suggested that antibodies to secretory-excretory antigens are more likely to be related to active migration and infection than are antibodies evoked by somatic antigens—for example, in the indirect fluorescent antibody test[7]—and may therefore indicate a truer causal relation. De Savigny and Tizard found, however, in studies on animals that antibody to secretory-excretory antigens persisted for a long time,[6] and other workers have drawn similar but indirect conclusions about persistence of antibody in man.[3] Clearly further work is required on the serological features of toxocariasis, and this is being undertaken in this department and elsewhere. We hope that doctors in the British Isles will continue to send specimens of serum for toxocara serology from appropriate patients providing as much clinical information as possible for further analysis.

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Influenza surveillance: November 1982-June 1983

As in the previous four winters the prevalence of influenza remained low throughout the 1982-3 season. Most of the indices used in surveillance suggested that influenza was most active from early January to early March. For the first 10 weeks of 1983 clinical influenza was reported to the Royal College of General Practitioners at rates which were about twice the seasonal average for non-epidemic years; unlike the previous winter's influenza there was no definite peak. By the end of the winter 0·5% of the practice populations had consulted with influenza, compared with 1·1% in the previous winter (1981-2), when much of the illness was caused by influenza B virus. Outbreaks were reported in boarding schools, hospitals, and residential units for the elderly.

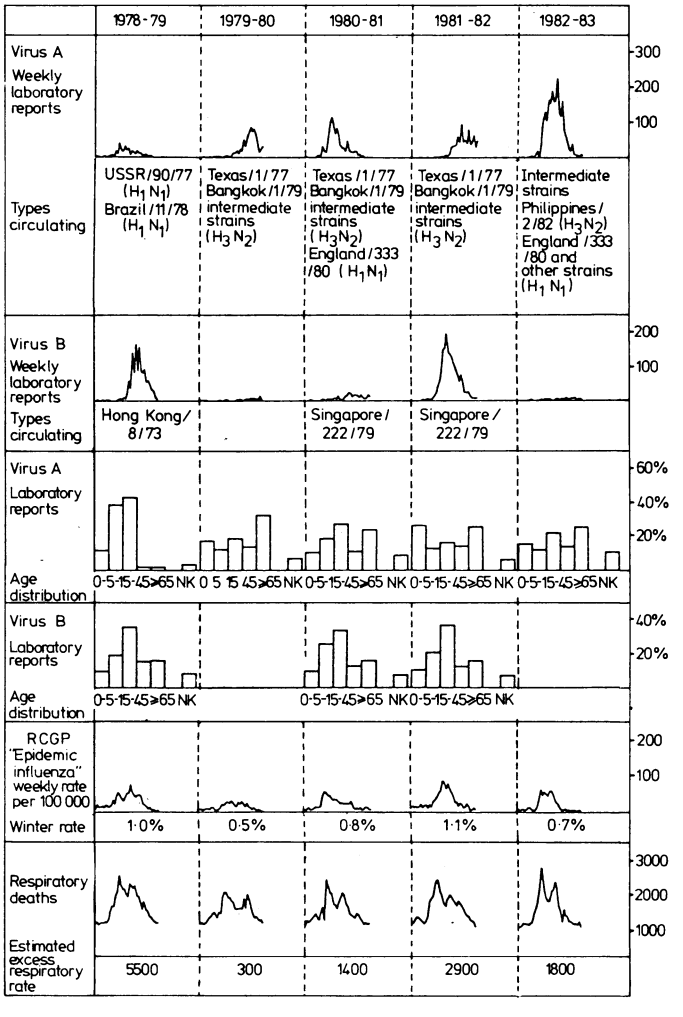
Influenza A viruses were identified from October 1982 to June 1983, and from the second week in January 1983 to the last week in March the numbers of reports exceeded 100 per week, with a peak of 229 in the 10th week of the year, the highest since 1977-8. Eighty per cent of the identifications were of subtype H_3N_2, and the predominance of this subtype was reflected in the wide age distribution of patients with influenza A infections (see figure). H_1N_1 infections tend mostly to attack those below about 25 years of age. Only a few influenza B viruses were identified.

Data for respiratory deaths (see figure) and for deaths from all causes were consistent with the other indices, showing that influenza activity was only fairly low in 1982-3. Mortality started to rise at the end of December, returned to the seasonal average early in March, and remained below average for the rest of the surveillance period. A retrospective estimate of 2000 "excess deaths" was made for the winter period, compared with 3000 in 1981-2 and 17 000 in 1975-6, the last major epidemic winter.

Scotland and abroad

In Scotland the indices for influenza were similar to those of England and Wales, with the largest weekly number of laboratory reports in March; influenza A virus subtype H_3N_2 was predominant.

Influenza A activity was noted in early December in parts of West Germany and Canada. By mid-January sporadic cases and localised outbreaks had also been reported from many European countries. In the United States, though there was widespread activity in several states, overall only a low to moderate peak incidence was reached, with maximum activity generally in February or March; as in Britain, subtype H_3N_2 predominated. Throughout the 1982-3 season in the northern hemisphere the most common strains were intermediate cross reacting viruses closely related to A/Bangkok/1/79 and A/Texas/1/77, but some reacted poorly with antisera to these two variants and resembled A/Philippines/2/82. Thus there was some evidence of antigenic drift. Influenza A H_1N_1 and influenza B were much less frequently reported.



Influenza in the last five winters.