hygienic and prevented our children eating each other's or their own faeces we began to nurture them more carefully and to reduce family size so that we had two or three children instead of 15 or 16. Poliomyelitis was then called 'infantile paralysis', because it was never seen except in young children. But as the years passed, so the age group of the persons affected advanced. I think we may be entering a period when some of what were regarded by the older of us as almost automatic infections (like herpes simplex in infancy and childhood) are going to emerge in the future as infections of considerable importance in older people who, because of a changed environment, escape earlier infection. In this respect inherited characteristics are important because the smaller our families the more likely we are to be careful in their nurturing and to delay their experience of the common viruses.

Last of all, one of the great epochs in medicine was the eradication of a group of infections following the remarkable improvement in sanitation that took place in the second half of the nineteenth century. All the magnificent methods that we now have for dealing with sewage and ensuring a safe water supply are very good for bacteria, but quite often they are useless in regard to viruses. The contamination of our water supply with viruses is something of which we are practically unaware. We may well have, in the second half of the twentieth century, just as big a problem in eliminating viruses from our water supply as we had with bacteria at the end of the nineteenth century.

Virus prevalence in Scotland

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You might be forgiven these days for wondering whether the enormous range of weird and wonderful new viruses with unfamiliar names is genuine or just a by-product of the fact that numerous virologists are working away with increasingly elaborate techniques, and more or less inventing the problems as they go along. Actually there is a bit of both. To get some idea of the prevalence of viruses we need initially to take a glance at the methods of detection that are available. First, evidence can be obtained from the traditional notification schemes: for example, that for Glasgow in 1963 shows emphasis on respiratory infections and some of the childhood exanthemata.

A similar analysis of infections in the Ruchill Infectious Disease Hospital, Glasgow in the same year again shows strong emphasis on pneumonia, then some of the rashes and a few cases recorded as polio, mumps and so on. In addition to these more traditional ways of recording infectious diseases, one must now depend also on laboratory data to get more aetiological detail. The WHO Virus Reports for 1964 show enteroviruses associated with neurological disease, numerous adenoviruses especially affecting the respiratory tract, influenza and para-influenza, and then herpes simplex, mumps in its proper relationship to the neurological disease, and respiratory syncytial virus appearing seventh in the list. Of course this is a biased sample; these are the kinds of things that laboratories were able to pick up at the time, but at least we are getting a little more detail.

Respiratory viruses pathogenic to man include the myxovirus group–influenza A B and C, the para-influenza viruses causing croup and various, usually minor, respiratory illnesses, and respiratory syncytal virus affecting infants. There are also

many types of adenoviruses, the common cold rhinoviruses, and the enteroviruses, the latter group containing polio, Coxsackie and Echo viruses, which sometimes cause acute respiratory illness. Coronavirus is being introduced as the name for one potentially important group of new viruses which can be cultivated in organ cultures but not normally in ordinary tissue cultures. And at the bottom of the list are organisms that are not true viruses; the psittacosis group, Q fever and mycoplasma.

Our own data over a number of years recorded age, number of cases studied, and percentage virus isolation rate. Respiratory syncytial virus showed the expected strong bias in the early childhood age groups, while influenza had a marked impact on ambulant children of school age and upwards into adult life. The rhinoviruses were far and away the commonest group associated with acute respiratory illness in adults. Para-influenza and adenoviruses were less tidily distributed while the enterovirus infections were mostly in young children.

The most important causes of epidemics are influenza and respiratory syncytial virus, the peak of children's pneumonia usually coming a month or so before that of the adults. It was not until techniques to diagnose respiratory syncytial virus became available that we were able to show that outbreaks of respiratory infection among children were caused by this organism, whereas the adult peaks coincided with outbreaks of influenza.

The story of influenza can be traced in terms of virus types since the appearance in 1957 of the A2 (Asian) strain which caused first a pandemic and then a second wave in winter. So many people acquired immunity that there were few cases in subsequent years until immunity had declined. Small outbreaks due to type B influenza have occurred from time to time while we had a most unusual winter in 1966–67 with no evidence of influenza at all. Influenza A2 came back in 1967–68, but this was not a particularly large outbreak, though it did produce the usual peak in pneumonia notifications, deaths and claims for sickness benefit. These are all reliable measures of the impact of influenza.

The pattern of rhinovirus isolations over the period 1962–66 was investigated, differentiating the various serological types, and showed a confused pattern with several types active at the same time. This illustrates the difficulty of preventing rhinovirus common colds by specific vaccines. With so many possible pathogens we should need chemotherapy or chemoprophylaxis; I do not see immunization coping with this sort of problem.

The incidence of polio in England and Wales from 1916 to 1962 increased until the introduction of vaccines brought a dramatic decline. But the really interesting and challenging question is, why did this original increase take place? The answer has to do with changing environmental conditions. Some years ago, shortly before polio-vaccination was introduced, we carried out antibody studies on children of various ages from central and west Scotland and many from Dundee. Age groups ranged from under 2 to 11 plus. Some children had no antibodies to any of the three types of polio, while at the other extreme a few had antibodies to all three, giving complete immunity. Most were intermediate, but those in social grades III to V acquired antibody rapidly from natural exposure to infection. Cases of polio are obviously more likely to be found in the age groups where antibody conversion takes place, that is the under fives, and this turned out to be true in practice. Under poor hygienic conditions with a more rapid circulation of viruses true *infantile* paralysis occurs, though many of the infections will be under the umbrella of maternal passive immunity, and most of them clinically hard or impossible to detect.

In social grades I and II, the children were more protected from infection in infancy, so that susceptibility extended right up into adult ages, and few had a full set of anti-

bodies. Under these circumstances a population of susceptibles big enough to support an epidemic can build up. So we have a paradox: The lower the prevalence of virus, the bigger the number of susceptibles in the population, and the greater the tendency to epidemics. We have to try to make up for this by immunization. The same is true of developing countries which are lowering their infant mortality and improving conditions. Their polio figures are now going up, and outbreaks of paralytic polio have been reported in conurbations in tropical Africa. The enteric viruses, those that spread in a polio-like way, include the enteroviruses (polio, the Coxsackies and the Echos), adenoviruses reoviruses and the hepatitis virus. I see no reason why the altered pattern of polio in relation to changing conditions should be unique to this infection. We are aware of epidemic emergence of various other viruses, for example, aseptic meningitis: in 1968 we have been finding Echo 6 and Echo 19 on both sides of the country, sometimes associated with rash, as with the Echo 9 virus in 1964. Bornholm disease due to Coxsackie B5 virus caused a European epidemic in 1965, and one sometimes runs into other manifestations—five per cent of the Coxsackie B5 infections recognized in this country in 1965 had evidence of cardiac disease (myocarditis or pericarditis). Hepatitis, like polio, may be showing some response to changing circumstances.

Cyclic recurrence of certain enteroviruses tends to follow a four-yearly pattern, rather less neat with Coxsackie B5. Polio type 1 used to do the same thing, but the pattern has now been spoiled by vaccination. But the typical pattern is of a virus which infects and immunizes a lot of people, disappears for some years, and then reappears.

The weekly report 'Communicable Diseases, Scotland' brings together data from all the virus and other microbiological laboratories in the country. In 1967 up to about half way through 1968, the different viruses isolated in Scotland numbered 547. The majority of polioviruses were isolated from children who had received oral vaccine shortly before, and we tried to follow up every one of these isolations especially if it did not follow vaccination. We found four cases of neurological disease in the latter category. One was a type 1 infection in a child who had flown to Glasgow from Pakistan during the incubation period. There were type 2 infections in three adults, two with aseptic meningitis and one with paralytic disease. This is a reminder that the poliovirus is still around.

I want to close with a brief mention of another example of changing ecology. German measles is after all the mildest and most trivial of fevers in children, but we have now reached the stage where something like 10–20 per cent of adult females have no antibodies and are therefore susceptible. This means that it is possible to have infection occurring in pregnancy, and reaching the foetus. It may or may not damage the foetus, but the child can be born still infectious and excreting large quantities of rubella from the respiratory tract, urine and faeces, for weeks or months. Faced with an ecologically difficult situation the virus seems to have come up with an ingenious method of bridging the generation gap. The young child is infectious to its (female) attendants, the nurses in hospital and so on, some of whom may be pregnant and at risk of foetal damage. This, I think, is an example of the new kind of problem which many viruses are likely to set us as we attempt to deal with the problems of the past. We cannot escape from the world of biology. We still have to live with it.