HYPERSENSITIVITY TO AEROALLERGENS IN CHILDREN WITH ATOPIC DERMATITIS

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SUMMARY – Atopic dermatitis (AD) is a chronically relapsing, inflammatory skin disease characterized by severe itch, rash and dry skin. Hypersensitivity to aeroallergens is found in 40%-50% of children with AD and it is the cause of intensive skin lesions. The aim of the study was to assess the presence of hypersensitivity to aeroallergens in AD children. The study included 114 children (56 boys and 58 girls), median age 27.5 months, who had been diagnosed with AD according to Hanifin and Rajka criteria. The severity of the disease was assessed by the SCORAD index. To recognize hypersensitivity to aeroallergens, the following parameters were analyzed: medical history, values of absolute eosinophil count, total IgE antibodies, specific IgE antibodies to aeroallergens, and results of the skin prick test for aeroallergens. A moderate form of the disease was present in 61.4% of study children, with a median SCORAD index score of 28.5 points; 12.3% of study children showed hypersensitivity to aeroallergens (history of hypersensitivity to aeroallergens in 27.2%, increased absolute eosinophil count in 53.5%, increased total IgE antibodies in 56.1%, positive skin prick test in 20.2%, and positive specific IgE antibodies to aeroallergens in 12.3% of children). The most common aeroallergens responsible were house dust in 6.1% and *Dermatophagoides pteronyssinus* in 3.5% of children with AD. Hypersensitivity to aeroallergens was recorded in the same number of children with mild and severe forms of the disease and in 5.7% of children with a moderate form. In conclusion, the presence of hypersensitivity to aeroallergens in children with AD is significant. By discovering and removing the aeroallergens responsible, it is possible to achieve reduction in the intensity of skin lesions and frequency of exacerbations.

Key words: *Atopic dermatitis; Children; Hypersensitivity; Aeroallergens*

Introduction

Atopic dermatitis (AD) is a chronic, relapsing, inflammatory skin disease caused by genetic predisposition (atopic) and characterized by severe itch and dry skin, with typical skin lesions depending on the age of the child and the severity of the disease[1]. In infants, skin lesions are exudative with erythema, vesicles and crusting, and are most often localized on the face, neck, trunk and extensor areas of the extremities. After infancy, dry skin lesions are most frequently erythematous based lichenification and excoriation, and are more common on the dorsal and flexor regions of the extremities[1,2].

AD is also the most common skin disease in childhood; the number of affected children is constantly rising, its rate having reached 10% to 20% in developed countries[3,4]. It rarely occurs in the first six weeks after birth, and the median of disease onset is around the third month of life in 47.5%-100% of cases. It occurs by age 5 in 75% of these children, with a higher prevalence in boys than girls, with the male to female ratio of 1.3-1.5:1[5]. In only 10% of those suffering from AD it begins after age five[6]. A mild or moderate form of the disease is present in most affected children (84%), whilst the severe form of the disease is present in 2% of children under age five[7].

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Received March 29, 2012, accepted September 12, 2012

The disease is one of the initial allergic manifestations characteristic of the typical sequence of clinical signs of atopic disease known as the "atopic march". Actually, it most often occurs as the first illness in the atopic march, since a significant number of children with AD later develop asthma (40%-50%) and allergic rhinitis (80% of children)[8].

Opinions on the actual role of aeroallergens in the pathophysiology of AD still differ, although research shows that in 40%-50% of children with AD positivity is present to at least one aeroallergen and these children more often have associated respiratory atopy[9-12]. It has been proven that exposure to aeroallergens (through direct contact with aeroallergens on the skin or by inhalation) significantly increases the risk of occurrence of the first skin lesions of AD, but also deterioration of the existing ones[13,14]. The aeroallergens most frequently responsible for hypersensitivity in children are house dust, *Dermatophagoides pteronyssinus,* animal hair and pollens[15,16]. A great deal of research has shown that removal of the responsible aeroallergens from the child's environment leads to improvement in skin lesions in a significant number of children with AD[15-17].

The discovery that hypersensitivity to aeroallergens has an important role in the occurrence and maintenance of the pathological process in the skin of children with AD has led to awareness of the necessity of recognizing the allergens responsible, so that avoiding them precisely would contribute to reduction in the intensity of skin lesions, lower number of exacerbations and longer remission of the disease.

Therefore, the aim of this study was to assess the presence of hypersensitivity to aeroallergens in children with AD.

Subjects and Methods

This prospective study was conducted at the University Department of Pediatrics, Tuzla University Clinical Center (UCC) in Tuzla, in the period from December 1, 2009 to June 30, 2010. The inclusion criteria were meeting at least three major and three minor criteria according to Hanifin and Rajka[18] diagnostic criteria for AD, and being up to 15 years of age on the day of testing. The exclusion criteria were as follows: not meeting the diagnostic criteria, age above 15 years, use of antihistamines (e.g., cetirizine, loratadine, desloratadine) in the previous 5 days, use of systemic or local corticosteroids (e.g., methylprednisolone, alclometasone dipropionate) in the previous 4 weeks, acute and/or chronic disease without an atopic basis, an associated systemic disease, and refusal of the parents for their child to be included in the study; 114 children met the conditions for the study.

The following parameters were analyzed: age and gender of the children; age of the children when first skin lesions of AD appeared; severity of the disease assessed by the Severity Scoring of Atopic Dermatitis (SCORAD) index[19]; data on hypersensitivity to aeroallergens; personal and family history of atopy; values of total immunoglobulin (Ig)E antibodies, specific IgE antibodies to aeroallergens; and results of the skin prick test (SPT) for aeroallergens. The SCORAD index was used to assess the severity of the disease (Table 1).

Assessment included (A) spread and extent of skin lesions expressed in a range of 0-100. The intensity of skin lesions (B) evaluated on a 0-3 scale, where 0 signified absence of skin lesions, 1 mild skin lesions, 2 skin lesions of moderate intensity, and 3 skin le-

Table 1. SCORAD index parameters

Spread of changes		Intensity of changes	Subjective signs
	(A)	(B)	(C)
In front and back area:	Head Neck Upper extremities Trunk Genitals Lower extremities	Erythema Edema/papules Crust Excoriations Lichenification Dry skin	Scratching Interrupted sleep (due to scratching for the previous three days and nights)

sions of severe intensity. Subjective signs (C) were presented by a 0-10 scale, where 0 denoted subjective assessment "never better" and 10 "never worse". The children themselves (in case of older children), or the child's care-giver expressed the intensity of subjective symptoms. The SCORAD index score was calculated according to the formula A/5+7B/2+C. The maximum SCORAD index score is 103. According to the SCORAD index scores, the severity of disease was assessed as mild (<15 points), moderate (15-40 points), or severe (>40 points)[19].

Testing for aeroallergens by use of SPT was undertaken in the allergy testing clinic of the University Department of Pediatrics, Tuzla UCC. Testing was performed for groups of aeroallergens (grass pollen, weed pollen, tree pollen, house dust, *Dermatophagoides pteronyssinus*, animal hair and skin, feathers, vegetable fibers, cloth, fungus, bacteria) using a dialyzed extract of the allergen dissolved in a solvent, which is a mixture of 50% of glycerol solvent in a buffer physiological solvent (Immunological Institute, Zagreb, Croatia). Urtica of >3 mm was considered a positive test result.

The absolute eosinophil count was determined in peripheral blood. The smear was dyed according to May-Grünwald stain, the number of eosinophils calculated *per* 100 leukocytes, and the number reached was multiplied by the total number of leukocytes in the patient. An eosinophil count of ≥450x10^6/L was considered a pathologic result.

The blood for determination of total values of IgE antibodies and specific IgE antibodies to aeroallergens was obtained by the standard procedure and analyzed at the Polyclinic of Laboratory Diagnosis, Department of Immunology, Tuzla UCC. Blood samples were centrifuged at 2000 rpm for 10 minutes. In order to establish total IgE antibody count, the samples were analyzed over 24 hours by obtaining samples and using immunonephelometry method with a nephelometer (Dada Behring, Marburg, Germany). The values of total IgE antibodies 0-100 IU/mL were considered normal.

Specific IgE antibodies to aeroallergens (taking the most common aeroallergens, i.e. house dust, *Dermatophagoides pteronyssinus*, animal hair, grass pollen and weed pollen) and to aeroallergens for which the children showed positive SPT were determined. Specific IgE antibodies were also determined for aeroallergens which, according to their medical history, were suspected of being responsible for the hypersensitivity. The separated serum was stored at -80 °C until the procedure had been conducted on all samples. Determination was performed using the enzyme linked immunosorbent assay (ELISA), with Hy Tec 288 Plus apparatus from Agilent Technologies Company, Biomedica. Values >0.35 IU/mL were considered positive.

The study protocol was approved by the Ethics Committee of Tuzla UCC, no. 01/1-37-492/10.

Statistical analysis

Statistical data analysis was conducted using the MedCalc for Windows, Version 114.4 biomedical software. Numerical data were shown by the central tendency measure and the relevant dispersion measure. The variables with distorted distribution were shown with median as a measure of the central value and interquartile range

Results

In the period from December 1, 2009 to June 30, 2010, 126 (19.5%) of 646 children examined and/or hospitalized at specialized outpatient clinic or Department of Allergology, Immunology with Rheumatology, University Department of Pediatrics, Tuzla UCC, were found to have AD. Twelve children with AD were excluded from the study: seven were using antihistamines, three had received systemic corticosteroids, one child had an associated systemic ailment, and for one child written consent was not given for inclusion in the study.

In 114 AD children, median age was 27.5 months (interquartile range: 12.5-66 months), minimum 1.5 months and maximum 14.9 years. There were 56 (49.1%) boys and 58 (50.8%) girls.

The most common major criteria were: itchy feeling on the skin and scratching, recorded in 109/114 (95.6%), typical localized skin lesions in 98/114 (85.9%) (on the face in 37.7%, on the face and skin folds of the neck in 9.6%, on the face and upper extremities in 22.8%, on lower extremities in 14.9%, and on the trunk in 10.5%), and chronic skin lesions in 88/114 (77.2%) children with AD. Positive fam-

ily medical history of atopic diseases was recorded in 29.8% and existence of an associated atopic disease (asthma in 7 and allergic rhinitis in 16 children) in 23/114 (20.2%) children with AD.

Analysis of minor criteria revealed that the largest number of AD children had dry skin (103/114; 90.3%) and deterioration of skin lesions caused by emotional factors (94/114; 84.4%). Early age at onset of skin lesions (before 2 years of life) was recorded in 92/114 (80.7%) children, with median age at the appearance of first lesions at 6 months (interquartile range: 3-12.5, minimum 0.5, maximum 48 months). Thirty-one of 114 (27.2%) of AD children had a positive history of the appearance and/or deterioration of skin lesions from AD after exposure to environmental factors.

The median SCORAD index value in AD children was 28.5 points (interquartile range 17.4-38 points), minimum 4.7 and maximum 102.8 points. The largest number of AD children had a moderate form of the disease (70/114; 61.4%), whilst there was an almost equal number of children with a mild form (23/114; 20.2%) and severe form (21/114; 18.4%) of the disease.

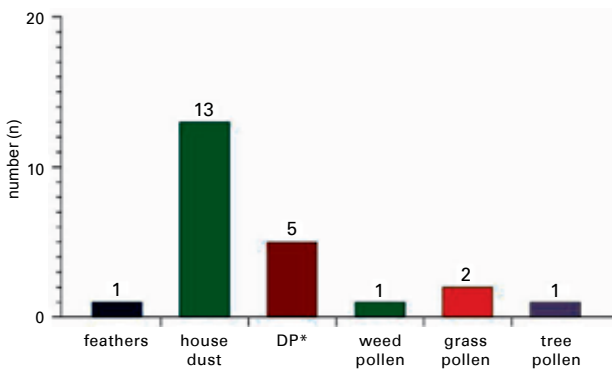
The absolute eosinophil count in 114 AD children ranged from 10 to 1230x10^6/L, mean ± SD 620±264.66x10^6/L. This parameter was increased in 61/114 (53.5%) AD children. Increased values of total IgE antibodies, median 288.5 IU/mL (interquartile range: 42-730 IU/mL), minimum 7.6 and maximum 3280 IU/mL were found in 64/114 (56.1%) AD children.

Positive SPT to aeroallergens was recorded in 23/114 (20.2%) AD children. The most common aeroallergens that produced positive SPT were house dust in 13/114 (11.4%) and *Dermatophagoides pteronyssinus* in 5/114 (4.4%) cases. The number of positive SPT to aeroallergens is shown in Figure 1.

Analyzing SPT results in AD children according to the form of disease, we found positive SPT to aeroallergens in 6/23 (26.0%) children with mild form (house dust in three, *Dermatophagoides pteronyssinu* in two, and feathers in one), 12/70 (17.1%) children with moderate form (house dust in eight, *Dermatophagoides pteronyssinus* in two, grass pollen in one, and weed pollen in one), and 5/21 (23.8%) children with severe form of the disease (house dust in two, *Dermatophagoides pteronyssinus* in one, tree pollen in one, and grass pollen in one).

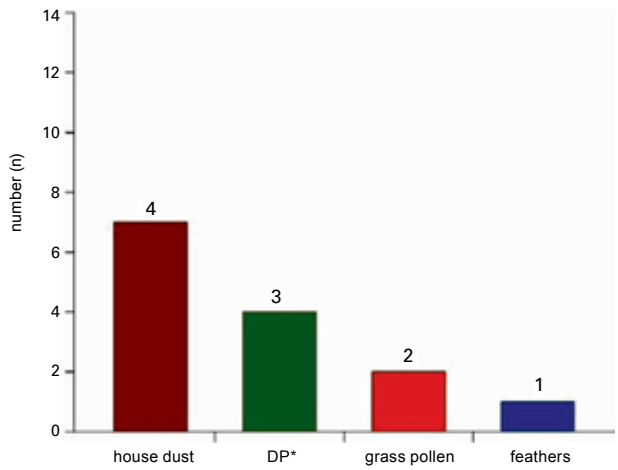
Positive finding of specific IgE antibodies to aeroallergens was noted in 14/114 (12.3%) AD children. The most common aeroallergens that produced positive SPT were house dust in 7/114 (6.14%) and *Dermatophagoides pteronyssinus* in 4/114 (3.5%) cases (Fig. 2).

Analysis of the findings of specific IgE antibodies to aeroallergens in study children according to form of disease revealed positive results in 5/23 (21.7%) children with mild form (house dust in two, *Dermatophagoides pteronyssinus* in two, and feathers in one), 4/70 (5.7%) children with moderate form (grass pollen in two and house dust in two), and 5/21 (23.8%) children with severe form of the disease (house dust in three and *Dermatophagoides pteronyssinus* in two).



**Dermatophagoides pteronyssinus*

Fig. 1. Prevalence of positive skin prick test for aeroallergens.



**Dermatophagoides pteronyssinus*

Fig. 2. Prevalence of positive findings of specific IgE antibodies to aeroallergens.

Hypersensitivity to aeroallergens was found in 14/114 (12.3%) AD children. These children had a positive history of hypersensitivity to aeroallergens, elevated absolute eosinophil count, elevated total IgE antibody values, positive SPT for aeroallergens and positive values of specific IgE antibodies to aeroallergens. The most common aeroallergens responsible were house dust in 7/114 (6.14%) and *Dermatophagoides pteronyssinus* in 4/114(3.5%) cases, whilst hypersensitivity to grass pollen was detected in 2/114 (1.7%) cases and to feathers in one child with AD. No hypersensitivity to aeroallergens could be demonstrated in 100/114 (87.7%) AD children.

Upon analyzing the presence of hypersensitivity to aeroallergens in AD children according to the form of the disease, we demonstrated hypersensitivity in an equal number of children with mild 5/23 (21.7%) and severe forms 5/21 (23.8%) of the disease, whilst hypersensitivity to aeroallergens was found in only 4/70 (5.7%) children with a moderate form of the disease. Numerical relationship between the positive and negative results of the parameters tested for assessment of the presence of hypersensitivity to aeroallergens in AD children according to the form of the disease is shown in Table 2.

House dust was the aeroallergen most frequently responsible for hypersensitivity in AD children according to the form of the disease. We found hypersensitivity to this aeroallergen in two children with a mild and moderate form each and in three children with a severe form of the disease.

Discussion

Over a 7-month period, we assessed the presence of hypersensitivity to aeroallergens in AD patients analyzing their history of hypersensitivity to aeroallergens, absolute eosinophil count, total IgE antibodies, specific IgE antibodies to aeroallergens, and SPT results.

Atopic dermatitis is a disease with a constantly growing prevalence and the number of children affected has tripled in the past three to four decades[3]. It seems that changes in lifestyle are one of the significant causes of the so-called "epidemic of atopic diseases". That is to say, over the last few decades, a lifestyle has been present which assumes a "hygienically cleaner environment", the microbe environment has changed, children are significantly less exposed to infective agents, and as a result the vital natural stimulation of the immune system is lacking at all levels[20,21]. Of the atopic diseases, AD manifests earliest, followed by allergic rhinitis and asthma[11,12]. This atopic march is the natural history of atopic manifestations, characterized by typical gradual appearance and progression of clinical signs of atopic diseases, whereby some signs become more expressed than others[9-12].

Table 2. Numerical relationship of positive and negative results of the parameters for assessment of hypersensitivity to aeroallergens in children with atopic dermatitis

Parameters of hypersensitivity to aeroallergens	Children with atopic dermatitis according to the form of the disease											
	Mild form (n=23)				Moderate form (n=70)				Severe form (n=21)			
	Present		Absent		Present		Absent		Present		Absent	
	n	%	n	%	n	%	n	%	n	%	n	%
Positive history of hypersensitivity	8	34.8	15	65.2	16	22.8	54	77.2	7	33.3	14	66.7
Increased absolute eosinophil count	13	56.5	10	43.5	28	40	42	60	20	95.2	1	4.8
Increased total IgE* antibodies	17	73.9	6	26.1	33	47.1	37	52.9	14	66.7	7	33.3
SPT** for aeroallergens	6	26.1	17	73.9	12	17.2	58	82.8	5	23.8	16	76.2
Specific IgE antibodies for aeroallergens	5	21.7	18	78.3	4	5.7	66	94.3	5	23.8	16	76.2

*IgE = immunoglobulin E; **SPT = skin prick test

Research has shown that children with AD (who have a positive family history of atopy, early age at onset of the first skin lesions, hypersensitivity to allergens, and a more severe form of the disease) have a high risk of developing asthma and allergic rhinitis[22,23]. In our study, we found associated atopic diseases in 20.2% of AD children (7 children had asthma and 16 allergic rhinitis). These children had the moderate or severe form of the disease. None of the children with a mild form of the disease had any associated atopic disease. Gustafsson *et al.*[23] report similar results as part of a longitudinal study in which 94 children with AD were monitored over seven years. Children with the mild form of AD did not develop asthma or allergic rhinitis in this period. In 84 children, improvement of skin lesions occurred, but 43% of the children suffered from asthma and 45% from allergic rhinitis. Illi *et al.*[24] found associated manifestations of AD and breathing difficulties in 56% of children, and Carmi *et al.*[25] found associated asthma and allergic rhinitis in 20.3% of children with AD.

Direct contact with the skin, and intranasal or intrabronchial introduction of aeroallergens may lead to the appearance of the first or deterioration of the existing skin lesions of AD[11,15-17]. After patch application of aeroallergens, eczematous skin lesions appeared on the previously unchanged skin in 30%-50% of children with AD[26]. Our results are in line with this: 31 of 114 (27.2%) AD children had a positive history of the appearance and/or deterioration of skin lesions from AD after exposure to environmental factors. In normal skin, aeroallergens do not succeed as easily in reaching the antigens in the presenting cells, whilst, due to the disturbed skin barrier, aeroallergens are able to penetrate the skin of children with AD more easily and in greater quantities, which initiates an immune reaction[9,10,27].

We noticed increased absolute eosinophil count in 61/114 (53.5%) AD children. This result was expected because an increased number of eosinophils is one of the important characteristics of AD, since eosinophils participate by their potent inflammatory function in the occurrence and maintenance of skin lesions in AD[28,29]. Similarly, Kaczmarski *et al.*[30] found increased absolute eosinophil counts in 87% of AD children, and Ogbuanu *et al.*[31] in 56.2% of AD children.

A significant number of children with AD (80%) have high values of total IgE antibodies, although some children (20%) have normal values[15,32]. The presence of increased values of total IgE antibodies in the first years of the child's life is seen as an important risk factor for the occurrence of AD precisely at that time of life[33]. Higher total IgE antibody values are more frequent in younger children, but in older children with AD, higher total IgE antibody values are present when it is a case of a persistent form of skin lesions and when the lesions are present over a larger area of the body[33,34]. We noticed increased total IgE antibody values in 56.1% of AD children, whilst other researchers report somewhat different results. Illi *et al.*[24] found increased total IgE antibody values in 35.7% of AD children, whilst Pourpak *et al.*[35] found them in 71.2% of AD children.

When suspicion exists of the presence of IgE antibodies caused by hypersensitivity, SPT is a reliable method for recognizing the allergen to which the child is hypersensitive, and against which the child will create specific IgE antibodies. Skin prick test is the most often performed test because of its simplicity, safety and fewer false-positive results. Severe reactions in prick testing are extremely rare[36]. These tests indicate the existence of allergen specific IgE antibodies, whilst a negative SPT essentially confirms the absence of IgE antibodies caused by hypersensitivity, with a negative predictive value of >95%[37]. In recognizing hypersensitivity to aeroallergens, SPT has significant sensitivity (68%-100%), but lower specificity (33%-71%)[38,39]. Research has shown that the most frequent aeroallergens to which AD children have positive SPT are house dust and *Dermatophagoides pteronyssinus*[37,40]. Our results are also in line with this, as the most frequent aeroallergen to which the AD children showed positive SPT was house dust (11.4%), followed by *Dermatophagoides pteronyssinus* (4.4%) and pollen (3.49%). Different results have been reported by Kam-Lun[41]: in children with AD, the SPT findings were positive for *Dermatophagoides pteronyssinus* in 25%, *Dermatophagoides farinae* in 17% and house dust in 8% of cases, whereas none of the AD children in his study had positive SPT for cat or dog hair. In our study, none of the children had positive SPT for animal hair either, although just animal hair is often thought to be the aeroallergen responsible for hypersensitivity in children with AD[11]. Over seven-year monitoring, Gustafsson *et al.*[23] found positive SPT to

at least one aeroallergen in 80.7% of AD children tested. Samochocki *et al.*[42] report on positive SPT to dust, grass pollen and cat hair in 52.3%, 32.1% and 35.8% of children, respectively. Raos *et al.*[40] found positive SPT for *Dermatophagoides pteronyssinus* in 43.9%, for *Dermatophagoides pteronyssinus* and various pollen in 45%, and for pollen alone in 7.7% of AD children.

House dust (mites and cockroach allergen) are also the most responsible aeroallergens in children with asthma and allergic rhinitis. Out of 140 children included in their study (98 with asthma, 36 with allergic rhinitis and 8 children suffering from both diseases), Salehi *et al.*[43] found 36 children with positive SPT to these aeroallergens.

Aeroallergens related to specific IgE antibodies cause the release of histamines and other inflammatory mediators, which results in tissue damage. Precisely histamine contributes to exacerbation of the cycle of itch and skin scratching, which contributes to deterioration of AD skin lesions[10,44].

The sensitivity of specific IgE antibodies in demonstrating hypersensitivity to aeroallergens is significant, ranging from 70% to 100%, with lower specificity of 40%-75%[38]. Since the positive predictive value is >95%, a positive finding of specific IgE antibodies to aeroallergens practically confirms hypersensitivity to the tested allergen and may, along with SPT, be used as a reliable parameter in recognizing the existence of hypersensitivity[43]. Research has shown that specific IgE antibodies to aeroallergens are found in 40%-50% of children with AD[11,45,46]. Our results are slightly different, but expected, in view of the fact that determination of specific IgE antibodies was undertaken for a small number of aeroallergens. So, we found positive result for specific IgE antibodies to aeroallergens in 12.3% of AD children, and the most common aeroallergens were house dust in 6.1% and *Dermatophagoides pteronyssinus* in 3.5% of children. Similar results have been reported by Platts-Mills *et al.*[46], who found positive specific IgE antibodies to one or more aeroallergens in 12.7% of AD children, with antibodies to house dust detected in 5.6% of children. Shefer *et al.*[47] report on specific IgE antibodies to *Dermatophagoides pteronyssinus* detected in 14.7% of children.

We demonstrated hypersensitivity to aeroallergens in 12.8% of AD children. We explain this result by the age of the study children with AD. Their median age was 27.5 months and it has been shown that the role of aeroallergens in the appearance and/or deterioration of skin lesions from AD is more pronounced in children aged 2-5 years and in adults, thus the possibility of sensitization to these allergens being greater at this age[48]. On the other hand, this result may also be explained by the fact that both SPT and determination of specific IgE antibodies were performed for a small number of aeroallergens.

Research results indicate that just the allergens of house dust and *Dermatophagoides pteronyssinus* are the most common aeroallergens responsible for hypersensitivity in children with AD[11,42,49]. Similarly, in our study, the most common aeroallergens responsible for hypersensitivity were house dust and *Dermatophagoides pteronyssinus* (in 6.1% and 3.5% of AD children, respectively). A possible reason why these aeroallergens are most often responsible for the appearance and/or deterioration of skin lesions from AD may be the principles of their action, mediated through three mechanisms: incomplete proteolytic enzyme activity activation of the protease-activated-receptor 2 and binding of IgE antibodies, which all results in an increase in inflammation[38,11]. Proteolytic activities directly contribute to damage to the skin barrier and delayed recovery of its function[11,27,38]. Since they contain cysteine and serine protease, they thereby directly damage the epithelial links, they also cause degranulation of eosinophils and activate keratinocytes, causing a rise in the production of proinflammatory cytokines[39,44]. These effects contribute to the appearance of defects in the skin barrier and local inflammation, and the exogenous proteases in changed skin (as is the case in children with AD) alter the natural balance between the endogenous proteases and the endogenous protease inhibitors, causing delayed, extended recovery of the skin barrier in the stratum corneum[50].

Hypersensitivity to individual aeroallergens, especially the allergens of house dust and *Dermatophagoides pteronyssinus*, is connected to a significant extent to the severity of the disease. The degree of hypersensitivity mediated by IgE antibodies to these aeroallergens, as shown by research results, is directly linked to the severity of the disease[15-17]. So, it has been shown that hypersensitivity to aeroallergens is more often present in children with moderate and severe forms of the disease, and in children that react less well to

usual therapy. These children more often have associated respiratory atopy, whilst it is less often found in children with the mild form of the disease[11,12].

Our results are slightly different. We demonstrated hypersensitivity to aeroallergens in the same number of children with the mild and severe forms of the disease (5/21 each), whilst in children with the moderate form of the disease, hypersensitivity was found in only 4/70 (5.7%) cases, although the largest number of our AD children had this form of the disease.

Conclusion

The aeroallergens recognized are an important factor in the occurrence and continuance of the pathologic processes in the skin of the children with AD. Exposure of AD children to aeroallergens may prompt the occurrence of the first skin lesion but may also cause deterioration of the existing skin lesions. Discovering the responsible aeroallergen (through an exhaustive medical history of hypersensitivity, determining total IgE antibodies, SPT and determining specific IgE antibodies to aeroallergens) and removing them from the living environment of the child, it is possible to reduce the intensity of skin lesions and the frequency of exacerbations, along with longer remission of the disease.

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Sažetak

PREOSJETLJIVOST NA AEROALERGENE U DJECE S ATOPIJSKIM DERMATITISOM

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Atopijski dermatitis (AD) je kronično-recidivirajuća upalna bolest kože obilježena izrazitim svrbežom, osipom i suhoćom kože. Preosjetljivost na aeroalergene prisutna je u 40%-50% djece s AD i uzrokom je intenziviranja kožnih promjena. Cilj ove studije bio je procijeniti prisutnost preosjetljivosti na aeroalergene u djece s AD. U istraživanju je sudjelovalo 114 djece (56 dječaka i 58 djevojčica), medijan dobi 27,5 mjeseci, s dijagnozom AD prema kriterijima Hanifina i Rajke. Težina bolesti procijenjena je indeksom SCORAD. Za prepoznavanje preosjetljivosti na aeroalergene analizirani su anamnestički podaci, vrijednosti apsolutnog broja eozinofila, ukupnih IgE protutijela, specifičnih IgE protutijela na aeroalergene i rezultati kožnog ubodnog testa (engl. *skin prick test*, SPT) na aeroalergene. Umjeren oblik bolesti zabilježen je u 61,4% ispitivane djece, uz medijan vrijednosti indeksa SCORAD od 28,5 bodova. Preosjetljivost na aeroalergene prepoznata je u 12,3% ispitivane djece (anamneza preosjetljivosti na aeroalergene u 27,2%, povišene vrijednosti apsolutnog broja eozinofila u 53,5%, povišene vrijednosti ukupnih IgE protutijela u 56,1%, pozitivan SPT u 20,2%, pozitivna specifična IgE protutijela na aeroalergene u 12,3% djece). Najčešće odgovorni aeroalergeni bili su kućna prašina u 6,1% i *Dermatophagoides pteronyssinus* u 3,5% ispitivane djece. Preosjetljivost na aeroalergene prepoznata je za isti broj djece s blagim i teškim oblikom bolesti, te u 5,7% djece s umjerenim oblikom. Zaključuje se da prisutnost preosjetljivosti na aeroalergene u djece s atopijskim dermatitisom nije zanemariva. Kroz otkrivanje i uklanjanje odgovornih aeroalergena moguće je utjecati na smanjenje intenziteta promjena i učestalosti egzacerbacija.

Ključne riječi: *Atopijski dermatitis; Djeca; Preosjetljivost; Aeroalergeni*