% wheeze in past yé	26 24 22 20 18 16 14 12 10 8 6 4 2		Boys													
0		11			12		13		14	1	1	5		16	3	
		Age of child (years)														
Prevalence in boys (%)		19.0		18.5		1	17.1		17.0		14.5			14.5		
Prevalence in girls (%)		17.2		20.4		2	20.9		22.4		23.0			22.0		
Relative risk (boys v girls)		1.10		0.91		0	0.82		0.76		0.63			0.66		
Self reported pre	evale	nce	of	whe	eze	in pa	ast <u>y</u>	yea	r b	y a	ge a	nd s	ex			

in boys. A tendency for girls to overreport asthma symptoms and boys to deny them may have overestimated the size of the sex difference. However, the effect persisted when we used parental responses and also when we restricted the analysis to current wheezers with a doctor's diagnosis of asthma, both of which measures may be less affected by sex. We found no difference in response rates among boys or girls of different ages except for slightly lower rates by both sexes in year 11, which would not have affected the overall trend. Parental response rates did fall with age, but this does not appear to have biased the estimates since the pattern of parent reported prevalence is similar to that of self reported prevalence. It therefore seems unlikely that the sex effect is solely the result of reporting or selection bias.

Hormonal changes occurring in early puberty could have a role in the changing prevalences. Troisi et al reported a positive dose-response relation between

oestrogen use and the risk of adult onset asthma in women.³ Also there is evidence that girls are more likely than boys to develop asthma in adolescence,⁴ which could be hormone related or due to girls experiencing different exposures to triggers for wheeze at this age, such as smoking. The decrease in wheezing prevalence in boys may be related to the increase in size of airways, which are on average smaller than those of girls in infancy.⁵ The effects of hormones and other factors operating during early adolescence on measures of airway inflammation and other markers of asthma deserve further investigation.

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Contributors: SL, JH, and JB designed the study. AV, SL, JH, and MC collected the data. AV analysed the data and wrote the paper. All authors helped with interpreting the data and preparing the manuscript. JB is guarantor for the paper.

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Conflict of interest: None.

Appendix: Respiratory questions asked

 Have you ever had attacks of wheezing in the chest? (a noisy whistling sound from the chest, not the throat, causing tightness and breathlessness)

Have you had any wheezing attacks in the last year?

• Have you ever been told by a doctor that you have asthma?

- Luyt DK, Burton PR, Simpson H. Epidemiological study of wheeze, doctor diagnosed asthma, and cough in preschool children in Leicestershire. BMJ 1993;306:1386-90
- 9 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
- 3 Troisi RJ, Speizer FE, Willett WC, Trichopoulos D, Rosner B. Menopause, postmenopausal estrogen preparations and the risk of adult-onset asthma. A prospective cohort study. Am J Respir Crit Care Med 1995-152-1183-8
- Schachter J, Higgins MW. Median age at onset of asthma and allergic 4 Thintis in Tecumseh, Michigan, J Allergy Clin Immunol 1976;57:342-51. Tepper RS, Morgan WJ, Cota K, Wright A, Taussig LM. Physiological 5
- growth and development of the lung during the first year of life. Am Rev Respir Dis 1986;134:513-9 (Accepted 16 December 1997)

Seasonal variation in coronary artery disease mortality in Hawaii: observational study

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continued over BMJ 1998;316:1946-7 A seasonal variation in cardiac mortality has been noted in both the northern¹² and southern³ hemispheres, with higher death rates during winter than summer. Previous studies reporting seasonal variation in mortality from coronary artery disease examined data from regions with distinct seasonal changes in temperature. To determine whether seasonality in mortality exists in a tropical climate with little variation in temperature we examined the monthly mortality from coronary artery disease among residents of Hawaii.

Methods and results

Hawaii consists of six main islands, with a population of 1.1 million.⁴ We obtained monthly rates of deaths from coronary artery disease (ICD-9 410-414) as recorded on death certificates during 1984-93 from the state of Hawaii. All non-residents of Hawaii were excluded. Because the likelihood of a diagnosis of a respiratory infection might vary by season, we used mortality from respiratory infection (ICD-9 480-487) as a proxy for incidence of respiratory infection. We

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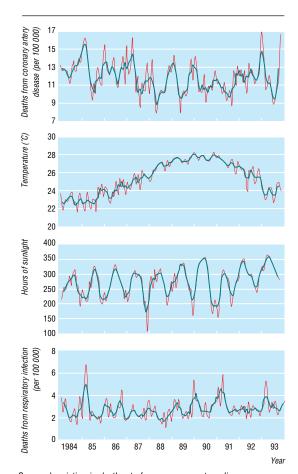
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Seasonal variation in death rate from coronary artery disease, average temperature, death rate from respiratory infection, and hours of sunlight by month 1984-93. Grey lines represent monthly rates and black lines three month moving averages. All curves display significant annual periodicity (P<0.001)

used the average 24 hour temperature and hours of sunlight in Honolulu for each year as an estimate for the entire state.

We used the Cochrane-Orcutt regression analysis to test for annual periodicity. Results were fitted to the equation: $y=b_0+b_1\times\cos(2\pi\times \mathrm{month}/12)+b_2\times \mathrm{sin}$ $(2\pi\times \mathrm{month}/12)$. Goodness of fit was evaluated by the *F* test, testing the significance of b_1 and b_2 . Because secular trends in the management of coronary artery disease might affect death rates, we included the year of death (1986-7, 1988-9, 1990-1, and 1992-3 compared with 1984-5) in the regression models. Pearson correlation coefficients were reported for all comparisons.

During 1984-93 there were 11 010 deaths from coronary heart disease, with average mortality varying from 10.4 deaths per 100 000 population in August to 13.3 per 100 000 in March. Death rates were significantly higher during winter than summer, with the rate in March 22% higher than in August. The average temperature was 25.1°C (range 22.8°C to 27.8°C). Despite the narrow range of temperatures, there was significant annual periodicity with the highest average temperature in August and the lowest in February (figure). The monthly death rate from coronary artery disease and average monthly temperature were inversely correlated (r = -0.55, P < 0.001). Because sunlight and respiratory infections have been postulated to affect mortality from coronary artery disease we examined the monthly hours of sunlight and mortality from respiratory infection. Both sunlight and mortality from respiratory infection showed annual periodicity (figure). There were 2558 deaths from respiratory infection, with the June rate $(2.29/100\ 000)\ 33\%$ higher than the February rate $(3.4/100\ 000)$. The monthly mortality from coronary artery disease was directly associated with the monthly mortality from respiratory infection (r=0.41,P<0.001) and inversely associated with the hours of sunlight per month (r=-0.27, P=0.002).

Comment

We examined the monthly mortality from coronary artery disease in Hawaii, which is closer to the equator than previously studied locations and has a tropical climate with little seasonal variation in temperature. We found a 22% increase in mortality during winter, similar to that noted in Scotland $(30\%)^2$ and New Zealand (33%).³

Seasonal variation in mortality from coronary artery disease in Hawaii is consistent with reports of a greater mortality increase with a given fall of temperature in regions with warm winters.¹ Our results contrast with a report from New Orleans which noted higher mortality from coronary artery disease during summer than winter.⁵ Although the relation between coronary artery disease mortality and seasons could be U shaped, with higher mortality associated with extremely hot or cold weather, our data suggest that even relatively small changes in weather may affect mortality.

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- Eurowinter Group. Cold exposure and winter mortality from ischaemic heart disease, cerebrovascular disease, respiratory disease, and all causes in warm and cold regions of Europe. *Lancet* 1997;349:1341-6.
 - Douglas AS, Allan TM, Rawles JM. Composition of seasonality of disease. Scot Med J 1991;36:76-82.
 - Douglas AS, Russell D, Allan TM. Seasonal, regional and secular variations of cardiovascular and cerebrovascular mortality in New Zealand. Aust NZ J Med 1990;20:669-76.
- Famighetti R, ed. *The world almanac and book of facts 1994*. Mahwah NJ: Funk and Wagnalls, 1993:407.
- DePasquale NP, Burch GE. The seasonal incidence of myocardial infarction in New Orleans. Am J Med Sci 1961;242:468-74.

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Correction

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Cumulative risk adjusted mortality chart for detecting changes in death rate: observational study of heart surgery

An editorial error occurred in this paper by J Poloniecki et al (6 June, pp 1697-700). The authors and contributors were Jan Poloniecki, Oswaldo Valencia, and Peter Littlejohns. Tom Treasure chose not to be an author and was not a contributor.