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# Prevalence of sensitisation to oilseed rape and maize pollens in France: a multi-center study carried out by the Allergo-Vigilance Network

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## KEY WORDS

*Oilseed rape, maize, pollen, seed, sensitization, prevalence, atopy, exposure, GMO*

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## SUMMARY

**Background:** Oilseed rape and maize crops represent a large part of agriculture fields in European countries. **Objective:** To establish the actual prevalence of sensitization to oilseed rape and maize pollen, and to determine if this is correlated to the amount of exposure as well as to the patient's history of atopy or asymptomatic atopy. **Methods:** The study was conducted by 69 allergists belonging to the Allergo-Vigilance Network, in collaboration with the French Agency for Safety of food, and compiles the results of skin prick-tests using oilseed rape and maize pollens and seeds, as well as common aeroallergens. The patients were classified into 3 groups: non-atopic, asymptomatic atopy, and actual atopic diseases. **Results:** Among the 5372 subjects studied (2515 children, 2857 adults), 62.3% had an atopic disease, 10.2% had an asymptomatic atopy, and 27.5% were non-atopic. The level of sensitization was higher in the subjects with atopic disease, as compared to those with asymptomatic atopy: oilseed rape pollen: 11.8% vs 8%, maize pollen, 26% vs 19%, oilseed rape seeds, 7.7% vs 6.9%, corn seeds: 8.3% vs 4.8% ( $p < 0.001$ ). The rate of sensitization was significantly increased in those living in high crop density regions. The association of an atopic disease with a high rate of exposure yielded a higher rate of sensitization of 13.8% and 21.3% for rapeseed pollen, and 22.9% and 30.7% for maize pollen in both children and adults, respectively. **Conclusions:** The incidence of sensitisation to rapeseed and maize pollen is positively correlated to the level of exposure. This prevalence is higher in patients with actual atopic disease as compared to those with asymptomatic atopy. The frequency of sensitization confirms the allergenicity of these plants destined for food supply and demonstrates the importance of monitoring for respiratory allergies to these pollens, not only in workers exposed to these types of crops, but also in atopic patients living in regions that contain a high density of rapeseed and maize fields. Cross-reactivities between pollens and seeds could potentially elicit cross-reacting food allergies.

## Introduction

Sensitisation to the pollens of plants grown for food, such as oilseed rape (*Brassica napus* var. *napus* and *Brassica rapa* subsp. *oleifera*) and maize (*Zea mays*), has not been extensively studied (1-4), despite the fact that these crops have increased considerably in Europe. The area sown with oilseed rape continued to increase in 2003 because market demands for oilseed rape oil have increased with nutritionists highlighting the benefits of this oil, which is rich in omega 3 (5, 6). The development of bio-fuels is likely to increase cultivated areas even more. Furthermore, these species are associated with many of the requests for authorisation of genetically modified organisms (GMO) in Europe. Future GMOs for food may have even further modified proteomes than do current GMOs, accompanied by an unforeseeable allergic risk (7). In Europe, the culture of GMOs has led to a public debate and a moratorium on planting such crops. One of the worrying issues concerns their pollen, which could be dispersed into neighbouring crops and create new sensitisation risks. Therefore the present study was carried out by allergists of the Allergo-Vigilance Network, as recommended by the French Agency for Food Safety and Environnement (ANSES) (8, 9). The study aimed to assess the rate of sensitisation to pollen and seeds of oilseed rape and maize in the French population, and to seek its relationship to the degree of exposure. This was based indirectly on crop density and compared the prevalence of sensitisation with age-related characteristics and the presence of latent atopy or current atopic disease. The data could be used in further studies evaluating the frequency of sensitisation to these pollens in areas where GMOs may be planted in future.

## Methods

### *Study design*

One of the missions of the Allergo-Vigilance Network, currently consisting of 345 French and Belgian allergologists, is the monitoring of new allergic risks (8, 9). In July 2004, AFSSA (French Agency for Food Safety) and the network coordinator provided information to the members on the aim of the study. 69 participants were recruited until November 2004. Pollen and seed extracts were sent to network members in February 2005, together with the instructions for reporting findings. The participants were informed on the subsequent distribution of the data according to the

density of crops, but they were unaware of the classification of the areas. The study was carried out between March and June 2005. Each member carried out the tests in all referred patients over a period of one month to six weeks. The last data were collected in June 2005.

### *Study population*

The study population included patients attending allergology departments of hospitals or private practices for various reasons: seasonal or perennial allergic rhinitis, allergic asthma, atopic eczema/dermatitis syndrome (AEDS), food allergy, allergy to hymenoptera, adverse drug reactions, non allergic rhinitis, nasal polyposis, intrinsic asthma, chronic urticaria, contact dermatitis and suspected ocular allergies. Included patients were those having to undergo skin tests to common airborne allergens in order to diagnose their condition. Informed consent was obtained to carry out four extra prick-tests. Clinical histories and test results were used to classify the patients according to age (children up to 15 years, adults) into one of the following three groups:

- Group I:* Non atopic subjects who were referred for hymenoptera allergy, adverse drug reactions, non-allergic rhinitis, having no past history of atopy and negative prick-tests to 12 common inhalants.
- Group II:* Patients consulting because of various symptoms, having no current atopic disease, but with positive prick tests to one or more common inhalants. They were considered as having latent atopy.
- Group III:* Patients with atopic disease (AEDS, rhinitis, asthma) and positive prick-tests to one or more inhalants.

Patients not fitting strictly these criteria were excluded from this study.

### *Skin tests*

Atopic status was evaluated by skin prick testing using a panel of common aeroallergens: house dust mites, dog and cat epithelia, grass pollens, birch, mugwort, ash, plantain, cypress pollens, and in Southern France, olive tree pollen. The extracts were supplied by Allerbio (Varenes en Argonne, France) and Stallergènes (Antony, France). The control reagent for skin reactivity was 9% codeine, while the negative control was saline.

Four prick-tests to oilseed rape and maize pollens and seeds were performed. Forty-six allergists chose Allerbio extracts: W/V 1/20 (oilseed rape pollen), W/V 1/100

(maize pollen). Twenty-two chose extracts from Stalergènes: 100 IC/ml. The laboratory was not quoted by one allergist.

Prick-tests were defined as positive if the wheal was greater or equal in size to 2/3 of that of the codeine control, the latter being at least 2.5 mm.

#### *Areas of maize and oilseed rape crops*

After the collection of the results from the allergists, in a second step, these results were classified according to the area where they had been recorded.

Data concerning the areas planted with maize and oilseed rape in 2000 were available on the web site: [agreste.agriculture.gouv.fr](http://agreste.agriculture.gouv.fr). Areas were classified separately for oilseed rape and maize, and were divided into three crop density groups: low, medium and high (Table 1, Fig. 1).

#### *Statistical analysis*

Chi-Squared tests were used to compare the prevalence of sensitisation in the different areas and to test the association between sensitisation and atopy.

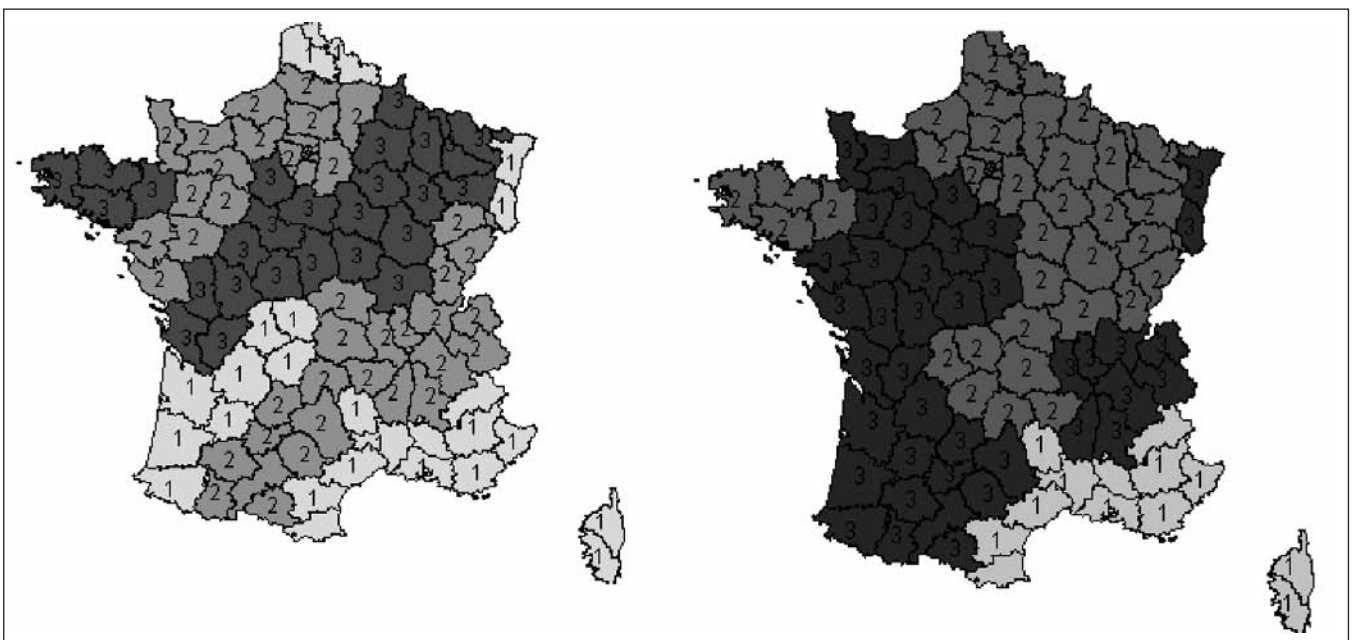
#### **Results**

A total of 5,372 people benefited from this study which was carried out by 69 allergists (hospitals and private practice). There were 2,515 children and 2,857 adults. Sex was not recorded. 62.3% of patients had an atopic disease, 10.2% had latent atopy and 27.5% had no atopy (Table 2). The overall prevalence of prick-tests positive to maize pollen was 13.1% (children) and 15.9% (adults) and to oilseed rape pollen 5.6% (children) and 7.1% (adults). There was less sensitisation to maize seeds: 3.6% (chil-

**Table 1** - Classification of the regions into 3 catégories of crop density (source Agreste 2000)

Culture	Low density (Ha)	Medium density (Ha)	High density (Ha)
Oilseed rape	0 - 6853	18121 - 55561	98442 - 266010
Maize	0 - 7815	33352 - 81151	147070 - 432207

**Figure 1** - Areas of crops of rapeseed (left) and maize (right) according to the density



**Table 2** - Sensitization to rapeseed and maize pollens: classification of patients.

group	children	adults
Non atopic patients	536	944
Atopic patients	273	273
Current atopic disease	1706	1640

dren) to 5.7% (adults).

Where there was no latent atopy, there was no sensitisation to oilseed rape and maize pollen, even in areas of high crop density. Prick tests were negative to oilseed rape pollen in 533/536 children and 936/944 adults, and negative to maize pollen in 536/536 non atopic children and 944/944 non atopic adults.

Rates of sensitisation to pollen and seed, in both children and adults, were significantly higher in those with atopic disease at the time of testing, compared with those with latent atopy: oilseed rape pollen: 11.8% vs 8%; maize pollen: 26% vs 19%; maize seed 8.3% vs 4.8%; oilseed rape seed 7.7% vs 6.9%.

Sensitisation rates varied significantly according to crop density in the area where people lived (tables 3, 4). Comparing high with low crop density areas, sensitisation to oilseed rape pollen increased from 0% to 6% in children and from 1.4% to 9.7% in adults, while sensitisation to maize pollen increased from 3.7% to 6.4% in children and from 1.6% to 6.3% in adults.

The duration of exposure influenced the frequency of sensitisation in atopic patients: sensitisation to oilseed rape pollen was always more frequent in adults than children.

Correlating data for latent atopy or clinical manifestations with crop density in the areas where patients lived suggested that the association of atopic disease with high exposure produces high rates of sensitisation (13.8% and 21.3% to oilseed rape pollen and 22.9% and 30.7% to maize pollen in children and adults respectively) (tables 3 and 4; figures 2 and 3).

## Discussion

### *Pollination period of oilseed rape pollens*

Grains are oval and measure 45 x 20 µm. A quarter are dispersed by wind and the rest by insects (1, 4, 10). The pollination period starts after that of Betulaceae and during the

first part of the pollination period of Grasses, i.e. April, May and the beginning of June. Oilseed rape pollen counts relate to crop density and are on the increase. Between 1985 and 1995 there was a 6-fold increase in the Vienne area, corresponding to an intensification of crop production in the surrounding countryside (4). A pollen calendar documenting the English countryside in May estimated between 10 to 100 pollen grains/m<sup>3</sup> (3).

### *Oilseed rape pollen allergens*

Numerous allergens have been described: a 14 kD profilin, cross-reacting with Bet v 2 (11), three analogues of *Arabidopsis thaliana* allergens: a cyanocobalamin-independent methionin synthase, a receptor-like kinase protein, a berberin bridge protein (12). Bra r 1 is a calcium-binding protein crossing with a grass pollen allergen, Cyn d 7 (13). A 6-8 kDa allergen crosses with alder pollen and celery homologues (14). It has a high homology to the allergens of olive and *Chenopodium* pollens (15). Other allergens include a 43 kD polygalacturonase (4, 16) and a 7.5 kD lipid transfer protein (table 5) (17) and finally carbohydrate determinants (18). Cross reactions have been identified with rye and birch pollens (14). Possible cross reactivity with grass pollens remains controversial (19, 20).

### *Sensitisation to oilseed rape pollen*

It is frequent: 7.1% of 4,468 subjects, co-sensitised to grasses (88%), birch (67%) and other pollens (78%). Mono-sensitisation to oilseed rape pollen is less than 0.25% (3, 4, 20), but specific sensitisation to oilseed rape pollen does occur (21,22). This depends on exposure. In a study of 4,468 town and country dwellers, a 7.1% sensitisation rate increased to 23% in a Swedish population living in a crop area (1, 4). The same rate was found in crop areas in Scotland (2). The present study carried out in France demonstrates the correlation between the rate of sensitisation of children and adults with atopic disease and the crop density: from 0% to 9.4% in children, from 4.5% to 15.1% in adults, in areas with high and low crop density (tables 3, 4). These observations suggest a sensitisation to allergens specific to oilseed rape pollen.

### *Allergy to oilseed rape pollen*

There are conflicting opinions concerning oilseed rape allergy with some authors considering it of minimal importance (10, 20). It is, however, well documented in people in

**Table 3** - Effect of latent atopy and existing disease on the prevalence of sensitization to rape seed pollen and seeds, in relation to the rape crop density

<b>a. Children</b>				
Maize Crops	Group	Patients	Positive prick-test to maize pollen	Positive prick-test to maize seeds
		(n)	%	%
Low density (0-6853)	No atopy	58	0	0
	Latent atopy	55	0	0
	Atopic disease	224	0	0
	Total	398	0	0
Medium density (18121-55561)	No atopy	306	1%	0
	Latent atopy	165	0	0
	Atopic disease	1056	7%	6.9%
	Total	1527	5%	4.8%
High density (98442-266010)	No atopy	172	0	0
	Latent atopy	52	3.8%	0
	Atopic disease	426	13.8%	9.4%
	Total	650	9.4%	6.1%
<b>b. Adults</b>				
Maize Crops	Group	Patients	Positive prick-test to maize pollen	Positive prick-test to maize seeds
		(n)	%	%
Low density (0-6853)	No atopy	69	0	0
	Latent atopy	21	0	0
	Atopic disease	199	6.5%	2%
	Total	289	4.5%	1.4%
Medium density (18121-55561)	No atopy	586	1.4%	0.7%
	Latent atopy	187	0	3.2%
	Atopic disease	995	8.6%	6.2%
	Total	1768	5.3%	4.1%
High density (98442-266010)	No atopy	289	0	0
	Latent atopy	65	4.6%	4.6%
	Atopic disease	446	21.3%	13.5%
	Total	650	15.1%	9.7%

occupational contact with crops (3, 21). For the general population, there is no specific season for diagnosing oilseed rape allergy since, in the event of seasonal rhinitis, routine diagnosis is pollinosis to Betulaceae and/or grasses, with oilseed rape pollen arriving simultaneously at the end of April to June (4). Patients with hay fever to grass pollens often incriminate oilseed rape crops when they live in the vicinity of such fields. There is a higher prevalence of cough, wheeze, headaches (but not of rhinitis) in people living in areas densely planted with oilseed rape (23, 24). During their flowering period these crops emit a characteristic, un-

pleasant odour due to over 20 volatile organic compounds (VOC). Monoterpene beta-myrcene and sesquiterpene alpha-farnesene account for more than 59% (25). Several VOCs irritate the respiratory mucosa, and oilseed rape is often considered irritant for individuals allergic to grass pollens (10, 26). Other factors associated with oilseed rape crops are often implicated, such as fungal spores (*Cladosporium*, *Sporobolomyces* and *Tilletiopsis*) which may be more common than with other crops (26). Some cases of seasonal asthma have been diagnosed by nasal challenge test (27) or by correlating mono-sensitisation with concentrations of

**Table 4** - Effect of latent atopy and existing disease on the prevalence of sensitization to maize pollen and seeds in relation to the maize crop density

<b>a. Children</b>				
Maize Crops	Group	Patients	Positive prick-test to maize pollen	Positive prick-test to maize seeds
		(n)	%	%
Low density (0-7815)	No atopy	63	0	0
	Latent atopy	59	0	0
	Atopic disease	225	4%	5.8%
	Total	347	2.6%	3.7%
Medium density (32352-81151)	No atopy	205	1%	0
	Latent atopy	91	2.2%	2.2%
	Atopic disease	654	19.1%	6.3%
	Total	950	13.4%	4.5%
High density (147070-432207)	No atopy	265	0	1.1%
	Latent atopy	122	3.3%	2.4%
	Atopic disease	831	22.9%	8.7%
	Total	1218	16%	6.4%
<b>b. Adults</b>				
Maize Crops	Group	Patients	Positive prick-test to maize pollen	Positive prick-test to maize seeds
		(n)	%	%
Low density (0-7815)	No atopy	55	0	0
	Latent atopy	21	0	0
	Atopic disease	180	8.3%	2.2%
	Total	256	5.9%	1.6%
Medium density (32352-81151)	No atopy	426	1.4%	0
	Latent atopy	128	9.4%	2.3%
	Atopic disease	694	25.6%	7.3%
	Total	1248	15.7%	4.3%
High density (147070-432207)	No atopy	463	0.9%	0.9%
	Latent atopy	124	4.8%	0
	Atopic disease	766	30.7%	10.6%
	Total	1353	18.1%	6.3%

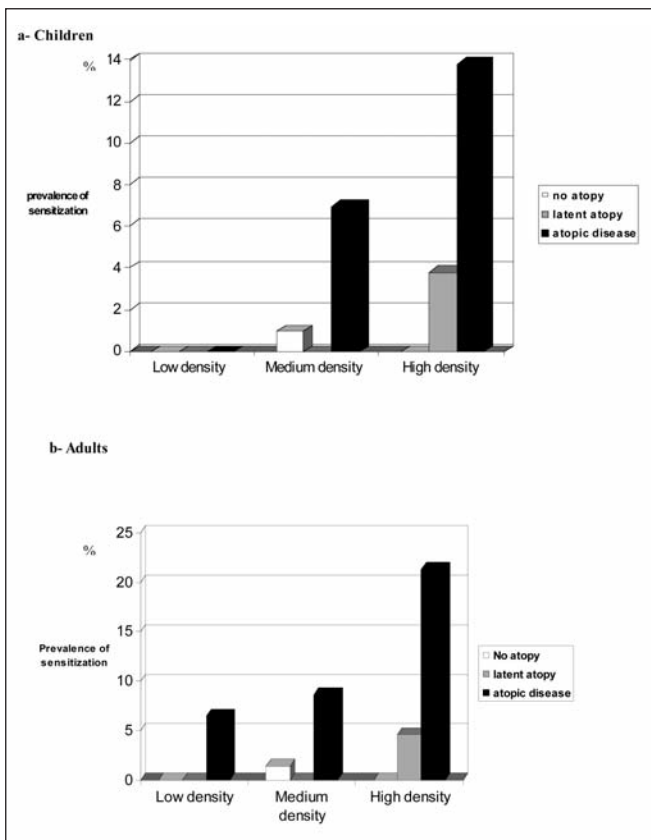
pollens at 30 pollen grains/m<sup>3</sup> (28). The extent of sensitisation to oilseed rape pollen in seasonal respiratory diseases and in exacerbating seasonal atopic dermatitis should be evaluated by challenge testing in areas where crops are grown (29). In a Swedish study by Bucur, 81% of conjunctival challenge tests were positive (1). Etiological links could be based on double-blind, nasal and bronchial challenge tests with non-irritant, VOC-free substances (3, 30). Another disease related to oilseed rape pollen sensitisation could be food allergy to mustard: oilseed rape

pollen-mustard seed cross-reactivity is well documented (31) and the prevalence of this allergy is markedly higher in South West France (6% vs 1%), an area extensively planted with oilseed rape (over 400,000 hectares) (32, 33).

#### *Pollination period of maize pollens*

The maize pollen grain is large, with a mean size of 99 µm (80 to 140 µm). The pollination period ranges from 6 to 18 days in July. Dispersal by wind depends on the degree of

**Figure 2** - Effect of latent atopy, and existing disease on the prevalence of sensitization in relation to the rape crop density



hydration. Maize pollen can be dispersed considerable distances (1000 m) downwind from maize crops (34).

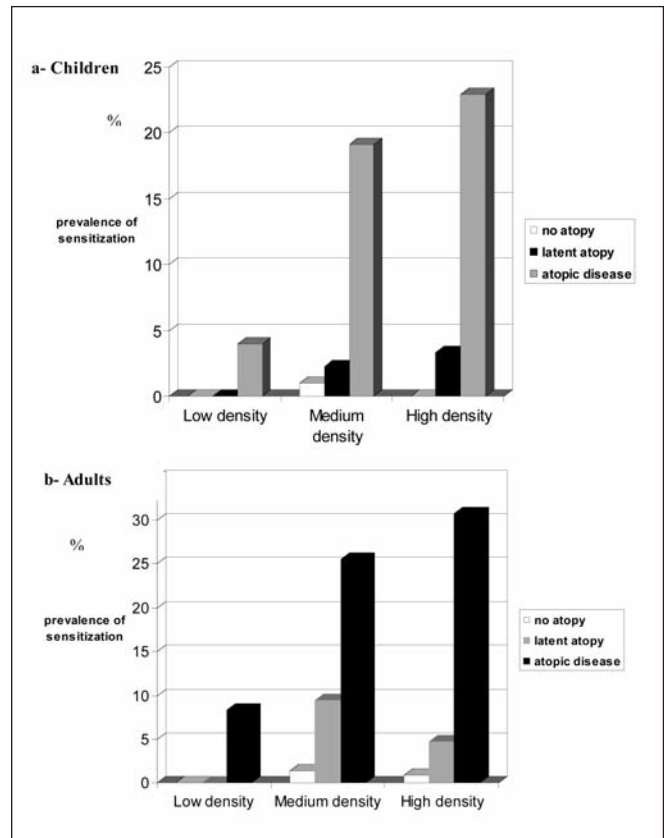
#### *Maize pollen allergens*

It contains many groups of allergens: Zea m 1 (expansins), Zea m 12 (profilins), Zea m 13 (polygalacturonase) and Zea m CBP (polcalcin) (table 5).

Group 1 allergens, (Zea m 1) belonging to the beta-expansin family are 70% homologous with major grass pollens (35): 72% identity between Zea m 1 and Phl p 1 (36). Zea m 13 has a 70% identity with Phl p 13. Using immunoblot inhibition tests the timothy pollen extract completely blocks the IgE-binding to maize pollen (36, 37). However, when specific IgEs for 14 grass and cereal pollens were sought, values for maize pollen were the lowest and overall cross reactivity was low (38).

A case of seasonal asthma by mono-sensitization to maize pollen was successfully treated with specific immunotherapy (39). Food allergy to maize pollen, ingested ritually by

**Figure 3** - Effect of latent atopy, and existing disease on the prevalence of sensitization to maize pollen in relation to the maize crop density



Navajo Indians, has been reported (40). Our study confirmed frequent sensitization to maize pollen, greater in adults, and related to crop density.

#### *Sensitisation and allergy to oilseed rape seed*

Occupational asthma has been reported (30, 41, 42). 2S albumin and 5-6 kD and 10 kD allergens cross-react between oilseed rape seeds and pollen and mustard seeds (31, 43). Oilseed rape seed is not ingested as food, but its allergens could be present in oilseed rape oil, used extensively in baby foods for infants. Adults regularly eat oilseed rape margarine and oil. The frequency of prick-tests positive to oilseed rape seed could reflect either cross reactivity between pollen and seed, or sensitization to seed proteins present in oilseed rape oil and margarine. Poikinen demonstrated that 10.9% of 1,987 children with suspected food allergy were sensitized to oilseed rape seed (versus 4.8% to 6.1% in our study). He

**Table 5** - Allergens described in rapeseed and maize pollens and seeds.

Species	Allergens	In silico homology	In vitro cross reactivity
	Bra n 1 (calcium binding protein)	Cyn d 7 (Smith)	Rye and birch pollens (Focke)
	Allergen 6 – 8 kDa	Alder pollen and celery (Focke) Olive tree pollen and chenopodiaceae pollen (Barderas)	
Rapeseed pollen	Profilins 14 kDa		Grass pollens (Astwood, Welch)
	80 kDa (pI : 5)	Cobalamine independent methionine synthetase of <i>A. thaliana</i> (Chardin)	
	70 kDa (pI : 8,5-10)	Berberin-bridge protein of <i>A. thaliana</i> (Chardin)	
	40 kDa (pI : 10)	Receptor-like protein kinase of <i>A. thaliana</i> (Chardin)	
	Polygalacturonase 43 kDa (Hemmer, Chardin)		
	LTP 7,5 kDa (Torriama)		
Rapeseed seeds	Allergènes 5 – 6 kDa		Rapeseed pollens Mustard seeds (Dominguez, Monsalve)
	Allergens 10 kDa		
	2S Albumine		
	Zea m 1 : expansin	Grass pollen major allergens : 70% homology (Lian – Chao)	
Maize pollen	Zea m 12: profilin		
	Zea m 13: polygalacturonase	Grass pollens (Heiss)	
	Polcalcin		
Maize seeds	LTP 9 kDa (Pastorello)		
	50 kDa (Pasini)		

confirmed potential food allergy in 89% of these children by DBPCFC (44).

#### *Sensitisation and allergy to maize seed*

Maize seed, a potential source of food allergy in countries where its consumption is common, contains major allergens: a 9 kD thermo-resistant LTP and a 50 kD protein (45, 46). The allergenic repertoire of maize seed has recently increased with the addition of thioredoxin and other proteins (47). The prevalence of sensitisation to maize seed noted in this study probably reflects cross reactivity

with the pollen, or cross sensitisation with cereal glutelins but probably reflecting non clinically relevant allergens, since maize allergy is not common in France (48, 49).

There are two hypotheses for the fact that sensitisation to oilseed rape and maize pollens occurs more frequently in subjects with ongoing atopic disease than in subjects with latent atopic disease: atopic disease may signify that the propensity to develop sensitisation to environmental allergens is greater in these subjects, or alternatively, atopic disease may be, at least partially, dependent on the sensitisation observed with these pollens.



## Conclusion

Sensitisation to oilseed rape pollen is common, particularly where crop production has increased considerably. Both the intensity and duration of exposure are important factors, as confirmed by this study. The risk of respiratory allergy has to be feared, and the future use of bio-fuels in agricultural machines will create a further risk for farmers who may inhale these allergens. Overlapping pollination periods of oilseed rape, maize, grasses and Betulaceae hinders the identification of oilseed rape pollinosis. Another confounding factor is the irritant effect of oilseed rape VOCs on respiratory tract mucosae that may exacerbate any pollinosis due to grasses (26, 50). The consequences of cross reactivity with other Brassicaceae, particularly an allergy to mustard, justify further investigation. Pollen calendars in areas with high crop densities may help to document the relationship between crop density and respiratory allergy. Immunotherapy could be recommended in some cases of mono-sensitisation. Preliminary experiments with an hypoallergenic allergen have been started in mice (51).

Food allergy to oilseed rape seed has recently been described in children (44). Allergy to maize flour remains rare. However, a systematic search for specific IgEs in sera of children allergic to other foods showed they were present in over 10% of cases (52), comparable to the rate of positive prick-tests in this study: 8% to 8.3%.

It would therefore be wise to monitor carefully any changes in the prevalence of sensitisation to oilseed rape and maize seed, to study the allergome of their pollens and seeds with sera from patients with a positive double-blind challenge (nasal, ocular or oral challenge) and to determine whether there is a clinically relevant cross sensitisation between pollen and seed. Data on the current prevalence of sensitisation to maize and oilseed rape pollens would provide a basis for further evaluations of sensitisation when GMO crops become more common (51, 52).

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## Declaration of interest

The authors report no conflicts of interest.

## References

1. Bucur I, Arner B. Rape pollen allergy. *Scand J Resp Dis* 1978; 59:222-7.
2. Paratt D, Thomson G, Saunders C, Mc Sharry C, Cobb S. Oilseed rape as a potent antigen. *Lancet* 1990;335:121-5.
3. Fell PJ, Soulsby S, Blight MM, Brostoff J. Oilseed rape – a new allergen? *Clin Exp Allergy* 1992;22:501-5.
4. Hemmer W, Focke M, Wantke F, Jäger S, Götz M, Jarisch R. Oilseed rape pollen is a potentially relevant allergen. *Clin Exp Allergy* 1997;27:156-61.
5. Bima (Bulletin d'Information du Ministère de l'Agriculture) anonymous. Les oméga 3 relancent l'huile de colza. La vogue des bons acides gras a-t-elle un impact sur la production agricole ? 2004;1509:12.
6. Martin A. The « apports nutritionnels conseillés » for the French population. *Reproduction, nutrition, développement* 2001;41:119-28.
7. Lack G, Chapman M, Kalsheker N, King V, Robinson C, Venables K; BSACI working party. Report on the potential allergenicity of genetically modified organisms and their products. *Clin Exp Allergy*. 2002;32:1131-43.
8. Moneret-Vautrin DA. Present aspects of food allergies: the need for allergo-vigilance. *Bull. Acad Natl Med* 2001;185:943-62.
9. Moneret-Vautrin DA, Kanny G, Morisset M, Rancé F, Fardeau MF, Beaudouin E. Severe food anaphylaxis: 107 cases registered in 2002 by the Allergy Vigilance Network. *Eur Ann Allergy Clin Immunol* 2004; 36: 46-51.
10. Murphy DJ. Is rapeseed really an allergenic plant? Popular myths versus scientific realities. *Immunol Today* 1999;20:511-4.
11. Focke M, Hemmer W, Valenta R, Götz M, Jarisch R. Identification of oilseed rape (*Brassica napus*) pollen profiling as a cross-reactive allergen *Int Arch Allergy Immunol* 2003; 132: 116-23.
12. Chardin H, Mayer C, Sénéchal H, Tepfer M, Desvaux FX, Peltre G. Characterization of high-molecular-mass allergens in oilseed rape pollen. *Int Arch Allergy Immunol* 2001;125:128-34.

13. Smith PM, Xu H, Swoboda I, Singh MB. Identification of a Ca<sup>2+</sup> binding protein as a new Bermuda grass pollen allergen Cyn d 7: IgE cross-reactivity with oilseed rape pollen allergen Bra r 1. *Int Arch Allergy Immunol* 1997;114:265-71.
14. Focke M, Hemmer W, Hayek B, Gotz M, Jarisch R. Identification of allergens in oilseed rape (*Brassica napus*) pollen. *Int Arch Allergy Immunol* 1998;117:105-112.
15. Barderas R, Villalba M, Pascual CY, Batanero E, Rodriguez R. Profilin (Che a 2) and polcalcin (Che a 3) are relevant allergens of *Chenopodium album* pollen: isolation, amino acid sequences, and immunologic properties. *J Allergy Clin Immunol* 2004; 113: 1192: 8.
16. Chardin H, Mayer C, Sénéchal H, Poncet P, Clément G, Wal JM, Desvaux FX, Peltre G. Polygalacturonase (pectinase), a new oilseed rape allergen. *Allergy* 2003; 58: 407-11.
17. Toriyama K, Hanaoka K, Okada T, Watanabe M. Molecular cloning of a cDNA encoding a pollen extracellular protein as a potential source of a pollen allergen in *Brassica rapa*. *FEBS Lett* 1998; 424: 234-8.
18. Chardin H, Sénéchal H, Wal JM, Desvaux FX, Godfrin D, Peltre G. Characterization of peptidic and carbohydrate cross-reactive determinants in pollen polysensitization. *Clin Exp Allergy* 2008; 38: 680-5.
19. Astwood JD, Mohapatra SS, Ni H, Hill RD. Pollen allergen homologues in barley and other crop species. *Clin Exp Allergy* 1995;25:66-72.
20. Galloway D. Oilseed rape - allergen or irritant? *Clin Exp Allergy* 2000;30:308-9.
21. Welch J, Jones MG, Cullinan P, Coates OA, Newman Taylor AJ. Sensitization to oilseed rape is not due to cross-reactivity with grass pollen. *Clin Exp Allergy* 2000;30:370-5.
22. McSharry C. New aeroallergens in agricultural and related practice. *Clin Exp Allergy* 1992;22:423-6.
23. Soutar A, Harker C, Seaton A, Brooke M, Marr I. Oilseed rape and seasonal symptoms: epidemiological and environmental studies. *Thorax* 1994;49:352-6.
24. Parratt D, Macfarlane Smith WH, Thomson G, Cameron LA, Butcher RD. Evidence that oilseed rape (*Brassica napus* ssp. *oleifera*) causes respiratory illness in rural dwellers. *Scott Med J* 1995;40:74-6.
25. Butcher RD, Macfarlane-Smith W, Robertson GW, Griffiths DW. The identification of potential aeroallergen/irritant(s) from oilseed rape (*Brassica napus* ssp. *oleifera*): volatile organic compounds emitted during flowering progression. *Clin Exp Allergy* 1994; 24: 1105-14.
26. Soutar A, Harker C, Seaton A, Packe G. Oilseed rape and bronchial reactivity. *Occup Environ Med* 1995;52:575-80.
27. Colldahl H. Rape pollen allergen; report of a case. *Acta Allergol* 1954; 7: 367-9.
28. Fiorina A, Scordamaglia A, Guerra L, Passalacqua G. Aerobiologic diagnosis of brassicaceae-induced asthma. *Allergy* 2003; 58: 829-30.
29. Reekers R, Busche M, Wittmann M, Kapp A, Werfel T. Birch pollen-related foods trigger atopic dermatitis in patients with specific cutaneous T-cell responses to birch pollen antigens. *J Allergy Clin Immunol* 1999;104:466-72.
30. Suh CH, Park HS, Nahm DH, Kim HY. Oilseed rape allergy presented as occupational asthma in the grain industry. *Clin Exp Allergy* 1998;28:1159-63.
31. Monsalve RI, Gonzalez de la Pena MA, Lopez-Otin C et al. Detection, isolation and complete amino acid sequence of an aeroallergenic protein from rapeseed flour. *Clin Exp Allergy* 1997; 27: 833-41.
32. Morisset M, Moneret-Vautrin DA, Maadi F, Fremont S, Guegnard L, Croizier A, Kanny G. Prospective study of mustard allergy: first study with double-blind placebo-controlled food challenge trials (24 cases). *Allergy* 2003;58:295-9.
33. Rancé F. Mustard allergy as a new food allergy. *Allergy* 2003; 58: 287-8.
34. Jarosz N, Loubet B, Durand B, Foueillassar X, Huber L. Variations in maize pollen emission and deposition in relation to microclimate. *Environ Sci Technol* 2005;39:4377-84.
35. Li LC, Bedinger A, Volk C, Jones D, Casgrove DJ. Purification and characterization of four beta-expansins (*Zea m 1* isoforms) from maize pollen. *Plant Physiology* 2003;132:2073-85.
36. Petersen A, Dresselhaus T, grobe K, Becker WM. Proteome analysis of maize pollen for allergy-relevant components. *Proteomics* 2006; 6: 6317-25.
37. Heiss S, Flicker S, Hamilton DA, Kraft D, Mascarenhas JP, Valenta R. Expression of Zm13, a pollen specific maize protein, in *Escherichia coli* reveals IgE-binding capacity and allergenic potential. *FEBS Lett* 1996;381:217-21.
38. Van Ree R, Van Leeuwen WA, Aalberse RC. How far can we simplify in vitro diagnostics for grass pollen allergy? A study with 17 whole pollen extracts and purified natural and recombinant major allergens. *J Allergy Clin Immunol* 1998;102:184-190.
39. Gonzalo-Garijo MA, Pérez-Calderon R, Munoz-Rodriguez A, Molina-Tormo R, Silva-Palacios I. Hypersensitivity to maize pollen. *Allergy* 2004;59:365.
40. Freeman GL. Oral corn pollen hypersensitivity in arizona native americans: some sociologic aspects of allergy practice. *Ann Allergy* 1994;72:415-7.
41. Alvarez MJ, Estrada JL, Gozalo F, Fernandez-Rojo F, Barber D. Oilseed rape flour: another allergen causing occupational asthma among farmers. *Allergy* 2001;56:185-8.
42. Di Giacomo GR, Boschetto P, Maestrelli P, Moro G. Asthma and rhinoconjunctivitis caused by rape flour: description of a clinical case. *Med Lav* 1998;89:226-31.
43. Dominguez J, Cuevas M, Urena V, Munoz T, Moneo I. Purification and characterization of an allergen of mustard seed. *Am Allergy* 1990;64:352-7.
44. Poikonen S, Puumalainen TJ, Kautiainen H, Burri P, Palosuo T, Reunala T, Turjanmaa. Turnip rape and oilseed rape are new potential food allergens in children with atopic dermatitis. *Allergy* 2005;DOI:10.1111/j.1398-9995.2005.00929x.
45. Pastorello EA, Pompei C, Pravettoni V, et al. Lipid-transfer protein is the major maize allergen maintaining IgE-binding activity after cooking at 100°C, as demonstrated in anaphylactic patients and patients with positive double-blind, placebo-controlled food challenge results. *J Allergy Clin Immunol* 2003;112:775-83.
46. Pasini G, Simonato B, Curioni A, Vincenzi S, Cristaudo A, Santucci B et al. IgE-mediated allergy to corn: a 50 kDa protein, belonging to the reduced soluble proteins, is a major allergen. *Allergy* 2002;57:98-106.
47. Weichel M, Vergoossen NJ, Bonomi S, Scibilia J, Ortolani C, Ballmer-Weber BK, Pastorello EA, Cramer R. Screening the allergenic repertoires of wheat and maize with sera from double-blind, placebo-controlled food challenge positive patients. *Allergy* 2006;61:128-35.

48. Moneret-Vautrin DA, Kanny G, Beaudouin E. Food allergy to corn—does it exist? *Allerg Immunol* 1998;30:230.
49. Borghesan F, Borghesan N. Maize flour-induced rhinitis. *Eur Ann Allergy Clin Immunol* 2005;37:283-4.
50. Seaton A, Soutar A. Oilseed rape and seasonal symptoms. *Clin Exp Allergy* 1994;24:1089-90.
51. Okada T, Swoboda I, Bhalla PL, Toriyama K, Singh MB. Engineering of hypoallergenic mutants of the Brassica pollen allergen, Bra r 1, for immunotherapy. *FEBS Lett* 1998;434:255-60.
52. Batista R, Nunes B, Carmo M, Cardoso C, Jose HS, de Almeida AB, et al. Lack of detectable allergenicity of transgenic maize and soya samples. *J Allergy Clin Immunol* 2005;116:403-10.