

Prevalence and incidence of asthma and rhinoconjunctivitis in Swedish teenagers

Norrman E, Nyström L, Jönsson E, Stjernberg N. Prevalence and incidence of asthma and rhinoconjunctivitis in Swedish teenagers. Allergy 1998; 53: 28–35. © Munksgaard 1998.

In 1987, we studied the prevalence of asthma and rhinoconjunctivitis in 1112 grade-eight students in the town of Umeå, northern Sweden. The present study is based on the same cohort, reinvestigated in 1991. We aimed to estimate the changes in the prevalence of asthma and rhinoconjunctivitis and to examine some presumed risk factors of asthma and rhinoconjunctivitis. A postal questionnaire on present asthma, rhinoconjunctivitis, symptoms from the upper and lower airways, and the need for medication was used. Questions about living conditions, smoking, and present education were also included. The questionnaire was answered by 89% (990) of the subjects. The prevalence of self-reported asthma was 10.8% (boys 7.9%, girls 13.7%). The incidence rate/year was 1.1%, the remission rate/year was 5.7%, and the relapse rate/year was 10%. The risk factors for developing asthma appeared to be atopy, having a family history of asthma or rhinoconjunctivitis, and smoking. The prevalence of self-reported rhinoconjunctivitis was 14.3% (boys 15.6%, girls 13.1%). The incidence rate/year was 1%, the remission rate/year was 22%, and the relapse rate/year was 11%. Stopping smoking and having no family history of rhinoconjunctivitis or asthma appeared to favor remission in rhinoconjunctivitis.

**E. Norrman¹, L. Nyström²,
E. Jönsson³, N. Stjernberg³**

¹Department of Pulmonary Medicine and Allergology, University Hospital of Northern Sweden, Umeå; ²Department of Epidemiology and Public Health Research, Umeå University, Umeå; ³National Institute of Occupational Health, Umeå, Sweden

Key words: allergic rhinoconjunctivitis; asthma incidence; remission.

Eva Norrman, MD
Department of Pulmonary Medicine and Allergology
University Hospital of Northern Sweden
S-901 85 Umeå
Sweden

Accepted for publication 23 July 1997

Reports of the increasing prevalence of asthma and atopy are being received from most parts of the world (1–6). The fact that allergic heredity and atopy increase the risk of asthma and allergic disease is common knowledge (7, 8). There is some evidence that asymptomatic persons with IgE antibodies to an allergen risk developing symptoms within 2 years (9, 10). On the basis of information from cross-sectional prevalence studies, several causes of the increasing prevalence have been discussed, such as increasing air pollution, infections, a poor indoor climate, and smoking (11–18). However, for investigation of the causes of the increasing prevalence, point-prevalence studies offer insufficient information. Although the prevalence of asthma has been investigated in most industrialized countries, studies of incidence and remission rates are less common (19–28). In 1987, we studied the prevalence of asthma and rhinoconjunctivitis in 1112 grade-eight students (most of whom were 14 years old) in Umeå, northern Sweden (29, 30). A short description of this study is given below. In the present study, the same cohort was reinvestigated after 3 years. We aimed to

estimate the incidence and remission rates and present prevalence of self-reported asthma, allergic rhinoconjunctivitis, and airways symptoms. The influence of smoking and some other presumptive risk factors was evaluated.

Material and methods

Former study (29, 30), study cohort (Fig. 1)

In 1987, all grade-eight students in Umeå were invited to participate in this study. Of 1163 subjects, 1112 participated, most of whom were 14 years old (range 13–16 years, 14-year-olds, $n=1058$). The study consisted of two parts. All the subjects were asked to answer a questionnaire and participate in a skin prick test (SPT) by Phazet. Ten allergens common in Sweden were used, birch, timothy grass, mugwort, cat, dog, horse, two mites (*Dermatophagoides farinae* and *D. pteronyssinus*), and two molds (*Alternaria* and *Cladosporium*), as well as histamine as positive control and a blank needle as negative control. A resulting wheal with a mean diameter of 3 mm or more was taken as a positive

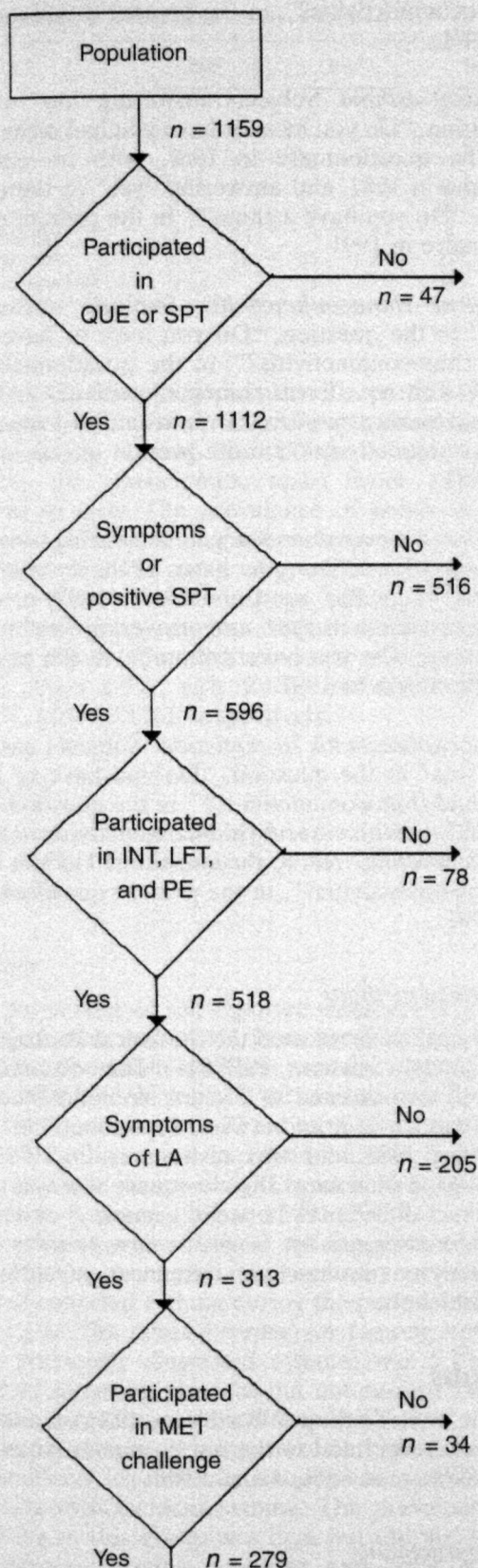


Fig. 1. Flowchart of study design. QUE: questionnaire, SPT: skin prick test, INT: interview, LFT: lung-function test, PE: physical examination, LA: lower airways, MET: methacholine.

reaction. Atopic and symptomatic subjects were then further investigated by structured interview, spirometry, and physical examination. In subjects with symptoms from the lower airways, a methacholine provocation test was carried out. After the interview, subjects with asthma were told not to smoke and to avoid occupations involving welding or exposure to severe irritants of the airways. Symptomatic atopic subjects were told to avoid the allergen in question. Nonsymptomatic atopic subjects were not given any advice on the avoidance of irritants or allergens.

Present study

All the subjects participating in the first study (1112 total, 562 boys, 550 girls) were invited to answer a postal questionnaire. The questionnaire was mailed in February 1991. It was followed by three reminders.

Questionnaire

The questionnaire consisted of 25 questions. Five were the same as in the 1987 questionnaire:

Do you often cough abnormally, more often than your friends?
Do you usually have wheezing in your chest?
Do you usually get attacks of dyspnea?
Do you get more out of breath than your friends when exercising?
Do you get wheezing in your chest when exercising outdoors in the winter?

In two questions, the wording was changed from "do you have or have you ever had?" to "do you have?": Do you have asthma? Do you have hay fever? The questions relating to symptoms from contact with certain allergens were rephrased from the 1987 questionnaire. In addition, we included questions relating to the subjects' use of antiasthmatic or antiallergic drugs; smoking habits, both among the subjects and their families; the subjects' choice of education or work; and living conditions (including former or present pets at home, visible mold at home, and living in a flat or house). These questions were worded in the same way as in the former interview.

Definitions

Previous self-reported asthma. Subjects answering "yes" to the question, "Do you have or have you had asthma," in the questionnaire in 1987.

Previous self-reported rhinoconjunctivitis. Subjects answering "yes" to the question, "Do you have or

have you had rhinoconjunctivitis," in the questionnaire in 1987.

Previous diagnosis of asthma. Subjects who were given a diagnosis of *current* asthma in 1987, fulfilling our definition of *current* asthma in 1987; subjects within the preceding year with recurrent attacks of dyspnea with or without wheezing, triggered by known stimuli (not including infections), and at least one of the following: $PC_{20} \leq 8$ mg/ml or signs of obstructive airways at examination. Eight subjects, four men and four women, who did not participate in the second part of the study in 1987, but reported a doctor diagnosis of asthma, current symptoms from lower airways, and current medication against asthma at the time of the study were also classified as having a previous diagnosis of asthma.

Previous diagnosis of allergic rhinoconjunctivitis. Subjects who were given a diagnosis of *current* allergic rhinoconjunctivitis in 1987, fulfilling our definition of *current* allergic rhinoconjunctivitis in 1987; subjects within the preceding year with symptoms of rhinitis or rhinoconjunctivitis from contact with an allergen and positive SPT to that allergen. Eleven subjects, nine men and two women, who did not participate in the second part of the study in 1987 but with symptoms from contact with certain allergens in the preceding year and a doctor diagnosis of rhinoconjunctivitis, were also classified as having a previous diagnosis of allergic rhinoconjunctivitis.

Present self-reported asthma. Subjects answering "yes" to the question, "Do you have asthma?", in the present questionnaire in 1991.

Present self-reported allergic rhinoconjunctivitis. Subjects answering "yes" to the question, "Do you have allergic rhinoconjunctivitis?", in the present questionnaire in 1991.

Atopy. Subjects who, in 1987, had at least one SPT ≥ 3 mm.

Relapsing asthma. Subjects answering "yes" to the question, "Do you have or have you had asthma?", in the questionnaire in 1987, with no current asthma in 1987, and answering "yes" to the question, "Do you have asthma?", in the present questionnaire in 1991.

Relapsing rhinoconjunctivitis. Subjects answering "yes" to the question, "Do you have or have you had rhinoconjunctivitis?", in the questionnaire in 1987, with no current rhinoconjunctivitis in 1987, and answering "yes" to the question, "Do you have

rhinoconjunctivitis?", in the present questionnaire in 1991.

Incident asthma. Subjects answering "no" to the question, "Do you have or have you had asthma?", in the questionnaire in 1987, with no current asthma in 1987, and answering "yes" to the question, "Do you have asthma?", in the present questionnaire in 1991.

Incident rhinoconjunctivitis. Subjects answering "no" to the question, "Do you have or have you had rhinoconjunctivitis?", in the questionnaire in 1987, with no current rhinoconjunctivitis in 1987, and answering "yes" to the question, "Do you have rhinoconjunctivitis?", in the present questionnaire in 1991.

Asthma in remission. Subjects answering "yes" to the question, "Do you have or have you had asthma?", in the questionnaire in 1987, or with current asthma in 1987, and answering "no" to the question, "Do you have asthma?", in the present questionnaire in 1991.

Rhinoconjunctivitis in remission. Subjects answering "yes" to the question, "Do you have or have you had rhinoconjunctivitis?", in the questionnaire in 1987, or with current rhinoconjunctivitis in 1987, and answering "no" to the question, "Do you have rhinoconjunctivitis?", in the present questionnaire in 1991.

Statistical methods

Statistical analyses used the Statistical Package for the Social Sciences (SPSS). "The observation period" was defined as 3 years, since the study in 1987 was carried out between September 1987 and February 1988, and the questionnaire in 1991 was sent out in February. The chi-square test was used to detect differences between groups; $P < 0.05$ was considered significant. Logistic regression was used to analyze simultaneous influences of different determinants.

Results

Of the invited group, 990 subjects, 493 men and 497 women, responded to the questionnaire (response rate 89%, men 88%, women 90%).

Nonparticipants

In the nonresponding group (122 subjects, 69 boys and 53 girls), one had died, three were abroad, and the rest refused to answer.

Table 1. Self-reported smoking in 1987 and 1991 ($n=990$)

	Boys		Girls		Total	
	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%
Smoker 1987	6	1.2	13	2.6	19	1.9
Smoker 1991	29	5.9	59	11.9	88	8.9
New smoker 1991	26		53		79	
Ex-smoker 1987	2	0.4	3	0.6	5	0.5
Ex-smoker 1991	8	1.6	15	3.0	23	2.3
New ex-smoker 1991	8		15		23	

The prevalence of previous self-reported and/or diagnosed asthma and rhinoconjunctivitis was generally lower among the nonparticipants than among the participants, apart from reported asthma in girls. The prevalence of previous self-reported asthma among nonparticipants was 7.3% (1.4% boys, 9.4% girls); of previous self-reported rhinoconjunctivitis, 5.2% (boys 7.2%, girls 3.8%). The prevalence of previous diagnosis of asthma and allergic rhinoconjunctivitis in nonparticipants was 5.7% (boys 2.9%, girls 9.4%) and 13.1% (boys 11.6%, girls 15.1%), respectively.

Smoking

The prevalence of self-reported smoking in 1991 was 8.9%, as compared with 1.9% in 1987 (Table 1).

Asthma

The prevalence of self-reported asthma was 10.8% (8.6% in men and 14.4% in women). Of these, 10 subjects, seven men and three women, had relapsed since 1987, and 26 subjects, 10 men and 16 women, were incident cases. In 33 subjects, 19 men and 14 women, asthma was in remission. Of these, 11 subjects, three men and eight women, had gone into remission between 1987 and 1991 (Fig. 2).

If subjects with previous self-reported or diagnosed asthma were excluded, the *incidence rate/year* for self-reported asthma during the period 1987–91 was 1.1%. The *remission rate/year* (among subjects with previously diagnosed asthma) was 5.7%. In subjects reporting previous but not current asthma in 1987, the *relapse rate/year* was 10.4%.

The prevalence of some presumed risk factors is summarized in Table 2. Atopy was common in subjects with incident asthma. The prevalence of smoking in this group was high but did not reach significance. Having no atopy and being a boy appeared to favor remission. Subjects with atopy appeared to risk relapse, but the numbers were too small to reach significance.

The most important risk factors for "present self-reported asthma" ($n=97$) were atopy, family history of asthma or rhinoconjunctivitis, and being a girl. Visible mold or dampness at home also increased the risk significantly (Table 3). The subjects with asthma in remission tended to be more nonatopic than those with persistent asthma (atopy vs no atopy; OR=0.1, 95% CI 0.0–0.8), and more men than women had gone into remission (women vs men; OR=0.06, 95% CI 0.01–0.51) (Table 3). In the incident and relapse groups, the number of subjects was small, thereby reducing the power of the model. When backward elimination of variables was used, the only independent variables left in the equation in the incident group were atopy (OR=2.5, 95% CI 1.0–5.6), previous smoking (OR=2.8, 95% CI 0.4–23.0), and smoking (OR=3.4, 95% CI 1.2–9.3). In the relapse group, the number of subjects was too small to create a significant model.

Rhinoconjunctivitis

The prevalence of self-reported rhinoconjunctivitis was 14.3% (15.6% in men and 13.1% in women). Of these, six subjects (one man, five women) were relapse cases and 22 subjects (14 men, eight women) were incident cases. In 82 subjects (39 men, 43 women), rhinoconjunctivitis was in remission. Of these, 13 subjects had been in remission since 1987 (Fig. 3).

Between 1987 and 1991, the incidence rate/year was 1.0%, the remission rate/year was 22%, and the relapse rate/year was 11%.

The prevalence of atopy in subjects with self-reported rhinoconjunctivitis was 91%; 93% in men, 89% in women.

Considering "present self-reported hay fever" heredity (OR 3.9, 95% CI 2.4–6.1) and visible mold at home (OR 3.5, 95% CI 1.6–7.9) appeared to increase the risk significantly. The risk pattern in the *incident* cases was similar to that for present self-reported rhinoconjunctivitis. In the *relapsing* cases, the numbers were too small. Having no heredity (heredity vs no heredity OR 0.4, 95% CI 0.2–0.8) and making the mothers stop smoking appeared to favor *remission*, although only the absence of heredity reached significance (Table 3).

Atopy

Of 177 atopic subjects (90 men, 87 women) not reporting asthma or rhinoconjunctivitis in 1987, 8.5% (15 subjects, nine men, six women) reported rhinoconjunctivitis or asthma in 1991. The corresponding figure in nonatopic subjects (435 total;

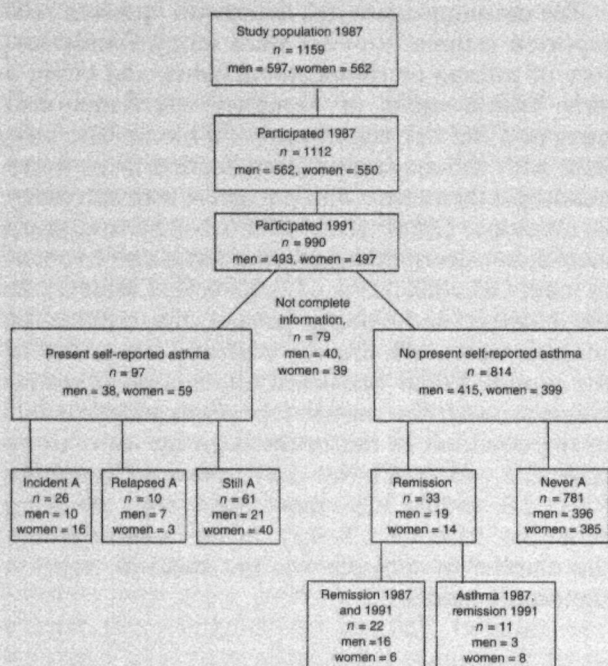


Fig. 2. Flowchart of asthma (A).

211 men, 222 women) was 2.1% (nine total; three men, six women).

When we looked at subjects with asthma or rhinoconjunctivitis among the atopic subjects, heredity increased the risk of having asthma or rhinoconjunctivitis (OR 3.4, 95% CI 1.8–6.5). Subjects who had previously smoked ran a greater risk without reaching significance (OR 6.7, 95% CI 0.7–63).

Discussion

In the present study, we found the same incidence of self-reported asthma as previously reported from northern Sweden (19). As stated in that study, the incidence appeared to be higher than in the UK and the USA (Minnesota, Michigan, Arizona, and Connecticut) (20, 23, 25–27). The incidence rates in the Swedish studies could be influenced by the intervention performed in the samples; i.e., conducting a further investigation of subjects reporting symptoms in the first study. This intervention is likely to have increased the probability of subjects identifying their symptoms as asthma or allergic rhinoconjunctivitis during the next study period. However, the intervention also diagnosed symptomatic subjects who had not yet received a diagnosis, thereby probably decreasing the incidence during the next period. Moreover, in the period 1987–91, the news media focused on the problem of the increasing prevalence of asthma and allergy, thereby making the

subjects more aware of the symptoms. These factors could also influence remission rates and relapse rates. The relapse rate/year in this age group is seemingly higher than that reported from Tucson, Arizona (24), but the number of subjects in comparable ages in the Tucson study was small, making the calculations unreliable. The high number of relapsing subjects during the observation period presents a target for possible intervention. The remission rate in our study was slightly lower than in Arizona, Michigan, and Connecticut (24–26), but, again, the number of observations was small.

For allergic rhinoconjunctivitis, both the relapse rate and the remission rate were high. The incidence of allergic rhinoconjunctivitis during this period was low, although 30% of the subjects without allergic rhinoconjunctivitis in 1987 were atopic. Of the nonsymptomatic atopic population in 1987, 8.5% developed symptoms of asthma or allergic rhinoconjunctivitis within 3 years, a percentage which is low compared with the 17.6% reported from Rhode Island, USA (10).

The prevalence of smoking increased between 1987 and 1991, mainly among girls, but the total number of smokers was still low in 1991. In subjects with a previous diagnosis of asthma, the prevalence of smoking was very low (two subjects), indicating some effect of the information given in 1987.

When we analyzed factors associated with the risk of having asthma, the risk pattern in previous asthma and incident asthma appeared to be similar. Some tendencies could be seen, however; heredity, mothers who smoked, and female sex appeared to become less important, while the impact of smoking oneself and dampness in the home appeared to increase. There is some evidence that, in adolescence, being female involves a high risk of developing asthma. The reasons for this are not known (19, 20). Atopy and heredity have been shown to be associated with an increased risk of developing asthma (28). In our study, subjects with these factors appeared to develop their symptoms at an early stage, and if they experienced remission periods, they appeared to run a high risk of relapse. The impact of smoking has been reported in several studies (7, 11–13, 16–19, 22, 31), starting as an indoor pollutant (mothers smoking) at home, and extending to the subjects' own smoking later. There have been several reports on the respiratory health effects of home dampness, molds, and mites (14–16, 18, 32). In this study, dampness or visible mold at home appeared to increase the risk of both asthma and rhinoconjunctivitis.

Unlike the study of Brunekreef et al. of Dutch children (33), the risk of asthma, allergic rhinocon-

Table 2. Prevalence of some presumed risk factors in subjects who never had asthma and in subjects with still, incident, relapsing asthma and asthma in remission

Risk factors	Internal nonresponse (%)	Never A		Still A		Incident A		Relapsed A		Remission A	
		%	n	%	n	%	n	%	n	%	n
Heredity of asthma or rhinoconjunctivitis	8	25	183	51 ¹	31	42	10	80	8	36	10
Month of birth Oct–March	0	47	364	58	31	54	14	50	9	58	19
Atopy	9	41	295	69 ¹	42	61 ¹	14	75	6	48 ²	15
Female sex	0	49	385	65 ¹	40	62	16	30	3	42 ²	14
Smoker	0.4										
Present		9	66	3	2	19	5	0	0	15	5
Former		2	16	5	3	4	1	10	1	0	0
Never		89	695	92	48	77	20	90	9	85	28
Mother smoker	2	28	215	30	18	46	12	20	2	34	11
Mother former smoker	2	10	74	16	10	8	2	10	1	19	6
Living in house	0	71	558	49	30	65	17	50	5	49	16
Visible mold at home	2	4	91	15 ¹	9	8	2	0	0	9	3
Furry pets at home	13										
Present		62	424	33	17	64	14	14	1	39	11
Former		31	208	47	24	23	5	71	5	54	15
Never		7	49	20	10	14	3	14	1	7	2
Total no. of subjects			781		61		26		10		33

¹ Higher prevalence as compared with healthy ($P < 0.05$). ² Higher prevalence as compared with persistent asthma ($P < 0.05$).
A: asthma.

Table 3. Multiple logistic regression analysis of present self-reported asthma (A, $n = 97$) and rhinoconjunctivitis (Rc, $n = 128$), with subjects with no present self-reported A ($n = 893$) and Rc ($n = 762$) as referents, and subjects with Rc and A in remission with subjects with Rc ($n = 89$) and A ($n = 53$) in both 1987 and 1991 as referents, by month of birth, sex, heredity of asthma or rhinoconjunctivitis, parental smoking, own smoking, type of home, and visible mold at home. Atopy has been included as independent variable in analysis of A. Odds ratio (OR) and 95% confidence intervals (CI)

Independent variables		Dependent variables							
		Asthma				Rhinoconjunctivitis			
		Present self-reported		In remission		Present self-reported		In remission	
	Categories	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Month of birth	April–Sept	1		1		1		1	
	Oct–March	1.2	0.7–2.1	0.7	0.1–3.9	1.1	0.7–1.8	1.3	0.6–3.0
Sex	Men	1		1		1		1	
	Women	3.5	1.9–6.6	0.06	0.01–0.51	0.9	0.5–1.4	1.2	0.5–2.6
Heredity of A or Rc	No	1		1		1		1	
	Yes	2.7	1.5–4.8	6.5	0.9–45	3.9	2.4–6.1	0.4	0.2–0.8
Smoking	No	1		1		1		1	
	Yes, former	2.1	0.46–9.8	–		2.8	0.9–8.5	0.6	0.1–5.2
	Yes, present	1.0	0.34–2.8	3.3	0.2–57	0.4	0.2–1.3	1.2	0.2–8.7
Mother smoking	No	1		1		1		1	
	Yes, former	2.1	0.8–5.5	0.4	0.0–21	1.3	0.6–3.1	2.3	0.6–9.5
	Yes, present	1.5	0.8–3.0	11.3	0.5–253	1.0	0.6–1.7	1.7	0.6–4.4
Father smoking	No	1		1		1		1	
	Yes, former	1.0	0.6–2.5	8.3	0.2–421	0.7	0.3–1.6	1.7	0.4–6.4
	Yes, present	1.2	0.4–2.7	0.2	0.0–4.2	0.7	0.4–1.3	2.4	0.9–6.6
Type of residence	Apartment	1		1		1		1	
	House	1.1	0.7–2.9	0.1	0.0–1.4	1.0	0.6–1.8	0.7	0.3–1.9
	Undefined	2.5	0.8–7.3	0.03	0.0–4.3	1.4	0.6–3.5	0.2	0.0–1.3
Visible mold at home	No	1		1		1		1	
	Yes	3.0	1.2–7.4	0.06	0.00–1.03	3.5	1.6–7.9	0.3	0.1–1.7
Atopy	No	1		1					
	Yes	4.5	2.5–8.3	0.1	0.0–0.8				
Furry pets at home	No	1		1		1		1	
	Yes, formerly	0.5	0.2–1.1	19.4	0.7–407	0.4	0.2–0.9	1.2	0.4–4.1
	Yes, present	0.3	0.1–0.6	16.4	0.4–878	0.3	0.2–0.6	9	0.6–5.9

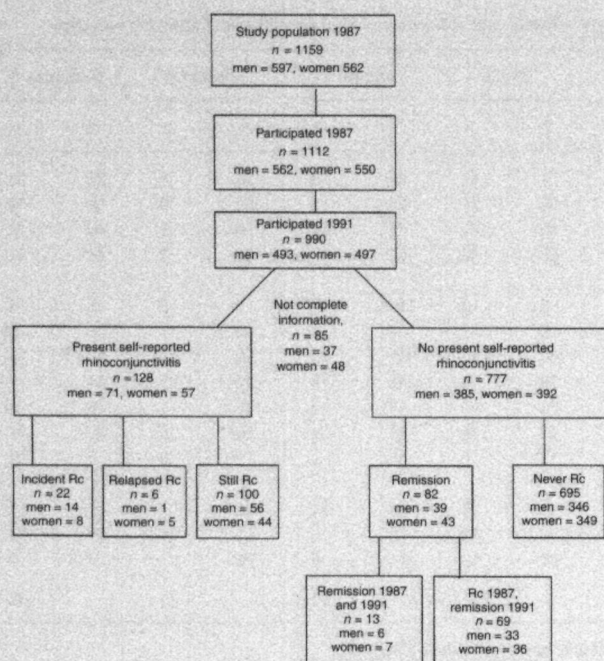


Fig. 3. Flowchart of rhinoconjunctivitis (Rc).

junctivitis, and atopy in the present study was lower in subjects who previously or currently had furry pets at home. These results agree with the results of a study of wheezing and asthma in New Zealand, Wales, South Africa, and Sweden, in which wheezing was positively associated with pet ownership in every country except Sweden (34). The reasons for this are not obvious. A healthy worker effect (subjects with a family history of asthma or rhinoconjunctivitis might be less prone to acquire furry pets) is the most probable explanation (35). In the present study, the total prevalence of previously or currently having furry pets at home was 92%. In families which previously or currently did not have furry pets, 75% of the investigated subjects had never had asthma, while 87% in families with pets had never had asthma.

Our results appear to support the theory that smoking affects the immune system, thereby enhancing the contact between allergen and the immune system and increasing the ability of forming IgE antibodies to common allergens, especially in younger subjects (31). The expression of symptoms in the sensitized subject appears to be linked to susceptibility (heredity) in the subject in combination with an environmental pollution load. The confusing fact that furry pets (previous or current) did not increase the risk of either symptoms or atopy in our study does not contradict the theory. The allergen may not cause symptoms if the environmental pollution is kept at a low level, or else

it may not be recognized as a risk factor as it is "endemic".

In conclusion, we found a high incidence of asthma in young adults. The incidence rate appeared to be influenced by atopy and smoking

Acknowledgments

This study was made possible by grants from the Swedish Heart and Lung Foundation and the Swedish Association against Heart and Lung Diseases.

References

- Morrison-Smith J, Harding LK, Cumming G. The changing prevalence of asthma in schoolchildren. *Clin Allergy* 1971;1:57-61.
- Mitchell EA. Increasing prevalence of asthma in children. *N Z Med J* 1983;96:463-4.
- Gergen PJ, Mullally DI, Evans R. National survey of prevalence of asthma among children in the United States, 1976 to 1980. *Pediatrics* 1988;81:1-7.
- Robertson CF, Heycock E, Bishop J, Nolan T, Olinsky A, Phelan PD. Prevalence of asthma in Melbourne schoolchildren: changes over 26 years. *BMJ* 1991;302:1116-18.
- Omran M, Russel G. Continuing increase in respiratory symptoms and atopy in Aberdeen schoolchildren. *BMJ* 1996;312:34-1.
- Peat JK, van den Berg RH, Green WF, Mellis CM, Leeder SR, Woolcock AJ. Changing prevalence of asthma in Australian children. *BMJ* 1994;308:1591-6.
- Jenkins MA, Hopper JL, Flander LB, Carlin JB, Giles GG. The associations between childhood asthma and atopy, and parental asthma, hay fever and smoking. *Paediatr Perinat Epidemiol* 1993;7:67-96.
- Croner S, Kjellman N-IM. Development of atopic disease in relation to family history and cord blood IgE levels. Eleven year follow up in 1654 children. *Pediatr Allergy Immunol* 1990;1:14-20.
- Hatlevig G, Kjellman B, Björkstén B, Johansson SGO. The prevalence of allergy and IgE antibodies to inhalant allergens in Swedish schoolchildren. *Acta Paediatr Scand* 1987;76:349-55.
- Hagy GW, Settupane GA. Prognosis of positive allergy skin tests in an asymptomatic population. *J Clin Immunol* 1971;48:200-11.
- Infante-Rivard C. Childhood asthma and indoor environmental risk factors. *Am J Epidemiol* 1993;137:834-44.
- Magnusson CGM. Maternal smoking influences cord serum IgE and IgD levels and increases the risk for subsequent infant allergy. *J Allergy Clin Immunol* 1986;78:898-904.
- Cogswell JJ, Mitchell EB, Alexander J. Parental smoking, breast feeding and respiratory infection in development of allergic diseases. *Arch Dis Childhood* 1987;62:338-44.
- Verhoeff AP, van Strien RT, van Winjen JH, Brunekreef B. Damp housing and childhood respiratory symptoms: the role of sensitization to dust mites and molds. *Am J Epidemiol* 1995;141:103-10.
- Dales RE, Zwanenburg H, Burnett R, Franklin C. Respiratory health effects of home dampness and molds among Canadian children. *Am J Epidemiol* 1991;134:196-203.
- Wickman M, Nordvall SL, Pershagen G. Risk factors in early childhood for sensitization to airborne allergens. *Pediatr Allergy Immunol* 1992;3:128-33.

17. Bråbäck L, Breborowicz A, Julge K, Riikjäv M-A, Vasar M, Björkstén B. Risk factors for respiratory symptoms and atopic sensitization in the Baltic area. *Arch Dis Child* 1995;72:487-93.
18. Newman-Taylor A. Environmental determinants of asthma. *Lancet* 1995;345:296-9.
19. Larsson L. Incidence of asthma in Swedish teenagers: relation to sex and smoking habits. *Thorax* 1994;50:260-4.
20. Anderson HR, Pottier AC, Strachan DP. Asthma from birth to age 23: incidence and relation to prior and concurrent atopic disease. *Thorax* 1992;47:537-42.
21. Burrows B, Sears MR, Flannery EM, Herbison GP, Holdaway MD, Silva PA. Relation of the course of bronchial responsiveness from age 9 to age 15 to allergy. *Am J Respir Crit Care Med* 1995;152:1302-8.
22. Strachan DP, Butland B, Ross Anderson H. Incidence and prognosis of asthma and wheezing illness from early childhood to age 33 in a national British cohort. *BMJ* 1996;312:1195-9.
23. Yunginger JW, Reed CE, O'Connell EJ, Melton LJ, O'Fallon WM, Silverstein MD. A community-based study of the epidemiology of asthma. Incidence rates 1964-1983. *Am Rev Respir Dis* 1992;146:888-94.
24. Bronniman S, Burrows B. A prospective study of the natural history of asthma. *Chest* 1990;4:480-4.
25. Schachter EN, Doyle CA, Beck GJ. A prospective study of asthma in a rural community. *Chest* 1985;5:623-30.
26. Broder I, Higgins MW, Mathews KP, Keller JB. Epidemiology of asthma and allergic rhinitis in a total community in Tecumseh, Michigan. *J Allergy Clin Immunol* 1974;54:100-10.
27. Dodge RR, Burrows B. The prevalence and incidence of asthma and asthma-like symptoms in a general population sample. *Am Rev Respir Dis* 1980;122:567-75.
28. Blair H. Natural history of childhood asthma. *Arch Dis Child* 1977;52:613-19.
29. Norrman E, Rosenhall L, Nyström L, Bergström E, Stjernberg N. High prevalence of asthma and related symptoms in teenagers in northern Sweden. *Eur Respir J* 1993;6:834-9.
30. Norrman E, Rosenhall L, Nyström L, Jönsson E, Stjernberg N. Prevalence of positive skin prick tests, allergic asthma, and rhinoconjunctivitis in teenagers in northern Sweden. *Allergy* 1994;49:808-15.
31. Holt PG, McMenamin C, Nelson D. Primary sensitization to inhalant allergens during infancy. *Pediatr Allergy Immunol* 1990;1:3-13.
32. Dowse GK, Turner KJ, Stewart GA, Alpers MP, Woolcock AJ. The association between *Dermatophagoides* mites and the increasing prevalence of asthma in village communities within the Papua New Guinea highlands. *J Allergy Clin Immunol* 1985;75:75-83.
33. Brunekreef B, Groot B, Hoek G. Pets, allergy and respiratory symptoms in children. *Int J Epidemiol* 1992;21:338-42.
34. Burr ML, Limb ES, Andrae S, Barry DMJ, Nagel F. Childhood asthma in four countries: a comparative survey. *Int J Epidemiol* 1994;23:341-7.
35. Kjellman B, Pettersson R. The problem of furred pets in childhood atopic disease. *Allergy* 1983;38:65-73.

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.