Influenza A of Human, Swine, Equine and Avian Origin: Comparison of Survival in Aerosol Form

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SUMMARY

Strains of Influenza A representative of human, avian, swine and equine sources were examined for decay when in aerosol form. Strains having their origin in avian and equine hosts were considerably more resistant to decay. Strains derived from human and swine sources were less resistant and bore a similarity in this property.

RÉSUMÉ

Le taux de dégénérescence de souches d'influenza A sous forme d'aérosol, d'origine humaine, aviaire, porcine et équine fut étudié. On a constaté une résistance à la dégénérescence beaucoup plus grande chez les souches d'origine aviaire et équine. La résistance à la dégénérescence est moins grande chez les souches d'origine humaine et porcine quoique similaire chez ces dernières.

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INTRODUCTION

In a paper published previously (1) attention was drawn to the decay of influenza viruses (Type A) of human and avian origin when aerosolized in small particle size. It was shown that strains of avian origin decayed more slowly than those of human origin. This suggested that strains havings their origin in other species should be examined. Therefore, strains having their origin in swine and horses were obtained and compared with several strains of human and avian origin.

MATERIALS AND METHODS

The materials examined were samples of Type A influenza virus representative of strains of human, avian, swine and equine origin. Those of human origin were the historic W.S./33 strain, the F.M./47 and the more recent isolate Japan 170/62. The avian strains were composed of Duck (Canada)/52, Turkey/Eng/63, Tern/62 and Quail/65. The swine strains were the one originally propagated by Shope (the first influenza strain isolated from any species), one isolated in 1961 and a strain isolated in 1968. Six strains of equine origin were included. They were isolated in several geographic areas of Europe and America.

The method of examination described in

the former paper (1) was used and is briefly as follows. Virus contained in allantoic fluid is diluted 10^{-6} in Earles balanced salt solution. Into the allantoic sac of twelve chick embryos (ten day development) is inoculated 0.2 ml of the diluted fluid. The eggs are allowed to incubate for 48 hours, at which time the allantoic fluids are harvested. Each is tested by the hemagglutination test (HA) in a dilution of 1:20, using 0.25 ml of fowl erythrocytes. Those fluids giving well-marked reactions are chosen, pooled and a serial passage carried out. The allantoic fluid of those giving well-marked HA reactions are selected, pooled, bottled in 15 ml amounts and stored at -80°C. When about to be tested, they are removed to room temperature, allowed to melt and transferred to the Collison spray apparatus which is adjusted to deliver an aerosol — the majority of the particles being one to two microns when the apparatus is operated at 40 lbs pressure. The aerosol is forced into a Goldberg rotating drum having a humidity reduced by dry air washing to relative humidity 15 and the temperature maintained at 70°F. Samples are collected at three hour intervals by drawing under negative pressure for three minutes a sample of the aerosol into a Shipe impinger charged with ten ml of Earles solution.

Tenfold dilutions of the collecting fluid containing the virus is made in normal salt solution. Using a dose of 0.2 ml, the undiluted and diluted samples are inoculated into embryonated eggs. After incubating for 48 hours, the fluids are harvested and examined by the HA test. The end-point of decay is based on the last three hour time interval when live virus particles are present. The titer of the initial sample which serves as the base line is usually 10^{-4} or 10^{-5}; if lower, the run is discarded and the work repeated.

Using the above method, each strain was examined twice and excepting those of equine origin, no difficulty was found in reproducing results.

RESULTS

The results of these examinations are indicated in Fig. 1. It will be noted that there is some variation in the time of survival, when in aerosol form, of all strains except those of swine origin. What is particularly striking is that those of equine and avian origin are much more resistant to decay than the others examined. Strains of swine origin approximate those of human origin. In addition, every swine strain had precisely the same resistance to decay although many years separated the dates when they were isolated.

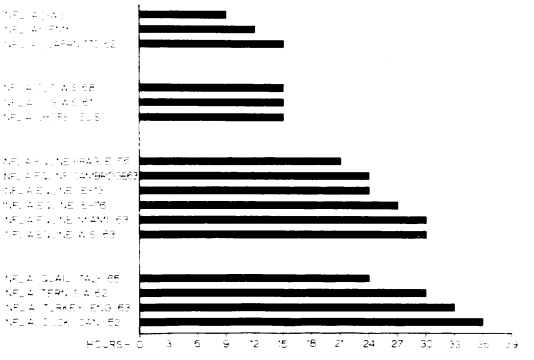

Fig. 1. Survival in aerosol form of influenza type A of human, swine, equine and avian origin.

DISCUSSION

The similar behaviour of swine and human strains in relationship to decay in aerosol form brings to mind the possible genesis of influenza in swine. This disease was unknown until approximately 1918 when it appeared in the mid-west of the United States as a respiratory disease of swine and was soon recognized as a separate disease entity apparently being different from other infections of this species. Dr. J. S. Keon (2), an Inspector of the Bureau of Animal Industry was so impressed with the clinical and epidemiological similarity of the porcine ailment with that of human influenza then prevalent that he considered these two conditions to be caused by the same agents. At the time, neither the cause of human nor swine influenza had been demonstrated although the cause of the former was thought to be Pfeiffer's or influenza bacillus (3,4). It is

curious how the future of each dove-tailed and led to an understanding of the cause of illness in man and swine. An investigation of swine influenza was undertaken by Shope, a scientist of great ability and imagination. A search was made for Pfeiffer's Bacillus which we now know as *Hemophilus influenza.* A similar organism was found in swine which has been named *Hemophilus influenza suis.* During the course of these experiments it was demonstrated that a virus was also present (5). Finally, it was shown that to produce a disease similar to that found in the field both agents were required.

About the same time, Smith, Andrews and Laidlaw were investigating influenza in England and, two years after Shope's discovery, they demonstrated that a virus was involved in influenza of man (6). Subsequently, it was found that these two viruses belong to the present Type "A" and have many properties in common. These two discoveries aroused increased interest in the genesis of swine influenza. While there is no concrete evidence that the human strain which swept through the world at the time of the appearance of the infection in swine is the parent of the causative agent of the disease in these creatures, it does seem a remarkable coincidence. It is also interesting that the decay of the human and swine strains should be similar.

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