8. Allergy and epidemiology

Say what the use, were finer optics given,
To inspect a mite not comprehend the heaven?
Or touch, if trembling alive all o’er,
To smart and agonise at every pore.

Alexander Pope, 1733,
An Essay on Man.

8.1 Introduction
This chapter deals with the allergic disorders associated with dust mite allergens, principally allergic asthma, atopic eczema and allergic perennial rhinitis. The main theme is the natural history of allergen exposure, concentrating on allergen levels and mite population densities in relation to temporal and spatial variation in the prevalence of these disorders, particularly atopic asthma. I do not review epidemiology of asthma or allergy more generally. This topic has been investigated in two large, long-term, international studies of asthma and allergies in adults and children respectively: the European Community Respiratory Health Survey (ECRHS; Burney et al., 1994; European Community Respiratory Health Survey, 1996; Janson et al., 2001; Sunyer et al., 2004; www.ecrhs.org) and the International Study of Asthma and Allergies in Childhood (ISAAC; International Study of Asthma and Allergies in Childhood Steering Committee, 1998; Asher et al., 2006; Pearce et al., 2007; Ait-Khaled et al., 2007; http://isaac.auckland.ac.nz). Several ‘environmental’ factors have been examined in these studies, including effects of climate (Verlato et al., 2002; Weiland et al., 2004), economic status (Stewart et al., 2001), diet (Ellwood et al., 2001) and mite allergen exposure (Zock et al., 2006). Although there were differences in absolute prevalence values between the two studies, in general the patterns of prevalence were similar: low in Eastern Europe and higher in Western Europe with a strong northwest-southeast gradient, and highest in English-speaking countries (Pearce et al., 2000).

The story of the relationship between dust mites, allergy and asthma is complicated by the fact that atopy, the genetic predisposition to make IgE antibodies to common allergens, may or may not be associated with the presence of disease, and that asthma, eczema and rhinitis can be provoked by agents other than allergens and may not be associated with atopy. Therefore, the disease states of relevance to this chapter are those combining allergen-specific IgE antibodies and/or a positive skin-prick test to dust mite and other common airborne allergens; where there is a family history of atopy; and where patients have active asthma, eczema and rhinoconjunctivitis. Nomenclature of allergy and allergic diseases was revised by Johansson et al. (2001) and Figure 8.1 indicates the overlap between atopy and allergic disease...
based on this classification. For practical purposes in epidemiological studies, atopy has been defined by a positive skin-prick test to common airborne allergens such as those of dust mites, cats, dogs, pollens, cockroaches and various moulds. If the proportion of people with asthma who have specific IgE or positive skin-prick tests is known, the population attributable fraction of asthma due to atopy can be calculated (Pearce et al., 1999, 2000; Sunyer et al., 2004; Weinmayr et al., 2007).

### 8.2 Diseases associated with dust mites

Over the years, dust mites have been implicated in several diseases and disorders, mostly with an allergic basis. The evidence for the associations ranges from very high to possible for most of the allergic conditions, and low to non-existent for the non-allergic ones.

#### 8.2.1 Allergic diseases

**a. Allergic asthma**

Asthma is probably not a single disease. It is hard to characterise unambiguously, but it involves wheeze, shortness of breath, airway narrowing and inflammation. However, the wide variety of provoking agents and the variable time course of symptoms in childhood and adulthood point towards the existence of different types of asthma – so-called phenotypes. The recognition and classification of different phenotypes has been around for many years, and non-allergic asthma and allergic asthma is one of the better known broad classifications. But it is not clearly understood whether these phenotypes represent clinical manifestations of different underlying diseases or whether they are different stages in the progression of the pathology of a single disease – inflammation of the airways – that presents differently in different people according to their susceptibility to different provoking agents. Classifications of asthma phenotypes are starting to emerge, with improved characterisation derived from large-scale epidemiological studies and clinical trials of patients with particular phenotypes (Wenzel, 2006).

A large proportion of spending on healthcare is devoted to asthma and it accounts for massive loss of time from school and work, with associated productivity losses (Weiss et al., 1992). Asthma has been the subject of billions of dollars worth of research funding involving thousands of researchers, and yet we still have no clear understanding about how to prevent it, despite many developments in our knowledge, despite it being the subject of tens of thousands of research papers and several major textbooks (e.g. Barnes et al., 1997; Naspitz et al., 2001; Gershwin and Albertson, 2001; the British Library Catalogue has over 1500 records of books with the word ‘asthma’ in the title). What has happened is that we have realised that it is complex, challenging, elusive, and that many of the concepts that were received wisdom 10 years ago have since been discarded or are being re-evaluated.

Asthma is among the most common chronic diseases of childhood, particularly in urbanised, English speaking countries with a high standard of living. While treatable, asthma has increased in prevalence.