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Apparent but not real increase in asthma prevalence during the 1990s

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Apparent but not real increase in asthma prevalence during the 1990s. R. Barraclough, G. Devereux, D.J. Hendrick, S.C. Stenton. ©ERS Journals Ltd 2002.

ABSTRACT: The authors investigated changes in asthma prevalence and perception of bronchoconstriction over 6 yrs in adults of Newcastle-upon-Tyne.

Postal questionnaires were sent to 6,000 subjects aged 20–44 yrs in 1992–1993 and 1998–1999. Random samples of 600 responders had assessments of atopy, airway responsiveness, and their ability to perceive methacholine-induced bronchoconstriction. The prevalences of asthmatic symptoms, physician-diagnosis, and medication use increased by an average of 4.4%, particularly in subjects aged <30 yrs (8.7 *versus* 2.7). Atopy prevalence increased from 25% to 31% but atopics and nonatopics had similar mean changes in questionnaire data (5.2 *versus* 3.4). The probability of a positive methacholine test decreased as did the mean methacholine dose/response slope (0.00527 to 0.00379), indicating lower levels of airway responsiveness. This can be largely explained by an increase in use of inhaled corticosteroids (5.0–9.3%). The proportion of subjects perceiving bronchoconstriction during methacholine tests increased from 63 to 77%.

The authors conclude that current changes in asthma epidemiology in adults may result from increased awareness of symptoms (and/or an increased willingness to report them), and from an increased willingness of physicians to make the diagnosis and prescribe treatment, not from increased disease prevalence.

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Asthma prevalence appears to have increased dramatically in industrially developed countries over the last two decades as judged by hospital admission rates, diagnoses, use of medication, and certified deaths. An increase in disease prevalence has been supported by a number of studies in which the same population has been surveyed more than once at intervals of ≤25 yrs [1–4]. Observed rates of change for those affected within UK populations have been >6% per annum for 8–13-yr-old children [1, 2], 7% per annum for university entrants [3], and 8% per annum for 12-yr-old children [4]. These findings were based on questionnaire responses, and so may have been influenced by increased awareness of asthmatic symptoms and an increased likelihood of reporting them. There is a possible source of confusion in expressing the magnitude of change in terms of percentage; if prevalence itself is quantified as a percentage (as in the present study) and increases from, for example, 4.5–4.8, the net change of 0.3 represents a 7% increase in the number of affected subjects.

Few studies have included objective features of asthma severity or prevalence, such as airway responsiveness measurements, and no clear picture has emerged from those that have. BURR *et al.* [4] reported a greater than two-fold increase in "current asthma" amongst schoolchildren between 1973 and 1988, but there was only a 15% increase in the proportion who showed a bronchoconstrictor response to exercise (decrement in peak expiratory flow of >15%). By contrast, PEAT *et al.* [5] showed that an 87% increase in "current asthma" amongst 8–10-yr-old children between 1982 and 1992 was paralleled by an 83% increase in the prevalence of airway hyperresponsiveness to histamine [5]. However, the type of nebuliser used for the inhalation challenge tests was changed over the period of their study (with a probable change in aerosol output) and this might have confounded the result [6]. The same group showed a relatively modest 21% increase in "current asthma" between 1981 and 1990 in 18–55 yr olds, and found that the prevalence of airway hyperresponsiveness fell over the period of the study [7]. These discrepancies raise the possibility that recent increases in symptom reporting, physician diagnoses, and medication (which involve countries as widely separated as Australia, Finland, and the USA [5, 8, 9]) do not reflect a true increase in the pathophysiological abnormalities of asthma. They led a recent reviewer to conclude that the evidence for an increase in asthma prevalence is weak and that further studies of defined populations using objective data are necessary [10].

The current authors have conducted surveys of the adult population of Newcastle-upon-Tyne in the north east of England in 1992–1993 and 1998–1999 using the same methodology and equipment. In these

surveys, objective measurements of spirometry, airway responsiveness, and atopy were made in addition to questionnaire assessments of symptoms, physician diagnoses, and medication. The perception of bronchoconstriction reported by participants undergoing methacholine tests was also assessed.

Methods

The study followed closely, but not exactly, the design of the European Community Respiratory Health Survey (ECRHS) [11]. An initial survey was carried out in 1992–1993 and a repeat survey in 1998–1999. On each occasion a postal questionnaire was sent to 6,000 adult residents of Newcastle-upon-Tyne aged 20–44 yrs, randomly selected from the Health Authority register and stratified equally by age and sex. Each subject received up to three mailings of a short questionnaire comprising nine items about asthmatic symptoms and treatment, and a further item related to hay fever (table 1). All mailings were carried out between July and September. To assess response bias attributable to an incorrect address, a random sample of nonresponders was investigated by comparing details on the Health Authority register with those on the Electoral Roll.

A random sample of responders was then invited to attend the laboratory for an interviewer-administered questionnaire, measurements of height and weight, skin-prick tests with six common aeroallergens, spirometric tests, and a methacholine-challenge test. The aim was to recruit 600 subjects for these laboratory studies of each survey. The methods were the same in the two surveys and have been published in detail elsewhere [12, 13]. All laboratory studies took place between October and April to avoid the pollen season.

Methacholine was administered by nebuliser in doubling cumulative doses over the range 3.125–6,400 μg. Airway responsiveness was expressed both as the dose/response slope (DRS) and as the dose of methacholine provoking a 20% fall in forced expiratory volume in one second (FEV_1, PD_{20}) [14]. Using this methodology, PD_{20} values <200 μg are usually associated with active asthma, values 200–1,000 μg are sometimes associated with active asthma, and values >1,000 μg are seldom associated with active asthma [12, 15]. The precision of the method is described fully elsewhere [13].

To assess perception of bronchoconstriction, all subjects were asked: whether they were aware of any abnormal respiratory sensation at the point of maximal bronchoconstriction at the end of the methacholine test, even if the decrement in FEV_1 was of a negligible degree; whether the sensation could be described as wheeze, chest tightness, or breathlessness; and whether it had ever been experienced before [16]. Local ethics approval was obtained and all subjects gave written informed consent.

The data from the two surveys were pooled and Chi-squared tests, unpaired t-tests, and stepwise multiple linear and logistic regression techniques were used to identify predictors of participation in the study, symptoms, and airway responsiveness.

Table 1. – Response prevalences from postal and laboratory questionnaires

Item	Questionnaire	1992–1993	1998–1999	Change	p-value
Responses n	Postal	3047	2803		
	Laboratory	626	615		
Wheezing ever	Postal	42.6	45.8	3.2	0.014
	Laboratory	43.0	54.3	11.3	<0.001
Wheezing in last year	Laboratory	29.8	36.4	6.6	0.013
Ever wheeze no cold	Postal	29.6	33.7	4.1	0.001
Ever short of breath and wheeze	Postal	22.8	28.0	5.2	<0.001
Woken wheezing in last year	Postal	9.0	12.4	3.4	<0.001
	Laboratory	12.7	17.7	5.0	0.01
Woken breathless in last year	Postal	10.0	13.6	3.6	<0.001
	Laboratory	8.2	8.7	0.5	NS
Woken with chest tightness in last year	Postal	19.7	23.9	4.2	<0.001
	Laboratory	12.2	13.5	1.3	NS
Woken coughing in last year	Postal	30.9	37.8	6.9	<0.001
	Laboratory	24.5	30.4	5.9	0.02
Asthma attack in last year	Postal	5.4	8.8	3.4	<0.001
	Laboratory	7.5	10.2	2.7	NS
Mean change in symptom prevalence	Postal			4.3	
	Laboratory			4.8	
Current asthma medication	Postal	7.5	12.8	5.3	<0.001
	Laboratory	8.3	10.7	2.4	NS
Asthma ever diagnosed by doctor	Laboratory	12.7	16.9	4.2	0.03
Overall mean change	Postal			4.4	
	Laboratory			4.4	
Hay fever	Postal	28.3	34.4	6.1	<0.001
	Laboratory	26.1	37.8	11.7	<0.001

Data are present as % unless otherwise stated. NS: nonsignificant.

Results

Response rates

The response rates to the postal questionnaire were similar in the two surveys, with 3,047 valid replies in 1992–1993 (51%) and 2,803 in 1998–1999 (47%). The adjusted response rate, which took account of concordance between the Health Authority register and the Electoral Roll, fell from 84% in 1992–1993 to 69% in 1998–1999. The response rate was lower in the younger age groups in both surveys, and this effect was more marked in 1998–1999, resulting in an increase in the mean age of the population from 32.2–34.0 yrs ($p<0.001$). Females were more likely to respond (55.1% of responders in 1992–1993 and 58.1% in 1998–1999), and the change over 6 yrs was significant ($p=0.02$).

The response rate amongst those invited to participate in the laboratory studies was 35% in 1992–1993 and 28% in 1998–1999, giving 626 and 615 participants, respectively. They were slightly older in 1998–1999 (35.0 *versus* 33.2 yrs, $p<0.0001$) reflecting the increased age of the responders to the postal questionnaire, but similar proportions were current smokers (28% in 1992–1993 and 27.5% in 1998–1999, $p=0.8$) or exsmokers (25.5% in 1992–1993 and 21% in 1998–1999, $p=0.2$), and total cigarette consumption was similar (6.3 pack-yrs in 1993–1994 and 5.4 in 1998–1999, $p=0.2$). In both surveys, increasing age and response to an early mailing of the postal questionnaire were the strongest predictors of participation in the laboratory study (odds ratio (OR) 1.03 yrs·older^{-1}, 95% confidence interval (CI) 1.02–1.04; 0.64·mailing^{-1}, 95% CI 0.57–0.72). A positive response to any of the nine respiratory items on the postal questionnaire was also a significant predictor of laboratory participation (OR for any item 1.18, 95% CI 1.04–1.35), as was hay fever (OR 1.23, 95% CI 1.07–1.41).

Questionnaires

The postal and laboratory questionnaires identified substantial increases from 1992–1993 to 1998–1999 in the prevalences of asthmatic symptoms, physician diagnosis, and use of medication (table 1). The prevalences of symptoms increased by a mean of 4.3% on the postal questionnaire and 4.8% on the laboratory questionnaire. There were very similar prevalence increases in physician-diagnosed asthma (4.2%) and the use of medication (4.8%, weighted mean from both questionnaires), giving an average overall change of 4.4% over the 6 yrs. The use of inhaled corticosteroids rose from 5.0% to 9.3% ($p=0.003$), and inhaled β-agonists from 10.5% to 14.1% ($p=0.054$). No dosage information was obtained. In subjects with a physician-diagnosed asthma, inhaled corticosteroid use increased from 37.2% to 52.9% ($p=0.035$), but there was no change in β-agonist use (68% *versus* 64%, $p=0.6$).

The increases were more marked in participants aged <30 yrs (overall 8.7%) compared with those who were older (2.7%). For the laboratory study, in which corresponding measurements of airway responsiveness were obtained, the increases in positive questionnaire responses from these younger participants were especially striking for "asthma attacks in last year" (7.2%, $p=0.025$), "physician-diagnosed asthma" (13.7%, $p<0.0001$), and "current medication" (fig. 1).

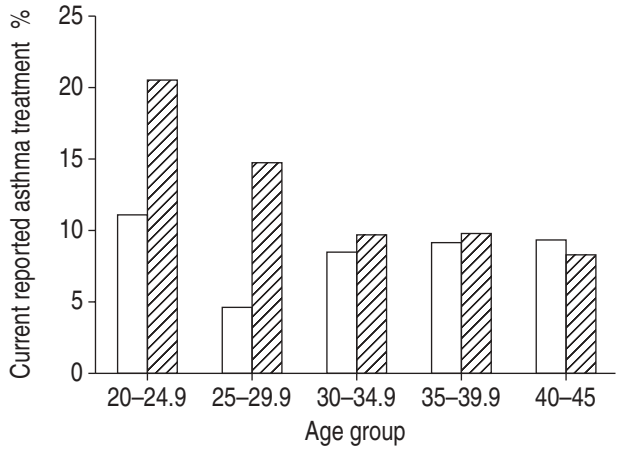

Fig. 1. – Percentages of laboratory populations reporting current asthma treatment in 1992–1993 (□) and 1998–1999 (▨): effect of age.

Airway responsiveness

Methacholine tests were carried out in 591 subjects (302 males and 289 females) in 1992–1993, and 580 (303 males and 277 females) in 1998–1999. The remaining 68 participants in the laboratory studies (from both surveys) either declined or were considered unsuitable because of pregnancy, cardiac disease, epilepsy or baseline FEV_1 <60% of the predicted value.

The means for the baseline measurements of FEV_1 exceeded 100% pred and did not differ significantly between the two surveys (table 2). The baseline FEV_1 is not, therefore, likely to have differently influenced PD_{20} measurements between the two surveys. A positive methacholine test (*i.e.* PD_{20} <6,400 μg) was obtained from 37.2% of subjects in 1992–1993

Table 2. – Lung function, airway responsiveness levels, and atopy

	1992–1993	1998–1999	p-value
Mean FEV_1 % pred (SEM)	102.9 (0.59)	104.3 (0.58)	0.09
PD_{20} <6400 μg	37.2	31.6	0.041
PD_{20} <1000 μg	22.5	16.6	0.01
PD_{20} <200 μg	9.1	7.6	0.338
Geometric mean DRS	0.00527	0.00379	0.004
Atopic[#]	24.8	31.1	0.015

Data are present as % unless otherwise stated. FEV_1: forced expiratory volume in one second; PD_{20}: provocative concentration causing a 20% fall in FEV_1; DRS: dose/response slope. [#]: at least one positive allergen skin test (wheal ⩾4 mm greater than that of saline control) [10].

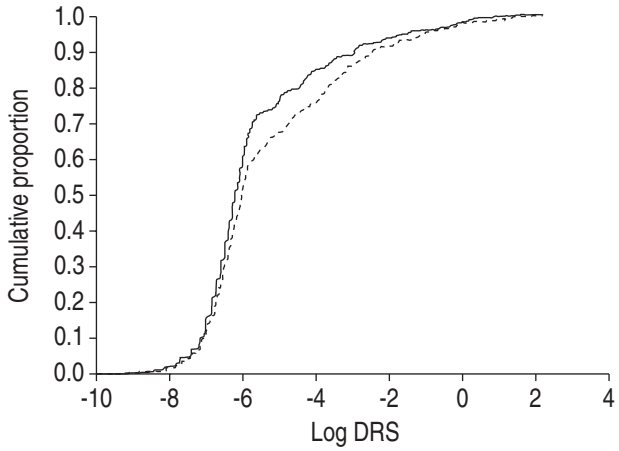

Fig. 2. – Distribution of airway responsiveness (dose/response slope (DRS)) in the population samples of 1992–1993 (—) and 1998–1999 (---). Geometric mean DRS 1992–1993=0.00525, 1998–1999=0.00377, p=0.002.

compared with 31.6% in 1998–1999, indicating that airway hyperresponsiveness was less prevalent in the later survey (table 2 and fig. 2). The geometric mean DRS lessened in parallel from 0.00527 to 0.00379 (p=0.004). Multiple regression analyses showed that this association with survey year was of borderline statistical significance after adjusting for the effects of sex, smoking, and baseline FEV1 expressed as a percentage of the predicted value (table 3). The OR for a positive methacholine test in 1998–1999 compared with 1992–1993 was 0.77 (95% CI 0.58–1.01). Similar results were obtained for the analysis of PD_{20}<1,000 μg (OR 0.64, 95% CI 0.49–0.90), PD_{20}<200 μg (OR 0.79, 95% CI 0.49–1.26), and log DRS (t=-3.17, p=0.002).

For the younger participants, aged <30 yrs, the prevalence of airway responsiveness at a level consistent with active asthma (PD_{20}<1,000 μg) fell from 26.9% to 22.7%, despite the increases in positive responses to the questionnaires.

Table 3. – Logistic regression analysis of airway responsiveness: provocative concentration causing a 20% fall in forced expiratory volume in one second (FEV_1, PD_{20}) <6400 μg methacholine

	Odds ratio	95% Confidence interval	p-value
Atopy per positive allergen	2.05	1.71–2.46	<0.001
Sex female *versus* male	2.43	1.84–3.22	<0.001
Smoking status yes *versus* no	2.26	1.68–3.03	<0.001
Baseline FEV_1 per % less than pred	0.94	0.93–0.95	<0.001
Survey 1998–1999 *versus* 1992–1993	0.77	0.58–1.01	NS

NS: nonsignificant; pred: predicted.

Table 4. – Predictors of perception of bronchoconstriction during methacholine tests

	Odds ratio	95% Confidence interval
Year of study 1998–1999 *versus* 1992–1993	2.78	2.04–3.78
Age per year	0.97	0.94–0.99
Sex female *versus* male	1.55	1.14–2.10
Degree of bronchoconstriction per % fall of FEV_1	1.09	1.06–1.13
PD_{20} per band[#]	0.64	0.47–0.89
Atopy[¶] yes *versus* no	1.77	1.21–2.59

Data drawn from both surveys. FEV_1: forced expiratory volume in one second; PD_{20}: provocative concentration causing a 20% fall in FEV_1. [#]: <200 μg, 200–1000 μg, 1001–6400 μg, >6400 μg; [¶]: at least one positive allergen skin test (wheal ⩾4 mm greater than that of saline control) [10].

Perception of bronchoconstriction

Younger age, female sex, the presence of symptoms on the laboratory questionnaire, the degree of bronchoconstriction during the methacholine test, a lower band of PD_{20} measurement (<200 μg, 200–1,000 μg, 1,001–6,400 μg, >6,400 μg methacholine), atopy, and involvement in the 1998–1999 survey were all predictors of the ability to perceive (and report) methacholine-induced bronchoconstriction when the results from both surveys were combined (table 4). In 1992–1993, 62.5% of subjects reported the development of an abnormal respiratory sensation at the end of the methacholine test compared with 77.2% in 1998–1999 (p<0.001, fig. 3). The associations with younger age, symptoms, and the degree of methacholine-induced bronchoconstriction were apparent within both the 1992–1993 and the 1998–1999 populations separately, but the association with sex was only significant in the 1992–1993 population. The association with atopy was of borderline statistical significance in 1992–1993 (OR 1.75, 95% CI 0.99–3.08) but was clearly significant in 1998–1999

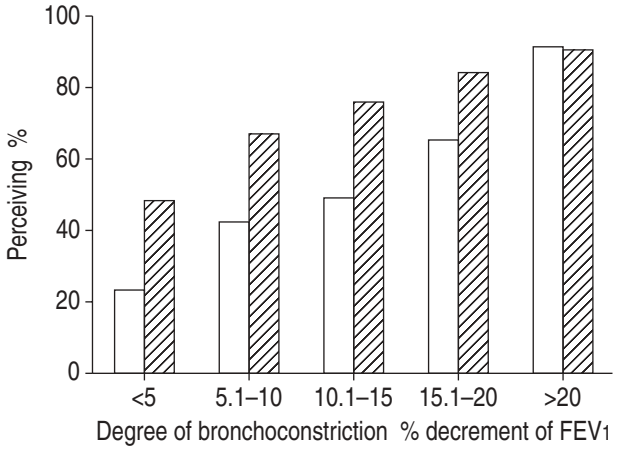

Fig. 3. – Perception of bronchoconstriction during methacholine test in 1992–1993 (□) and 1998–1999 (▨). FEV_1: forced expiratory volume in one second.

(OR 1.99, 95% CI 1.15–3.43). There were no differences between the two surveys in the terms used to describe the respiratory sensation induced by bronchoconstriction, or in the proportion of subjects who reported having experienced this previously.

Atopy

The proportion of subjects with at least one positive allergen skin test (defined as a mean wheal diameter $\geqslant$4 mm greater than the saline control [10]) increased between 1992–1993 and 1998–1999 from 24.8% to 31.1%. This was mostly attributable to an increase in responses to Timothy grass pollen, with a nonsignificant increase in the responses to *Dermatophygoides pteronyssinus*. The significant predictors of atopy were younger age (OR 0.95 yr^{-1}, 95% CI 0.93–0.97), male sex (OR 1.7, 95% CI 1.31–2.20), involvement in the 1998–1999 survey (OR 1.56 for 1998–1999, 95% CI 1.20–2.03) and nonsmoking (OR 0.67 for smokers, 95% CI 0.50–0.90). Symptom prevalences increased almost as much in nonatopics as in atopics (3.8% *versus* 5.4%, table 5), and the predictive value of atopy for physician-diagnosed asthma fell slightly between the two surveys (OR 4.3, 95% CI 2.6–6.9 in 1992–1993 and OR 3.7, 95% CI 2.2–6.1 in 1998–1999). Both nonatopics and atopics showed decreases in the levels of airway responsiveness, with that in the atopics being no less than that in the nonatopics (table 6). For the logistic regressions that assessed the predictors of symptoms and airway responsiveness, the year of survey (*i.e.* change from 1992–1993 to 1998–1999) did not show an interaction with atopy (or sex, or smoking). This indicates that atopic status (such as sex and smoking) was not a significant determinant for the changes that were observed.

Table 5. – Absolute changes in prevalence from the laboratory study between 1992–1993 and 1998–1999 for symptoms, physician diagnosis and treatment, by atopic status[#]

	Nonatopic	Atopic
Wheezing ever	10.4	9.0
Wheezing in last year	4.7	8.7
Woken wheezing in last year	3.5	6.6
Woken breathless in last year	0	1.5
Woken with chest tightness in last year	-0.8	5.2
Woken coughing in last year	7.0	3.0
Asthma attack in last year	1.7	3.9
Mean change in prevalence rates	3.8	5.4
Current asthma medication	1.4	3.1
Asthma ever diagnosed by doctor	2.5	5.7
Overall mean increase	3.4	5.2
Hay fever ever	6.9	13.9

Data are presented %. [#]: participant numbers, nonatopic *versus* atopic: 1992–1993, 471 *versus* 155; 1998–1999, 424 *versus* 191.

Discussion

The current authors have identified significant increases relevant to asthma in the prevalences of symptoms, physician diagnosis, and use of medication in the young adult population of Newcastle-upon-Tyne between 1992–1993 and 1998–1999. The rates of increase are substantial and equivalent to a ~5% increase per annum in the number of subjects affected, but they are similar to those reported in other studies in which the same population has undergone repeated investigation [1–5]. The rates of increase are more striking in subjects aged <30 yrs. A recent study of general practice consultation rates suggested that the UK asthma "epidemic" might have peaked around 1993 [17], but the findings of that study could be explained by organisational changes or by changes in treatment. Other data from UK general practice suggest an increase in asthma prevalence between 1994 and 1996, similar to that found in the current study [18].

The increases in asthmatic symptoms in the current study were paralleled by an increase in the prevalence of atopy, as judged by skin-prick test results and questionnaire reporting of hay fever (table 1). The greatest effect was on the skin test response to Timothy grass pollen, which might be the most labile from year to year due to variations in the extent of pollen exposure. However, similar though smaller changes were seen with *D. pteronyssinus* and cat, which suggests a real increase in atopy similar to that found in other studies [1, 2, 19]. As with the asthmatic symptoms, there was a tendency for the increase in atopy to be more marked in the younger subjects, after adjusting for sex and smoking.

Although asthma and atopy were associated in both 1992–1993 and 1998–1999 surveys, the increases in asthmatic symptoms were not much greater in the atopic than the nonatopic groups, and the proportion

Table 6. – Ventilatory function and airway responsiveness in 1992–1993 and 1998–1999, by atopic status

	Nonatopic			Atopic		
	1992–1993	1998–1999	p-value	1992–1993	1998–1999	p-value
Mean FEV_1 % pred (SEM)	103.4 (0.69)	104.9 (0.72)	0.13	101.6 (1.2)	103.1 (0.99)	0.32
PD_{20} <6400 μg	30.6	26.5	0.187	57.5	43.2	0.01
PD_{20} <1000 μg	16.4	10.6	0.014	41.1	30.7	0.052
PD_{20} <200 μg	5.8	3.5	0.115	19.2	17.1	0.62
Geometric mean DRS	0.00377	0.00272	0.004	0.0145	0.00832	0.038

Data are presented as % unless otherwise stated. FEV_1: forced expiratory volume in one second; PD_{20}: provocative concentration causing a 20% fall in FEV_1; DRS: dose/response slope.

of physician-diagnosed asthma that could be attributed to atopy fell slightly from 42% to 38% over the period of the study. Other investigations have identified a similar lack of association between changes in asthma and changes in atopy. For example, the prevalence of atopy among Leipzig schoolchildren increased from 19 to 27% between 1991 and 1996 but there was no change in the prevalence of apparent asthma or airway responsiveness over the same period [19]. Conversely, Australian schoolchildren showed a more than two-fold increase in asthma prevalence over the 1980s with no significant changes in atopy prevalence [5, 7]. Asthma and atopy are not as closely associated as is commonly supposed, even amongst children [20], and these epidemiological studies suggest that factors other than increased atopy or increased exposure to common environmental aeroallergens have been making important contributions to the changing epidemiology of asthma.

UPTON *et al.* [21] recently reported that increased diagnostic awareness could have been entirely responsible for a 2.5-fold increased asthma prevalence in nonatopic subjects between 1972 and 1996. They found no evidence that a similar bias influenced the diagnosis of atopic asthma, but the extent to which the increases in estimates of asthma prevalence generally are related to increased awareness and increased willingness to make the diagnosis is uncertain. The present authors' failure to identify increases in airway responsiveness in parallel with the increased prevalence of symptoms supports the possibility that these factors have exerted an important influence.

The current findings are not likely to have been due to a measurement artefact. The dosimeter and nebulisers were identical in the two surveys, the dosimeter was calibrated for aerosol output before each survey using an ion tracer method, and there was considerable overlap in the research personnel [22]. Furthermore, the increase in symptom prevalence noted from the questionnaires in 1998–1999 was accompanied by an increased, not decreased, reported awareness of bronchoconstriction in association with the methacholine tests. The increase was seen only with decrements of FEV_1 <20% and was most marked at the lowest level of bronchoconstriction. Thus, reported perception of FEV_1 decrements <5% increased in prevalence from 22.9 to 55.1%, while for decrements of 16–20% the increase was from 74.7 to 86.5% (fig. 3). Decrements in FEV_1 of <5% indicate levels of airway responsiveness well below those conventionally associated with asthma, and they suggest that symptoms are now being reported after relatively trivial changes in airway calibre.

The prevalence of inhaled corticosteroid use increased from 5.0 to 9.3% over the period of the current study. Such medication can be expected to increase mean PD_{20} values 3–4 fold [23, 24], and is therefore likely to be the chief explanation for the observed decrease in the measured mean level of airway responsiveness in 1998–1999. In an attempt to adjust for these differences in corticosteroid use, the data was re-analysed after lowering the PD_{20}s of those using inhaled corticosteroids by a factor of four. Thus, the levels of airway responsiveness that would have been present at the times of both surveys had it not been for corticosteroid use were estimated. There were then similar proportions of the population in the PD_{20} <200 μg category in 1992–1993 (9.6%) and 1998–1999 (9.1%). The corresponding figures for the PD_{20} category <1,000 μg were 22.8% in 1992–1993 and 17.4% in 1998–1999. Similar outcomes were observed when the atopics and nonatopics were analysed separately. These analyses strengthen the current authors' beliefs that the diminished level of airway responsiveness in 1998–1999 can be largely explained by the increased use of inhaled corticosteroids, and that this increase in corticosteroid use did not mask an actual increase in airway responsiveness. The findings are consistent with other recent studies. PEAT and colleagues [5, 7] noted an increase in asthma symptoms but a nonsignificant decrease in the distribution of airway responsiveness in young adults in Australia, and, in a case-controlled study, RICHTER *et al.* [25] showed almost a doubling of asthma diagnoses in Hamburg from 1990–1991 to 1995–1996 but a slight decrease in airway responsiveness [25].

The overall importance of selection biases to the present findings is difficult to estimate. They are potentially important, as the presence of respiratory symptoms is a recognised determinant of participation in such studies [26]. To the present authors' knowledge, the current study is the first repeat survey of a population following the ECRHS template and so the repeatability of the methodology is unknown. The response rates in these surveys were within the range reported by centres participating in the ECRHS, but they were low and the response rate in the second survey was lower than the first. The method of DRANE [27] was used to estimate overall symptom prevalences in the population. This assumes a linear relationship between symptom prevalence and the probability of responding to a postal questionnaire, and it extrapolates results from a series of mailings to estimate the prevalence in nonresponders. It did not lead to any reduction in the estimates of the prevalence increases in asthma symptoms, physician diagnosis, or treatment. There were also no systematic differences in response to the postal questionnaire between those who did and did not take part in the subsequent laboratory studies. The critical point is that recruitment bias is not likely to explain the discrepant findings of increased symptoms yet decreased airway responsiveness.

VAN SCHYACK *et al.* [28] demonstrated that individuals with poor perception of bronchoconstriction are less likely than others to seek medical attention. The converse is also likely to be true, and the increased perception of bronchoconstriction reported in the current population over 6 yrs in association with the methacholine tests is likely to have contributed to the increased prevalences of asthmatic symptoms and physician-diagnosed asthma. Little is known about the mechanisms that underlie the perception of bronchoconstriction and the explanation for the change in this population is unclear. Bronchoconstriction may not have been the true cause in all the subjects responding positively, since measured decrements in FEV_1 were of trivial degree in some. It is

likely, however, that there were more subtle physiological changes, perhaps at the small airway level. The authors have consequently used the term "bronchoconstriction" somewhat loosely to indicate the maximum effect generated by the administration of methacholine.

Females, younger subjects, and atopics are recognised to have an increased ability to perceive bronchoconstriction [29] and these relationships were apparent in the present study. The increase in the prevalence of atopy was not, however, a major factor because this increased ability to perceive methacholine-induced bronchoconstriction was seen almost equally amongst both atopics and nonatopics, and there was no interaction between atopic status and the influence of the second survey of 1998–1999. Treatment with inhaled corticosteroids has been reported to increase the perception of bronchoconstriction [30] but this is unlikely to have had an effect as substantial as was seen in the present study population. Psychological factors can influence the perception of most sensory stimuli and it is likely that increased education and awareness of asthma among the current population was responsible. About 15% of the young adult population have intermediate levels of airway responsiveness (PD_{20} between 200 and 1,000 μg using the current authors′ techniques) but in only ~20% of these have physician-diagnosed asthma [16]. These 20% show few, if any, differences from the 80% majority in terms of airway physiology, so changes in the ability to perceive bronchoconstriction (and report it) have the potential to make a substantial contribution to estimates of asthma prevalence.

The current authors believe that the results presented here are consistent with the hypothesis that the current rise in asthma symptom recognition, diagnosis, and treatment in adults is largely a consequence of better education, increased awareness of symptoms, and/or increased readiness to report them, and an increased willingness of physicians to make the diagnosis and initiate treatment. No objective evidence of any increase in the prevalence or severity of the pathophysiological features of asthma was found.

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