Asthma control in obesity-associated asthma phenotype in East Croatia

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ABSTRACT

Aim To determine if obese asthmatics represent a distinct clinical phenotype than non-obese and the level of asthma control in obese asthmatics.

Methods The study was conducted in the pulmonary clinic of the Department of Pulmonary Diseases, Osijek University Hospital Centre during 201, on 201 outpatients with asthma, who came to a regular examination. Each patient underwent a clinical examination with an extensive anamnesis and lung auscultation. During the examination internally made questionnaire, spirometry, fractional exhaled nitric oxide (FeNO), Asthma Control Test and Asthma Control Questionnaire were used to estimate the level of asthma control, whereas the Hospital Anxiety and Depression scale was used to estimate the presence of mood disorder.

Results The severity of asthma was increased in obese patients. There was no significant difference in the number and frequency of hospitalizations in obese compared to non-obese asthmatics. Non-obese asthmatics had more frequent visits to their general practitioner/Emergency medical services (GP/EMS) during the last month compared to obese asthmatics, which is a new finding inconsistent to any study conducted before. Obese asthmatics had more symptoms and had them almost every day. Lung function of obese asthmatic was worse compared to non-obese, especially in women. Values of exhaled nitric oxide showed no significant difference for obese asthmatics. No significant difference in intake of corticosteroids or combination therapy was found. Obese patients with asthma had more frequent and multiple comorbidities.

Conclusion Results confirmed that obese asthmatics represent a different phenotype of asthma, more severe and poorly controlled.

Key words: depression, anxiety, severity, body mass index
INTRODUCTION

Recent literature suggests that asthma is different in the obese from the normal weight asthmatics. Obesity affects lung mechanics and has significant effects on asthma control and response to medication. These differences may justify adding a new phenotype – obesity-associated asthma (1). Studies of risk factors for adult asthma have concluded that increased body mass index is, among others, a significant risk factor for asthma, independent of gender and allergic status (2). Obesity has become a serious health problem due to changes in eating habits and decreased physical activity (3). Although asthma may promote weight gain through an increased sedentary life style and occasional use of oral corticosteroids, this does not fully explain the association of asthma and obesity in the majority of patients. Relative risk of asthma in obesity ranges from 1.0 to 3.5 (1). Obesity has been reported to be associated with asthma (6-8), but a convincing relationship between asthma and obesity has not been recognized yet. The physiological changes associated with obesity can contribute to respiratory symptoms, and these should be differentiated from those caused by asthma. Obesity can possibly influence the development of asthma through genetic, hormonal, neurogenic or mechanical influences (9). Recent investigations regarding genetic base for these two disorders have identified polymorphisms in specific regions of chromosomes 5q, 6p, 11q13 and 12q (8). Also, adipose tissue in obese people secretes regulatory adipokines, chemokines, acute phase proteins and energy regulating hormones including leptin (8). Elevated leptin levels in obese may be important to systemic inflammation (8,9). It is well known that C-reactive protein (9), IL-6 (10) and tumor necrosis factor-alfa (11) are increased in the plasma of obese patients. All adipokines have a proinflammatory effect, leading to an association between obesity and chronic inflammation. Adipokines may alter T helper 1 (Th1)–T helper 2 (Th2) balance, immune tolerance, lung development, airway smooth muscle and airway responsiveness, which are associated with asthma development. The mechanical effect of obesity on the chest and abdominal wall affects respiratory function, and may increase the work of breathing. That reduces lung volume and airway diameter and increases asthma (8). Smaller lung volumes and increased breathing frequency may cause an increase in airway smooth muscle shortening with enhanced airway hyperresponsiveness. Finally, obesity is a risk factor for obstructive sleep apnea and gastroesophageal reflux disease (12). Taken together, these data raise the possibility that the relationship between obesity and asthma may be mediated, at least by overlapping or interacting pathogenic mechanisms. Obesity may directly affect the asthma phenotype by mechanical effects including airway latching and increasing in airway resistance and responsiveness to methacholine (13). Obese asthmatics represent a specific asthma phenotype (14). It is associated with low lung volumes breathing and a less eosinophilic inflammatory process (15). Moreover, obese patients referred for severe asthma (16,17) often seem to have poorly controlled asthma (18,19) and show a reduced response to standard controller therapy (20-22). Obesity as a risk factor for difficult-to-control asthma increases the risk of incident asthma (19). This theory of a new phenotype includes the fact that weight loss is associated with a significant reduction of asthma symptoms (4,23). In the last few years, several asthma phenotypes have been described, but were not put into the use in everyday clinical practice. Guidelines for asthma therapy generally use a “one size fits all” approach, so that phenotypic details are not used in treating patients (2). This approach works well in the majority of patients. However, when an asthmatic reaches a specialist, it is often because the response to “commonly used asthma therapies” has not been adequate. A better understanding of the complexities of the disease and its presentation are required (24), as well as developing new therapies specifically targeted to this unique patient population – obesity-associated asthma phenotype.

The aim of this study was to determine if obese asthmatics represent a different phenotype of asthma than non-obese.

PATIENTS AND METHODS

A total number of 201 outpatients were included, obese and non-obese asthmatics, who came for a standard outpatient pulmonology visit in tertiary care in the pulmonary clinic of the Department of Pulmonary Diseases, Osijek University Hospital Centre. The data was collected between January 2012 and January 2013. At first, 284 patients in-
cluded, but 83 patients (29.2%) excluded who did not satisfy the required criteria. Participants were eligible if they had a newly physician-diagnosed asthma with airway responsiveness to metacholine 16 mg/mL, adults older than 18, obese (body mass index - BMI, over 30 kg/m²) or non-obese (BMI between 18.5–24.9 kg/m²) according to the criteria of the current international classification (25). The bronchoprovocation test to metacholin was considered positive if there was an increase in forced expiratory volume in 1 second (FEV₁) of more than 12% and more than 0.2 L or an increase in maximal expiratory flow at 50% (MEF₅₀) more than 25% and more than 0.2 L. The patients who were only overweight but not obese, with respiratory diseases other than asthma were excluded. Patients could have comorbidities such as rhinitis, nasal polyposis, sinusitis, pneumonia, diabetes or cardiovascular disease. Each patient was subjected to a detailed anamnesis and subjective symptoms were evaluated: daily and nocturnal asthma symptoms like cough, shortness of breath, wheezing, production of mucus and waking up or walking at night. Family and social history (living conditions, exposure to dust mites, molds, pets, tobacco smoke, level of education, employment, etc.) was recorded. Each respondent was asked about physical activity and possible inability to perform daily school or work obligations, and the data about regular medication use.

After evaluating the severity of the symptoms, a clinical examination with lung auscultation was made. Pulmonary function of asthma patients was measured by spirometry which was repeated three times. The evaluation was based on the best values of 3 acceptable attempts before, and 20 minutes after the application of salbutamol 0.4 mg through inhalation, which is equivalent to the standard test (26). Evaluated spirometry parameters were: forced vital capacity (FVC), forced expiratory volume in 1st second (FEV₁), ratio of forced expiratory volume and vital capacity x100 (Tiffeneau index) and the peak expiratory flow rate (PEF). Obstructive ventilatory disorder was considered if the results were expressed as the standard value in relation to the reference according to The European Community of Coal and Steel/European Respiratory Society (ECCS/ERS) standards (27). Furthermore, measurement of nitric oxide (NO) in exhaled air (FeNO) chosen as an objective parameter of eosinophilic inflammation in the airway, has been done in the Department of Pulmonology in Osijek University Hospital Center (Ninox Mino, Medisoft, Belgium) (28). FeNO test considered positive if the value was 25 ppb or more. After examination, spirometry and FeNO, each patient was given four self-assessment instruments, which were completed in a random order, and the respondents could consult the examiner about any ambiguities. General socio-demographic, psychosocial data and variables and data relevant for asthma phenotype were gathered using a questionnaire about general and psychosocial data, made internally for this study. The collected data include: age, gender, body mass index (BMI), employment and marital status, smoking, atopic status, socioeconomic status, atopy, family history of asthma, provoking factors, occurrence and duration of illness, deterioration, doctor visits or hospital services, hospitalization, earlier mental disorders and comorbid diseases, and drug use. Each patient was tested on subjective symptoms that make up a significant portion of the indicators of asthma control and satisfaction with the quality of life. We evaluated the day and night symptoms as follows: early morning cough, shortness of breath, wheezing, short or rapid breathing, chest pressure, creating sewage which is difficult to release and wake up and walk at night. Using the reported subjective symptoms of asthma and information of taking the recommended therapy, patients were classified into one of four groups of intensity, according to the International scale of intensity (2): intermittent asthma, mild persistent, moderate persistent and severe persistent asthma. Hospital Anxiety and Depression scale (HAD) (29) was used to measure anxiety and depression symptoms. It consists of 14 claims, 7 of which are related to anxiety and 7 to depression. The value 7 or less on the HAD scale was taken as normal, 8-10 indicated presence of anxiety and depression symptoms, and a value of 11 or more a definitive presence of anxiety and depression symptoms.

Two questionnaires were used for the evaluation of asthma control: Asthma Control Test-ACT (30) and Asthma Control Questionnaire (ACQ) - Juniper’s test (31). The ACT test includes five questions (shortness of breath, asthma control score by respondents, use of medications, activity limitations due to asthma and nocturnal asthma symptoms) for
getting the result on the scale of 5-25, and thereby gain insight into the disease and control measures needed for its improvement. The Asthma Control Questionnaire (ACQ) - Juniper’s test measures the adequacy of asthma treatment according to international guidelines. It consists of seven counts: five counts related to the symptoms, the use of a bronchodilator and a value of FEV₁% predicted compared to the norm value. All participants gave written informed consents. The research was approved by the Ethics in Research Committee of the Osijek University Hospital Center and research was conducted according to the Helsinki guidelines.

To describe numerical figures, basic measures of the medium and scattering was used. A normality of distribution of the observed numerical variables was tested by Kolmogorov-Smirnov’s test. Categorical variables were described in absolute and relative frequencies. Kruskal-Wallis’s test was used for comparing more than two independent groups. Differences between categorical variables were tested with χ²-test or Fisher’s exact test. For assessing the importance of the results, the level of the importance of alpha = 0.05 was chosen.

RESULTS

The study included 201 asthmatic participants, 126 (62.7%) of which were obese. Obese asthmatics were more frequently less educated, 40 (31.3%), compared to non-obese (p=0.028). Most of obese patients had moderate persistent asthma, 34 (27%) (p=0.046) compared to non-obese asthmatics, 11 (14.7%) (Table 1).

Table 1. Asthma severity according to Global Initiative for Asthma (GINA) guidelines

<table>
<thead>
<tr>
<th>Body Mass Index</th>
<th>No (%) of patients</th>
<th>Normal weight asthmatics</th>
<th>Obese asthmatics</th>
<th>Total</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asthma severity score</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Intermittent</td>
<td>8 (10.7)</td>
<td>21 (16.7)</td>
<td>29 (14.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild persistent</td>
<td>47 (62.7)</td>
<td>64 (50.8)</td>
<td>111 (55.2)</td>
<td>0.046</td>
<td></td>
</tr>
<tr>
<td>Moderate persistent</td>
<td>11 (14.7)</td>
<td>34 (27)</td>
<td>45 (22.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe persistent</td>
<td>9 (12)</td>
<td>7 (5.6)</td>
<td>16 (8)</td>
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</tr>
</tbody>
</table>

There was no significant difference in number and frequency of hospitalizations between obese and non-obese asthmatics, although obese asthmatics had asthmatic attacks almost every day (p=0.017). On the contrary, a larger number of non-obese asthmatics, 91 (59.5%) visited general practitioner/emergency medical services (GP/EMS) because of asthma during the last month (p=0.020) (Table 2).

Table 2. Frequency of asthma symptoms, number of hospitalizations, general practitioner/emergency medical services (GP/EMS) visits because of asthma during the last month and values of forced expiratory volume in 1st second (FEV₁)

<table>
<thead>
<tr>
<th>Body Mass Index</th>
<th>No (%) of patients</th>
<th>Frequency of asthma symptoms</th>
<th>Number of hospitalizations because of asthma</th>
<th>Visiting GP/EMS because of asthma during last month</th>
<th>FEV₁ before bronchodilator and FEV₁% predicted value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal weight</td>
<td>39 (60.8)</td>
<td>28 (58.3)</td>
<td>121 (60.2)</td>
<td>110 (54.7)</td>
<td>0.020</td>
</tr>
<tr>
<td>Obese</td>
<td>22 (39.2)</td>
<td>20 (41.7)</td>
<td>71 (39.8)</td>
<td>100 (45.3)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>61 (100)</td>
<td>48 (100)</td>
<td>192 (100)</td>
<td>210 (100)</td>
<td>0.020</td>
</tr>
</tbody>
</table>

Early morning or persistent cough, wheezing and chest pressure were more common in obese patients, but not statistically significant. Regarding the symptoms, in the group of obese asthmatics the most common symptom was creating a secretion difficult to expel (p=0.011). Waking up and walking at night was more present in non-obese asthmatics. Asthma interfered somewhat more with the daily activities of the obese in comparison to subjects with normal body weight, but this is not statistically significant (p=0.197).

Lung function was lower in obese asthmatics compared to non-obese. There were significantly lower values of FEV₁ before bronchodilator (p=0.042) and FEV₁% predicted values (p=0.009) in obese compared to non-obese asthmatics (Table 2). FEV₁ values before bronchodilator (p<0.001) and FEV₁% predicted values (p<0.001) were significantly lower in females. Comparing these two groups there was no difference in FeNO values. According to the ACQ test, more obese asthmatics had poor and very poor asthma control, but when comparing obese to non-obese asthmatics the difference was not statistically significant (p=0.036). According to the ACT test, uncontrolled asthma was more prevalent in obese asthmatics, but the differen-
ce was not significant (p=0.014). In the group of obese asthmatics, ACT test differed significantly between genders. Statistically more females 16 (47.1%) had uncontrolled asthma compared to males (p=0.014) (Table 3).

### Table 3. Asthma control according to Asthma Control Test (ACT) and gender

<table>
<thead>
<tr>
<th>Asthma control – ACT test</th>
<th>No (%) of patients</th>
<th></th>
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<tbody>
<tr>
<td></td>
<td>Obese asthmatics</td>
<td></td>
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<tr>
<td></td>
<td>Males Females Total p</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Controlled</td>
<td>2 (14.3) 11 (22.4) 13 (27.1)</td>
<td></td>
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</tr>
<tr>
<td>Good</td>
<td>9 (64.3) 7 (20.6) 16 (33.3) 0.014</td>
<td></td>
<td></td>
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<tr>
<td>Uncontrolled</td>
<td>3 (21.4) 16 (47.1) 19 (39.6)</td>
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</table>

There was no difference in treatment due to weight. Inhaled corticosteroids were used more frequently in the group of non-obese asthmatics and oral bronchodilators in the group of obese, but not significantly (p=0.359).

According to comorbidities, significantly more obese asthmatics had diabetes (p=0.013), hypertension (p<0.001) and gastroesophageal reflux (p=0.031) (Table 4). According to the results of HAD scale, anxiety was more prevalent in obese asthmatics, 22 (45.8%) than in non-obese, 56 (36.6%). Depression as comorbidity, ranked high second place and it was significantly more prevalent in obese asthmatics, 17 (35.4%) than in non-obese, 27 (17.6%) (p=0.009).

### Table 4. Comorbidities in obesity-associated asthma

<table>
<thead>
<tr>
<th>Comorbidity</th>
<th>No (%) of patients</th>
<th>Body Mass Index</th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Normal weight asthmatics Obese asthmatics Total p</td>
<td></td>
<td></td>
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<tr>
<td>Diabetes</td>
<td>4 (2.6) 6 (12.5) 10 (5) 0.013</td>
<td></td>
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<tr>
<td>Arterial hypertension</td>
<td>32 (20.9) 25 (52.1) 57 (28.4) &lt;0.001</td>
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</tr>
<tr>
<td>Gastroesophageal reflux disease</td>
<td>8 (5.2) 7 (14.6) 15 (7.5) 0.031</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nasal polyps</td>
<td>15 (9.8) 5 (10.4) 20 (10) 0.902</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Sinusitis</td>
<td>17 (11.1) 9 (18.8) 26 (12.9) 0.216</td>
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<tr>
<td>Rhinitis</td>
<td>25 (16.3) 9 (18.8) 34 (16.9) 0.665</td>
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</table>

In both groups, an equal number of respondents were smokers. But if we look only at the group of obese asthmatics, males smoked significantly more cigarettes per day (p=0.012).

Most respondents had had asthma for more than 5 years, but it started after they had reached their 30-ties and there was no family history of asthma in those patients (Table 5).

### Table 5. Obesity-associated asthma – late onset in non-atopic individuals

<table>
<thead>
<tr>
<th>No (%) of patients</th>
<th>Body Mass Index</th>
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<th></th>
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</thead>
<tbody>
<tr>
<td>Age of asthma onset</td>
<td>Normal weight asthmatics Obese asthmatics Total p</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;12</td>
<td>29 (38.7) 17 (13.5) 46 (22.9)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>12 – 18</td>
<td>6 (8) 11 (8.7) 17 (8.5) 0.001</td>
<td></td>
<td></td>
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<tr>
<td>19 – 30</td>
<td>14 (18.7) 28 (22.2) 42 (20.9)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 30</td>
<td>26 (34.7) 70 (55.6) 96 (47.8)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family history of asthma</td>
<td>Yes 34 (45.3) 36 (28.6) 70 (34.8) 0.021</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>No 41 (54.7) 90 (71.4) 131 (65.2)</td>
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</table>

### DISCUSSION

In this study, among 201 asthmatics recruited, the greater part (62.7%) were obese. The height and weight for all respondents was measured to get the correct BMI result to avoid classification bias and did not rely on self-reported measurements (25). It is not the best measure of adiposity, particularly if looking at the effects of obesity on the lungs, because there may be some gender differences between muscle and fat distribution, so maybe it is better to use alternative measures of abdominal adiposity to look at the effects on pulmonary function. The BMI was used only for practical purposes because BMI is by far the most commonly used measure of obesity (25).

Obesity-associated asthma has been reported more in women (2,5,24) in most studies, but not in all (26). According to our results, obesity-associated asthma was not more prevalent in women, it was nearly the same in both obese and non-obese women.

Most clinical trials found that obesity changes the severity of asthma, meaning that the asthma severity increases with increasing BMI (2). According to our results, most of the obese patients had moderate persistent asthma. Asthma incidence and frequency of hospital admissions for asthma are higher among women compared to men; women report more symptoms, especially dyspnea and have more bronchial hyperresponsiveness, and in general have more severe asthma than men (32,33).

We did not confirm a significant difference in number and frequency of hospitalizations of obese asthmatics, but our results showed that non-obese asthmatics visited GP/EMS because of asthma during the last month significantly more.
than obese patients. We did not find any similar data in studies conducted so far.

According to the results of HAD scale, obese asthmatics are more depressive than non-obese. That could be the reason why obese asthmatics visited GP/EMS far less than non-obese asthmatics. Because of their depression, they could be less motivated for taking care of their asthmatic problems. Obesity and depression together perhaps make them more tired or just lazier to note their symptoms.

We monitored the patients using of short-acting beta-agonist (SABA) with ACT and Juniper’s test and we noticed that obese use SABA less frequent than non-obese asthmatics, although they have more severe symptoms. This result supports our theory that obese asthmatics are less interested or too tired to take proper care of their illness.

The most common symptom in obese asthmatics was creating mucus difficult to expel, which can be explained through mechanical effects of obesity on lungs. This result also confirms that asthma in non-atopic adults is likely caused by obesity and its effects.

Bronchial asthma is characterized by reversible airway obstruction and obesity has various effects on lung function (34), which is lower and worsen in obese patients (35). The primary effect of obesity on lung function is a reduction in functional residual capacity (FRC) and a reduced expiratory reserve volume (ERV) (1,36). These reductions are detectable even with a modest increase in weight and that occurs because of chest wall restriction. Obesity also causes a reduction of both FEV₁ and FVC with a preserved FEV₁/FVC ratio (14). Also, asthmatics have higher breathing frequencies and reduced tidal volumes (7), reduction in lung volumes and increased oxygen cost of breathing (36,37). It has been previously reported that obese non-asthmatics have lost the protective effect of a deep inspiration observed in non-obese non-asthmatics (37). Although it is noticed that lung volumes are reduced and asthma symptoms are increased in obesity, airflow obstruction and airway hyper responsiveness are not altered (4). These results are consistent with ours.

Obesity has a significant effect on asthma control. Our results showed that obese asthmatics have poorer asthma control than non-obese, which is consistent with findings of Lavoie (18) and Saint-Pierre (19). Several investigations found poorer control of asthma in obese despite good medication (20-22). Taylor (16) showed that obese individuals with asthma had more severe symptoms and increased medication use, and Vortmann and Eisner (37) found that obese asthmatics had increased symptoms and decreased quality of life but not increased emergency health care utilization. Mosen (17) showed that obesity had significant adverse effects on symptoms, medication use, and quality of life, and that obese individuals with asthma had a 4.6-fold increased risk of hospitalization for asthma compared with non-obese individuals with asthma.

Poor asthma control in obese individuals could have been due to their having an altered perception of asthma symptoms. However, Lessard (14) found no significant difference in asthma symptom perception between individuals in both groups with the same degree of bronchoconstriction. A different phenotype of asthma, reduced response to treatment, or more severe asthma could explain the poorer asthma control in obese asthmatics. Kajbaf (38) found a statistical association between obesity and the prevalence of current wheezing, night cough and exercise-induced wheezing. The main goal of asthma treatment is to achieve adequate control of the disease, as reflected by minimal symptoms and rescue bronchodilator use, no nighttime symptoms, normal daily activities, rare and mild exacerbations and optimal pulmonary function (39). Reduced asthma control may be related to the effects of obesity in itself on asthmatic airways, to the contribution of obesity-related comorbid conditions, or reduced response to medication. Obesity causes reduced response to asthma medications such as inhaled corticosteroids (ICS) and to a combination of ICS and a long-acting bronchodilator (21,22) and worsens asthma control with theophylline (20). The mechanisms underlying this reduced response to various medications remain to be determined. An obvious therapeutic intervention that should be evaluated is weight loss, which improves asthma symptoms and lung function (as measured by FEV₁, FVC, and peak flow), spirometric values, asthma control and reduce its severity, as assessed mainly by medication needs (2,35) but not necessarily in airflow obstruction or airway hyperresponsiveness (23). A better understanding of the pathogenesis of asthma in the obese is needed to improve therapies for this population.
Asthma and obesity are often related to comorbid conditions such as gastroesophageal reflux (12) and sleep apnea which can result in dyspnea and wheezing and might be mistaken for asthma by patients and clinicians (40,41). These results are consistent with ours.

An elevated BMI has been proposed as a risk factor for atopy, the major risk factor for asthma. However, studies show that association between asthma and obesity is not mainly mediated through atopy – association was found among subjects with both positive and negative skin-prick results (42). A family history of asthma was a significant risk factor (43). Our results confirm the theory of later onset of asthma in the non-atopic individual which is likely caused by obesity.

The association between asthma and smoking is unclear. Several studies have shown smoking at any time or ex-smoking status to be associated with asthma (44). It is not clear whether or not the subjects experienced their onset of asthma before or after they had stopped smoking. One study found only ex-smoking status to be a significant risk factor for asthma.

FUNDING
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TRANSPARENCY DECLARATIONS
Competing interests: none to declare.

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Kontrola astme u fenotipu astme povezanom s debljinom u istočnoj Hrvatskoj

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SAŽETAK

Cilj Odrediti pripadnost pretilih astmatičara u poseban klinički fenotip u odnosu na astmatičare normalne tjelesne mase i razinu kontrole astme u pretilih astmatičara.

Metode Istraživanje je provedeno na 201 bolesniku s astmom, koji su se javili na redovni pregled u pulmološku ambulantu Odjela za pulmologiju Kliničkog bolničkog centra Osijek, tijekom 2012. godine. Svaki ispitanik podvrgnut je detaljnom kliničkom pregledu s opsežnom anamnezom i auskultacijom pluća. Tijekom ispitivanja korišten je intern apotrof ren upitnik, spirometrija, test frakcije izdahnutog dušikovog oksida, test kontrole astme, upitnik o kontroli astme za procjenu kontrole bolesti, te hospitalna ljestvica anksioznosti i depresije za određivanje prisutnosti poremećaja raspoloženja.


Zaključak Rezultati potvrđuju da pretili astmatičari čine zaseban fenotip astme, teži i lošije kontrolirani.

Ključne riječi: depresija, anksioznost, težina, indeks tjelesne mase