Oilseed rape and bronchial reactivity

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Abstract

Objectives—To investigate atopy and changes in symptoms, peak flow rate, and bronchial reactivity in people complaining of symptoms during the oilseed rape flowering season.

Methods—37 people who had given positive answers to questions about the presence of symptoms in relation to the flowering season of oilseed rape and 24 controls with no such symptoms were studied, although not all took part in all parts of the study. All had been previously identified in a cross sectional survey of a random sample of a rural population. Atopy was assessed by means of skin prick tests, total immunoglobulin E (IgE), and radioallergosorbent test (RAST) assays. Bronchial reactivity (PC₂₀) was measured by histamine challenge. Total IgE and bronchial reactivity were measured both before and during the oilseed rape flowering season. Day to day changes were monitored by subjects who kept a record of their symptoms and peak flow morning and evening, starting before the flowering season and continuing during it and into the grass pollen season.

Results—Only two cases, of 23 tested, showed evidence of allergy to oilseed rape and only 10 of 23 tested, including these two, were atopic. Eye, nasal, and headache symptoms increased in the season in cases, which validated the questionnaire used in the previous cross sectional survey. 12 of 16 cases tested and seven of 15 controls showed a seasonal fall in PC₂₀; the fall in the cases was significantly greater than in the controls. Peak flow charts showed no evidence of fall or of increased variability during the season.

Conclusions—People who complained of symptoms in relation to the flowering of oilseed rape were rarely allergic to the plant and fewer than half were atopic. Nevertheless, they usually showed increased bronchial reactivity during the season, which may have been due in some cases to other allergens but in others to non-specific irritant effects of the air. Whether these are due to chemicals released by the crop, to rising summer ozone levels, or to other factors remains unclear.

(*Occup Environ Med* 1995;52:575-580)

There has been much interest expressed in the potential of oilseed rape to cause seasonal epidemics of respiratory symptoms in people living close to the crop. Claims have been made that the plant is a potent allergen. Although oilseed rape pollen has been shown to cause asthma, there has been much debate about the frequency with which this occurs.¹⁻³ The debate has been fuelled by media interest and many patients consult their general practitioners with eye and respiratory symptoms that they attribute to oilseed rape.

We have previously reported a cross sectional study of a random sample of a rural population in the Grampian region to compare the prevalence of seasonal symptoms in people living in a rape growing area with that in people in a non-rape growing area.⁴ The results showed spring and summer seasonal symptoms to be common in rural communities, whether or not oilseed rape is grown locally. Only a small excess of such symptoms occurred in areas of rape cultivation.

We now report the results of a study of symptomatic and asymptomatic people from the area of oilseed rape cultivation, designed to investigate the relevance to such symptoms of atopy and bronchial reactivity and to assess the validity of the questionnaire used in the initial study.⁴

Methods

SELECTION OF SUBJECTS

The study protocol was approved by the local ethics committee and all subjects gave fully informed consent to their participation. Cases and controls were all selected from those living in an area of high oilseed rape cultivation who had completed the questionnaire in the original study, in which they had been randomly selected from the lists of rural general practitioners.⁴ Cases were defined as those who reported conjunctivitis or respiratory symptoms, or both, only or predominantly in May and June. Controls were those with no seasonal symptoms. In all, 56 cases and 35 controls were invited to volunteer for these further studies, but were not required to participate in all aspects; 37 cases and 24 controls were ultimately able to take part between 1991 and 1993.

INVESTIGATIONS

All subjects were first studied before the oilseed rape flowering season, when a short questionnaire to confirm the presence or absence of symptoms, as determined in the previous questionnaire,⁴ was given by one of us. A clinical history was also taken. Subjects were then examined by skin prick tests, total

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Accepted 25 May 1995

Keywords: oilseed rape, bronchial reactivity, allergy

immunoglobulin E (IgE) concentrations, radioallergosorbent test (RAST) assays, serial peak flow rate records, and a histamine challenge test. The histamine challenge test and total IgE concentrations were repeated where possible during the oilseed rape flowering season. Both tests were carried out at the same time of day in all subjects.

ALLERGY TESTS

Skin prick tests were performed to five common allergens: *Cladosporium herbarum*, house dust mite (*Dermatophagoides pteronyssinus*), grass pollen mix, birch pollen, and oilseed rape (*Brassica napus*). Also positive (histamine dihydrochloride) and negative controls were used. The skin test responses were read after 15 minutes and were regarded as positive if the diameter of the weal was 3 mm or more, corrected for the diameter of the negative control weal if necessary. The RASTs for the above allergens were performed and total IgE was also measured. Subjects using antihistamines were asked to stop them 48 hours before the test.

HISTAMINE CHALLENGE TEST

Subjects who were taking bronchodilators were requested not to use them for six hours before the test. Also, subjects were asked to refrain from smoking for at least two hours beforehand. None of the subjects had had symptoms of a viral illness during the preceding six weeks. The forced expiratory volume in one second (FEV₁) was measured as the best of three blows with a Vitalograph spirometer and no subject had a baseline FEV₁ less than 70% of that predicted. Histamine challenge was performed with a Wright nebuliser driven by compressed air, calibrated to an output of 0.13 ml/min. Saline, followed by successive incremental doses of histamine from 0.03 to 16 mg/ml were inhaled by tidal breathing for two minutes with the nose clipped. The FEV₁ was measured at 30 seconds and 90 seconds after inhalation of each solution. The starting concentration of histamine varied according to baseline FEV₁, the response to the control inhalation, and recent treatment required to control symptoms.⁵ The test was stopped when the FEV₁ had fallen by more than 20% or until the highest concentration had been given. The percentage decrease in FEV₁ was calculated for each concentration with the lower of the 30 and 90 second values each time. The histamine concentration provoking a 20% fall in FEV₁ (PC₂₀) was calculated by interpolation of the last two points on the dose response curve. If a fall of 20% or more was not attained, PC₂₀ was recorded as >16 mg/ml and a value of 16 mg/ml was used in subsequent data analysis. On completion of the test, affected subjects were given inhaled salbutamol if the FEV₁ had fallen by 20% or more, or if they felt wheezy or tight in the chest. Tests were supervised by a physician.

PEAK FLOW AND SYMPTOM DIARIES

All people participating in this part of the

study were carefully instructed in the use of the peak flow meter and in how to complete the symptom diaries. All measurements were recorded twice daily, first thing in the morning and last thing at night. Cough, wheeze or shortness of breath, sneezing or blocked nose, sore or itchy eyes, itching of the skin, and headache symptoms were recorded on a scale of 0 to 2 (0 = no symptom, 1 = mild to moderate symptoms, 2 = bad to severe symptoms). The best of three peak flow readings with a mini-Wright peak flow meter (Clement Clark International) was recorded at the same time. Subjects were instructed that the test should be performed in a standing position and that the marker should be at the bottom of the scale before starting. The test was repeated twice and the highest of the three values recorded in the diary. The first two days were regarded as learning days and readings from these days were omitted from subsequent analyses. Subjects were encouraged to perform the tests at roughly the same times each day and were asked to note the time when recordings were made to check this. Also, any daily medication taken was noted and space for any comments people wished to make was provided. Subjects were also asked to indicate any periods of time spent away from their home area and also if they had a cold or other respiratory infection. Results from any periods of time spent away from home were omitted from analyses. Completed diary sheets were returned weekly by freepost. Diaries were started well before the flowering season and continued throughout the flowering season and into the grass pollen season.

STATISTICAL ANALYSIS

Statistical analysis was carried out with SPSS/PC+ and BMDP software. Data from the peak flow diaries were initially input into a spreadsheet with Quattro Pro v3.0. Daily variation in peak flow was defined as ((maximum PF - minimum PF) / mean PF) × 100. A program written and compiled with Prospero Fortran 77 was used to calculate mean values for each season for peak flow, variability, and symptom scores. For the purposes of analysis individual FEV₁ values were expressed as a percentage of their predicted values calculated from the equations given by Quanjer *et al.*⁶ Mean peak flow values were expressed as a percentage of their predicted values in the equations proposed by Nunn and Gregg.⁷ Mean FEV₁ values as a percentage of the predicted, PC₂₀ values, total IgE concentrations, mean peak flow values as a percentage of the predicted, and mean variability values were analysed with repeated measures analysis of variance. The factors were, case or control, season (before and during flowering), and interaction between season and case or control. The PC₂₀ values, total IgE values, and mean amplitude percentage were normalised by taking natural logs. Symptom scores were analysed with the Wilcoxon paired test to compare scores before and during the flowering season and the Mann-Whitney U test was used to compare cases and controls.

Results

Table 1 shows the characteristics of 37 cases and 24 controls who participated in the studies. By definition, none of the control group had a doctor's diagnosis of hay fever or asthma, and of the cases 40.5% and 10.8% reported diagnoses of hayfever and asthma, respectively.

Forty one subjects, 23 cases and 18 controls, volunteered to participate in allergy tests and histamine challenge tests, but seven cases and three controls were unable to complete both the histamine tests before and during the season for various reasons, including change of mind, lack of time, or medication taken before the test. It was not possible to obtain a blood sample for one case.

ALLERGY TESTS

Nine out of 22 (41%) cases had a positive skin prick test to one or more allergens compared with four of 18 (22%) controls. People were most frequently positive to grass pollen (seven of 22 *v* none of 18) and house dust mite (seven of 22 *v* three of 18) for cases and controls, respectively (table 2).

Only one subject, a case, had a weakly positive reaction to oilseed rape as well as to grass pollen. The RAST tests generally corresponded with the skin tests (table 3). Discrepancies between the two tables are due to two people from whom blood samples were not obtained (one case and one control). Again, only one subject, a case, had a positive reaction to oilseed rape. This was not the same subject as the one with a positive skin prick reaction. She had, however, taken anti-

histamines before testing and so the skin tests were invalid; she also had high concentrations of IgE to many other allergens including grass pollen. Overall, therefore, 10 of 23 (43%) cases were atopic as shown either by positive skin tests or positive RAST tests. Total IgE values were slightly higher in cases than controls (fig 1), although these differences were not significant. There were no differences between seasons.

HISTAMINE CHALLENGE

The FEV₁ as a percentage of the predicted values remained stable over the period of the study and there was no difference between cases and controls (fig 2). Histamine challenge was carried out successfully both before and during the season on 16 cases and 15 controls. Only one case dropped out because of hayfever symptoms. Twelve of the 16 cases showed an increase in histamine sensitivity during the season compared with seven out of 15 controls (fig 3). Bronchial reactivity measured by PC₂₀ was significantly greater in cases than controls (*P* < 0.001) and increased during the season (*P* = 0.004).

PEAK FLOW AND SYMPTOM DIARIES

Thirty four cases and 25 controls were originally recruited into this part of the study. Three people (one case and two controls) did not send back any records, six dropped out (five cases and one control), eight had unsatisfactory records (five cases and three controls), and four withdrew for personal or family illness (one case and two controls). Twenty two cases and 17 controls completed satisfactory

Table 1 Characteristics of the study population

	Cases	Controls
Number	37	24
Males	9	6
Females	28	18
Age:		
Mean	35	37
SD	10.09	10.72
Range	17-54	18-51
Smoking:		
Smokers	4	5
Exsmokers	7	2
Never smokers	26	17
Previously diagnosed:		
Hayfever	15	0
Asthma	4	0
Allergy	9	4

Table 2 Positive skin test results

Allergen	Cases (22) n (%)	Controls (18) n (%)
Grass pollen	7 (31.8)	0 (0.0)
House dust mite	7 (31.8)	3 (16.7)
Cladosporium	0 (0.0)	0 (0.0)
Birch pollen	1 (4.5)	1 (5.6)
Oilseed rape pollen	1 (4.5)	0 (0.0)

Table 3 Positive RAST results

Allergen	Cases (22) n (%)	Controls (17) n (%)
Grass pollen	8 (36.4)	0 (0.0)
House dust mite	7 (31.8)	2 (9.1)
Cladosporium	1 (4.5)	0 (0.0)
Birch pollen	2 (9.1)	0 (0.0)
Oilseed rape pollen	1 (4.5)	0 (0.0)

Figure 1 Total IgE values before and during the oilseed rape flowering season.

Figure 2 FEV₁ values as percentage of predicted values in relation to the oilseed rape flowering season.

Figure 3 Bronchial reactivity before and during the oilseed rape flowering season.

records before and during the oilseed rape flowering season. One of the 22 cases did not complete the symptoms section satisfactorily. Table 4 shows mean symptom scores along with ranges and peak flow results.

Mean peak flow values as percentages of the predicted values did not differ in relation to the season and there were no significant differences between cases and controls. Variability in peak flow was less during the season than before ($P = 0.022$), although the difference was very small.

Recorded symptom scores were all very low with a highest mean score of 0.50 being recorded by cases for rhinitis during the flowering season. They were, however, generally higher in cases both before and during the season than in controls, with the exception of headache before the flowering season when values were the same. Significantly higher differences in symptom scores during the flowering season were found for rhinitis ($P = 0.044$) and headaches ($P = 0.005$) for cases and for eye symptoms for both cases and controls ($P = 0.011$ and $P = 0.018$, respectively). Cases had significantly higher scores than controls for cough ($P = 0.011$), wheeze ($P = 0.007$), rhinitis ($P = 0.001$), and eye symptoms ($P = 0.040$).

Discussion

In our previous study, we showed that residence in an oilseed rape area is only associated with a small excess risk of seasonal symptoms over that in a comparable rural area without oilseed rape, and that such symptoms are remarkably common in both

areas.⁴ The purpose of the present study was to look for clinical evidence that those people who did complain of symptoms during the oilseed rape flowering season were allergic to or irritated by oilseed rape. In so doing, we have validated the questionnaire used in the previous cross sectional survey⁴ by showing that those who complain of seasonal symptoms do, on average, have higher symptom scores and greater bronchial reactivity during the season, even though the general level of severity of symptoms was low. Only two people with symptoms were found to have an allergy to oilseed rape, one with a positive skin prick test to rape and grass pollen and one with a positive RAST test. This subject had a total IgE of more than 3700 kU/l both before and during the season and it is possible that this positive reaction may have been the result of a cross reaction, as extremely high concentrations of total IgE can also cause false positive results.⁸ Meding in 1985 reported a case where a woman who showed sensitivity to mustard was also allergic to crushed rape seeds.⁹

The absence of evidence of allergy to oilseed rape in most people who attribute their seasonal symptoms to the crop supports our previous suggestion that it is not a common cause of allergic sensitisation. Those with symptoms were, however, more likely to be atopic as shown by positive skin prick tests and RAST to other allergens, which suggests that in some cases symptoms due to other causes such as grass pollen may wrongly be attributed to oilseed rape. Others seem to have symptoms and bronchial hyperreactivity in the absence of atopy, and in these cases it is likely that they are reacting to irritants in the air. We have shown in a previous paper that oilseed rape produces terpenes,⁴ and others have found a wide range of other potentially irritant chemicals produced by the plant.¹⁰⁻¹² It is possible that these may cause some seasonal symptoms of irritation rather than allergy, but there is as yet no direct evidence that this is so.

We found significant decreases in PC_{20} during the season in spite of no parallel changes in peak flow measurements. Indeed peak flow became somewhat less variable during the season in cases. We were disappointed at the lack of discriminating power of peak flow readings in our study. This may in part be a consequence of studying an essentially normal population in which there would not be much variation in peak flow. Secondly, as others have found, we encountered the problem of drop out^{13,14} and the difficulties subjects encountered in completing their records may well have led to invented or retrospective values being written in^{15,16} reducing the reliability of the results. In contrast to their value in clinical studies, serial peak flow readings in our hands have proved disappointing in epidemiology.

Our main finding is that people who have seasonal symptoms that occur during the oilseed rape flowering season do not usually show evidence of allergy to the plant. This is

Table 4 Seasonal measurements of mean symptoms and peak flow

	Cases (n=22)		Controls (n=18)	
	Before	During	Before	During
Cough (range)*†	0.17(0.0-0.56)	0.20(0.1-0)	0.07(0.1-0)	0.14(0.1-0)
Wheeze (range)*†	0.15(0.1-0)	0.12(0.1-0.12)	0.01(0.0-0.05)	0.02(0.0-0.17)
Rhinitis (range)*†	0.35(0.1-0.35)	0.50*(0.1-0.98)	0.13(0.1-0)	0.20(0.1-0)
Eye (range)*†	0.10(0.1-0)	0.18*(0.1-0)	0.01(0.0-0.08)	0.08*(0.0-0.48)
Itchy skin (range)‡	0.06(0.1-0)	0.07(0.1-0)	0.01(0.0-0.07)	0.01(0.0-0.07)
Headache (range)‡	0.02(0.0-0.17)	0.11*(0.0-0.8)	0.02(0.0-0.15)	0.06(0.0-0.44)
Peak flow (SEM) (%)				
predicted, l/min ⁷	92.3(2.37)	92.2(2.47)	92.8(2.26)	92.6(2.44)
Peak flow (SEM)				
(amplitude % mean)§	4.2(0.18)	3.6(0.14)	3.8(0.16)	3.3(0.17)

* $P < 0.05$, for differences in symptoms overall between cases and controls;

† $P < 0.05$, for differences between seasons;

‡ n=21 for cases;

§ SEMs are derived from the log, transformed means because of skewed distribution.

based on the skin and blood tests of all those requested to participate who were prepared to do so. It is unlikely that any systematic bias occurred resulting in selection of people who were not allergic; indeed, any bias might intuitively be thought to err in the other direction. Our secondary finding, of increases in bronchial reactivity during the season unrelated to peak flow changes, simply supports the validity of the original questionnaire in selecting people with symptoms and showing that those symptoms are a manifestation of measurable physiological change in the group studied. Again, we think it unlikely that the fact that a number of subjects failed to participate influenced the results. Such an influence could have arisen if people who had symptoms failed to take part, but this would also have had the opposite effect to our findings. In fact the reasons for non-participation were those expected and found by others, largely related to the personal inconvenience of the rather complex and prolonged series of investigations.

The first report of oilseed rape allergy, in 1954,¹⁷ was of a patient with rhinitis, conjunctivitis, and asthma associated with the flowering of the crop. This patient showed both positive skin and nasal provocation tests to rape. Subsequently the prevalence of oilseed rape allergy was investigated in 366 consecutive patients with bronchial asthma and other allergies who attended an allergological department in southern Sweden, and 85 (23%) had a positive skin test to oilseed rape.¹⁸ Fifty four of these were subjected to a conjunctival provocation test and 44 showed a positive reaction.

The first reports of oilseed rape allergy in Britain were in 1990.¹ RAST tests were carried out on serum samples collected from 175 people in Tayside from May to September and from 82 people in November and December. Serum samples from 141 people from Glasgow collected during February, June, and July in 1988 were also tested. Of those tested in rural Tayside 26.3% were defined as having a positive response to oilseed rape compared with 7.8% in Glasgow, a highly significant difference ($P < 0.005$), likely to have been explained in part on the basis of selection bias. It was concluded that oilseed rape was a highly potent allergen. A study reported in 1992 of skin prick tests carried out in Aberdeen, on 81 atopic children in whom rape allergy was suspected, detected only four with a weakly positive reaction to oilseed rape.² It was concluded that allergy to oilseed rape was uncommon, and that the plant was not a potent sensitiser.

In the most extensive study published to date,³ 1478 people who were naturally exposed to oilseed rape were studied in 1989. They comprised 70 living in a village surrounded by rape and 1470 attending their local health centre for any complaint during the peak oilseed rape flowering season. Twenty four people had symptoms that they attributed to oilseed rape; however, of these, only three were found to have true allergy to

oilseed rape as shown by skin test, RAST, and nasal provocation tests. These people were also susceptible to other seasonal allergens. On the other hand, of 29 symptomatic people working in a research laboratory with oilseed rape, 16 had at least one positive skin test, nine had a positive skin test to rape, and of these five had positive RAST and nasal provocation tests. With one exception, all those found to be allergic to rape in that study had existing allergies to other pollens; these authors also concluded that oilseed rape pollen is not a potent sensitiser, mostly affecting people already sensitised to other allergens.

It has also been suggested that other animals are similarly affected by oilseed rape.¹⁹ In a study of asymptomatic horses and horses with chronic obstructive lung disease, it was concluded that exposure to oilseed rape may exacerbate existing pulmonary disease in some animals. None of these horses showed a positive reaction to intradermal tests, which suggests that the effects of oilseed rape were due to non-specific irritation rather than to allergy.

In summary, there is evidence that few of those people who experience symptoms during the oilseed rape flowering season are allergic to oilseed rape and less than half are atopic. We have shown that there is an increase in bronchial reactivity during the oilseed rape flowering season, especially among cases. This is likely to be due in some cases to non-specific irritation, probably related to chemical changes in the air, or in others to a reaction to other seasonal allergens. Whether the irritant reaction is due to terpenes released by the crop, to the natural rises in ozone in the air in spring and summer, or to other chemical causes remains to be investigated.

The study was supported by the Chief Scientist's Office of the Scottish Home and Health Department. We are also most grateful to the general practitioners for their assistance with the study, to the local branch of the National Asthma Campaign for their additional financial support, Allen and Hanbury's for supplying peak flow meters at cost price and the Department of Immunopathology, Aberdeen University, for total IgE and RAST analysis.

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Correspondence and editorials

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