ≤ 24 h, 38% vs 47%; p = 0.05), obese participants did not differ by ED management or clinical response (eg, change in PEF, hospital admission).

Because women were almost twice as common as men, and comprised 77% of the obese group (Table 1), we paid particular attention to potential confounding or interaction by sex in all subsequent analyses. We first explored potential sex differences in the major asthma factors. Compared to men, women reported more severe symptoms (71% vs 81%, p = 0.007) despite a higher PEF (45% vs 53%, p < 0.001). Other important factors (eg, median number of inhaled β-agonist puffs within 6 h of presenting to the ED, hospital admission, ED length of stay, and the composite admission measure) did not differ by sex (data not shown).

Tables 3, 4 show the relation of BMI to the more important asthma factors, with analyses stratified by

![Figure 1. Distribution of BMI among adults presenting to the ED with acute asthma.](http://publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21998/)}
sex. Obesity was not related to initial PEF among men \( (p = 0.72) \) or among women \( (p = 0.27) \). Among men, obese patients were more likely to report recent use of inhaled steroids and were least likely to be admitted to the hospital, but did not differ from normal/underweight or overweight patients according to other acute or chronic asthma severity measures. Among women, obese patients were least likely to report duration of symptoms of \( \leq 24 \text{ h} \), while overweight women had the shortest ED length of stay. Other acute and chronic asthma measures did not differ across BMI groups among women.

### Table 2—Acute Asthma Presentation and ED Course According to BMI*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Normal/Underweight</th>
<th>Overweight</th>
<th>Obese</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe symptoms</td>
<td>107 (75)</td>
<td>132 (78)</td>
<td>203 (80)</td>
<td>0.21</td>
</tr>
<tr>
<td>No. of inhaled ( \beta )-agonists in past 6 h, median (IQR)</td>
<td>4 (2–10)</td>
<td>4 (0–12)</td>
<td>6 (2–13)</td>
<td>0.07</td>
</tr>
<tr>
<td>Duration of symptoms</td>
<td>0.03</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( \leq 3 ) h</td>
<td>19 (13)</td>
<td>13 (7)</td>
<td>18 (7)</td>
<td></td>
</tr>
<tr>
<td>4–24 h</td>
<td>59 (40)</td>
<td>56 (33)</td>
<td>78 (31)</td>
<td></td>
</tr>
<tr>
<td>1–7 d</td>
<td>61 (42)</td>
<td>79 (47)</td>
<td>120 (48)</td>
<td></td>
</tr>
<tr>
<td>( \geq 8 ) d</td>
<td>7 (5)</td>
<td>21 (12)</td>
<td>34 (14)</td>
<td></td>
</tr>
<tr>
<td>Initial PEF (% predicted), median (IQR)</td>
<td>44 (33–58)</td>
<td>45 (32–60)</td>
<td>51 (38–67)</td>
<td>0.049</td>
</tr>
<tr>
<td>No. of inhaled ( \beta )-agonists in first hour</td>
<td>1.6 ± 1.2</td>
<td>2.0 ± 1.2</td>
<td>1.8 ± 1.2</td>
<td>0.05</td>
</tr>
<tr>
<td>Received steroids in ED</td>
<td>111 (75)</td>
<td>140 (82)</td>
<td>193 (76)</td>
<td>0.21</td>
</tr>
<tr>
<td>Change in PEF (% predicted), median (IQR)</td>
<td>49 (15–89)</td>
<td>48 (24–57)</td>
<td>44 (20–86)</td>
<td>0.84</td>
</tr>
<tr>
<td>Hospital admission</td>
<td>29 (20)</td>
<td>21 (13)</td>
<td>46 (19)</td>
<td>0.16</td>
</tr>
<tr>
<td>ED length of stay in hours, median (IQR)</td>
<td>3.6 (2.6–5.8)</td>
<td>3.2 (2.3–4.7)</td>
<td>3.9 (2.7–5.5)</td>
<td>0.02</td>
</tr>
<tr>
<td>Admitted or ED length of stay &gt; 6 h, %</td>
<td>47 (32)</td>
<td>36 (22)</td>
<td>79 (32)</td>
<td>0.06</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SD or No. (%) unless otherwise indicated. Missing values in some variables.
To further examine the relation of obesity to initial PEF and the composite admission measure, multivariate regression was performed. The overall finding of an increased initial PEF among obese patients (5.47; 95% CI, –0.31 to 11.25; p = 0.06) was attenuated when controlling for sex in a linear regression model (4.18; 95% CI, –1.58 to 9.94; p = 0.16). Potential obesity-related differences in the severity of symptoms and number of inhaled β-agonists before ED presentation also were attenuated by statistical adjustment for sex (data not shown). By contrast, multivariate modeling of the composite admission end point suggested little influence of sex on risk of admission for overweight or obese patients. The risk of admission for overweight did not vary from the unadjusted model (OR, 0.6; 95% CI, 0.4 to 1.5) and the sex-adjusted model (OR, 1.0; 95% CI, 0.6 to 1.5). In other words, obese individuals with acute asthma had the exact same risk of asthma hospitalization as their normal/underweight counterparts.

**Discussion**

Many epidemiologic studies have noted the striking increase in both obesity and asthma, and both cross-sectional and longitudinal studies have attempted to document a link between these two chronic disorders. Our study is the first to examine the association between BMI and acute asthma severity among adults presenting to the ED. The prevalence of obesity among asthmatic adults in the ED was significantly higher than that of adults from the general population. More importantly, however, our data demonstrate that the asthma exacerbations of obese individuals do not materially differ from those experienced by nonobese people.

**Table 3**—Relation of BMI to Important Asthma Factors in Men Only*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Normal/Underweight (n = 59)</th>
<th>Overweight (n = 79)</th>
<th>Obese (n = 58)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of ED visits in past year</td>
<td>3 (0–5)</td>
<td>2 (0–5)</td>
<td>4 (1–10)</td>
<td>0.11</td>
</tr>
<tr>
<td>Admitted for asthma in past year</td>
<td>17 (29)</td>
<td>17 (22)</td>
<td>21 (37)</td>
<td>0.15</td>
</tr>
<tr>
<td>Inhaled steroids in past 4 wk</td>
<td>16 (28)</td>
<td>29 (37)</td>
<td>31 (53)</td>
<td>0.02</td>
</tr>
<tr>
<td>Current smoker</td>
<td>27 (47)</td>
<td>27 (34)</td>
<td>19 (34)</td>
<td>0.26</td>
</tr>
<tr>
<td>Severe symptoms</td>
<td>37 (65)</td>
<td>59 (75)</td>
<td>43 (74)</td>
<td>0.41</td>
</tr>
<tr>
<td>Number of inhaled β-agonists in 6 h before ED presentation</td>
<td>4 (0–10)</td>
<td>6 (2–10)</td>
<td>6 (0–16)</td>
<td>0.15</td>
</tr>
<tr>
<td>Duration of symptoms ≤ 24 h</td>
<td>31 (53)</td>
<td>36 (46)</td>
<td>26 (45)</td>
<td>0.58</td>
</tr>
<tr>
<td>Initial PEF (% predicted)</td>
<td>40 (30–54)</td>
<td>41 (29–53)</td>
<td>45 (35–55)</td>
<td>0.72</td>
</tr>
<tr>
<td>Change in PEF (% predicted), median (IQR)</td>
<td>25 (7–35)</td>
<td>20 (9–28)</td>
<td>26 (15–43)</td>
<td>0.39</td>
</tr>
<tr>
<td>Hospital admission</td>
<td>26 (26)</td>
<td>9 (12)</td>
<td>5 (9)</td>
<td>0.02</td>
</tr>
<tr>
<td>ED length of stay, h</td>
<td>3.4 (2.7–5.8)</td>
<td>3.3 (2.3–5.4)</td>
<td>3.5 (2.7–5.9)</td>
<td>0.46</td>
</tr>
<tr>
<td>Admission to hospital or ED length of stay &gt; 6 h</td>
<td>20 (35)</td>
<td>19 (25)</td>
<td>16 (28)</td>
<td>0.43</td>
</tr>
</tbody>
</table>

*Data are presented as No. or median (IQR).

**Table 4**—Relation of BMI to Important Asthma Factors in Women Only*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Normal/Underweight (n = 59)</th>
<th>Overweight (n = 91)</th>
<th>Obese (n = 196)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of ED visits in past year</td>
<td>2 (1–6)</td>
<td>2 (0–4)</td>
<td>3 (1–5)</td>
<td>0.36</td>
</tr>
<tr>
<td>Admitted for asthma in past year</td>
<td>27 (30)</td>
<td>25 (27)</td>
<td>35 (39)</td>
<td>0.82</td>
</tr>
<tr>
<td>Inhaled steroids in past 4 wk</td>
<td>47 (53)</td>
<td>44 (49)</td>
<td>97 (50)</td>
<td>0.68</td>
</tr>
<tr>
<td>Current smoker</td>
<td>33 (37)</td>
<td>35 (39)</td>
<td>63 (32)</td>
<td>0.48</td>
</tr>
<tr>
<td>Severe symptoms</td>
<td>70 (79)</td>
<td>73 (81)</td>
<td>160 (83)</td>
<td>0.69</td>
</tr>
<tr>
<td>Number of inhaled β-agonists in 6 h before ED presentation</td>
<td>6 (2–9)</td>
<td>4 (0–12)</td>
<td>6 (2–12)</td>
<td>0.25</td>
</tr>
<tr>
<td>Duration of symptoms ≤ 24 h</td>
<td>47 (53)</td>
<td>33 (37)</td>
<td>70 (36)</td>
<td>0.02</td>
</tr>
<tr>
<td>Initial PEF, % predicted</td>
<td>47 (34–62)</td>
<td>50 (36–63)</td>
<td>55 (39–71)</td>
<td>0.27</td>
</tr>
<tr>
<td>Change in PEF, % predicted</td>
<td>22 (7–39)</td>
<td>27 (16–35)</td>
<td>19 (11–33)</td>
<td>0.39</td>
</tr>
<tr>
<td>Hospital admission</td>
<td>14 (16)</td>
<td>12 (13)</td>
<td>41 (22)</td>
<td>0.21</td>
</tr>
<tr>
<td>ED length of stay, h</td>
<td>3.7 (2.5–6.0)</td>
<td>3.1 (2.4–4.5)</td>
<td>4.0 (2.7–5.5)</td>
<td>0.02</td>
</tr>
<tr>
<td>Admission to hospital or ED length of stay &gt; 6 h</td>
<td>27 (31)</td>
<td>17 (19)</td>
<td>63 (33)</td>
<td>0.06</td>
</tr>
</tbody>
</table>

*Data are presented as No. or median (IQR).
For example, we found no association between obesity and chronic asthma severity, using several measures such as history of steroid use, hospitalizations, intubations, number of ED visits, and hospital admissions. Although data are sparse, if anything, prior studies suggest that obese participants with asthma reported increased severity of symptoms. Taken together, the medical literature does not support contentions that the asthma of obese individuals is of a lesser chronic severity or simply reflects misdiagnosed dyspnea. 

Although our study largely confirmed the absence of any important obesity-related differences, we did find that obese individuals had a higher initial percentage of predicted PEF at presentation to the ED. This finding was juxtaposed against a nonsignificant trend in tendency for obese subjects to rate their symptoms more severely and to use more inhaled β-agonists in the 6 h prior to ED presentation. Might obese persons have a heightened awareness of their bronchoconstriction? Or is this evidence for a relatively greater dyspnea unrelated to bronchoconstriction, as posited by some groups? The comparable response to bronchodilator therapy, with documented airway reversibility in all three BMI groups, provides real-world evidence against claims that many obese individuals do not have “true” asthma. At least in the ED setting—with > 2 million annual visits in the United States alone—the asthma exacerbations of obese individuals appear quite real and respond appropriately to bronchodilator therapy.

Several observations led us to explore potential confounding by sex of the obesity-PEF finding. As previously reported in another cohort of ED patients, we confirmed that women were almost twice common as men among ED patients with acute asthma, and that women presented with higher initial percentage of predicted PEF. In another prior publication by our group, we found that women were more likely than men to report severe asthma symptoms and activity limitations at similar levels of airflow obstruction. Since 77% of obese individuals in the current study were women, the potential for confounding was clear. Indeed, the relationship between obesity and initial PEF was due largely, but not entirely, to confounding by sex. In stratified analyses, the association between obesity and initial PEF was not significant in both men (p = 0.72) and women (p = 0.27), without evidence of any significant interaction by sex.

This study has some potential limitations. As in most prior studies, the temporal relation between obesity and asthma cannot be established using our study design. Although this issue was not our focus, we note that prospective studies of asthma incidence provide temporally correct evidence for a causal relationship. Our hypothesis, supported by other investigators, is that exercise intolerance from asthma does not lead to obesity. Beckett et al investigated the relationship between obesity and incident asthma in a prospective study of 4,547 participants followed up for 10 years, and concluded physical inactivity did not explain the association between a gain in BMI and incident asthma found in women. Additionally, Chen et al assessed the relation between energy expenditure and asthma in 16,813 patients, and determined that physical inactivity determined by energy expenditure failed to explain the relationship between obesity and asthma.

We focused instead on the remarkably high prevalence of ED patients with comorbid obesity and asthma. The addition of a nonasthmatic ED comparison group would have nicely complemented the national survey data since it is possible that ED patients, in general, are more obese; the prevalence of obesity is higher among individuals with a low socioeconomic status. However, the prevalence of obesity among ED patients with asthma was high regardless of socioeconomic status. Moreover, the presence or absence of additional comparison groups in no way detracts from our observation that three of every four ED patients with asthma are overweight or obese. Although these data may not be generalizable outside the acute setting, greater awareness of this public health problem might lead urgent-care staff to initiate a multidisciplinary approach for the prevention and treatment of both conditions.

In addition, because asthma is a common disease in the general population that still lacks a universally accepted definition, asthma overdiagnosis may have been common. Overdiagnosis of asthma among obese individuals would complicate interpretation of the apparent relationship between obesity and asthma. Although bronchoprovocation studies would be inappropriate in the ED setting, we provide evidence that clearly demonstrates comparable airway reversibility in all three BMI groups. This novel finding suggests, at least in the ED setting, that obese individuals with doctor-diagnosed asthma very likely have “asthma” by traditional definitions.

Another potential limitation of our study is reliance on self-reported height and weight to calculate BMI. Studies that have examined the accuracy of self-reported height and weight to determine overweight and obesity prevalence among adults have shown that this approach may lead to an underestimation of the prevalence in men and women. However, the relatively large sample size and almost complete absence of an association between BMI and several markers of chronic and acute asthma severity is consistent enough for us to believe that we have not missed any major associations.
CONCLUSION

In conclusion, our study supports the assertion that obese adults present to the ED with asthma exacerbations that are remarkably similar to those of nonobese adults. Compared to normal/underweight and overweight individuals, obese individuals were treated similarly and responded similarly to treatment. At least in the ED setting, these real-world data refute claims that the asthma of many obese individuals is not true asthma but instead an exaggerated dyspnea without evidence of airway hyperreactivity. The only significant difference was a higher initial PEF among obese patients, and this was due, in large part, to confounding by sex. Clearly, these are complex issues that merit further exploration. Future work might focus on the primary prevention of both obesity and asthma, and, for the millions that are currently afflicted, how co-management of these two conditions might improve outcomes. The ED setting provides an excellent opportunity for the enrollment of subjects with both obesity and moderate-to-severe persistent asthma. With additional research and a focused public health initiative, the ED could partner with other groups and play an important role in the control of both epidemic conditions.

ACKNOWLEDGMENT: We thank the MARC Investigators for their ongoing dedication to emergency airway research.

APPENDIX

EMNet Steering Committee

Edwin D. Boudreaux, PhD; Barry E. Brenner, MD, PhD; Carlos A. Camargo, Jr, MD (Chair); Rita K. Cybulka, MD; Theodore J. Gaeta, DO, MPH; Michael S. Radeos, MD, MPH.

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Keith Brinkley, MA; Carlos A. Camargo, Jr, MD (Director); Sunday Clark, MPH; Jennifer A. Emond, MS; Jessica L. Hohrmann, MPH; Sungbye Kim, MD (all at Massachusetts General Hospital, Boston).

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