The impact of environmental and agricultural pollutants on the prevalence of allergic diseases in people from Qassim, KSA

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Background: There are multiple environmental factors that influence a sensitized (IgE antibody positive) patient's predisposition to manifest allergic symptoms following allergen exposure. The majority of allergens are known to induce morbidity with chronic symptoms such as rhinitis, pruritis, dermatitis and urticaria.

Aim: To study the impact of environmental and agricultural pollutants with different pollens on the immunological, hematological and biochemical markers and to determine the prevalence of sensitization to allergens among exposed individuals as well as to identify the eliciting allergens.

Subjects and Methods: Ninety six highly exposed individuals to environmental and agricultural pollution in addition to 20 as controls were selected. A solid phase enzyme-linked immunosorbent assay and the EUROLINE test kit were used for the quantitative determination of total IgE concentration and semi-quantitative in vitro assay of human IgE antibodies to some of the inhalant, ingestant and contactant allergens in serum samples, respectively. Percentage and absolute eosinophil counts and biochemical parameters were analyzed.

Results: Thirteen (13.5%) out of the 96 studied highly exposed subjects were manifesting allergic symptoms. Higher significant total serum IgE levels and absolute eosinophil counts in groups 1 and 3 of the highly exposed individuals compared to the control group were found (p1=0.00, p3=0.001 and p1=0.016, p3=0.028, respectively). Higher sensitization with inhalant Timothy grass, *Aspergillus fumigatus*, Der. Farinae and Olive; ingestant Egg yolk, Mango, Strawberry and Codfish and with contactant Latex/plastic and Crude oil was found in the studied groups compared with the controls.

Conclusion: The present data suggest that the highly exposed subjects to pollution are at high risk of developing an allergy. For the screening of those with suspected allergen sensitization, the determination of specific IgE antibodies is a suitable marker of type I allergy.

Key words: Environmental and agricultural pollens, Recombinant allergens, Specific IgE antibodies.

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Introduction

The prevalence of allergic diseases has increased worldwide, a phenomenon that can be largely attributed to environmental effects and it is agreed that genetic changes in populations would be too slow to account for such a rapid change in prevalence. [1] Among environmental factors, air pollution due to traffic is thought to be a major threat to health. Residing near busy roadways is associated with increased asthma hospitalization, decreased lung function, and increased prevalence and severity of wheezing and allergic rhinitis and bronchitis. Particulate matter and ground level ozone are the most frequent air pollutants that cause harmful effects, and the mechanisms underlying these effects may be related to oxidative stress. [2]

Inhalant allergens from grass or tree pollens, house dust mite and animal dander are the major substances that are capable of provoking type I hypersensitivity. An individual who has hypersensitivity to pollen often suffers from seasonal allergic rhinitis or extrinsic asthma. [3, 4]

Ingestant allergens often refer to substances inducing allergy after the sensitized individuals eating a certain food. Typical symptoms of this type of allergy include mouth or throat itching and lip swelling. [5, 6] Latex is the most important contactant allergens in plants. Since the late 1980's, this immediate-type allergy provoked by natural rubber products has been reported around the world. [7, 8]

In atopic individuals, elevated total serum IgE levels have also been associated with a predisposition for airway hyper-responsiveness and asthma. [9] Detection of allergen-specific IgE has been generally considered the more clinically useful analyte, since it is a specific biomarker for the atopic state which allows linkage of symptoms to particular allergen exposure. Detection of allergen-specific IgE antibodies in serum has been used clinically to confirm sensitization to a particular allergen in support of a history-based diagnosis of allergic forms of rhinitis, asthma, dermatitis, urticaria, angioedema, conjunctivitis, anaphylaxis and food and insect sting allergy. [10]

The precise knowledge of the patient's allergenic profile helps to choose a better targeted immunotherapy. The selection of an appropriate allergen extract containing precisely defined amount of an individual active molecule and/or molecules might lead to optimization of immunotherapy and reduction of adverse side effects of possible newly formed specific IgE antibodies. [11, 12]

The work aimed to study the impact of environmental and agricultural pollutants with different allergens on the immunological, hematological and biochemical markers and to determine the prevalence of sensitization to different inhalant, ingestant and contactant allergens among exposed individuals as well as to identify the eliciting allergens. Also, correlate the degree of allergens exposure with deterioration of respiratory health in exposed individuals.

Subjects and Methods:

Study subjects:

This is a case control study in which the 96 highly exposed individuals to pollution in addition to 20 with no or minimal level of exposure (with age ranged from 20 to 43 years) were selected within the period from May 2012 to November 2012 according to the following criteria: job-related allergic disorders either with previous history or manifesting symptoms; response to anti-allergy therapy and the work duration at fields of exposure. All subjects of the studied groups and control group were males. Informed consent was obtained from all participants and the study was approved by ethics and scientific committees in the university. Complete demographic data including: age, job description and history of allergic disorders; type; duration; family history and drug intake were taken from every subject.

According to site of pollution's exposure, four groups were selected and divided into:

(1) Thirty two people from the farm area (workers and constant living populations) and forestry workers.
(2) Thirty two workers in petrol stations.
(3) Thirty two road construction workers.
(4) Twenty healthy age- and sex-matched subjects were included as a control group.

Blood samples:

One venous blood sample (10 ml) was obtained from each subject and control and

processed at research laboratory; One ml of EDTA-blood samples for complete blood count and the remaining of samples were centrifuged to obtain serum samples that frozen at –20°C until analysis for measurement of other parameters. Also, heparin-arterial blood samples were taken, transported on ice and analyzed for blood CO_2 level. [1]

Immunological, hematological and biochemical markers were investigated and included: [1]

(1) Total serum IgE level:
A solid phase enzyme-linked immunosorbent assay for the quantitative determination of total IgE concentration in human serum samples was used (Pishtaz Teb Diagnostics, ID Consulting Services Ltd. Korbach/Germany) according to the manufacturer's guidelines. Reading was done using Robonic Readwell Strip ELISA Analyzer, India.

(2) Serum levels of allergen-specific IgE:
The EUROLINE test kit was used for semi-quantitative in vitro assay of human allergen-specific IgE antibodies (EUROIMMUN, Medizinische, Labordiagnostika AG) against some of the common commercially available inhalation, plant and animal food and contact allergens in serum samples. The test kit contains test strips coated with parallel lines of 21 different allergens extract. The test strips were first moistened and then incubated in the first reaction step with patient serum. In positive samples, specific antibodies of class IgE were bound to the allergens and detected by second incubation using an enzyme-labelled monoclonal anti-human IgE catalyzing a colour reaction. The band positions and intensity of staining must be taken into consideration. By comparing the incubated test strips with the printed evaluation strip, the allergens against which IgE antibodies are present could be identified. The signals can be divided into four classes which correspond to the bands.

(3) Total and differential leukocytic counts:
Total and differential leukocytic counts were determined from EDTA-venous blood samples using an autoanalyzer and absolute eosinophilic count was calculated.

(4) Biochemical parameters including kidney function tests, liver function tests and blood glucose test were measured by using Spectrum Diagnostic Reagent, The Creative Approach to Bioscience (Robonic Prietest Autobiochemistry analyzer, India). Blood CO_2 level was measured using blood gas analyzer.

Results:

Thirteen (13.5%) out of the studied highly exposed subjects were manifesting allergic symptoms. All subjects of the studied groups and control group were males.

The immunological and hematological markers of the studied subjects in relation to the control group were summarized in Table 1. There was higher significant total serum IgE level in groups 1 and 3 of the highly exposed individuals compared with the control group (p1=0.00; 95% CI, 122.2-164.9 and p3=0.001; 95% CI, 100.4-147.1, respectively). The total leukocytic count was significantly higher in group 1 of the study subjects than the controls (p1=0.015). Regarding the percentage and absolute eosinophil counts, there were higher significant counts in groups 1 and 3 of the highly exposed individuals than the controls (p1=0.028, p3=0.007 and p1=0.016, p3=0.028; 95% CI, 0.5-0.89, 0.4-0.89, respectively).

As summarized in Table 2, there were slightly higher significant levels of serum bilirubin and serum creatinine in group 1 of the highly exposed subjects than the controls (p1=0.005; 95% CI, 0.96-1.13 and p1=0.004; 95% CI, 0.98-1.13, respectively) with no statistical differences in other parameters between the studied groups and the control group (p1, p2, p3>0.05).

The serum samples of the studied subjects were tested with a panel of 21 common commercially available allergenic extracts to each of inhalant, ingestant and contactant allergens for determination of the rate of occurrence of specific IgE antibodies against these different allergens.

As shown in Table 3, high sensitization to inhalant allergens was observed with Timothy Grass in 25% (8/32) of group 1 of the studied subjects, with *Aspergillus fumigatus* in 15.6% (5/32) of group 2 and equally with Timothy Grass, Der. Farinae and Olive in 12.5% (4/32)

of group 3. High sensitization to ingestant Egg yolk in 18.8% (6/32) of group 1, equally to Mango, Strawberry and Egg yolk in 12.5% (4/32) of group 2 and to Codfish in 21.9% (7/32) of group 3 was demonstrated. Regarding contactant allergens, high sensitization to Latex/plastic in 28.1% (9/32) and 15.6% (5/32) of group 1 and 2, respectively and equally to Latex/plastic and Crude oil in 12.5% (4/32) of group 3 was found. In contrast, only one of 20 healthy controls reported sensitization to Egg yolk. Significant differences were detected concerning the sensitization to inhalant, ingestant and contactant allergens between the studied highly exposed groups and the control group (p=0.00, 0.00 and 0.002, respectively).

Semiquantitative analysis of sensitization to inhalant, ingestant and contactant allergens in the studied groups of highly exposed subjects compared to the controls was demonstrated in Tables 4, 5 and 6 according to band density as the following: negative (no band or 0), weak band (+), clear band (++) and intense band (+++). The results were evaluated by comparing the incubated test strips with the printed evaluation strip.

Table (1): Immunological and hematological markers of the studied highly exposed subjects in relation to the controls

Immunological and hematological markers	**Group 1** (n=32) mean±SD	**Group 2** (n=32) mean±SD	**Group 3** (n=32) mean±SD	**Control** (n=20) mean±SD	***P value***
Total serum IgE (IU/ml)	143.6± 59.2	94.3± 56.2	123.7± 64.8	69.1± 32.7	P1=0.00 p3=0.001
Total leukocytic count (10^3/ul)	10.1±3.2	9.3±2.4	8.5±4.1	7.3±2.0	P1=0.015
Eosinophils (%)	6.0±3.4	4.8±2.6	6.5±3.4	4.1±2.6	P1=0.028 P3=0.007
Absolute eosinophils (10^3/ul)	0.7±0.5	0.5±0.5	0.6±0.6	0.3±0.2	P1=0.016 p3=0.028

P1: Control versus group 1
P2: Control versus group 2
P3: Control versus group3

Table (2): Biochemical parameters of the studied groups in relation to the control group

Biochemical parameters	**Group (1)** mean±SD	**Group (2)** mean±SD	**Group (3)** mean±SD	**Control** mean±SD	***P value***
Liver function tests:					
SGPT	37.6±6.5	35.8±8.8	31.2±5.5	34.8±6.6	P1, P2, p3>0.05
SGOT	31.8±7.2	29.6±9.1	25.6±6.6	29.7±7.0	P1, P2, p3>0.05
S. albumin	3.8±0.60	3.9±0.59	3.8±0.58	3.9±0.47	P1, P2, p3>0.05
S. bilirubin	1.04±0.24	0.97±0.14	0.87±0.15	0.92±0.14	P1=0.005
Kidney function tests:					
S. urea	38.6±10.0	31.5±8.6	37.6±11.9	33.3±8.2	P1, P2, p3>0.05
S. creatinine	1.08±0.2	0.8±0.2	0.9±0.3	0.8±0.1	P1=0.004
Random blood glucose	113.2±11.3	99.0±18.0	107.7±16.4	104.6±19.7	P1, p2, p3>0.05
Blood CO2	23.2±9.6	19.2±6.3	22.3±8.3	19.3±4.7	P1, p2, p3>0.05

P1: Group 1 versus control
P2: Group 2 versus control
P3: Group 3 versus control

Table (3): Monosensitization or/and polysensitization to tested allergens in the studied subjects compared to the controls

Allergens	The studied groups				*P value*
	Group 1 (n=32)	**Group 2** (n=32)	**Group 3** (n=32)	**Control** (n=20)	
Inhalant:					
Aspergillus fumigatus	4 (12.5%)	5 (15.6%)	0 (0%)	0 (0%)	
Timothy Grass	8 (25%)	4 (12.5%)	4 (12.5%)	0 (0%)	
Cultivated rye	3 (9.4%)	4 (12.5%)	0 (0%)	0 (0%)	0.00
Alternaria alt.	0 (0%)	0 (0%)	3 (9.4%)	0 (0%)	
Der. Farinae	0 (0%)	0 (0%)	4 (12.5%)	0 (0%)	
Olive	0 (0%)	0 (0%)	4 (12.5%)	0 (0%)	
Ingestant:					
Banana	5 (15.6%)	3 (9.4%)	0 (0%)	0 (0%)	
Mango	4 (12.5%)	4 (12.5%)	0 (0%)	0 (0%)	
Egg yolk	6 (18.8%)	4 (12.5%)	4 (12.5%)	1 (5%)	0.00
Codfish	4 (12.5%)	0 (0%)	7 (21.9%)	0 (0%)	
Tomato	3 (9.4%)	0 (0%)	0 (0%)	0 (0%)	
Strawberry	0 (0%)	4 (12.5%)	0 (0%)	0 (0%)	
Chicken Meat	0 (0%)	0 (0%)	2 (6.2%)	0 (0%)	
Soya bean	0 (0%)	0 (0%)	4 (12.5%)	0 (0%)	
Contactant:					
Latex/plastic	9 (28.1%)	5 (15.6%)	4 (12.5%)	0 (0%)	
Fabrics	3 (9.4%)	4 (12.5%)	0 (0%)	0 (0%)	0.002
Furniture	4 (12.5%)	4 (12.5%)	2 (6.2%)	0 (0%)	
Crude oil	0 (0%)	0 (0%)	4 (12.5%)	0 (0%)	

Table (4): Semiquantitative analysis of sensitization to inhalant allergens in the studied groups of subjects compared to the control group

Inhalant allergens	The studied groups			
	Group 1 (n=32)	**Group 2** (n=32)	**Group 3** (n=32)	**Control** (n=20)
Aspergillus fumigatus				
0	28	27	32	20
+	1	2	–	–
++	3	2	–	–
+++	–	1	–	–
Timothy Grass				
0	24	28	28	20
+	3	1	2	–
++	4	2	1	–
+++	1	1	1	–
Cultivated rye				
0	29	28	32	20
+	2	–	–	–
++	1	2	–	–
+++	–	2	–	–
Alternaria alt.				

0	32	32	29	20
+	–	–	1	–
++	–	–	2	–
+++	–	–	–	–
Der. Farinae				
0	32	32	28	20
+	–	–	3	–
++	–	–	1	–
+++	–	–	–	–
Olive				
0	32	32	28	20
+	–	–	2	–
++	–	–	1	–
+++	–	–	1	–

0: Negative
+: Weak band
++: Clear band
+++: Intense band

Table (5): Semiquantitative pattern of sensitization to ingestant allergens in the studied groups of subjects compared to the control group

Ingestant allergens	**Group 1** (n=32)	**Group 2** (n=32)	**Group 3** (n=32)	**Control** (n=20)
Banana				
0	27	29	32	20
+	2	1	–	–
++	2	1	–	–
+++	1	1	–	–
Mango				
0	28	28	32	20
+	2	1	–	–
++	1	2	–	–
+++	1	1	–	–
Egg yolk				
0	26	28	28	19
+	2	2	1	1
++	3	2	1	–
+++	1	–	2	–
Codfish				
0	28	32	25	20
+	2	–	3	–
++	1	–	2	–
+++	1	–	2	–
Tomato				
0	29	32	32	20
+	1	–	–	–
++	1	–	–	–
+++	1	–	–	–
Strawberry				
0	32	28	32	20
+	–	1	–	–
++	–	2	–	–
+++	–	1	–	–

Chicken Meat				
0	32	32	30	20
+	–	–	1	–
++	–	–	1	–
+++	–	–	–	–
Soya bean				
0	32	32	28	20
+	–	–	1	–
++	–	–	2	–
+++	–	–	1	–

Table (6): Semiquantitative pattern of sensitization to contactant allergens in the studied subjects compared to the controls

Contactant allergens	**Group 1** (n=32)	**Group 2** (n=32)	**Group 3** (n=32)	**Control** (n=20)
Latex/plastic				
0	23	27	28	20
+	3	1	1	–
++	5	2	3	–
+++	1	2	–	–
Fabrics				
0	29	28	32	20
+	1	1	–	–
++	1	1	–	–
+++	1	2	–	–
Furniture				
0	28	28	30	20
+	1	–	1	–
++	2	3	1	–
+++	1	1	–	–
Crude oil				
0	32	32	28	20
+	–	–	1	–
++	–	–	1	–
+++	–	–	2	–

Discussion

The prevalence of allergic diseases has increased worldwide, a phenomenon that can be largely attributed to environmental effects. [(2)] Oxidative stress and epigenetic mechanisms are considered as possible mechanisms for developing allergic disease and sensitization. A linkage between oxidative stress and inflammation is believed to contribute to the pathogenesis of some allergic diseases. Many cells and mediators are thought to modulate the formation of reactive oxygen species, which in turn may mediate cellular signaling pathways related to inflammation. [(13)]

The reactive oxidative species (ROS) produced in response to air pollutants can overwhelm the redox system and damage the cell wall, lipids, proteins, and DNA, leading to airway inflammation and hyper-reactivity. Elevated levels of ROS may induce a variety of pathological changes that are highly relevant in nasal and airway mucosa. These include lipid peroxidation, increased airway reactivity, increased nasal mucosal sensitivity and secretions, production of chemoattractant molecules, and increased vascular permeability. [(14)] Pollutants may also cause harmful effects via epigenetic mechanisms,

which control the expression of genes without changing the DNA sequence itself. These mechanisms are likely to be a target for the prevention of allergies. [15, 16, 17]

In the present study, thirteen (13.5%) out of 96 studied highly exposed workers were manifesting allergic symptoms and starting anti-allergic therapy while, the remaining with no symptoms. There are multiple personal and environmental factors that influence a sensitized (IgE antibody positive) patient's predisposition to manifest allergic symptoms following allergen exposure. Among these are the magnitude and quality of the patient's humoral immune response to the offending allergens in question. The concentration, affinity and clonality of the IgE antibody response all influence how effectively the effector cells are armed with relevant IgE antibody and how well the IgE binds to allergenic molecules following inhalation, ingestion or injection. [9]

IgE-mediated occupational allergic diseases manifest clinical symptoms after a certain period of exposure, which varies according to the offending allergen. For example, it typically takes one year of exposure to develop allergies to rice powder, 5 years for olive pollen, 5 years for sunflower pollen, and 7 years for wheat. The incubation period can range from several weeks to ≥20 years. The reasons for this may include the transport time and the settling time of pollen. [18, 19, 20] The fraction of the total IgE that is allergen specific may vary as a function of the allergen specificity and thus in principal contribute to the greater or lesser severity of allergic symptoms.

The finding of significantly higher level of total serum IgE among groups 1 and 3 of the studied subjects compared with the controls (p1=0.00; 95% CI, 122.2-164.9 and p3=0.001; 95% CI, 100.4-147.1, respectively) as shown in Table 1 stresses the importance of atopy in the expression of asthma in this community. Immunological recognition of environmental allergens may have an important role in the subsequent development of asthma as the allergens may play a dual role by inciting episodes of wheezing and causing asthma to develop in the first instance.

It is known that eosinophils are the most dominant inflammatory cells in allergic disease. [21] Our results showed that the total leukocytic count was significantly higher in group 1 of the study subjects than the controls (p1=0.015). Regarding the percentage and absolute eosinophil counts, there were higher significant counts in groups 1 and 3 of the highly exposed individuals than the controls (p1=0.028, p3=0.007 and p1=0.016, p3=0.028; 95% CI, 0.5-0.89, 0.4-0.89, respectively), Table 1. In corroboration with other study, Kartasamita *et al*. [22] reported statistically significant increased levels of eosinophils among asthmatic children. While, Ige *et al*. [1] observed no statistically significant differences in the total and percentage eosinophil counts between the asthmatic patients and the controls although the asthmatic patients have higher eosinophil counts. The presence and extent of eosinophilia in the peripheral blood may reflect the severity of the asthma and the presence of no significant eosinophilia may reflect that the asthma control is optimal as the levels of peripheral eosinophils tend to fall with better control in stable asthma. [1]

There were higher significant levels of serum bilirubin and serum creatinine in group 1 of the highly exposed subjects than the controls (p1=0.005; 95% CI, 0.96-1.13 and p1=0.004; 95% CI, 0.98-1.13, respectively) but within the normal limits with no statistical differences in other parameters between the three studied groups and the control group (p1, p2, p3>0.05) as summarized in Table 2. Most of the highly exposed subjects of group 1 were clinically manifesting allergic symptoms and starting their anti-allergic treatment so evaluation of serum levels to these parameters must be continue in allergy consultation after treatment, to know their clinical evolution, the apparition of adverse reactions and the use of other asthmatic medications in the period of the treatment.

This study summarized the results concerning the rate of occurrence of specific IgE antibodies against different commercially available inhalant, ingestant and contactant allergens in a group of individuals living and or working in the highly exposed areas in comparison to the controls (Table 3). Moreover, the present study demonstrated the semiquantitative pattern of sensitization to different allergens according to the band density and described it as the following: negative (no band or 0), weak band (+), clear

band (++) and intense band (+++) as shown in Tables 4, 5 and 6.

Our results explored high sensitization to inhalant allergens with Timothy Grass in 25% (8/32) of group 1 of the studied subjects, with *Aspergillus fumigatus* in 15.6% (5/32) of group 2 and equally with Timothy Grass, Der. Farinae and Olive in 12.5% (4/32) of group 3. The high sensitization to ingestant Egg yolk in 18.8% (6/32) of group 1, equally to Mango, Strawberry and Egg yolk in 12.5% (4/32) of group 2 and to Codfish in 21.9% (7/32) of group 3 was demonstrated.

In a similar study done by Ebo *et al.* [23] who reported that a specific IgE antibody to at least one of the investigated inhalant and animal food allergens was found in respectively 76% and 12% of the serum samples. A plant food-specific IgE antibody was observed in 88% of the serum samples. The higher sensitization to inhalant and plant food allergens in this study compared with our study could be attributed to the studied subjects were allergic adults and all had a history of natural rubber latex allergy confirmed by a positive skin test for latex and a positive latex-specific IgE. On the other hand, similar and /or slightly higher sensitization to animal food allergens in the current study compared to Ebo *et al*. [23]

In a case of occupational rhinitis induced by maize pollen exposure in a farmer, Sung *et al*. [24] found that the specific IgE antibodies to crude and commercial maize pollen extracts were higher in the patient than in the healthy subjects.

In this study as regard with contactant allergens, higher sensitization to Latex/plastic in 28.1% (9/32) and 15.6% (5/32) of group 1 and 2, respectively and equally to Latex/plastic and Crude oil in 12.5% (4/32) of group 3 was found. Tucke *et al*. [25] demonstrated circulating IgE antibodies to latex in 16.2% of studied patients. Additionally, the children with latex-specific IgE antibodies were older, had significantly higher (p=0.003) total IgE values than atopic patients without sensitization to latex. The prevalence of latex sensitization and/or allergy apparently varies widely, depending on the population examined. In atopic children, Liebke *et al*. [26] reported that 20.8% (60 of 306) had increased latex-specific IgE antibodies, whereas Cremer *et al*. [27] found a prevalence rate of 13.6% (six of 44) for atopic children. The identification and characterization of allergens and their structures and biological functions will be benefit for the diagnosis and treatment of pollen related allergic diseases.

Of the allergens causing considerable sensitization rates, a strong association between Latex and some food allergen extracts most frequently to Mango and Tomato in group 1 and Codfish in group 2 was observed. Furthermore, co-association between Latex and some inhaled allergens extracts especially to Timothy grass in groups 1 and 3 while, to Olive in group 2 of the studied highly exposed subjects was demonstrated and this may be explained on the bases of cross-reactivity between Latex and some foods and inhaled allergens as identified by Tucke *et al*. [25]

Holgate *et al*. and Stenfors *et al*. [28, 29] attempted to clarify whether bronchial epithelial cells (BECs) from asthmatic individuals were more sensitive to DEPs than those from non-asthmatics with regard to production of pro-inflammatory mediators, but the data failed to reveal exaggerated airway inflammation in asthmatic individuals after diesel exhaust exposure. These findings demonstrated that diesel exhaust exposure has evident inflammatory effects on the airways of non-asthmatic individuals, but this does not occur in the presence of asthmatic airway inflammation.

Conclusion

The present data suggest that the highly exposed subjects to environmental and agricultural pollution are at high risk of developing an allergy and it is important to develop methods of identifying susceptible individuals within a large population. For the screening of those with suspected allergen sensitization the determination of specific IgE antibodies is a reliable marker of type I allergy. Future studies should evaluate whether measurement of airway inflammatory biomarkers in bronchial epithelial cells would be an appropriate method of assessment.

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