Climate Change and the End of the Respiratory Syncytial Virus Season

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The seasons associated with laboratory isolation of respiratory syncytial virus (RSV) (for 1981–2004) and RSV-related emergency department admissions (for 1990–2004) ended 3.1 and 2.5 weeks earlier, respectively, per 1°C increase in annual central England temperature ($P = .002$ and .043, respectively). Climate change may be shortening the RSV season.

In temperate regions, respiratory disease adds greatly to the workload in general practice facilities and hospitals during the winter. This is partly because of cases of bronchiolitis in young children caused by infection with respiratory syncytial virus (RSV). The marked seasonality of these annual epidemics of RSV infection could be associated with meteorological factors influencing either viral survival or infectivity, or it could be indirectly associated because of seasonal variations in social behavior and consequent cross-infection. Inhalation of cold air will slow the mucociliary escalator and reduce phagocytic activity of leukocytes, thereby increasing susceptibility to infection [1]. However, whole-body chilling does not precipitate the common cold, and there is little evidence to suggest that modern urban dwellers alter their normal habits and crowd together indoors in response to changes in the temperature of a few degrees [2]. It is possible that people stay indoors more when ice and snow make the pavement slippery, but this would not affect the onset of the RSV season, which starts during the autumn. In extreme climates in Russia, the proportion of Russian adults who venture outside for >10 min per day for outdoor activity changed little changed between temperatures of 20°C and −20°C [3]. Cross-infection of children at the beginning of the school year has been ruled out as a trigger of the RSV season onset, and it is unclear whether atmospheric pollution has had an influence on the onset of epidemics of RSV infection [4, 5]

Epidemiological evidence of a link between RSV and temperature is also controversial: 2 studies reported an inverse association between temperature and RSV-related hospital admissions [4, 6]. However, 2 other studies did not find a close relationship between temperature and the season's onset [7, 8]. The strongest argument against the suggestion of a causal link with temperature is that epidemics of RSV infection occur in tropical regions, where temperatures are higher when these epidemics occur (which is normally in the rainy season) [9]. However, in the United Kingdom and Scandinavia, no link has been found between RSV season and humidity or rainfall [4, 7].

Global warming has increased temperatures in the United Kingdom during the past few decades [10]. A wide variety of organisms have responded, with winter moth eggs hatching and buds bursting from the oak tree earlier than usual [11]. If epidemics of RSV infection are influenced by temperature, then this increase in the temperature resulting from global warming should alter them, demonstrating that the epidemics are not just spuriously linked with temperature because they occur at the same time of year.

Methods. Weekly data on laboratory reports of RSV isolation by the Health Protection Agency and National Health Service hospital laboratories in England and Wales for 1981–2004 were obtained with permission from the *Communicable Disease Report Weekly.* Identification of RSV was made by any method (e.g., culture, PCR, immunofluorescence, 4-fold increase in the antibody titer, and a single high serology titer).

The number of emergency department admissions to National Health Service hospitals in England and Wales for bronchiolitis was calculated from hospital episode statistics data provided by the Department of Health for the period from January 1990 through December 2004. For the financial years starting in 1989–1994, admissions were identified using the *International Classification of Diseases, Ninth Revision,* code for bronchiolitis of 466.1; thereafter, the codes J21.0 and J21.9 from the *International Classification of Diseases, Tenth Revision,* were used.

Meteorological data were the annual mean daily data from 4 surface stations (Ringway, located at Manchester Airport; Squires Gate, located at Blackpool Airport; Malvern, located within the town of that name; and Rothamsted, located on the edge of the village of that name). These sites are used to con-

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struct the well-known central England temperature series and are representative of a roughly triangular area of the United Kingdom enclosed by Preston, London, and Bristol.

In accordance with International Standard ISO 8601, the end and start of the year's RSV seasons were identified as follows: the end of the season was the first week of the year in which the number of viral isolations and hospital admissions were less than a particular threshold; the start of the next season was the first week in the year in which the number of viral isolations and hospital admission was higher than that threshold. The threshold was set at 60% of each year's average weekly number of isolations and admissions, and that number peaked in January. This approach allowed for large changes in the reporting of laboratory detection of RSV and of hospital admissions for bronchiolitis. The rate of RSV isolations increased from an average 32 isolations per week in 1981 to 239 isolations per week in 1997, but it then decreased to 157 isolations per week in 2004. Similarly, the rate of hospital admissions for bronchiolitis increased from 29 admissions per week in 1990 to 66 admissions per week in 2004. These changes are probably the result of improvements in laboratory techniques and modifications in diagnostic practices and care by physicians. The findings of this study were unchanged if the threshold was set at 50% or 70%, except that the relationship between the week of the disappearance of the RSV epidemic and hospital admission data and temperature was no longer statistically significant ($P = .13$) with a 50% threshold. It was unnecessary to express the RSV data as a prevalence rate because (1) only timings for the start and the end of the RSV seasons were required, and (2) the denominator used to calculate the rate would alter the threshold to the same degree.

Lines were fitted by least-squares regression. Significance was defined as a P value of <.05. Statistical analyses were performed using Stata software, version 5.0 (Stata Corporation).

Results. Annual mean daily temperatures for central England increased by 0.05°C per year (95% CI, 0.019°C–0.080°C; $P = .002$) over the 24 years, from 9.2°C in 1981 to 10.5°C in 2004. Figure 1*A* shows how the RSV season ended earlier as the annual mean daily temperature increased between 1981 and 2004, with the season ending at a rate of −3.1 weeks earlier per 1°C increase in the temperature (95% CI, −4.9 to −1.2 weeks; $P = .002$). No relationship was observed between the start of each season and temperature (regression coefficient, −0.8 weeks per 1°C decrease; 95% CI, −2.1 to 0.48 weeks per 1°C decrease; $P = .212$). The interval between seasons increased by 2.28 weeks per 1°C increase (95% CI, 0.2–4.4 weeks; $P = .034$), indicating that the RSV season has become shorter. No relationships were observed with wind speed, amount of rainfall, hours of sunshine, and relative humidity, with or without allowance for temperature (data not shown).

Figure 1*B* shows how the end of the season for hospital

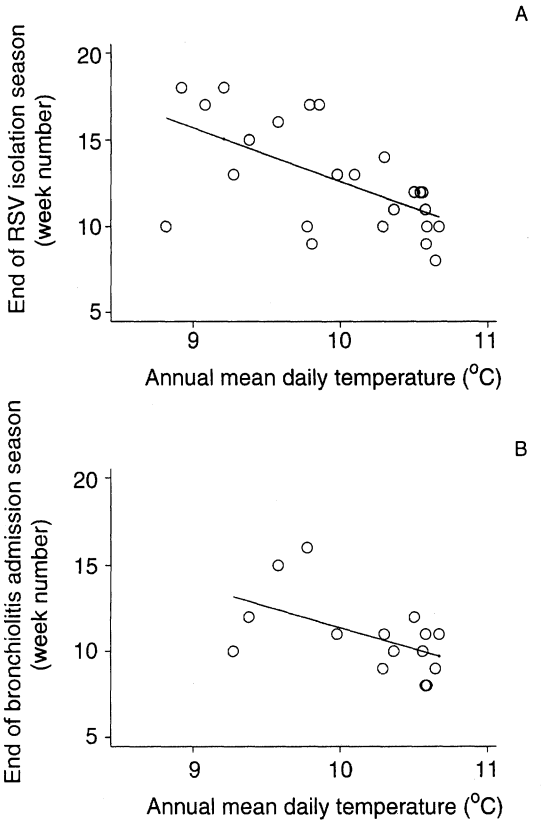

Figure 1. Annual mean daily temperatures in central England and week at which the respiratory syncytial virus (RSV) season ended for laboratory isolations of RSV in 1981–2004 *(A)* and hospital admissions for bronchiolitis in 1990–2004 *(B)*.

emergency admissions for bronchiolitis arrived earlier as the annual mean daily temperature increased over the 15 years from 1990 to 2004. The end of the season arrived −2.5 weeks earlier per 1°C increase (95% CI, −4.9 to −0.1 weeks; $P = .043$). There was no relationship between the start of the season and temperature ($P = .255$) or between the interval between seasons and temperature ($P = .172$), although trends were in the same direction as those found with the RSV isolation data.

Discussion. This study has shown that the RSV season, in terms of both rates of laboratory isolation and hospital admission for bronchiolitis, has ended earlier and its duration has shortened as the climate in England and Wales has become warmer. This is consistent with global warming, which has mostly influenced spring-time events [11]. Why only the spring-time end of the RSV season and not the winter onset of the season has been affected by the increase in temperature is unclear. At first glance, one might argue against the role of temperature, as it could be expected to influence both events. However, these 2 times of the year may be different. People are more likely to be unexpectedly exposed to cold weather in the autumn when they are without their winter coats or thick sweaters, which remain in the wardrobe, or when they have

the central heating in their homes switched off, compared with in the late winter/early spring, when they take such precautions against cold weather. People might acquire immunity at the start of the season, affecting infection rates at the end of the season. These findings imply a health benefit of global warming in England and Wales associated with a reduction in the duration of the RSV season and its consequent impact on the health service.

Inaccuracies in disease coding for hospitalizations for bronchiolitis are unlikely to have biased the data in such a way as to cause a false association with annual temperature. Improvements in diagnostic or care practices over time are unlikely to explain why the timing of the end of RSV season was associated with changes in the annual temperature whereas the start of the RSV season was not. A reduction in the rate of cold-related infection is the corollary to the expected increase in heat-association infections, of which there should be concern.

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References

1. Eccles R. An explanation for the seasonality of acute upper respiratory tract viral infections. Acta Otolaryngol **2002**; 122:183–91.
2. Dowling HF, Jackson GG, Inouye T. Transmission of the experimental common cold in volunteers. II. The effect of certain host factors upon susceptibility. J Lab Clin Med **1957**; 50:516–25.
3. Donaldson GC, Tchernjavskii VE, Ermakov SP, Bucher K, Keatinge WR. Winter mortality and cold stress in Yekaterinburg, Russia: interview survey. BMJ **1998**; 316:514–8.
4. Martin AJ, Gardner PS, McQuillin J. Epidemiology of respiratory viral infection among paediatric inpatients over a six-year period in north-east England. Lancet **1978**; 2:1035–8.
5. Bhatt JM, Everard ML. Do environmental pollutants influence the onset of respiratory syncytial virus epidemics or disease severity? Paediatr Respir Rev **2004**; 5:333–8.
6. Mufson MA, Levine HD, Wasil RE, Mocega-Gonzalez HE, Krause HE. Epidemiology of respiratory syncytial virus infection among infants and children in Chicago. Am J Epidemiol **1973**; 98:88–95.
7. Sims DG, Downham MPPS, Mcquillin J, Gardner PS. Respiratory syncytial virus infection in north-east England. BMJ **1976**; 2:1095–8.
8. Reyes M, Eriksson M, Bennet R, Hedlund KO, Ehrnst A. Regular pattern of respiratory syncytial virus and rotavirus infections and relation to weather in Stockholm, 1984–1993. Clin Microbiol Infect **1997**; 3:640–6.
9. Chan PW, Chew FT, Tan TN, Chua KB, Hooi PS. Seasonal variation in respiratory syncytial virus chest infection in the tropics. Pediatr Pulmonol **2002**; 34:47–51.
10. Hulme M, Jenkins G, Brooks D, et al. What is happening to global climate and why? In: Maynard RL, ed. Health effects of climate change in the UK: report to the Department of Health. London: Department of Health, **2001**:18–49.
11. Visser ME, Holleman LJ. Warmer springs disrupt the synchrony of oak and winter moth phenology. Proc Biol Sci **2001**; 268:289–94.