VIRUSES FROM SPACE
AND RELATED MATTERS

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Preface

The first chapter of this book is an up-to-the-moment discussion of the concept that viruses from space are the primary cause of so-called infectious diseases. Often, after lecturing on this subject, we are asked the question: Where can we read about all this? General accounts were given some years ago by F. Hoyle and N.C. Wickramasinghe, in the books *Diseases from Space* (J.M. Dent, 1979) and *Space Travellers* (University College Cardiff Press, 1981). These accounts were based on more technical publications which are not now easily available. The opportunity has therefore been taken in the present volume to reprint a selection of these more technical publications, written by F. Hoyle and N.C. Wickramasinghe, unless otherwise stated.

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Introduction

It is generally thought that acute upper respiratory tract infections are caused by the intake of viral particles that were previously exuded by some other person, or in rare cases by some other animal\textsuperscript{1,2}. Yet little or no evidence capable of standing up to critical analysis has ever been presented in support of this widespread opinion, which appears to have arisen through historical accident rather than through accurate observation and experiment. Following Pasteur’s classic experiments on alcoholic fermentation and silkworm diseases it became established that some human diseases arise from the transmission of bacteria from person-to-person, and since in the later decades of the 19th century there was no appreciation of the difference between viral and bacterial diseases the concept of infection by person-to-person transmission became applied to all diseases, a point of view that appeared to gain support when in 1892 Pfeiffer\textsuperscript{3} mistakenly implicated the bacillus *H. Influenzae* as the causative agent of influenza. A few epidemiologists, notably Charles Creighton in Britain, continued to protest that the evidence contradicted the rising tide of medical opinion, but in an age when few students had the leisure, affluence and inclination to examine the facts for themselves, the 19th century belief became set rigid in the educational system.

Once a false belief becomes established it is very difficult to get it out, essentially because the system invents supposed facts in order to support it. Two of the present authors\textsuperscript{4} had an experience of this process in action following the peculiar epidemic outbreaks of influenza A in the winter of 1977-78, peculiar because of the return of influenza subtype H1N1 with a variant dating apparently from the year 1950. It is commonly
stated that the person-to-person transmissibility of influenza is proved by very high attack rates in institutions such as barracks and boarding schools. Yet a survey of boarding schools in 1977–78 involving a total of more than twenty thousand pupils with a number of victims estimated to be some 8880 for an average attack rate of about 30%, yielded the distribution of attack rates shown in the histogram of Fig.1. In fact, only three schools out of more than a hundred at the extreme upper end of an approximately exponential distribution had the very high attack rates which have been claimed to be the norm.

Figure 1.
Histogram showing distribution of influenza attack rate among independent schools in England and Wales during the 1977–78 pandemic.
All the diagnoses involved in these data were made by school medical staffs in advance of our enquiries. Possibly other respiratory infections became associated with influenza in the diagnoses, but since January and February 1978 were months of influenza epidemics, and since children of school age had no established immunity against influenza H1N1, the bulk of the reported cases were very likely of this disease. And even if, in the absence of isolates or serological tests, one were sceptical of explicit diagnoses, the cases were certainly of acute upper respiratory infection, to which just the same remarks and conclusions would apply regardless of the explicit viruses involved. The schools in question were fee-paying, all with boarders sleeping together in dormitories. The degrees of association of pupils in dormitories, classes and at meal times were not much different from one school to another, and if the virus or viruses responsible for the 8880 cases were passed from pupil to pupil, much more uniformity of behaviour would have been expected. Already in Fig. 1 we have evidence of great diversity, with a hint that the attack rate experienced by an individual school depended on where it was located, with some schools being in fortunate places and some in unfortunate places.

The alternative to the person-to-person transmission of a virus is that it falls from the air. For semantic convenience we refer to falling from the air as vertical incidence and to person-to-person transmission as horizontal transmission. Although in this article we are concerned to argue the case for vertical incidence as the cause of most acute upper respiratory infections, it is to be emphasised that we are not making this claim for all viral diseases. While we think that all viral diseases arise in the first place by vertical incidence, it is possible for a virus to establish a reservoir in the human population such that the chance $p$ of contracting the associated disease by human contact is greater than the chance $q$ of contracting the disease through vertical incidence. Normalising so that $p + q = 1$, there are the possibilities $p >> q$, $p \approx \frac{1}{2}$; $p \approx q \approx \frac{1}{2}$; $p << q$, $q \approx 1$. Diseases in the first of these categories are truly infectious and can be moderated greatly through the old-
fashioned method of isolating victims. Indeed one could say that it is just those diseases, as for example smallpox, which the medical profession found to be successfully treated by isolation, that constitute the truly infectious category $p \gg q$, $p \approx 1$. In this article we are concerned with the opposite more numerous category, $p \ll q$, $p \approx 1$, which includes most acute upper respiratory tract illnesses. Data for measles, the discussion of which goes beyond the scope of this article, suggests that measles belongs to the intermediate category $p \approx q \approx \frac{1}{4}$. We suspect it is the intermediate nature of measles which explains why the medical profession is divided in its opinions on whether the isolation of victims would or would not effectively stamp out the disease. If our assignment of measles is correct, isolation would appreciably reduce the number of cases but would not stamp out the disease. To stamp out a disease $q$ must be strictly zero, requiring that the input of a virus to the Earth's upper atmosphere shall have ceased.

As regards the input of viruses to the Earth's atmosphere, the particles responsible for the strong ultraviolet component of the zodiacal light must have radii of order 30 nm, the scale of viruses. The density of such particles necessary to explain the observed strength of the zodiacal ultraviolet is remarkably high, implying an addition $\sim 10^4$ tons per year to the Earth's atmosphere, a total $\sim 10^{26}$ particles added annually. This number may be compared with an epidemic of disease in which each of $\sim 10^9$ humans sheds $\sim 10^{11}$ viral particles, for a total shedding of $\sim 10^{20}$ particles. If only a small proportion of the small zodiacal particles are viruses, if only a small proportion maintain viability, and if only a small proportion interact pathogenically with terrestrial plants and animals, the incident number would nevertheless be so vast that it could dominate horizontal transmission, even under extreme epidemic conditions.

It is commonly assumed that viral diseases are caused by the input to a victim of particles that are substantially identical to the output of viruses from the victim. This assumption is normally made for horizontal transmission, but it is not necessary for vertical incidence. All our data and all our
arguments require a causative agent or trigger to fall from the air, but the resulting disease could be caused by the association of the causative agent with dormant viral particles present already in the victim. Or the whole virus could be involved as with horizontal transmission. The evidence we shall present does not distinguish these possibilities.

Bacterial diseases can also be thought of in terms of the categories $p > q$, $p \approx 1$; $p \approx q \approx \frac{1}{2}$; $p < q \approx 1$, but with the last category less common than for viral diseases, i.e. dominant vertical incidence being less common. One bacterial disease that is difficult to explain except by vertical incidence, however, is whooping cough. Pertussis has for long been known to occur in cycles of about 3.5 years, which used to be explained on the density of susceptibles theory, namely that after children susceptible to the disease become exhausted by a particular epidemic it was then supposed to take about three and a half years for new births to rebuild the density of susceptibles to the level at which a further epidemic would run. Thus the periodicity of this theory should have been a function of population density, with the shorter periods being found in inner city areas of very high density and either long periods or no periodicity at all in lightly populated country areas. But the periodicity was found to be everywhere the same, in town and country alike, and from one country to another. Figure 2 shows the record of notifications for England and Wales over the period 1940-82. If the theory had been correct, the sudden reduction in the density of susceptibles brought about in the 1950s by the introduction of an effective vaccine should have greatly disturbed the periodicity, or even destroyed it altogether. Yet the periodicity persisted exactly as before, but with the total number of cases much reduced.
Figure 2.
General Evidence Against the Horizontal Transmission of Certain Diseases

If infectious diseases were propagated from person-to-person according to the commonly-held view then people living in high-density city areas should be significantly more subject to disease than people living in lightly-populated areas. From normalised attack rates plotted as a function of population density it would be possible therefore to prove the correctness or otherwise of this point of view. The circumstance that such data do not appear to exist, despite the cogency they would have, is interesting psychologically. Whereas people are avid to collect the slightest scraps of information that support conformist opinion, they are unremitting in their determination not to collect, or even to notice when collected, data which prove the opposite. It really needs no more than the absence of this simple but critical information to see that the commonly-held view must be wrong.

One can say in general terms that if any major discontinuity existed between town and country the population at large would easily be aware of it. Also in general terms, one of the present authors has shifted on occasions from general practice among a major city population to standing locum in highly rural areas, without any difference in morbidity patterns being qualitatively apparent. Thousands of general practitioners must have experienced similar comparisons without any discontinuity of pattern being emphasised and reported. On a more quantitative level, Figure 3 shows data collected by Dr. P. Jenkins, the Community Health Officer for the City of Cardiff. It gives data covering the three diseases of so-called infective jaundice, whooping cough and measles, obtained quarterly from the heavily-populated Cardiff city area (after normalising to 100,000 population) and from the Vale of Glamorgan, much of which is very rural. Thus each disease in each quarter of a period of three years yields a point in Fig. 3. This is except for measles which was so prevalent in one particular quarter that the corresponding point for that quarter could not be plotted without prejudicing the scale of
Figure 3.
Quarterly incidence of whooping cough, measles and infective jaundice in the City of Cardiff vs The Vale of Glamorgan.

the figure. The one missing point lies on the line defined by the other eleven points, but far away to the right of the figure. Such bias as one can see in Fig. 3 goes the wrong way for horizontal transmission. It is the lightly populated Vale of Glamorgan that on a normalised basis appears worse affected. Of course one can always invent the hypothesis that standards of reporting are higher in country practices than in the cities, but one of us as a general practitioner in a city area would naturally dispute this suggestion. At all events, general experience, together with the data of Fig. 3, suggests that there is no marked difference between town and country, as one would
expect for vertical incidence but not as one would expect for horizontal transmission.

Ockham's razor warns us against inventing a 'multiplicity of hypotheses', a warning which some have seen fit to interpret as an edict proscribing the consideration of new ideas. What the warning really means is that we should be on our guard against the invention of a multiplicity of unsubstantiated hypotheses in order to defend conformist views against awkward facts. For example, it is in our opinion an *ad hoc* hypothesis to suppose that city populations possess greater immunity against infectious diseases than rural populations, and to such an extent that the greater exposure which city populations experience with respect to person-to-person transmission is almost precisely compensated by their greater immunity. A similarly *ad hoc* hypothesis would also be required to explain why individuals whose occupations involve exceptional hazards with respect to person-to-person transmission, for instance dentists and cashiers in banks, newsagents and large stores, nevertheless have records of upper respiratory infections that are not noticeably abnormal.

Looking over case notes dating from 1970 in a general practice, one of us (J.W.) identified 16 pairs of twins with ages between 6 months and 14 years. Of the 118 instances in which one twin was consulted for acute upper respiratory infection the corresponding twin succumbed to a similar infection in 28 instances. Since twins in the age range in question are found almost perpetually together, the opportunity for person-to-person transmission would be maximal in these twin-to-twin relationships. Yet in only 24 percent of instances did the second twin become a victim, which is not an impressive fraction, particularly as attack rates during epidemics of upper respiratory infections tend to run typically at about the 25 percent level among the general population. An epidemic will not run according to horizontal transmission unless each victim infects at least one other victim, thereby establishing a supercritical chain relation. A transmission probability as low as 0.24, which this data for twins yields on the horizontal transmission hypothesis, would therefore be quite insufficient to establish a
Figure 4.
Percentage attack rates in households where one member succumbs to influenza in the epidemics of 1968/69 and 1969/70 at Cirencester, England according to data from Hope-Simpson.
supercritical chain. Only if a stricken twin contrived to infect others much more readily than his or her own twin could adequate transmission be attained.

Following in the steps of Charles Creighton, Edgar Hope-Simpson was the first person in recent years to bring the hypothesis of person-to-person transmission rigorously under the hammer. Hope-Simpson had the idea of defining a set of households by the condition that one member succumbs initially to Influenza A. He then observed the subsequent fates of other members of the households thus defined, finding them to develop no greater proportion of attacks than would be expected for the population at large. Figure 4 gives Hope-Simpson’s results for epidemics of H3N2 in 1968/69 and 1969/70, shown in the histograms as I and II respectively. Besides the fraction of subsequent cases being normal for the population at large, no well-defined subsidiary peak occurred 2 days after the first cases were reported, as would be expected from incubation if horizontal transmission had been occurring. Hope-Simpson’s results have been fully confirmed by Mann et al., and by Philip et al.

The unusual circumstances in 1984-85 that few true cases of Influenza A were reported anywhere in the world up to mid-February 1985 (INFLU Centre, London) permitted the behaviour of other sources of upper respiratory infections to be examined in a manner similar to that used by Hope-Simpson. Starting in May 1984, a total of 80 households were defined, again by the criterion that one member presented themselves with an acute upper respiratory infection, and then subsequent histories of other household members were studied by one of us (J.W.), with the results shown in Fig. 5. The interesting point emerges that upper respiratory infections quite generally are like influenza, without evidence of person-to-person transmission, which if it had occurred would have caused an incubation peak of cases to occur two or three days after the initial attacks on day zero.

A proportion of the fee-paying schools in the survey already mentioned had both day pupils and boarders. The boarders were exposed to close person-to-person contacts for 24 hours a
Figure 5.
Percentage cases of acute upper respiratory infections in households where one member succumbs to influenza on day 0, from data collected by J.W. in 1984/85.

day, whereas the day pupils were only some 8 hours at school, with the remaining 16 hours spent under non-institutional conditions, conditions having fewer person-to-person contacts generally. If there were any substance to the claim of high attack rates in institutions, used to bolster the person-to-person transmission hypothesis, the overall attack rates on boarders should have been significantly higher than it was on the day pupils. With each of the schools in question represented by a point in Fig. 6, the results gave essentially a scatter diagram. Whatever slight bias there is about the 45° line in this
diagram disappears for a line of slope 40°, and this is within the expected statistical fluctuation. There are many instances in which the day pupils experienced considerably higher attack rates than boarders, a situation that defies the imagination to explain according to person-to-person transmission, for we would have to suppose that after leaving school the day pupils encountered more seriously infective contacts than were present at school, and that they did so systematically in order to explain high attack rates above 70 percent for the day pupils in some of the cases.

Figure 6.
Correlation of attack rates for Day pupils and Boarders for schools which had a mixture of both during the 1977/78 influenza pandemic in England and Wales.
In the next section, we shall see that vertical incidence is expected to lead to intricate patchy details in attack rates, with some localities relatively safe from attack and others relatively dangerous. On this view, schools that happened to be in relatively safe areas would have their boarders staying comparatively safe the whole time, whereas the day pupils would go out from the school into comparatively dangerous areas and so would experience significantly higher attack rates. And of course the opposite situation would occur for other schools, thereby producing the scatter shown in Fig. 6.

Attack rates of around 30 percent were found most useful for studying variations within school boundaries, since very high attack rates evidently preclude variations being found, while low attack rates gave inadequate statistical weight. Eton College had 441 victims among 1248 pupils, for an attack rate of 35 percent, with high statistical weight because of the large number of pupils. We were fortunate that Dr. J. Briscoe, the Medical Officer at Eton, had for long been puzzled to understand how his observations could be explained in terms of pupil-to-pupil transmission. Consequently, Dr. Briscoe had collected comprehensive information giving the distribution of victims in some 25 school houses. The houses averaged about 50 pupils each, with about 17 cases expected as the mean number of victims. Such numbers were very suitable for computing standards deviations, with the results shown in Fig. 7. Two houses had excess morbidities of 4 standard deviations, two had deficits of about 4 standard deviations, while one house (COLL) had a remarkable deficit of 6 standard deviations. Since pupils in the different houses were mixed in classes and at games these enormous fluctuations from a random distribution are quite inexplicable it seems to us in terms of horizontal transmission. The Eton College results imply that the school was hit vertically by the influenza virus during the night hours, or possibly at a weekend, and that the vertical incidence was patchy enough to distinguish between the locations of the various houses, some houses happening to lie in safe areas and others in dangerous areas. Dr. Briscoe informed us that similar effects had occurred in other influenza
Figure 7.
Deviations of attack rates of influenza above the mean attack rate for the 25 school houses at Eton College during the 1978/79 pandemic. The deviations are relative to the standard deviation computed house by house.
epidemics, with the identities of the lucky and unlucky houses being different from the situation in 1978. A patchy vertical incidence would of course not be reproducible in its details from one epidemic to another, so this too would accord with the vertical incidence hypothesis.

We end this section with a somewhat different issue. Younger children have sometimes been found to be more susceptible to influenza than older children. Usually it is not possible to distinguish how far the greater resistance of older children is inherent and how far due to already established immunities. Since no children of school age in 1978 had any established immunity to the H1N1 subtype, and since some schools in our survey had both junior and senior pupils, it was possible to compare attack rates that gave information largely free of the immunity factor. Results are shown in Fig. 8 where each point refers to a school having both junior and senior pupils. These data suggest that inherent resistance has little to do with age, implying that differences observed in other years were related to the immunity factor. Such asymmetry as one sees in Fig. 8 about the 45° line (after noting the two very low points marked heavily to catch the eye) would be removed by increasing the 45° slope to a slope of 50°. A slight bias in this sense could have arisen from a minority of cases where the virus subtype was still H3N2 rather than H1N1, with older children having better immunity to H3N2.

_The Vertical Incidence Theory_

According to medieval lore, diseases come from comets, and according to our view this is true, but only in a broad sense. We cannot maintain the dramatic position that ferocious new diseases come from spectacular comets, because for every spectacular comet there are almost certainly very many smaller ones. The smaller comets may not only evaporate more material collectively than large ones but the effect of the Earth crossing almost precisely the track of a small comet would lead to a greater addition of evaporated particles to the terrestrial atmosphere than would a more distant relationship to a large comet, as for instance a distant relationship to Halley's comet.
Support for this position comes from an analysis by Z. Sekanina\textsuperscript{10} of some 20,000 orbits of meteors, meteors being small particles typically with sizes $\sim 0.1$ mm also evaporated from comets. A minority of the orbits could be associated with known comets but the majority could not. It is perhaps possible to understand both the origin and the demise of the medieval lore in these terms. Over a time scale of historic length there must have been special situations with the Earth in close proximity to a large comet. If following such special situations