

The increasing trend of seasonal respiratory allergy in urban areas

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Man has always been exposed to airborne pollen which is potentially responsible for seasonal allergic symptoms and prehistoric pollen grains have been found in rocks millions of years old. However, pollinosis was identified and systematically described only in the 19th century and was rare until the 20th century. The leap from the identification of airborne pollen in the etiology of seasonal respiratory symptoms due to Charles Blackley in 1864 (1) to what, just one century later, has become the 'epidemic of the third millennium' is probably due to changes in lifestyle and the environment (2–4).

Now evidence suggests that respiratory allergic diseases are increasing in both prevalence and severity in most industrialized countries, and subjects living in urban areas are more likely to experience allergic respiratory symptoms, particularly those induced by pollen allergens, than subjects living in rural areas (3–5). This increase has been linked, among other factors, to urban air pollution which increases airway responsiveness to aeroallergens (6–8). Moreover, pollen grains dispersed in the atmosphere can interact with other airborne substances, such as components of air pollution. It has been hypothesized that pollutants can influence the way a pollen, once inhaled, is processed. In particular, T-cell responses to major allergen proteins may be skewed in a Th2 direction in the presence of pollutants such as diesel exhaust particles (DEP) (9). Furthermore, airway mucosal damage and impaired mucociliary clearance induced by air pollution may facilitate the access of inhaled allergens to the cells of the immune system. In addition, vegetation reacts with air pollution and

environmental conditions and influence the plant allergenicity. Several factors influence this interaction, including type of air pollutants, plant species, nutrient balance, climatic factors, degree of airway sensitization and hyper-responsiveness of exposed subjects.

Pollenosis represents a distinct phenotype of patients with rhinitis, conjunctivitis and asthma where symptoms at the level of various target organs usually occur together and are more influenced by immediate hypersensitivity than by significant inflammatory events *vs* other forms of asthma, rhinitis or conjunctivitis. Pollenosis is still the most frequent challenge for the practising allergist and we are convinced that the pollenosis patient represents a distinct clinical phenotype that requires a transversal and unitary clinical approach, with a specific diagnostic flowchart and treatment strategy (3). However, the last 25 years or so have seen tremendous progress in aerobiological and allergological studies. Phenological observations of allergenic plants and international aerobiological networks now provide pollen counts and hay fever forecasts. On the other hand, attempts to classify the most allergenic types of pollen have come from immunological and clinical studies. Pollen calendars are now commonly consulted by hay fever sufferers and physicians and they represent good indices of potentially troublesome pollen. Obviously, seasonal maps of pollen counts are merely 'a guide' to potential exposure in a given area and must be continually updated.

Among the 250 000 well-described, pollen-producing plant species, less than 100 are significant in terms of pollen allergy. In this context there

is Oleaceae family and Olive pollen (10,11), which has strong allergenic properties in the Mediterranean, where it reaches urban areas from suburbs, where this tree grows, contributing to the appearance of seasonal (from May to the end of June) allergic symptoms (oculorhinitis and/or bronchial asthma) in citizens besides subjects living in their proximity.

The Oleaceae family includes at least 400 species of woody, arboreal or shrubby, evergreen plants. They can be spontaneous (*Olea*, *Fraxinus*, *Phillyrea*, etc.) or cultivated, like privet, lilac, jasmine and olive, for ornamental, medicinal or food purposes. Olive is a long-lived tree that has been grown by man for more than 5000 years for its fruit, oil and wood. It probably originated from Asia Minor, from whence it spread across the Mediterranean basin. About four centuries ago, the *Olea* was introduced into the Americas (in North America, prevalently in California and Arizona, and in South America, mainly Chile). The olive tree is also grown in Australia and South Africa. In the last few years, increasing interest has been devoted to immunological, clinical and aeobiological aspects of this pollinosis. Extensive immunological cross-reactivity has been demonstrated between the

pollen extracts of the Oleaceae species such as olive, ash, privet and lilac (12). As a consequence, sensitivity to the pollen of one species may favour development of sensitivity to all four species in susceptible subjects. In addition there is a perennial pattern of clinical symptoms in patients, adults and children, monosensitized to *Olea europaea* pollen allergens in comparison with subjects with Gramineae and Parietaria pollinosis (13).

Olea is not present in Northern Europe where we find the ash tree (*Fraxinus*) and the privet (*Ligustrum*) (14). *Fraxinus* commonly grows in woods and is planted in gardens, parks and streets. *Ligustrum vulgare* is a shrub growing in light woods, and *L. ovalifolium* is very popular as a garden-separating hedge. *Ligustrum* pollen is present in very low concentrations. Only in conditions of local exposure (e.g. when cutting the *Ligustrum* hedge) can pollinosis or pollinosis-like symptoms develop. However, we need to consider that a pollinosis patient allergic to *Olea* pollen and living in the Mediterranean area may, when travelling to Northern Europe, present allergic symptoms due to inhalation of pollen from other Oleaceae, such as *Fraxinus* (14).

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