A "new" influenza virus

Last winter the influenza A virus behaved in a quite unexpected way. A new subtype, A/USSR/90/77 (H_1N_1), appeared and spread to most countries of the world; but its effects were confined almost entirely to children and teenagers. Though many human viruses survive and spread when only children are susceptible—measles and chickenpox, for example—the worldwide spread of an influenza A virus when most adults are immune is unique, at least since laboratory studies of the virus began in 1933.

The new virus closely resembles the H_1N_1 influenza viruses of 1947-57, and most people older than about 23 possess serum antibody to its surface haemagglutinin antigen. Influenza virus subtypes may reappear as serious human pathogens at long intervals but only when most of the world population is again susceptible. When the Asian virus (H_2N_2), for example, appeared in 1957 many people over 75 had specific serum antibody to the H_2 antigen but all younger age groups were susceptible.[1 2] The events of the last decade, which has seen the brief reappearance in man of the A swine virus in 1976 and the worldwide spread of so many variants of the A/Hong Kong (H_3N_2) subtype,[3] have not only stimulated research but also deepened the mysteries of influenza epidemiology.

The World Health Organisation sent out warning of the new virus in mid-December 1977, as soon as it had been identified as the cause of outbreaks of influenza among young people in the USSR and Hong Kong. The virus had, however, been recognised in China as early as May 1977 and in the Philippines in June.[4] Serological surveys soon showed that many people born before about 1957 possessed serum antibody to the haemagglutinin (H_1) of the virus, though those over the age of about 50 had it less often than did younger adults. Clinical protection appears to have been fairly well correlated[5] with having lived during the time when the $HswN_1$, H_0N_1, or H_1N_1 viruses were prevalent—that is, from 1918 to 1957; the haemagglutinin antigens of these viruses are now considered to be related.

Formerly, when new influenza A virus subtypes had emerged the preceding antigenic subtype rapidly disappeared throughout the world—but not on this occasion. The epidemic in Britain[6] was similar to that in other countries. Influenza was first reported in December 1977 in an outbreak (in a geriatric unit) caused by the A/Victoria/3/75 (H_3N_2) virus, which had been responsible for influenza in Britain in the winter of 1976-7. A further variant of the A/Hong Kong (H_3N_2) virus, designated A/Texas/1/77, was also detected, sporadically, in January 1978—the month when the H_1N_1 virus, A/USSR/77, was first isolated. All three influenza A viruses remained prevalent throughout the remainder of the winter, with a peak in late February. The epidemic was generally mild, and children were mostly affected; severe outbreaks, which were mainly in boarding schools, were often due to the H_1N_1 virus—though this subtype was isolated only occasionally in Scotland.

The origin of the new H_1N_1 virus is unknown. An antigenic shift to a new subtype seems to arise either from genetic recombination in a host coincidentally infected with more than one influenza virus (of human or animal origin) or from direct crossing of the species barrier by a strain from a non-human reservoir; the latter probably occurred in the A/New Jersey (swine flu) outbreak in Fort Dix in 1976. The selection of a mutant virus is theoretically possible, though the molecular biology of the influenza virus suggests that this is unlikely.[7] Could the present virus, however, simply have remained latent for some 25 years, either in a living host or, perhaps, in a laboratory deep freeze? The A/USSR/77 virus not only closely resembles genetically the A/FW/1/50 (H_1N_1) isolates of 1950 but is also similar to laboratory strains in its ability to grow well in eggs.[5] Although we have no other evidence for this possibility, the events underline the need in all countries for proper containment facilities in laboratories, a matter that has had considerable attention in Britain in recent years.[8]

Whenever antigenic variation is recognised in the influenza virus and a new strain seems capable of causing outbreaks, vaccines may need to be updated. Most people under 24 years are still susceptible to the H_1N_1 virus; and the elderly, with their low resistance to infection, may also be at some risk. H_1N_1 virus antigens should therefore be included in the vaccines for protecting both elderly and young people at special risk next winter. Children may need two spaced doses of H_1N_1 antigen.[9] For most adults, though vaccine containing A/Texas/77 (H_3N_2) antigens should suffice, H_1N_1 antigens in the next vaccine could provide a helpful boost to their immunity against possible further epidemics of this "new" virus.

[1] Mulder, J, and Masurel, N, *Lancet*, 1958, **1**, 810.
[2] Clarke, S K R, *et al*, *Lancet*, 1958, **1**, 814.
[3] *British Medical Journal*, 1976, **1**, 730.
[4] *Weekly Epidemiological Record*, 1978, **53**, 67.
[5] United States Public Health Service, *Influenza Virus Vaccine Workshop, 12 January 1978. Summary Report and Conclusions*. Bethesda, Department of Health, Education and Welfare, 1978.
[6] *British Medical Journal*, 1978, **1**, 1221.
[7] Stuart-Harris, C H, and Schild, G C, *Influenza. The Viruses and the Disease*. London, Arnold, 1976.
[8] *British Medical Journal*, 1978, **1**, 871.
[9] *Weekly Epidemiological Record*, 1978, **53**, 51.

Supplying the NHS

The NHS is not getting the best value for money for the goods it buys; nor are those who use the items getting the most efficient service from the supplies organisation. These unsurprising conclusions come from the report of the Supply Board Working Group[1] appointed last August (under the chairmanship of Mr Brian Salmon) to examine the supply of goods and equipment to the NHS. It represents the sixth inquiry of this kind since 1948, and many of its recommendations echo those of earlier reports.[2-5]

The NHS spends about £700m on items ranging from cornflakes to computers (£170m of it on medical equipment). About 80% of the total expenditure goes on items that are in common use throughout the NHS; but altogether 40% of the money is spent on uncoordinated purchases—often in ludicrously small orders—made by districts and hospitals. The working group considers this to be wasteful and inefficient.

The responsibility for supplies policy and implementation is split among the DHSS's supply division and regional and area health authorities. Because of this fragmentation there is a serious lack of information on purchases, stocks, and patterns of use and a tendency among authorities to disregard contracts made by higher authorities. Both these factors prevent the NHS from putting its enormous purchasing power to best use and providing the user with the best service.

Other shortcomings found by the working group include uncoordinated evaluation, especially for medical equipment; a lack of standard specifications for similar items; and the inability or reluctance of contracting authorities to commit themselves to buying specified numbers of items so that they can secure better terms. None of these findings are new. Nor are the recommendations, which are (among others) that supplies should be bought at the most economic level; that a national computer-based information system should be developed; and that authorities should whenever possible make firm, fixed-quantity contracts. Many of the report's suggestions have been made before and have not been challenged in principle: they just have not been implemented.

Because of the need for some element of compulsion and central co-ordination the working group has suggested that a central supply council should determine supplies policy, leaving staffs of supply division and NHS authorities to implement it. The council, to which Mr Ennals has given his approval, subject to consultation, would have 20 members drawn