

ALLERGY PRACTICE FORUM

Hayfever in the United Kingdom

Introduction

The first description of hayfever (seasonal allergic rhinoconjunctivitis) was probably in 865 AD, and recorded in Arabic text by Mohammed Al Razi. The first reported cases in Europe did not occur until the 16th century, with the first description in England by Dr John Bostock (1819) [1] who presented himself as a case of 'periodical affliction of the eyes and chest' to the Medical Society of London. At that time the condition was unknown to the Society and Bostock attributed his affliction to heat and rays from the sun. By 1873, Charles Blackley had established the cause of hayfever to be pollen [1]. By the time William Lloyd wrote 'Hayfever—its causes, diagnosis and treatment' (1907) hayfever was considered to be very common indeed and a consequence of urbanization and agricultural changes.

In the U.K., allergy to airborne pollen is very common with grass pollen allergy being predominant. Typical symptoms included conjunctival itching and redness, nasal and palatal itching, rhinorrhoea and sneezing with or without seasonal asthma. Difficulties can sometimes arise in distinguishing pollen allergy from the common cold. In these situations, the presence of eye symptoms will be the clue. Pollen grains are large (average 20 μm) and impact easily on to the eye from laminar air flow over the body [2]. As a result, pollens readily produce eye symptoms in contrast to smaller particles such as moulds or house dust mites which are respirable. Sunglasses can be effective in reducing eye symptoms during the summer.

The timing of a patient's symptoms can indicate the type of pollen allergy (see Fig. 1): tree pollen—March to May; grass pollen—June to July; weeds—August to September.

Tree pollen allergy affects a large number of people. During the spring, tree pollen counts are commonly over 1000 m^{-3} , in contrast to grass pollen which seldom exceeds 150 m^{-3} . Despite this, grass pollen allergy is predominant in the U.K.

Pollen counting

The Asthma Research Council (now known as the National Asthma Campaign) has published pollen counts since 1963. These have been obtained from the roof of St Mary's Hospital in London. The counts can only act as a reference, since they may vary considerably over short distances depending on local sources and conditions [3]. As a result, patients may fail to appreciate pollen

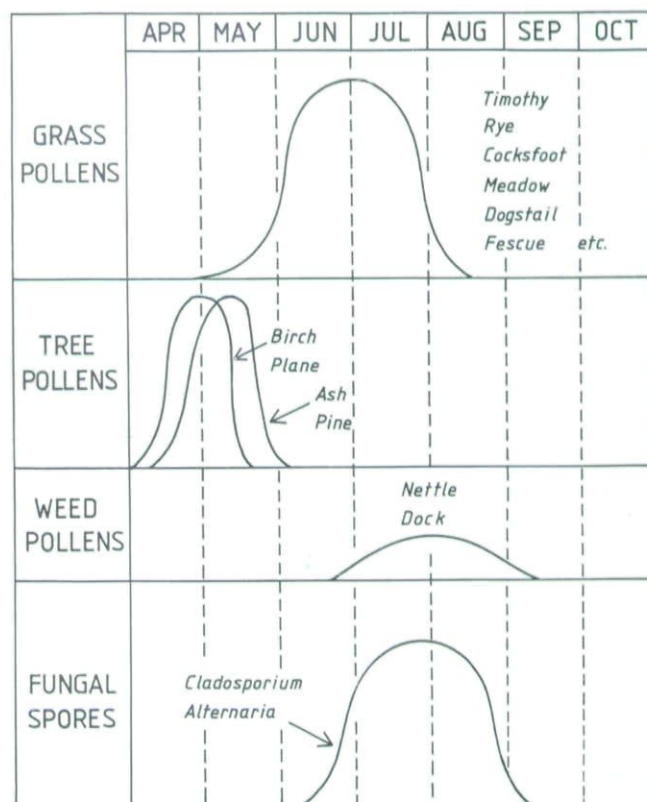


Fig. 1. A calendar of common seasonal aeroallergens in the U.K. (courtesy of Professor A. B. Kay).

exposure as a cause of their symptoms on days when the reported pollen counts are low.

For research purposes, hayfever symptoms should be related to local pollen counts. There are now many reference centres throughout the U.K. which record pollen counts, although few have recorded data for more than 10 years [4].

There are two devices (Hirst and Burkard) in current use for pollen counting [5]. Aerodynamically they are very similar, and draw air at a fixed rate (10 l/min) over a sticky slide or rotating drum, where particles from the air impact. After fixing and staining, the different pollens and spores can be readily identified. Counts are expressed per cubic metre of air with calculation from the duration of sampling and the rate of airflow through the trap. The Hirst spore trap provides only daily counts in view of the need to replace the slide every 24 hr. With the Burkard trap (which has a rotating drum), collections can continue

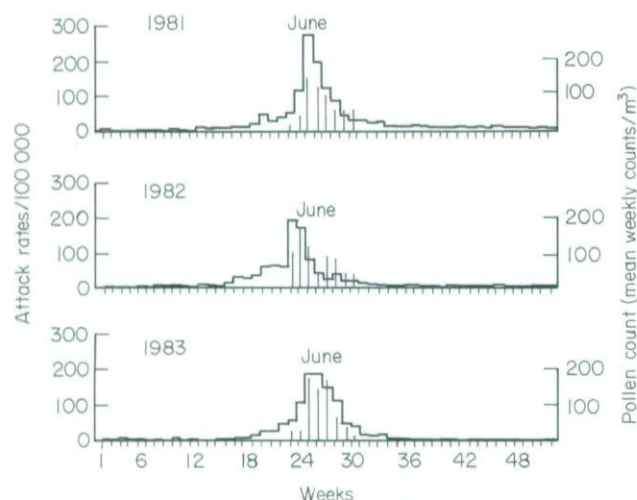


Fig. 2. Weekly attack rates for hay fever (1981–1983) and the grass pollen counts per week. Reproduced with permission from Ayres (1986) [6].

for seven days without attention. Thus all pollen counts are reported retrospectively.

Pollen counts above 50 m^{-3} are considered high [5], but not all potential sufferers will be symptomatic at this level. Patients with low sensitivities may only have symptoms for a few days of the season when the counts are at their highest. Patients with high sensitivity may suffer for 10–12 weeks depending upon the onset and duration of the season. In view of the wide range of individual sensitivities, and concurrent exposure to other allergens, it is difficult to establish a threshold level above which the majority of patients will be symptomatic [2]. There is, however, a unimodal increase in hayfever symptoms [6] which peaks at mid-late June each year (Fig. 2).

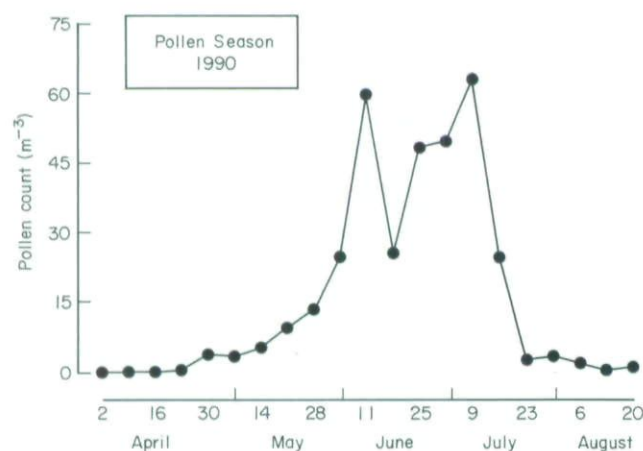


Fig. 3. Average weekly grass pollen counts for London 1990. Courtesy of Jane Norris Hill (Polytechnic of North London).

The pollen season

Figure 3 shows the grass pollen season for London in 1990. There is an early onset to the season (15 May) with peak counts occurring on 11 June and 9 July. The duration of the 1990 season was 10 weeks with a fall in pollen count due to rainfall observed during June. Many factors are known to influence pollen counts during the season. The ripening of grass pollen begins earlier in the south and west owing to the earlier rise in seasonal ambient temperatures. Pollen is shed in southern Cornwall 5 weeks earlier than northern Scotland. The pollen season in the north of the country is therefore shorter because the season finishes at the same time throughout the U.K. [7] (Fig. 4).

The pollen season usually begins between the last week in May and the second week in June. Studies in the south-east have shown that the onset may be predicted to within three days by the cumulative temperature [5]. Generally a mild spring, with daily temperatures in April and May averaging 21°C will ensure an early start to the season. Rainfall has not been shown to enhance the growing season, although drought conditions during the flowering season will destroy grass and therefore limit the season. Even if the seasonal influences are favourable, pollen

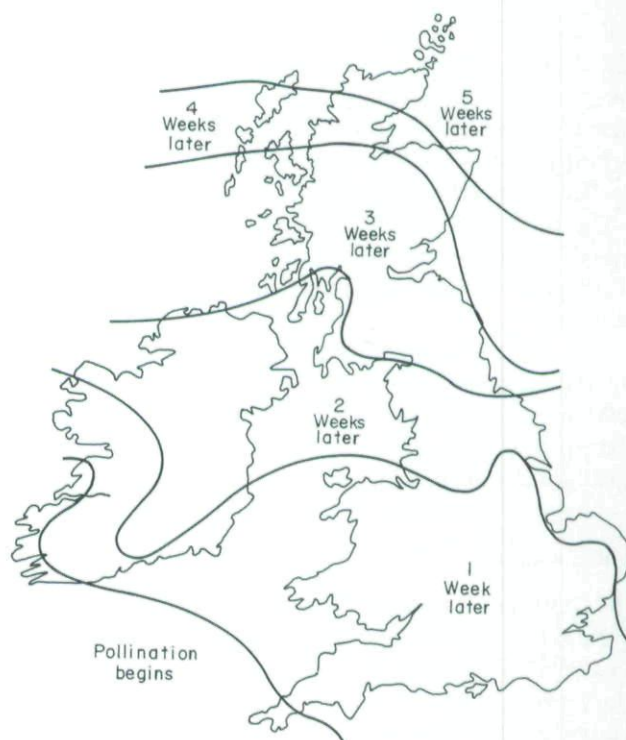


Fig. 4. Pattern of pollen release in the U.K. (courtesy of Fisons Pharmaceuticals).

counts in a collection of islands like the U.K. will vary considerably [7]. In coastal areas with onshore breezes, pollen counts will be reduced, since air currents over the sea will not collect pollen. In mountainous areas with intervening valleys, pollen counts tend to be low. This is due to the combined effect of high convection carrying pollen into the air [2] and winds carry the pollen moving across the mountains without descent into the valleys. In addition, the grassland of mountains and moorlands are generally lower pollen producers and rainfall is usually high [3]. In towns, rising warm air currents reduce pollen at street level whilst giving high exposure to those at open windows in tower blocks. Buildings provide shelter from the wind, thereby reducing pollen counts at street level. In parks and open spaces, pollen counts may be very high.

Climatic factors

Rainfall of more than 2 mm removes pollen from the air and the 'take-off' of pollen is reduced when vegetation is wet. Overnight rainfall is unlikely to reduce the next day's pollen count unless rainfall continues until after 6 a.m. [2]. Strong winds decrease pollen counts by removing pollen away from the earth's surface. Winds moving over grassland will carry high amounts of pollen to nearby towns. In London during the summer, there is a prevailing south-westerly wind [7]. The grassland distribution around London [5] is asymmetrical with the highest pollen counts occurring on days when the wind blows from the north-west. Warm dry conditions with bright sunshine trigger anthers to open and release pollen. A succession of sunny days will rapidly raise the pollen count early in the season when pollen production is plentiful. In contrast high humidity reduces pollen release.

Grass pollen release, in common with other flowers and plants, is maximal mid-morning and late afternoon [2]. Released pollen rises into the air producing peak counts

between 6–7 p.m. in the country and after 10 p.m. in urban areas. These differences relate to the higher temperatures in town which may result from industrial processes. In the evening, as the air cools, pollen settles on the ground. Here again, there is a time difference, with deposition after 8 p.m. in rural areas and after 2 a.m. on a warm evening in town. This accounts for the increase in hayfever symptoms during the evening and at night. Patients frequently report waking in the early hours with rhinitis and asthma. This effect can be reduced by avoiding country walks after dusk and closing windows during the evening and at night.

Prevalence of hayfever

Hayfever in the U.K. has changed from a disease which was virtually unrecognized 200 years ago [1], to one in which there are now over one million GP consultations per year. The current prevalence of hayfever is between 10–15% [8] with a male predominance (Table 1). Hayfever is diagnosed less frequently in Wales and north-west England compared with the east and south-east [9]. This difference is supported by reduced prescriptions for anti-allergic medication [10,11]. The reasons for these regional differences are unclear. A shorter pollen season in the north of England, with less favourable weather conditions may be partially responsible. Alternatively, social factors may play a role. Over the past 30 years there has been a fourfold increase in the prevalence of hayfever in the U.K. [8]. The reasons for this increasing prevalence are unclear, although it cannot be explained by changes in diagnostic fashion or a greater awareness of the condition alone. The figures reflect a true increase (Table 2) because the towns studied had stable populations and few general practitioner changes during the study period.

Pollen data shows that June grass pollen counts in London have tended to fall over this period (i.e. the last 20 years) [12] (Table 3). Changes in agricultural practice may, at least in part, account for this fall. For example, there has been a change in grassland composition from Timothy grass to the lower pollen producing rye grass. These changes occurred after the Second World War,

Table 1. Age distribution of hayfever consultation (per 1000 population). Data from the Office of Population Census and Surveys (1982). Royal College of General Practitioners.

0–4	12.6
5–14	48.0
15–24	47.2
25–44	29.8
45–64	12.6
65–74	8.9
75+	4.0

Table 2. Consultation per 1000 population for hayfever (male and female) in the U.K.

5.1/1000	1955–1956
10.6/1000	1970–1971
19.8/1000	1981–1982

Table 3. Average U.K. regional pollen counts for years 1986–1989

	Pollen count (m^{-3})
Cardiff	35 600
Glasgow	27 148
Isle of Wight	14 433
Rotherham	10 768
London	9 541

when excess agricultural land was reseeded without the usual Timothy/cocksfoot/rye grass mixture [12]. Animal stocking has doubled since 1940. The resulting increases in grazing prevent grass from flowering. In addition, silage production has increased tenfold over the past 20 years. Grass for silage is cut early in the season long before it flowers, and is stored in a pit or silo.

Hayfever and pollution

Another factor of possible importance in increasing the prevalence of hayfever may be pollution. For example, in the U.K. air pollution from cars has increased substantially in the past 30 years [13]. Exhaust fumes may have an adjuvant effect on pollen antibody production in animals. Pollens from industrial areas in Great Britain are coated with pollutants [14]. In addition, sulphur dioxide and nitrogen dioxide from car exhausts are associated with damage to the nasal mucosa [15]. While there are currently no studies in the U.K. which support an association, it is interesting to review studies from abroad. In Japan, where the Japanese cedar tree has flourished for a million years, there were no reported cases of allergy to cedar pollen before 1964. Since then, pollution has increased considerably, with the number of cars increasing from 15 000 to over 5 million. Recent studies of school children in urban polluted districts have shown 33% to have allergic symptoms and positive skin tests to Japanese cedar tree pollen compared with rural areas [16]. Studies of automobile exhaust particles show 65% to be of respirable size (below $5 \mu\text{m}$). Many of these particles are carbonaceous and have been shown to absorb the major allergens of Japanese cedar [16]. Diesel exhaust fumes have also been shown to be an adjuvant for antibody production *in vivo*. Intraperitoneal sensitization with a Japanese cedar pollen extract in mice failed to produce sensitization unless administered with diesel exhaust particles.

Pollen avoidance

Grass pollen avoidance is virtually impossible in every day life. Simple precautions such as wearing sunglasses, closing car windows and avoiding parks and evening country walks will help to reduce exposure. In addition, windows should be closed from the late afternoon onwards. Grass cuttings should be avoided by an affected person and some relief is obtained from holidays by the sea [17].

Skin tests and serological diagnosis

Diagnosis of hayfever can usually be made by a careful clinical history alone, without recourse to skin tests or serological investigations [17]. In the presence of a clear history, skin testing is not essential. However, a positive skin prick test provides helpful supportive evidence and may be reassuring for the patient. Skin prick tests are simple, quick and relatively inexpensive. They can be performed after a short period of instruction and may be applied to a wide range of allergens. In patients who are uncertain about the timing of their symptoms, prick testing may help to identify the relevant allergen and enable medication to be given before the onset of symptoms [18]. In the presence of extensive skin disease or dermatographia, skin prick testing may be unreliable and serological tests may be indicated [19]. Blood measurement of antigen-specific IgE can be carried out by RAST, ELISA or CAP etc. (radioallergosorbent test, enzyme-linked immunosorbent assay), but are rarely indicated in routine diagnosis and treatment [20]. Special circumstances include skin disease (as above), concurrent treatment with antihistamines, or when the relevant allergen for prick testing is not available.

Symptomatic treatment

Major therapeutic advances in the treatment of hayfever in recent years include the availability of H_1 -selective histamine receptor antagonists with a low sedative profile and the development of intranasal corticosteroid preparations for regular prophylactic use. For occasional symptoms, intermittent oral antihistamines may be all that is required. For more prolonged symptoms, prophylactic treatment with both intranasal and inhaled steroids or sodium cromoglycate (SCG) is indicated [21]. Preventive medication should be commenced 2–3 weeks before the season. In the majority of patients, this medication will be effective. Failure should raise the question of patient compliance with treatment. SCG or antihistamine eyedrops are effective for allergic eye symptoms. Palatal and throat itching responds best to oral antihistamines,

which will also reduce eye symptoms, nasal itching, sneezing and rhinorrhoea. Antihistamines have little effect on nasal blocking or asthma [20]. Patients inadequately controlled by the above measures may require an occasional course of oral corticosteroids. Prednisolone 20 mg daily for 7 days is usually very effective. Oral prednisolone is preferred to depot corticosteroids such as triamcinolone. Intramuscular doses are uncontrolled and tend to be high. Muscle atrophy can occur at the injection site. The potential risk of acute systemic side-effects may also be increased with intramuscular preparations. With oral steroids, careful adjustment of the dose (with discontinuation if necessary) is easily achieved. Intranasal corticosteroids when used twice daily have virtually no risk of systemic side-effects [22]. Aqueous formulations are preferred to the freon aerosols. Aqueous preparations have a reduced risk of mucosal drying, crusting or bleeding whilst producing a more uniform distribution of medication. Although SCG may be used prophylactically, patients often report immediate relief (within minutes) of both eye and nasal symptoms after topical use. The preparation is of particular value in children where even topical corticosteroids are usually avoided.

Terfenadine, astemizole, acrivastine, loratidine and cetirizine have all been shown to be as effective as conventional antihistamines but with a reduced risk of sedation [23]. Few comparative trials of these new drugs are available. Terfenadine is inexpensive, effective and available 'over the counter'. Cetirizine has a theoretical advantage through its additional 'anti-eosinophil' property [24], although the clinical relevance of this finding is not yet established. Cetirizine and loratidine have been shown not to interact with alcohol [24]. Acrivastine is short acting and therefore may be used as a 'rescue' medication, which may be an advantage when symptoms occur only infrequently and a regular antihistamine is not justified.

Topical sympathomimetics provoke vasoconstriction which may reduce mucosal oedema and therefore nasal blockage. Their use for longer than a few days will carry the risk of rebound rhinitis medicamentosa [22]. These drugs can successfully relieve temporary nasal blockage when it threatens to impede the delivery of corticosteroids or SCG, but they are not recommended for long term use. The use of sympathomimetics (even topical formulations) is not recommended in patients with cardiovascular or thyroid disease.

Immunotherapy

A minority of hayfever sufferers have extreme hypersensitivity to grass pollen and respond poorly to standard anti-allergic medication. In these individuals, immuno-

therapy (hyposensitization) would be the treatment of choice in many countries throughout the world including the U.S.A. and the continent of Europe [26]. In the U.K., allergen injection immunotherapy for the treatment of allergic disease including summer hayfever, has been largely discontinued as a result of recommendations by the Committee on Safety of Medicines Update in October 1986 [2]. Concerned about the number of deaths (largely in asthmatics), the Committee questioned the efficacy and safety of immunotherapy and recommended a 2 hr post-injection observation period with access to full resuscitation facilities. In addition, it recommended that the potential benefits of treatment must be weighed against its known risks. In Europe and the U.S.A. many studies of grass pollen immunotherapy have shown efficacy [25], and it is readily used in conjunction with standard medication. A recent double-blind placebo-controlled study in the U.K. using Alutard SQ (ALK, Horsholm, Denmark) (a partially purified and highly standardized grass pollen extract) showed marked efficacy in patients with severe hayfever uncontrolled by anti-allergic medication [27]. In this study, the safety and efficacy of immunotherapy was evaluated in 40 patients (mean age 35 years) with severe grass pollen allergy. Patients with perennial asthma or other clinically significant allergies were excluded from the study. Throughout the study symptom and medication requirements were recorded by diary cards, with visual analogue scores recorded during the grass pollen season. Objective tests, in the form of skin and conjunctival sensitivity, were also assessed before and after 8 months of treatment. The results showed a highly significant decrease in total symptom scores (median 61% decrease, $P=0.001$) in the Alutard SQ treated group with beneficial effect particularly in nasal blockage. Total medication usage was also significantly reduced (median 79% reduction, $P=0.002$) in the Alutard SQ group compared with placebo. Visual analogue symptom scores also showed a median 58% decrease ($P=0.02$) in the actively treated group. Provocation tests demonstrated a significant reduction for the active group for conjunctival allergen sensitivity (tenfold reduction, $P=0.001$) and in the early (median 40%, $P=0.02$) and late phase (median 57%, $P=0.001$) cutaneous response to intradermal allergen.

A total of 523 active injections were given. There was one systemic reaction at 10 minutes after injection which took the form of flushing and chest tightness and was rapidly reversed by intramuscular adrenaline. There was one mild delayed urticarial reaction at 2½ hr which was not life-threatening and responded promptly to treatment. Immunotherapy with Alutard SQ was considered to be effective with such treatment carried out by trained individuals who would quickly recognize and treat

adverse reactions. In the future, these new vaccines which are well standardized, may have a role in the treatment of pollen allergy in those who fail to respond to conventional treatment.

Summary

In the U.K. allergy to grass pollen is the single most important cause of seasonal allergic rhinoconjunctivitis (hayfever). The prevalence of the disease appears to be increasing, although the precise reasons for this are at present unknown. The introduction of non-sedating H₁-selective antihistamine drugs and local corticosteroids has been an important therapeutic advance. Immunotherapy (desensitization) has a part to play in the treatment of hayfever unresponsive to anti-allergic medication but because of the danger of systemic reactions its use is limited to specialized centres.

Acknowledgments

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