Prevalence of Adult Asthma Symptoms in Relation to Climate in New Zealand

Simon Hales, Simon Lewis, Tania Slater, Julian Crane, and Neil Pearce

Wellington Asthma Research Group, Department of Medicine, Wellington School of Medicine, Wellington, New Zealand

We conducted an ecological study linking prevalence of adult asthma symptoms with climate in the 93 New Zealand general electorates. For each electorate, the 12-month period prevalence of self-reported asthma symptoms was determined using a random sample of adults aged 20–44 on the 1991 New Zealand electoral roll. Long-term average climate was estimated using a national climate database and a geographic information system. Asthma prevalence was calculated within quartiles of the exposure variables. Independent effects of climate variables were assessed using linear regression models, with adjustment for confounding by climate, social deprivation, and geographic variables. There was a statistically significant association between asthma prevalence and mean temperature, with the lowest quartile of mean temperature having an approximately 2% lower asthma prevalence. After adjusting for confounding, there was a monotonic increase in asthma prevalence within quartiles of temperature. The results of this study are in agreement with other research suggesting a lower prevalence of asthma at low temperatures. Although on short (day-to-day) time scales, low temperatures may have a direct effect resulting in acute exacerbations of asthma symptoms, warmer average temperatures are associated with increased asthma prevalence. The reasons for this are unclear, although it is possible that on longer term (annual) time scales, higher temperatures are associated with higher levels of allergen exposure. *Key words*: adult, altitude, asthma, climate, epidemiology, geography, prevalence, temperature. *Environ Health Perspect* 106:607–610 (1998). [Online 17 August 1998]

http://ehpnet1.niehs.nih.gov/docs/1998/106p607-610hales/abstract.html

In New Zealand, adult asthma prevalence is relatively high, as in other English-speaking countries (*1*). A national survey, carried out during 1991–1993, found substantial regional variation in asthma prevalence among New Zealand general electorates (*2*). The prevalence of adult asthma symptoms appeared to be high in most urban areas and lower in most rural areas, but there was wide variation among rural areas.

There is evidence that asthma severity is related to seasonal and meteorological factors, and there has been considerable debate about the possible role of various environmental factors in explaining temporal and geographical patterns in asthma prevalence (*3–9*). It is therefore reasonable to hypothesize that climate may affect asthma symptom prevalence and frequency, either directly (for example, via an effect of air temperature on airway responsiveness), or indirectly (for example, via altered exposure to infections, aeroallergens, or air pollutants). In this study, we investigated the hypothesis that climatic factors have a role in the regional differences in adult asthma symptom prevalence in New Zealand. New Zealand is an ideal setting for the purposes of this investigation because the climate ranges from subtropical in the north to subarctic in the south.

Methods

Asthma prevalence. The European Community Respiratory Health Survey (ECRHS) measured adult asthma symptoms and severity in a number of countries, using standardized methods. New Zealand participated in the ECRHS, initially involving surveys in Auckland, Hawkes Bay, Wellington, and Christchurch in 1991–1992. The survey was subsequently extended to cover the whole country in 1993.

The methodology for the survey has been described in detail elsewhere (*10*). Briefly, a one-page questionnaire was mailed to 31,470 people aged 20–44, chosen from the 1991 New Zealand electoral roll, sampling at least 1 in 40 from each electorate.

Addresses of registered voters in the appropriate age range were obtained from the electoral office, and the questionnaire was sent to a random sample of these in each electorate, along with a letter explaining the purpose of the study. We made an attempt to telephone those people who had not responded after two reminders had been mailed to them. Respondents were asked to answer "yes" or "no" to seven questions relating to asthma and to provide basic demographic details. The overall response was 82% (excluding ineligibles). The only modification made to the ECRHS questionnaire for the purposes of the New Zealand study was the addition of a question on ethnicity. We defined asthma according to the ECRHS definition (the proportion of subjects who reported one or more of the following: had awakened with shortness of breath in the past 12 months, had an attack of asthma in the past 12 months, or was currently taking asthma medication). Asthma prevalence was directly standardized for age

and ethnicity, using the population profile of the New Zealand population (*2*).

Climate data. Monthly climate data for 1970–1995 were obtained from a national database (*11*). About 5,000 weather stations had records for one or more variables of interest (temperature, rainfall, relative humidity, atmospheric pressure, wind speed, sunshine hours, occurrence of fog) and the longitude and latitude of the weather station. For each weather station, we calculated means and standard deviations for each of the climate variables. Not all variables were recorded by all stations or for all years; data were only used where at least a full year's records were available. Because we were primarily interested in the effect of long-term average climate, all of the 25-year period of climate data were used.

Each weather station was assigned to 1 of the 93 electorates using the Mapinfo geographic information system (GIS) (*12*). A vector map of the 1991 electorate boundaries and the latitude and longitude of each station were imported into Mapinfo. Because seven electorates (7.5%) contained no weather stations, a 5-km buffer zone was created around each weather station, and a station was assigned to an electorate where the electorate contained any part of the station's buffer zone.

For each electorate, we calculated climate estimates by averaging the station-specific values for all weather stations assigned to them by the GIS. Where electorates still lacked data for one or more climate variables, these values were assumed to be the same as for an adjacent electorate, chosen by reference to a map of the electorate boundaries, without knowledge of the values for the climate variables there.

Geographic data and social deprivation. Data from a vegetation survey carried out in 1981–1983 were used to estimate the proportion of land area in each electorate devoted to urban areas. Although these data are quite old, the pattern of urban land use is unlikely to have changed greatly. The geographic

variables—urban land use, total land area, and the average altitude of each electorate—were calculated using Arcview "spatial analyst" (13).

Average levels of social deprivation in each electorate were estimated using the NZDep91 index of deprivation (14). NZDep91 deprivation scores for each mesh-block were aggregated by electorate.

Data analysis. The data were entered onto an IBM-compatible computer and analyzed using SAS software (15). We first calculated asthma prevalence within quartiles of the exposure variables. Two linear regression analyses were conducted. Initially, a linear regression was conducted using continuous values for all variables. A second analysis was based on quartiles of the exposure variables because some variables had associations that were not monotonically increasing in the univariate analysis (Fig. 1).

We conducted the analysis in three stages. In stage 1, means of climate variables were entered; the standard deviations (SDs) were then entered in stage 2. The standard errors (SEs) of the regression coefficients were compared with those of the same variables in the stage-1 analysis to check for multicollinearity. Finally, the geographic variables were entered in stage 3, and the occurrence of multicollinearity was checked once again. Multicollinearity was then minimized by removing the variables for urban land use and the standard deviations of atmospheric pressure, temperature, fog, humidity, sun, wind speed, and wind direction.

Results

Asthma prevalence by quartiles of climate and geographic variables. Table 1 and Figure 1 show the asthma prevalence by quartiles of the climate and geographic variables. The analysis involved both quartiles of means (e.g., mean temperatures) and quartiles of standard deviations (e.g., SD of temperature) to reflect the amount of month-to-month variation. The strongest findings were for mean temperature, for which the lowest quartile had an approximately 2% lower prevalence, and for altitude, for which the highest quartile had an approximately 2% lower prevalence.

Climate. There were approximately linear trends toward lower asthma prevalence with increasing frequency of fog, variability of fog, and variability of relative humidity. However, most of the relationships between climate and asthma prevalence appeared to be nonlinear (Fig. 1). Asthma prevalence tended to be lowest among those in electorates with the lowest quartile for mean temperature, highest quartile for rainfall, lowest quartile for atmospheric pressure, highest quartile for fog, and the highest quartiles for variability of these factors.
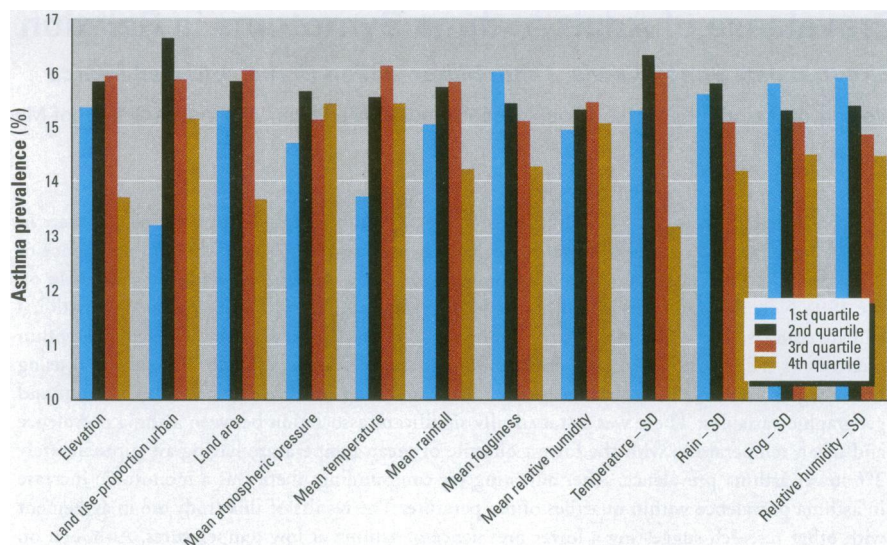

Figure 1. Asthma prevalence by quartiles of the explanatory variables.

Table 1. Quartiles of climate variables, mean asthma prevalence for each quartile, and *p*-values for linear trend (univariate analysis)

	Exposure quartile			Mean asthma prevalence (%)				
	1st	median	3rd	1st	2nd	3rd	4th	*p*-value
Elevation (m)	50	93	255	15.3	15.8	15.9	13.7	0.004
Urban land use (%)	0.4	9.8	75.9	13.2	16.6	15.8	15.1	0.72
Land area (km²)	9	62	1534	15.2	15.8	16.0	13.7	<0.001
Social deprivation index	964	988	1016	15.4	14.8	15.5	14.9	0.62
Monthly means								
Atmospheric pressure (hPa)	1013.8	1015.4	1016.1	14.7	15.6	15.1	15.4	0.98
Temperature (°C)	12.0	13.2	14.5	13.7	15.5	16.1	15.4	0.021
Rain (mm)	95	107	124	15.0	15.7	15.8	14.2	0.38
Fog (days)	0.75	1.0	1.8	16.0	15.4	15.1	14.3	0.25
Relative humidity (%)	80.5	82.1	83.9	14.9	15.3	15.5	15.1	0.55
Standard deviations								
Temperature (°C)	3.17	3.31	3.62	15.3	16.3	16.0	13.2	0.001
Rain (mm)	50.54	58.56	68.27	15.6	15.8	15.1	14.2	0.13
Fog (days)	1.09	1.44	1.92	15.8	15.3	15.1	14.5	0.27
Relative humidity (%)	4.8	5.3	6.2	15.9	15.4	14.9	14.5	0.025

Geography. The climate features described above are typical of sparsely populated, mountainous regions. Consistent with this, the lowest asthma prevalence was found in electorates in the highest quartile of altitude, the lowest quartile of urban land use, and the highest quartile of land area (therefore of low population density, as the electorates have similar total populations). Examination of tables of electorates ranked according to explanatory variables confirmed that these features tend to be clustered (data not shown).

Regression analysis. The results of the regression analysis are shown in Table 2. In the continuous analysis, there was a significant association between asthma prevalence (standardized for age and ethnicity) and mean temperature. An increase in mean temperature of 1°C was associated with an increase in asthma prevalence of almost 1%. Although mean temperature showed a nonmonotonic

association with asthma prevalence in the univariate categorical analysis (Table 1, Fig. 1), this was not apparent in the multivariate categorical analysis (Table 2). Asthma prevalence was 2% higher in the second quartile and 4% higher the third and fourth quartiles compared to the first quartile of temperature.

There were also significant inverse associations between asthma prevalence and both atmospheric pressure and land area (Table 2). The low asthma prevalence at high altitude in the univariate analysis (Table 1, Fig. 1) was not apparent in the multivariate analysis (Table 2).

Discussion

The main finding in this study is the association of mean temperature with asthma symptom prevalence. The reasons for this are unclear, although it could be related to levels of allergen exposure. Climatic factors such as

Table 2. Multivariate regression analysis (continuous and categorical) of climate variables and asthma prevalence

| | | | | Analysis by quartile | | | | | |
| | Continuous analysis | | | 2nd quartile | | 3rd quartile | | 4th quartile | |
Variable	Coefficient	SE	p-Value	Coefficient	SE	Coefficient	SE	Coefficient	SE
Elevation (m)	0.00004	0.00003	0.19	0.008	0.011	0.007	0.012	0.014	0.016
Area (km²)	-0.000006	0.000002	<0.01	0.011	0.011	0.002	0.014	0.005	0.017
Pressure (hPa)	-0.0079	0.0030	0.01	-0.010	0.014	-0.015	0.017	-0.015	0.018
Temperature (°C)	0.0067	0.0030	0.03	0.027	0.013	0.041	0.016	0.040	0.019
Rainfall, mean (m)	0.0004	0.0002	0.08	0.026	0.014	0.045	0.016	0.028	0.020
Fog (days)	-0.0003	0.0028	0.91	-0.017	0.011	-0.011	0.011	-0.024	0.013
Humidity (%)	0.0005	0.0012	0.69	-0.005	0.011	0.009	0.011	<0.001	0.012
Sun (hr/day)	-0.0002	0.0003	0.52	0.004	0.011	0.007	0.013	-0.004	0.013
Wind run (km)	-0.00001	0.00003	0.61	-0.006	0.013	-0.033	0.012	-0.011	0.011
Wind speed (m/sec)	0.0004	0.0018	0.83	-0.002	0.010	0.003	0.012	<0.001	0.014
Rainfall, SD (m)	-0.0007	0.0004	0.08	-0.043	0.014	-0.032	0.016	-0.042	0.021
Social deprivation index	0.0001	0.0001	0.28	-0.005	0.009	-0.012	0.011	-0.017	0.011

temperature and rainfall can affect respiratory function directly or via indirect mechanisms. Effects occurring over short time scales might be expected to give rise to relatively consistent associations between exposure and effect. A number of recent studies have examined asthma epidemics or acute exacerbations in relation to short term (day-to-day) changes in climate, air quality, and aeroallergens. These studies report reasonably consistent associations between low temperatures, exposure to air pollution or aeroallergens and acute worsening of asthma symptoms. Seven of the studies examined the effect of rainfall (16-22), and four of these reported a significant effect (17,18,20,22). In one, the direction of the effect depended on the season (18); in the other three, there was an association between asthma exacerbations and increased rainfall. Nonepidemic asthma was associated with fungal spores in three studies (17,21,23). One study reported associations with both humidity and wind direction (16), and another with humidity and atmospheric pressure (20).

The probable mechanism of thunderstorm-associated asthma is release of allergenic starch granules from grass pollen, triggered by rainfall (24). Other studies of asthma epidemics found evidence of unusual exposure to an aeroallergen, such as soybean dust or grass pollen. In addition, wind speed and direction were usually important in these studies, as might be expected if the main exposure pathway is atmospheric transport of allergen.

Since it is possible that related mechanisms influence the initiation of asthma and asthma exacerbations, it is plausible to assume that climate factors may also affect asthma prevalence. However, it should be stressed that the factors which affect asthma prevalence may not be the same as those that cause exacerbations and acute attacks. In addition,

indirect influences of climate on asthma prevalence may be difficult to demonstrate. For example, chronic exposure to aeroallergens of biological origin (such as pollens, spores, and insect allergens) will depend on diverse factors other than climate, and any climate relationships might be quite locally specific depending on the species involved.

Several recent studies have examined geographical patterns of asthma prevalence, but as yet none have studied the role of climatic factors explicitly (1,25-29). Two studies reported an increased prevalence of asthma among people living in coastal areas compared to inland areas (27,28) and two reported an increased sensitization to house dust mite allergens in asthmatic subjects living in coastal areas compared to inland areas (28). These authors suggested that their results might be explained by relatively high exposure to house dust mite allergen in humid coastal areas. In support of this, Charpin et al. (25,30) found an increased prevalence of asthma, exposure to house dust mites, and allergic sensitization to house dust mite allergen in coastal Marseille, compared to the town of Briancon situated at an altitude of 1,350 m (25,30). On the other hand, several studies have found population patterns of asthma prevalence to be unrelated to patterns of house dust mite exposure (31,32).

Exposure to house dust mite allergen is known to be an important factor in asthma etiology (9,33,34), and the growth of mite populations is sensitive to climate. Increasing temperature (up to 30°C) favors development of these mites, provided that the relative humidity is high enough (35,36). We found no evidence of a relationship between asthma prevalence and humidity, but levels were generally high (median 82%) and varied relatively little.

Global mean temperatures have increased in recent decades, but this could

only account for a small proportion of the concurrent trend in asthma prevalence, based on our results. Although it is possible that indoor temperatures have increased more substantially in recent decades, trends in asthma prevalence probably cannot be explained by increased exposure to insect allergens alone (4-6,34).

The possible role of air pollutants in asthma epidemiology is the subject of much debate (7,8). There is good evidence that air pollution can cause acute respiratory symptoms, but the evidence that polluted areas tend to have higher asthma prevalence is conflicting. This may reflect the difficulty of controlling adequately for confounding variables in the geographical studies. We found evidence of increased asthma prevalence in urban areas compared to rural areas. Although there is little information available about levels of air pollution in New Zealand, levels of most pollutants are probably low in comparison to other countries. The effect of climate on air pollution may be quite locally specific in New Zealand (for example, Auckland suffers from photochemical pollution in summer, while in Christchurch, the main problem appears to be winter smog due to temperature inversions). This will have limited our ability to detect an effect of air pollution based on proxy climate variables. There is evidence that, in combination, air pollution and exposure to aeroallergens may have synergistic effects (8,37). However, the asthma prevalence in Christ-church was similar to that in less-polluted urban areas. Thus, although we cannot exclude some role for air pollution in explaining the pattern of asthma prevalence in New Zealand, this seems unlikely to explain the extensive regional differences reported here.

The results of this study are in agreement with other research suggesting a low prevalence of asthma at high altitude and at low temperatures. Although on short (day-to-day) time scales, low temperatures may have a direct effect resulting in acute exacerbations of asthma symptoms, in New Zealand, warmer temperatures are associated with increased asthma prevalence (adjusting for the effects of other climate factors, altitude, land area, and social deprivation). The reasons for this are unclear, although it is possible that on longer term (annual) time scales, higher temperatures are associated with higher levels of allergen exposure.

REFERENCES AND NOTES

1. ECRHS. Variations in the prevalence of respiratory symptoms, self reported asthma attacks, and use of asthma medication in the European Community Respiratory Health Survey (ECRHS). Eur Respir J 9:687–695 (1996).
2. Lewis S, Hales S, Slater T, Pearce N, Crane J,

Beasley R. Geographical variation in the prevalence of asthma symptoms in New Zealand. New Zealand Med J 110:286–289 (1997).

3. Martinez F. Role of viral infections in the inception of asthma and allergies during childhood: could they be protective? Thorax 49:1189–1191 (1994).

4. Newman-Taylor A. Environmental determinants of asthma. Lancet 345:296–299 (1995).

5. Seaton A, Godden D, Brown K. Increase in asthma: a more toxic environment or a more susceptible population? Thorax 49:171–174 (1994).

6. Strachan D. Time trends in asthma and allergy: ten questions, fewer answers. Clin Exp Allergy 25:791–794 (1995).

7. Burr M. Pollution: does it cause asthma? Arch Dis Child 72:377–387 (1995).

8. Devalia J, Ruszniak C, Davies R. Air pollution in the 1990s—cause of increased respiratory disease? Respir Med 88:241–244 (1994).

9. Balfe D, Crane J, Beasley R. The worldwide increase in the prevalence of asthma in children and young adults. Continuing Med Educ 14:433–442 (1996).

10. Burney P, Luczynska C, Chinn S, Jarvis D. The European Community Respiratory Health Survey. Eur Respir J 7:954–960 (1994).

11. NIWA. Climate database (CLIDB) user's manual. Wellington, New Zealand: National Institute of Water and Atmospheric Research, 1997.

12. Mapinfo. Mapinfo Professional. Troy, NY:Mapinfo Corporation, 1995.

13. ESRI. Arcview Spatial Analyst. Redlands, CA: Environmental Systems Research Institute, 1996.

14. Crampton P, Salmond C, Sutton F. NZDep91: a new index of deprivation. Soc Policy J New Zealand 9:186–193 (1997).

15. SAS Institute. SAS Procedures Guide. Cary, NC:SAS Institute, 1988.

16. Carey M, Cordon I. Asthma and climate: experience from Bermuda, an isolated community. Br Med J 293:843–844 (1986).

17. Khot A, Burn R, Evans N, Lenney W, Storr J. Biometerological triggers in childhood asthma. Clin Allergy 18:351–358 (1988).

18. Bruce P. Asthma and weather: relationship of weather to asthma attacks in Wellington. Weather and Climate 9:15–22 (1989).

19. Rossi O, Kinnula V, Tienari J. Association of severe asthma attacks with weather, pollen and air pollutants. Thorax 48:244–248 (1993).

20. Celenza A, Fothergill J, Kupek E, Shaw RJ. Thunderstorm associated asthma: a detailed analysis of environmental factors. Br Med J 312:604–607 (1996).

21. Epton M, Martin I, Graham P. Climate and aeroallergen levels in asthma: a 12 month prospective study. Thorax 52:528–534 (1997).

22. Lipsett M, Hurley S, Ostro B. Air pollution and emergency room visits for asthma in Santa Clara County, California. Environ Health Perspect 105:216–222 (1997).

23. Delfino RJ, Zeiger RS, Seltzer JM, Street DH, Matteucci RM, Anderson PR, Koutrakis P. The effect of outdoor fungal spore concentrations on daily asthma severity. Environ Health Perspect 105:622–635 (1997).

24. Bauman A. Asthma associated with thunderstorms. Br Med J 312:590–591 (1996).

25. Charpin D, Kleisbauer J, Lanteaume A. Asthma and allergy to house dust mites in populations living in high altitudes. Chest 93:758–761 (1988).

26. von Mutius E, Fritzsch C, Weland S. Prevalence of asthma and allergic disorders among children in United Germany: a descriptive comparison. Br Med J 305:1395–1399 (1992).

27. Laor A, Cohen L, Danon Y. Effects of time, sex, ethnic origin and area of residence on prevalence of asthma in Israeli adolescents. Br Med J 307:841–844 (1993).

28. Peat J, Toelle B, Gray E. Prevalence and severity of childhood asthma and allergic sensitisation in seven climatic regions of New South Wales. Med J Aust 163:22–26 (1995).

29. Devereux G, Ayatollahi T, Ward R. Asthma, airways responsiveness and air pollution in two contrasting districts of northern England. Thorax 51:169–174 (1996).

30. Charpin D, Birnbaum J, Haddi E, Genard G, Lanteaume A, Toumi M, Faraj F, Van der Brempt X, Vervloet D. Altitude and allergy to house dust mites. A paradigm of the influence of environmental exposure on allergic sensitizations. Am Rev Respir Dis 143:983–986 (1991).

31. Martinez F. Complexities of the genetics of asthma. Am J Respir Crit Care Med 156:S117–S122 (1997).

32. Leung R, Ho P, Lam C, Lai C. Sensitization to inhaled allergens as a risk factor for asthma and allergic diseases in Chinese population. J Allergy Clin Immunol 99:594–599 (1997).

33. Peat J, Tovey E, Toelle B. House dust mite allergens—a major risk factor for childhood asthma in Australia. Am J Respir Crit Care Med 153:141–146 (1996).

34. Platts-Mills T, Carter M. Asthma and indoor exposure to allergens. N Engl J Med 336:1382–1384 (1997).

35. Arlian L, Dippold J. Development and fecundity of Dermatophagoides farinae (Acari: Pyroglyphidae). J Med Entomol 33:257–260 (1996).

36. Arlian L. Water balance and humidity requirements of house dust mites. Exp Appl Acarol 16:15–35 (1992).

37. Knox RB, Suphioglu C, Taylor P, Desai R, Watson HC, Peng JL, Bursill LA. Major grass pollen allergen Lol p1 binds to diesel exhaust particles: implications for asthma and air pollution. Clin Exp Allergy 27:246–251 (1997).