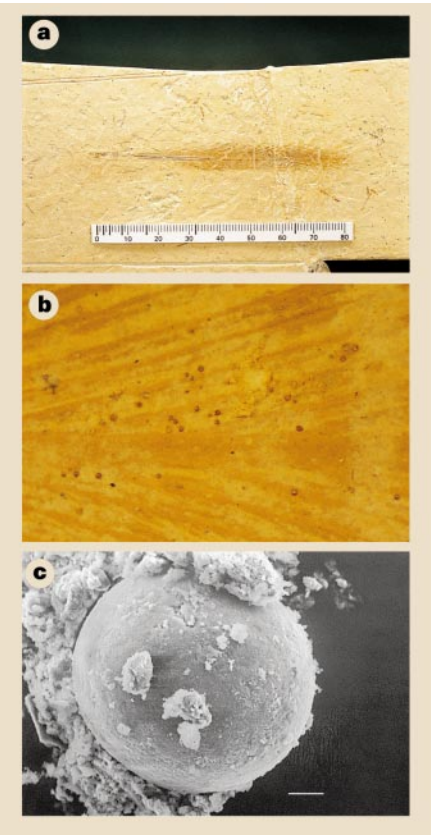

Figure 1 Fossil feather with ectoparasite eggs. **a,** Fossil feather from the Lower Cretaceous (Aptian) Crato Formation, Nova Olindá region, Ceara, Brazil. The feather is 85 mm long. **b,** Detail of the central portion of the feather showing loose clusters of probable ectoparasitic acariid eggs. Individual eggs are between 65 and 70 micrometres in diameter. **c,** Scanning electron micrograph of a probable ectoparasitic acariid egg from the feather. Scale bar, 10 micrometres.

described here are much smaller (70 micrometres in diameter) than the average size for eggs of phthirapterans (more than 500 micrometres), and are more similar to the eggs of small mites (Acari). Furthermore, the eggs are almost spherical, like those of Acari, rather than elongate and barrel shaped, like those of lice. However, several Acari families parasitize birds, and we are unable to identify the family to which the eggs belong.

The oldest record of feather-borne parasites is currently of a mallophagan (feather louse) egg on a feather entombed in Baltic (Oligocene) amber[12]. Our specimens indicate that feather-borne parasites have at least a Lower Cretaceous origin and may be even older, with a co-evolutionary history that perhaps spans more than 140 million years.

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Increased summertime heat stress in the US

In the past half century, the mean summertime temperature in the United States has increased[1–4], with nights warming more than days[5,6]. When humidity is high, hot weather can cause heat stress in humans. Here we show that the frequency of extreme heat-stress events in the United States, caused by extremely hot and humid days as well as by heatwaves lasting for several days, has increased over the period from 1949 to 1995.

We expect increases in mean summertime temperature to be accompanied by an increased frequency of days with extremely high temperature (T) (ref. 7), although there have been few attempts[8] to find observational evidence. Extremes of summertime heat have a greater impact on human health than any other severe weather in the United States[9], with elderly people being most at risk[10].

The combined effects of T and humidity in sultry weather are incorporated in the 'apparent temperature', A (ref. 11), which is a widely used measure of human heat stress. A can be expressed in terms of ambient T and water-vapour pressure (e, in kilopascals) as $A = -1.3 + 0.92\,T + 2.2\,e$ (ref. 11). In the United States, mean summertime A is increasing faster than mean summertime T because humidity has increased by several per cent per decade[4].

Analysis of mortality statistics and weather data for a few dozen cities in the United States has revealed that there are threshold T (ref. 10) and A (J. Detwiler, R. Livezey and L. Kalkstein, unpublished data) values above which mortality increases sharply. These threshold values are closely correlated with the 85th percentile values of T and A derived from climatological data (see Supplementary Information). Here we use climatology-based thresholds of A and T from 113 US weather stations (mainly at airports) to define events of extreme heat stress. The thresholds are locally defined on the basis of three-hourly temperature and humidity data for 1961 to 1990. Separate thresholds were computed for daily-average, daily-maximum and daily-minimum A and T (see Supplementary Information for a map of the thresholds of daily-average A). The thresholds are based on data for July and August (which are the hottest months), but they can be exceeded during any month of the year: they are typically exceeded about 12 times a year.

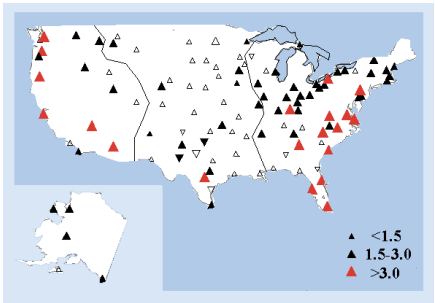

Figure 1 Trends in the annual frequency of the daily-minimum apparent temperature exceeding local threshold values from 1949 to 1995. The size and colour of the triangle indicates the magnitude of the trend (ranging from −2.7 to +5.2 per decade); its orientation (on its base or its apex) indicates the sign of the trend (positive or negative, respectively). Filled triangles indicate significant trends (at $P < 0.05$, using non-parametric methods)[4,14].

Table 1 **Trends in extreme heat events in the United States**

		Regional decadal trends		
	United States	Western	Central	Eastern
Days with $A_{ave} > A^*_{ave}$	**1.67**	**2.08**	**1.19**	**2.06**
Days with $A_{max} > A^*_{max}$	0.61	**1.13**	0.46	0.56
Days with $A_{min} > A^*_{min}$	**1.94**	**2.34**	0.79	**2.60**
Three-day heatwaves with $A_{ave} > A^*_{ave}$	**0.32**	**0.37**	**0.22**	**0.39**
Four-day heatwaves with $A_{ave} > A^*_{ave}$	**0.23**	**0.27**	**0.15**	**0.28**
Days with $T_{ave} > T^*_{ave}$	**1.56**	**2.14**	**0.92**	**1.81**
Days with $T_{max} > T^*_{max}$	0.29	**1.13**	0.20	0.03
Days with $T_{min} > T^*_{min}$	**1.79**	**2.41**	**0.41**	**2.48**

Decadal trends in the annual frequency of extreme apparent temperature, A, and temperature, T, for the United States as a whole and for three regions of the country from 1949 to1995. Trends significant for $P < 0.05$ or less are shown in bold. Extreme events are based on local thresholds, defined by the 85th percentile values of July and August daily averages (A^*_{ave} and T^*_{ave}), daily minima (A^*_{min} and T^*_{min}) and daily maxima (A^*_{max} and T^*_{max}). These climatologically based thresholds are closely correlated with thresholds associated with increased mortality (see Supplementary Information).

From 1949 to 1995, the annual frequency of days exceeding the thresholds increased at most stations. These increases are largest for daily-minimum *A*, and the number of high heat-stress nights increased by 25% or more at some locations. The largest and most statistically significant trends occurred in some of the most populated areas, in the eastern and western thirds of the United States (Fig. 1).

The spatial distribution of the trends allows us to present results for three regions of the country (Fig. 1) bounded approximately by the Mississippi River and the continental divide (Rocky Mountains). Regional trends in the frequency of extreme daily-maximum *A* are substantially smaller than those for extreme daily minima, and are statistically significant only in the western region (Table 1). In the eastern and central United States, trends in extreme *A* are larger than trends in extreme *T*. In the western region, where summertime humidity increases are less marked[4], the *A* and *T* trends are similar.

These increases in single-day heat stress events are associated with increases in heatwaves, defined as runs of three or four consecutive days with daily-average *A* exceeding the 85th percentile value. On average, each weather station experiences 1.7 three-day and one four-day heatwaves per year. Upward trends in the frequency of heatwaves are highly significant ($P < 0.01$) in the eastern and western regions (Table 1) and indicate an increase of about 20% in the number of heatwaves over the period from 1949 to 1995.

These trends may be partly associated with increased urbanization. If the spatial extent of urban heat islands has been growing, weather stations at airports near large cities might experience high temperatures more frequently, especially at night[12]. However, the regional consistency of the trends suggests that their origins are not strictly local.

If these climate trends continue they may pose a public health problem[13], particularly as there are increasing numbers of elderly people, who are most vulnerable to heat-related sickness and mortality[10].

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Supplementary information is available on *Nature*'s World-Wide Web site (http://www.nature.com) or as paper copy from the London editorial office of *Nature*.

p53 polymorphism and risk of cervical cancer

Storey and co-workers[1] recently presented results indicating that the allele encoding arginine in the codon-72 polymorphism of the *p53* gene represents a significant risk factor in the development of cancers associated with human papilloma virus (HPV). The form of the p53 protein carrying an arginine residue at this position was found to be significantly more susceptible to degradation by the HPV E6 protein than by the proline form. Genotype analysis of 30 cervical tumours and 12 skin carcinomas revealed that the homozygous Arg/Arg genotype was overrepresented compared with 41 controls. We have now analysed this polymorphism in leukocyte DNA from a larger sample of cancer patients and controls but have found no significant overrepresentation of this genotype.

We analysed leukocyte DNA from 77 cervical-cancer patients[2] who were positive for 'high-risk' HPVs and 92 patients with cervical intraepithelial neoplasia (CIN) grades II–III, of which 72 were positive for 'high-risk' HPVs. For controls, we used 225 females who were also tested for the presence of HPV in DNA from cervical smears, and 109 patients with breast cancer. The CIN patients and controls were from a population-based case-control study[3].

To analyse the codon-72 polymorphism, we digested a 199-base-pair (bp) product from polymerase chain reaction (PCR) with the restriction enzyme *Bst*UI and separated the fragments by polyacrylamide gel electrophoresis[4]. Only the arginine allele is cleaved, giving two fragments of 113 and 86 bp, whereas the proline allele is not cut (the fragment remains 199 bp long). More than 100 samples have been analysed both by this method and by constant denaturant gel electrophoresis, giving the same results. Cytological specimens from the CIN patients and from the controls were analysed for HPV using nested PCR and type-specific primers[3]. The patients with breast cancer were included as additional controls.

Our results on genotype distribution are presented in Table 1. We did not find any significant overrepresentation of homozygotes for the arginine allele either among the cervical-cancer patients or the CIN II–III patients compared with controls. The frequency in patients with breast cancer was similar to the other controls. Comparison of the total patient group with cervical malignancy with the 334 controls, regardless of HPV status, revealed no significantly increased risk for women carrying the Arg/Arg genotype (odds ratio, 1.09; 95% confidence interval, 0.73–1.61; $P = 0.74$). The power of this study to detect a twofold increase in the susceptibility to HPV-associated malignancy for Arg/Arg homozygotes was 92%.

HPV-16- or HPV-18-positive cervical-cancer patients revealed an odds ratio of 1.24 (95% confidence interval, 0.51–2.97; $P = 0.74$) for the Arg/Arg homozygotes, compared to HPV-positive controls. The probability of detecting a sixfold-increased risk among HPV-positive Arg/Arg homozygous individuals, as found by Storey *et al.*, is 96% in our study. We were therefore not able to confirm that HPV-positive women carrying the Arg/Arg genotype have an increased risk of developing cervical cancer.

The frequencies of the *p53* codon-72 genotypes vary according to ethnic group. The frequency in our control group is similar to that found in a Swedish study[5]. Storey *et al.*[1] report frequencies similar to those found in a Japanese population[6]. As infection with cancer-associated HPV types is relatively common among cytologically normal women as well[7], other environmental or genetic cofactors are required for cervical carcinogenesis. It may be that the virus load or the status of HPV integration influences the susceptibility to HPV-associated cancers. An association with HLA specificity has been found among both cervical-cancer and CIN patients[8,9], and a strong interaction between tobacco smoke and HPV-16 is indicated[10]. Further investigation is needed in different ethnic populations to

Table 1 **Genotype distribution in *p53* codon-72 polymorphism**

	Pro/Pro	Pro/Arg	Arg/Arg
Controls ($n = 225$)	13 (6%)	90 (40%)	122 (54%)
HPV-positive controls ($n = 29$)*	0 (0%)	14 (48%)	15 (52%)
Cervical carcinomas ($n = 77$)†	10 (13%)	23 (30%)	44 (57%)‡
HPV-positive CIN ($n = 72$)§	2 (3%)	29 (40%)	41 (57%)
HPV-negative CIN ($n = 19$)‖	1 (5%)	7 (37%)	11 (58%)
Breast carcinomas ($n = 109$)	6 (5%)	40 (37%)	63 (58%)

*Controls positive for 'high-risk' HPVs (12 controls were not HPV-typed).
†Cervical-cancer patients positive for HPV-16 or HPV-18.
‡Odds ratio was 1.24 for Arg/Arg homozygous cervical-cancer patients compared with controls; $P = 0.74$.
§CIN II–III patients positive for 'high-risk' HPVs.
‖CIN II–III patients negative for 'high-risk' HPVs (one CIN patient was not HPV-typed).