

occurrence of allergic reactions with two main peaks: in March-April and in May-June. The dates for these peaks vary by about one month from one year to the next. At the local level many phenomena can be observed: an increase in symptoms at the end of January around the Mediterranean region, a peak of activity in late August and early September in the Rhône valley. . . At their peaks, the IAS values are 50 to 100% greater than in periods where there is no pollen activity.

**Conclusion:** The peaks in allergic reactions described by the IAS are concomitant with the main peaks in pollen activity: Cypress in late winter around the Mediterranean, broadleaved trees and then grasses at the national level in spring, ragweed in the Rhône valley in late summer. These results are to be compared with those of general practitioner networks. They could be used to better understand the relative importance of the different allergens in terms of public health, as well as the interactions between pollination and pollution.

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### Early airborne allergen exposure is not associated with sensitization in young children

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**Background:** Allergen exposure is intuitively believed to be closely associated with the development of allergic sensitization in childhood. However, there is little evidence to substantiate such relationship. The objective of this study was to investigate the association between pre- and early postnatal airborne allergen exposure and sensitization to aeroallergens during childhood.

**Method:** Allergic sensitization to cat, dog, and house dust mites was diagnosed longitudinally from skin prick tests and specific IgE results at ages ½, 1½, 4, and 6 years in 392 children from the Copenhagen Prospective Study on Asthma in Childhood<sub>2000</sub> birth cohort born to mothers with asthma. Allergen exposure was defined as presence of dog or cat in the home during the 3<sup>rd</sup> trimester of pregnancy and the child's first year of life. Furthermore, objective measurements of dog, cat, and house dust mite allergen levels in bed dust samples were collected at age 1 year. Associations between exposure and allergic sensitization were analyzed by logistic regression.

**Results:** Allergic sensitization ever to dog was seen in 6.8% of the children ( $N = 27$ ), cat in 7.1% ( $N = 28$ ), *D. pteronyssinus* in 9.8% ( $N = 39$ ), and *D. farinae* in 9.1% ( $N = 36$ ). We found no association between pet exposure in early pre- or postnatal life and sensitization during childhood. Similarly, there was no association between levels of dog, cat, or house dust mite allergens in bed dust and sensitization during childhood. The results were adjusted for mother's sensitization and stratified for the child's eczema status.

**Conclusion:** Early life pre- and postnatal aeroallergen exposure does not seem to affect development of sensitization during childhood questioning the relevance of allergen avoidance.

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### Higher exposure to outdoor air pollution during infancy increase the risk of development of allergic rhinitis

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**Background:** The relationship between air pollution and allergic diseases in children is not yet fully understood, with epidemiological data being a matter of debate. The aim of this study was to investigate the association between exposure to outdoor air pollution during the first year of life and the development of allergic diseases in children.

**Method:** A nationwide cross-sectional survey was conducted in the first grade students from randomly selected 45 elementary schools. The participants of this survey were selected using a stratified two-stage cluster sampling design. Prevalence of physician-diagnosed atopic dermatitis (AD), allergic rhinitis (AR), and asthma was obtained through the Korean version of ISAAC (International Study of Asthma and Allergies in Childhood) questionnaire. Daily ambient concentrations of sulfur dioxide (SO<sub>2</sub>), nitrogen dioxides (NO<sub>2</sub>), ozone (O<sub>3</sub>), carbon monoxide (CO), and particulate matter with an aerodynamic diameter of 10 µm or less (PM<sub>10</sub>) during the first year of life were monitored from 235 monitoring sites throughout the nation. In this study, children who lived within 2 km from the nearest monitoring sites were selected and analyzed. Multiple logistic regression analyses were conducted to examine the association between the levels of air pollution and the prevalence

of allergic diseases, adjusting for the gender of the children, passive smoking, allergic diseases of the parents, education levels of parents, and income of family.

**Results:** Among the 4003 children, we finally selected 1828 subjects as study population through the exposure estimation process. For each increase of 0.1 ppm in daily mean and maximum CO, The adjusted odds ratio (aOR) for the presence of AR were 1.10 (95% CI: 1.03–1.19) and 1.05 (95% CI: 1.00–1.11), respectively. In addition, the aOR per increase of 1 ppb in daily maximum SO<sub>2</sub> was 1.05 (95% CI: 1.00–1.07) for the presence of AR. However, exposure to SO<sub>2</sub>, PM<sub>10</sub>, NO<sub>2</sub>, CO, and O<sub>3</sub> during the first year of life was not related to the development of AD and asthma.

**Conclusion:** Higher exposure to outdoor air pollution during the first year of life such as SO<sub>2</sub> or CO increase the risk of development of AR.

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### Particulate air pollution is associated with increased inflammatory and allergic symptoms in adolescents

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**Background:** The northern part of Belgium (Flanders) has among the highest annual concentrations of air pollutants (PM<sub>10</sub> and PM<sub>2.5</sub>) in Europe. The prevailing EU limit of 50 µg/m<sup>3</sup> has been exceeded on 35 days in 2013. This can mainly be attributed to Western civilization hallmarks such as dense traffic and urbanization. At the same time, the prevalence of allergic sensitization in children and adolescents keeps on rising.

**Method:** Six hundred adolescents were recruited as part of the Flemish environmental health surveillance program (FLEHS3). In this study, it was hypothesized that exposure to particulate matter was associated with respiratory and allergic symptoms and related biological markers. The individual exposure to particulate matter (PM<sub>2.5</sub> and PM<sub>10</sub>; ppb) at the home address was estimated based on an interpolation model and the monitored immission levels. pH of exhaled breath and exhaled nitric oxide (NO) was measured in 408 participants to monitor the inflammatory status of the airways. In urine samples of these 408 adolescents the concentration of 8-hydroxy-deoxyguanosine (8-OHdG) was

determined as a measure of systemic oxidative stress. Symptoms related to asthma, hay fever, eczema and respiratory infections were self-reported using a standardized questionnaire. Multiple linear or logistic regression models were used to calculate the change of effect for an increase of the exposure from P25 to P75.

**Results:** Multiple regression analysis showed 1.5% acidification of breath (95% CI = 0.1–3.0%) with an increase of PM10 exposure. The urinary concentration of 8-OHdG increased by a factor 1.065 (95% CI = 1.007–1.126) when PM2.5 changed. In addition, augmenting exposures to particulate matter (PM10 as well as PM2.5) were associated with more frequent reporting of allergies to household and personal care products (PM10: OR = 1.665, 95% CI: 1.182–2.344; PM2.5: OR = 1.834, 95% CI: 1.240–2.714), as well as allergies to pets (PM10: OR = 1.952, 95% CI = 1.284–2.967; PM2.5: OR = 1.875, 95% CI = 1.202–2.923). No significant associations were established between these air pollutants and exhaled NO. Prevalence of hay fever, eczema, asthma nor infections were correlated with PM exposure. All models were adjusted for gender, age, smoking status, familial allergy (only for respiratory and allergic symptoms) and a priori defined covariates.

**Conclusion:** The associated inflammatory and allergic symptoms confirm that the immune system is a target organ for particulate air pollutants.

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### Statistical analysis of the relationships between aeroallergens (pollen activities) and asthma admissions to hospitals in Abu Dhabi Emirate

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Environmental factors have significant effects on the prevalence of asthma that depend on their state. Primarily, the asthma is a problem that cannot be resolved easily without research to determine its causative agents. In Abu Dhabi the number of asthmatic patients is increasing significantly especially among young ages (<15 years

old). This paper aims to evaluate how urban air quality and vegetation cover conditions are affecting people with asthma in Abu Dhabi city. In this study, a scientific analytical research has been carried out to study the environmental factors that are triggering this chronic disease based on hospital admission data recorded between 2011 and 2013 in 12 public hospitals located in Abu Dhabi Emirate. Bi-weekly vegetation maps have been generated from NASA's high resolution satellite (LANDSAT) to assess the effects of vegetation (mainly pollen activities) on the number of asthmatics' admissions. In this regard, additional effort has been made to have better understanding on how pollen activities can be detected by Earth observation satellites. The importance of the monitoring has been discussed together with the other ways in which the monitoring becomes an effective technique of evaluation. It applies indexing strategies including the remote sensing from satellites. The Normalized Difference Vegetation Index (NDVI) has been used to classify the vegetation into different greenness indices. Seasonal variation of NDVI was found to have an effect on the variability of asthma cases. Descriptive analysis showing the correlation between vegetation variability and the admissions from various governmental hospitals and clinics was performed. Lastly, the vegetation variability was associated with rising cases of asthma admissions. It was concluded that vegetation condition has a direct effect on asthma-affected population with  $R^2 \approx 0.5$  and  $p$ -value ( $\leq 0.05$ ).

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### The effect of air pollutants on chronic cough

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**Background:** Chronic cough, defined as a cough of >8 weeks, is a common symptom for which patients seek the hospital. Three common underlying causes of chronic cough were asthma, reflux disease, upper

airway cough syndrome (UACS), however, some patients are not found to have a definable underlying cause. We investigated the association between the exposure of air pollutants and chronic cough.

**Method:** We reviewed retrospectively the clinical data of subjects who underwent skin prick tests to aeroallergens, induced sputum analysis, and methacholine bronchial provocation test between January 2011 and December 2013 to evaluate their chronic cough. We analyzed the effect of the ambient particulate matter (PM10), sulfur dioxide (SO<sub>2</sub>), nitrogen dioxide (NO<sub>2</sub>), ozone (O<sub>3</sub>), and carbon monoxide (CO) on chronic cough.

**Results:** A total of 444 subjects were analyzed (mean age: 49.5 ± 14.3, female 68.9%). Airway hyperresponsiveness (AHR) was demonstrated in 6.3%; sputum eosinophilia, in 13.1%; and sensitization to at least one aeroallergen in skin tests, in 25%. Their diagnoses were asthma or eosinophilic bronchitis (19.4%), UACS (66.0%), reflux disease (5.0%), chronic obstructive pulmonary disease (1.1%), and idiopathic chronic cough (31.3%). In the patients suffering from chronic cough, non-asthma patients were exposed the higher level of air pollutants, PM10 (48.8 vs 46.5 µg/m<sup>3</sup>,  $P = 0.014$ ), NO<sub>2</sub> (21.0 vs 19.7 ppm,  $P = 0.005$ ), and CO (4.1 vs 3.9 ppm,  $P = 0.013$ ), comparing to asthma patients. The pollutants exposure also was higher in idiopathic chronic cough patients; NO<sub>2</sub> (21.3 vs 20.5 ppm,  $P = 0.024$ ), and CO (4.2 vs 4.0 ppm,  $P = 0.017$ ). AHR and sputum eosinophilia were not associated with air pollutants. However, in idiopathic chronic cough patients, neutrophilic airway inflammation was related with PM10 ( $\rho = 0.209$ ,  $P = 0.033$ ), NO<sub>2</sub> ( $\rho = 0.269$ ,  $P = 0.006$ ), and CO ( $\rho = 0.308$ ,  $P = 0.001$ ).

**Conclusion:** Air pollutants had an effect on idiopathic chronic cough, and were related with neutrophilic airway inflammation.