Role of Interleukin-12, 13, 18 and IgE in Asthmatic Yemeni Children

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Abstract

Background: Asthma is the most common chronic respiratory disease among children causing considerable morbidity and mortality. The exact cause of asthma has not yet been identified but it is believed that multiple factors are involved with interplay between host factors (particularly genetics) and environmental factors that occur at a crucial time in the development of the immune.

Airway inflammation in asthma may represent a loss of normal balance between two opposing populations of T-helper lymphocytes; Th1 and Th2 either as overexpression of Th2 or under expression of Th1. Th1/Th2 imbalance plays an important role in the development of asthma and allergic diseases with an imbalance between Th1 and Th2 cytokine profiles and evidence that allergic diseases, and possibly asthma, are characterized by a shift toward a Th2 cytokine-like disease, either as over expression of Th2 or under expression of Th1. Cytokines play an important role in the coordination and persistence of inflammation in asthma, although the precise role of each cytokine remains to be determined.

Objective: To compare the serum levels of Interleukin-12 (IL-12), IL-13, IL-18 and total IgE (TIgE) in asthmatic children with age-matched healthy control group in Sana'a City.

Methods: The study was a hospital based prospective case control study of children 5-15 years old attending two referral Pediatric Hospitals in Sana'a, Yemen; Al-Sabeen Hospital for Maternity and Childhood and Al-Thawra General Modern Hospital. The data collection was for 12 months, from October 2013 up to November 2014. Seventy children were enrolled, 35 asthmatic children and 35 age matched (5-15y) healthy controls. Serum IL- 12, IL- 13, IL- 18 and TIgE concentrations were measured by enzyme-linked immunosorbent assay at the National Blood Transfusion and Researches Center (NB-TRC), Sana'a. Serum IgE levels were measured using: DRG Human IgE ELISA Kit (catalogue number (EIA- 1788), DRG International Inc., USA). Serum IL-12, IL-13 and IL-18: Using Glory Science Human IL- 12, IL- 13 and IL- 18 ELISA Kits (Glory Science Co., Ltd., USA) according to the manufacturer's instructions. Data were stored and analysed by Epiinfo 2011 version 3.5.3.

Results: Serum IL-12 and IL-18 levels in the asthmatic children were significantly lower than controls. On the other hand, serum IL-13 and serum TIgE levels in the asthmatic children were significantly higher than controls. The percentage as well as the absolute count of peripheral blood eosinophils was significantly elevated in the asthmatic children compared with the control group.

Conclusion: Th1-Th2 imbalance is an important characteristic of asthma with a predominance of Th2 cytokines such as IL-13 with a relative deficiency of Th1 cytokines, such as IL- 12 and IL- 18. Cytokines are critical in the pathophysiology of asthma raising the possibility that inhibition of Th-2 cytokines or promotion of Th1-skewing cytokines could be a logical approach to asthma therapy.

Key Words: Asthma – Children – IL12 – IL13 – IL-18 – children – Yemen.

Introduction

ASTHMA is the most common chronic respiratory disease among children causing considerable morbidity and mortality [1]. The exact cause of asthma has not yet been identified but it is believed that multiple factors are involved with interplay between host factors (particularly genetics) and environmental factors that occur at a crucial time in the development of the immune [2].

Airway inflammation in asthma may represent a loss of normal balance between two opposing populations of T-helper lymphocytes [3].

Th1/Th2 imbalance plays an important role in the development of asthma and allergic diseases with an imbalance between Th1 and Th2 cytokine profiles and evidence that allergic diseases, and possibly asthma, are characterized by a shift toward a Th2 cytokine-like disease, either as over expression of Th2 or under expression of Th1. Cytokines play an important role in the coordination and persistence of inflammation in asthma, although the precise role of each cytokine remains to be determined [4].
To date, more than 30 different cytokines involved in asthma pathology have been identified, and this number continues to grow. Among these cytokines are T cell-derived molecules such as the so-called Th 1 cells (IL-2, Interferon-γ (IFN-γ), and IL-12), Th2 cells (IL-4, -5, -10, -13, and -25), Th3 or T regulatory cytokines [IL-10 and transforming growth factor beta (TGF-β)], and Th-17 cells (IL-17A and -17F) [8].

Cytokines derived from the Th-2 lymphocytes are considered to orchestrate the asthmatic phenotype. Among the Th-2 cytokines, substantial evidence supports key roles for IL-4 and IL-13 in the pathogenesis of bronchial asthma. Nevertheless, a growing body of genetic and clinical evidence that these cytokines are critical in the pathophysiology of asthma both in mouse and human, raises the possibility that inhibition of Th-2 cytokines such as IL-4, IL-5, and IL-13 could be a logical approach to asthma therapy [6].

Two double-digit cytokines, IL-12 and IL-13, have been proposed to play pivotal roles in the Th2-polarized immune response to inhaled allergens. An impaired IL-12 production coupled to an overproduction of IL-13 by alveolar macrophages may underlie to a great extent the Th2-biased response in asthma. Recent studies indicate that when compared with control subjects, reduced numbers of IL-12-expressing cells, and elevated IL-13 mRNA and protein levels exist in bronchial biopsy specimens and bronchial lavage cells from asthmatic patients [7].

IL-12 is secreted by antigen presenting cells including lymphocytes, monocytes/macrophages, and dendritic cells [4]. IL-12 is an important regulator of Th 1 development promoting the differentiation and proliferation of Th1 cells and IFN-γ production by T cells and NK cells. IL-12 can inhibit TH2 cytokine production and suppress IgE synthesis suggesting immuno-modulatory role in asthma. IL-12 and IFN-γ antagonize Th2 differentiation and the production of IL-4, IL-5, and IL-13 [8]. The effects of IL-12 have been extensively studied in small animal models of allergic inflammation and consistently demonstrate that this cytokine is involved in reduction of allergen-specific IgE, abolition of AHR and airway eosinophilia [5]. In asthmatic patients, IL-12 level is significantly reduced in peripheral blood and in airway biopsy specimens in comparison with healthy controls. IL-12 mRNA levels have been shown to be increased in biopsy specimens from asthmatic patients following treatment with corticosteroids [9].

IL-13 is a pleiotropic 12-kDa product of a gene on chromosome 5 at q31 that is produced in large quantities by stimulated Th2 cells [10]. IL-13 is thought to be a central mediator of inflammation in asthma based on animal models and on findings of elevated levels of IL-13 in the airways of patients with asthma [11]. It has been shown that IL-13 directs many of the processes involved in the allergic asthmatic response. IL-13 triggers macrophage and eosinophil activation to accelerate airway inflammation, IgE production by B cells, smooth muscle cell activation contributing to AHR, mucus and growth factor production in airway epithelial cells, eotaxin production to stimulate eosinophil recruitment to the airway, and activation/proliferation/migration of airway fibroblasts to promote airway remodeling [12].

IL-18 is secreted by Kupffer cells of the liver and activated macrophages [13]. It is a pleiotropic cytokine with a unique capacity to induce Th1 or Th2 polarization, depending on the immunologic context [14]. IL-18 strongly induces IFN-γ production in Th1 cells and Natural Killer (NK) cells in synergy with IL-12. On the other hand, it was reported that IL-18, with IL-2 but not without IL-12, may be a strong cofactor for the expression of a Th2 cytokine, IL-13, in T cells and in a unique NK population [15].

Asthma is now regarded as an IgE-mediated sensitization to inhaled allergens with a Th2 cell response and subsequent eosinophilic inflammation and airway hyper responsiveness [16]. An elevation in serum IgE levels, a marker of allergic inflammation and atopy, contributes to asthma and is considered a potent predictor of the development of asthma [17]. Eosinophilia, defined as the presence of >450 eosinophils/L in peripheral blood, is the most common hematologic abnormality of allergic patients and the presence of eosinophils in the sputum of asthmatic patients is classic [18].

This study aims to compare the serum levels of IL-12, IL-13, IL-18, TlGE and serum eosinophil cells in asthmatic children of age group 5-15 years with age-matched healthy control group in Sana'a city to assess the role of Th1/Th2 cytokines in the pathogenesis of asthma.

**Patients and Methods**

The study is a prospective case control study. It was based in Sana'a Capital of Yemeni. Data collection was for a period of 12 month started on Oct. 2013 up to Nov. 2014. All children 5-15 years
of age with a clinical diagnosis of bronchial asthma presenting to the participating health facilities were eligible for entry into the study. Clinical and demographic data were recorded on a standard questionnaire. Based on prior studies [19-21] and EpiCalc 2000-Version 1.02 program used for sample size calculation. Assuming an 80% power, 90% confidence interval and 1:1 case control ration; the sample size required was 35 asthmatic children and an age matched 35 normal healthy controls. Cases enrolment included consecutive patients presenting to Emergency Department (ER) or the Out-Patient Department (OPD) of each of the participating health facilities with a diagnosis of Bronchial Asthma. Bronchial Asthma was confirmed by measuring the pulmonary function test showing obstructive lung pattern responsive to bronchodilator using Vitalgraph Spirotrac version 4.36 (UK). The controls were enrolled from the paediatric surgical OPD or inpatient department of the two participating hospitals attending for minor surgery such as tonsillectomy. Al Sabeen Maternity and Childhood Teaching Hospital, Sana’a, is one of the two main referral paediatric hospitals in Yemen. It has 150 in-patient beds including 2 nurseries, 2 medical wards, an infectious disease ward, nutrition ward, one paediatric surgical ward, an Emergency Department and 2 large outpatient clinics. Al-Thawra General Modern teaching Hospital, Sana’a, is also a main referral hospital in Yemen. It has all the specialities in paediatrics and adults. It has 570 beds overall with 40 beds in the Paediatric Department including the nursery, medical ward, paediatric surgical ward and intensive care. It has extensive outpatient clinics and Emergency Department.

The inclusion criteria for the patients were: Children aged 5-15 years old, children with a diagnosis of Bronchial Asthma confirmed by improvement of symptoms on treatment with bronchodilators or pulmonary function test showing an obstructive airway pattern that respond to bronchodilators.

The exclusion criteria for the patients were: Children aged <5 years >15 years, patients with wheezy chest of known diagnosis other than bronchial asthma and patient refuses to participate.

The inclusion criteria of the controls were: Children aged <5 years >15 years, patients with wheezy chest, any respiratory symptoms or chest infection within the previous four weeks and patient refuses to participate.

Any suspected child with bronchial asthma would be referred to the investigator by the doctor on duty (hospital staff) in either the E/A or the OPD. The investigator will screen those children whether they fulfil each of the inclusion and exclusion criteria. After necessary attention to airway, breathing and circulation, a baseline history and examination were obtained from the cases or his/her parents. The case was referred to a pulmonologist for measuring the Pulmonary Function Tests (PFT) to confirm the diagnosis of asthma: This was conducted by using the Vitalgraph Spirotrac version 4.36 (UK). All data were recorded on the baseline examination form. After confirming the diagnosis the immunological investigations were carried out for each patient. A 5-mL blood sample was taken under aseptic conditions and divided into 2 portions: 1.5mL of whole blood was collected in sterile EDTA-containing tubes for CBC and eosinophil counts, and the remainder was left for 30 to 60 minutes for spontaneous clotting at room temperature before being centrifuged at 3000rpm for 10 minutes. Then transferred to Eppendorf tubes which were labelled and stored at below −20°C at the National Blood Transfusion and Researches Center (NBTRC), Ministry of Health and population in Sana’a where quantitative assay of serum IgE, IL-13, IL-18 and IL-12 levels was done. Complete blood Count (CBC): Using Sysmex Automated Hematology Analyzer KX-2 1N (Sysmex corporation, Japan). The Absolute Eosinophil Count (AEC) calculated according to the formula AEC = differential eosinophil count X total WBC/100 [22]. Chest X-Ray was done to document hyperinflated lung and any sequelae also to exclude any pathology other than asthma such as pneumonia. The X-Ray was read by experienced radiologist and the researcher. Immunological markers: Serum TlGE done using DRG ELISA Kit (DRG International Inc., USA), according to the manufacturer’s instructions. Serum IL-12, IL-13 and IL-18: Done using Glory Science Human IL-12, IL-13 and IL-18 ELISA Kits (Glory Science Co., Ltd, USA) according to the manufacturer’s instructions. Analysis of the data was done by using Epi-Calc 2000-Version 1.02. Ethical approval was obtained from the Ethical Committee of the two participating hospitals.

Results

The current study compared 35 children with bronchial asthma and 35 healthy control children.
The means of age (years) (± SD) [range] of the asthmatic children was 8.6 (±3.2) [5-15] and the control children was 8 (±2.6) [5-14], (p-value=0.4). Twenty five children (71%) of the asthmatics were males while there were only 16 (46%) children in the control group.

In the present study comparison of the serum Levels of IL-12, 13, 18, TIgE and Absolute Eosinophils Count (AEC) in asthmatic children and healthy controls showed that the serum IL-12 levels in the asthmatic children (13.03 ±9.31 pg./mL; mean ± SD) were significantly lower than those in the controls (26.00 ±30.22pg/mL; means) (p = 0.02); the serum IL-13 levels in the asthmatic children (36.80±49.96pg/mL; mean ± SD) were significantly higher than those in the controls (16.37±10.34 pg/ml; mean ± SD) (p=0.02); the serum IL-18 levels were significantly lower in the asthmatic children (14.74 ±17.62pg./mL; mean ± SD) than in the controls (28.20 ±34.61pg./mL, mean ± SD) (p=0.04) and the serum total IgE level in the asthmatic children (143.71 ±153.19 IU/ml; mean ± SD) were significantly higher than those in the Controls (32.06 ±51.40; mean ± SD IU/ml; mean ± SD) (p=0.0001). Also the percentages as well as the Absolute Eosinophils Count (AEC) of peripheral blood were significantly elevated in the asthmatic children compared with the control group (p-value <0.05) (Table 1).

Based on linear regression analysis showed that the serum total IgE levels were positively correlated with serum IL-13 levels and that correlation was statistically significant (r=0.89, p=0.02) (Table 2). Also the Absolute Eosinophilic Count (AEC) was positively correlated with both serum IL-13 levels and serum total IgE levels and was statistically significant (r=1.10, p= 0.007, r=0.537, p=0.000015 respectively) (Table 3).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Asthmatic children N=35 Mean ± SD</th>
<th>Healthy control N=35 Mean ± SD</th>
<th>t-test</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum IL-12 (pg/ml)</td>
<td>13.03±9.31</td>
<td>26.00±30.22</td>
<td>2.43</td>
<td>0.02</td>
</tr>
<tr>
<td>Serum IL13 (pg/ml)</td>
<td>36.80±49.96</td>
<td>16.37±10.34</td>
<td>2.36</td>
<td>0.02</td>
</tr>
<tr>
<td>Serum IL18 (pg/ml)</td>
<td>14.74±17.62</td>
<td>28.06±34.61</td>
<td>2.05</td>
<td>0.04</td>
</tr>
<tr>
<td>Serum TIgE (IU/ml)</td>
<td>143.71±153.19</td>
<td>32.06±51.40</td>
<td>4.09</td>
<td>0.001</td>
</tr>
<tr>
<td>Eosinophils %</td>
<td>5.58±2.04</td>
<td>1.90±0.65</td>
<td>12.15</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>AEC (10² cells/µL)</td>
<td>4.03±1.77</td>
<td>1.29±5.9</td>
<td>10.38</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

**Table (2): Linear regression between serum total IgE and Serum IL-12, IL-13 and IL-18 in asthmatic children.**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>Correlation coefficient: r^2</th>
<th>Std error</th>
<th>F-test</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>IL-13</td>
<td>0.893</td>
<td>0.26</td>
<td>0.371</td>
<td>5.784</td>
<td>0.019</td>
</tr>
<tr>
<td>IL-12</td>
<td>−0.452</td>
<td>0.20</td>
<td>0.622</td>
<td>0.529</td>
<td>0.470</td>
</tr>
<tr>
<td>IL-18</td>
<td>−0.440</td>
<td>0.21</td>
<td>0.505</td>
<td>0.757</td>
<td>0.387</td>
</tr>
</tbody>
</table>

p≤0.05 is significant.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>Correlation coefficient: r^2</th>
<th>Std error</th>
<th>F-test</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>IL-13</td>
<td>1.10</td>
<td>0.65</td>
<td>0.397</td>
<td>7.698</td>
<td>0.007</td>
</tr>
<tr>
<td>Total IgE</td>
<td>0.537</td>
<td>0.70</td>
<td>0.115</td>
<td>21.78</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

p≤0.05 is significant.
Discussion

Asthma is a chronic airway inflammation caused by a defect in immune regulation involving Th lymphocytes, with an increase in Th2 lymphocytes and a compensatory decrease in Th1 lymphocytes [21].

It is well recognized that the Th 1-Th2 paradigm is an important characteristic of asthma. There is believed to be an imbalance in asthma, with a predominance of Th2 cytokines, including ILs-4, -5, and – 13, in patients with asthma, with perhaps a relative deficiency of Th1 cytokines, such as interferon-γ or ILs-12 and – 18 [23].

IL-12 is a key cytokine involved in regulating the balance between Th1 and Th2 cells by promoting Th1 response. A reduced capacity to produce this cytokine could lead to aberrant Th2 development [21]. In asthmatic patients, IL-12 is significantly reduced in peripheral blood and in airway biopsy specimens, compared with healthy controls [5]. The current study showed a significant low serum IL-12 levels in asthmatic children compared with the control group and this was in consistence with what have been found by Zedan et al., [21]; Rahi [24] and Zhang et al., [25]. In contrast to our findings Sultanova [26] and Wong et al., [27] found that serum IL- 12 levels in bronchial asthma patients were significantly higher when compared to controls. It has been postulated that low-level production of IL- 12 by antigen-presenting cells is associated with the risk of developing atopic asthma. Low IL-12 levels in dendritic Cells are associated with the risk of developing Th2 immunity to allergens, whereas high levels of IL-12 seem to offer protection from allergic diseases [28].

IL-13 is a pleiotropic cytokine, playing key roles in the pathogenesis of allergic diseases such as bronchial asthma [6]. It is claimed that the main pathophysiological features of asthma, i.e. Bronchial hyperresponsiveness, increase of mucous secretion and sub epithelial fibrosis may be induced by IL-13. The evidence for IL-13 participation in asthmatic inflammation was the presence of increased IL-13 expression in bronchial biopotates, induced sputum and nasopharyngeal aspirates in patients with asthma [29]. In the present study, we found that serum IL-13 levels were significantly high in children with asthma compared with the control group, and this was in consistence with what have been found by Gemou-Engesaeth et al., [30]; Al-Quraishi [31] and Machura et al., [29]. In contrast to our finding, Davoodi et al., [32] found that the serum IL-13 in asthmatic patients was lower than in healthy controls but the difference was not significant. Increased expression of IL-13 has been documented in the bronchial mucosa of asthmatics; therefore spillover of IL-13 from inflamed airways to peripheral blood cannot be discounted as a possibility for the raised serum IL-13 levels observed in asthmatics [31]. The source of elevated serum IL-13 levels in this cohort of asthmatics is not clarified by our cross-sectional study. However, there is evidence to suggest that the circulating Peripheral Blood Monocyte Cells (PBMCs) may be a major source for circulating IL-13 in asthmatics. In addition, the proportion of IL-13 producing PBMC’s has been shown to increase upon exposure to allergens. The contribution of Natural Killer T cells (NKT cells) and mast cells to the production of IL-13 is also increasingly recognized in asthmatics [33].

IL- 18 is a novel cytokine that plays an important role in the Th1 cell response, primarily through its ability to induce IFN-γ production, especially in collaboration with IL-12 [34]. IL-18 can inhibit Th2 immune response and that the reduced levels of IL-18 may contribute to the pathogenesis of asthma [35]. In the present study, it was found that significantly lower levels of serum IL- 18 in asthmatic children occur compared with the control group and this was in accordance with Abdel Naser et al., [20] and Cebeci et al., [36] who reported significantly lower serum IL- 18 levels in patients with asthma than in normal control subjects. On the other hand the result obtained from the present study was in contrast to Al-Quraishi [31]; Ando and Shima [37] who found that serum IL- 18 levels were significant higher in asthmatic children than healthy controls. The low levels of IL- 18 in asthma and reduced IL-18 dependent release of IFN-γ might be the reason for the development and expansion of Th2 cells and cytokine production. Thus low levels of IL-18 may be an important causative factor in the genesis of asthmatic inflammation [20].

It is a well-known fact that IgE plays a central role in the pathophysiology of allergic disorder such as asthma. An elevation in serum IgE levels contributes to asthma and is considered a potent predictor of the development of asthma [38]. Our study showed that serum TlG levels in asthmatic children were significantly high compared with the control group and this was in agreement with Al-Quraishi [31]; Shokry and Soliman [39] and Rahi [24] where they found that serum IgE concentrations were significantly higher in asthmatic patients compared to control subjects.
The presence of peripheral blood eosinophilia and activated eosinophils in the chronic inflammatory infiltrate of the airways is a characteristic of both allergic and non-allergic asthma [40]. In the present study, the Absolute Eosinophilic Count (AEC) was significantly high in asthmatic children compared with the control group which was in agreement with Shokry and Soliman [39] and Abdel Naser et al., [20] where they found that the eosinophil counts were significantly higher for the asthmatics when compared to the control children.

IL-13 may promote the differentiation and survival of eosinophils and mast cells and induce the isotype switching of IgE [41]. Our data showed a significant positive correlation between serum IL-13 and serum TlgE levels in asthmatic children. This finding was in accordance with Wang et al., [42] and Feleszko et al., [43] who found that serum IL-13 was positively correlated with serum TlgE in children with asthma. On the contrary to our finding, Joseph et al., [33] reported that there was no significant correlation between serum IL-13 level and IgE level in asthmatics or normal controls.

Peripheral blood eosinophil counts have been used for indirect assessment of airway inflammation and to aid the diagnosis of asthma. They are believed to reflect Th2-driven airway inflammation [44]. Blood eosinophils from patients suffering from bronchial asthma expressed IL-13 in contrast to blood eosinophils from control individuals which did not produce detectable IL-13 levels [45]. The present study showed a significant positive correlation between serum IL-13 and AEC in asthmatic children and this comes in accordance with Manise et al., [46] who found a positive correlation between IL13 and eosinophils either as a percentage or as absolute values. On the contrary to our finding, Joseph et al., [33] found no correlation between serum IL-13 levels and the peripheral eosinophil count in asthmatic children.

Blood eosinophilia and raised serum total IgE level are strong predictors of allergy in asthmatic children [47]. Our study showed that in asthmatic children, serum TlgE levels were positively correlated with AEC and this was in accordance with Lama et al., [38] and Mehmet et al., [48] where they found that raised eosinophil count showed significant association with the elevated level of serum TlgE in asthmatic children. However, Razi and Moosavi. [17] found that serum TlgE levels were not associated with changes in peripheral blood eosinophil counts. This correlation could be explained as eosinophil recruitment is attributed to IgE via cross-linking with high-affinity receptors on mast cells, by which IgE induces the release of preformed mediators, and thereafter newly formed mediators and cytokines such as IL-4 and IL-5, leading to the accumulation of eosinophils [49].

Conclusion:

Cytokines are critical in the pathophysiology of asthma raising the possibility that inhibition of Th-2 cytokines such as interleukin IL-13 or promotion of Th1-skewing cytokines, such as IL-12, IL-18 could be a logical approach to asthma therapy. Serum IL-13 could be considered as a key factor in bronchial asthma pathogenesis and hence its therapeutic manipulation may be of help in bronchial asthma management.

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الملخص العرabi

يعتبر الربوب القصبي من أكثر الأمراض في الجهاز التنفسي المزمنة شيوعًا بين الأطفال والتي تسبب في الكثير من الاعتلال والوفيات. لم يتم محددة السبب الدقيق لمرض الربوب ولكن يعتقد أن عوامل متعددة تشارك في التفاعل بين العوامل البيئية (ولسيا علم الوراثة) والعوامل البيئية التي تحدث في الوقت الحاسم لتظهر جهاز المناعة.

هذا نتائج الدراسة: مقارنة المستويات المضادة للإسطروريفين (IL-12، IL-10، وجميع IGFBP6 في الأطفال) في المصابين بالربوب مع مجموعة الأشخاص (المجموعة المهاجرة) منظومة العين في مدينة صنعاء.

منهجية البحث: تضمنت هذه الدراسة سبعين طفلا للفئة العمرية من (5-10 سنوات) منهم 35 طفلا مصابين بالربوب القصبي. تم حذف طفلا من الأطفال الأصحاء (المجموعة المهاجرة) وقد تم تجميع البيانات خلال 12 شهرا أبتداء من أكتوبر 2013 وحتى نوفمبر 2014 وتم أيضا قياس المستوي السهلي للإسطروريفين (IL-12، IL-10، وجميع IGFBP6) باستخدام فحوص تحليل مرتب مريض المناعي (enzyme-linked immunosorbent assay)

النتائج: أظهرت نتائج البحث أن المستويات المضادة للإسطروريفين (IL-12، IL-10، وجميع IGFBP6 في الأطفال الأصحاء) ومقدمة أخرى، وكانت المستويات المضادة للإسطروريفين في الأطفال المصابين بالربوب أعلى بكثير من الأطفال الأصحاء.

الخلاصة: هذا النوع من التوازن بين TH1-TH2 هو سمة هامة في الربوب القصبي مع غلبة السيتوكينات TH1-TH2. السيتوكينات TH1 وTH2 من السيتوكينات TH1-18 يمكن أن يكون نهجا منطقيا لعلاج الربوب.