ANTIBODIES TO INFLUENZA VIRUSES IN THE SERA OF NIGERIANS

By A. ISAACS

With the technical assistance of D. Busby and V. G. Law

National Institute for Medical Research, Mill Hill, London, N.W. 7

(With 4 Figures in the Text)

INTRODUCTION

Davenport, Hennessy & Francis (1953) studied the amount of antibody to different strains of influenza virus A in sera taken in Michigan, U.S.A. and a similar investigation was carried out in Sheffield (Davenport, Hennessy, Stuart-Harris & Francis, 1955). Pools of serum were prepared from people of different age-groups, and it was found that antibodies to a particular strain of virus were first detectable in sera corresponding in age to a time when that strain of virus is believed to have been prevalent. The deduction was made that the strain responsible for the initial infections of childhood had an effect on the antibody response to subsequent infections by antigenically different but related strains of influenza virus; and this deduction has received strong support from the results of serum absorption studies (Jensen, Davenport, Hennessy & Francis, 1956). This paper reports the results of a similar investigation with sera from Nigerians, in an attempt to see whether recent experience of infection with influenza A viruses in Nigeria could be inferred by this method.

MATERIALS AND METHODS

Sera

Sera were collected in Oji River Leper Settlement, near Enugu, Eastern Nigeria in July 1952 as part of a survey for yellow fever antibodies and were made available by courtesy of the Director of the West African Council for Medical Research Laboratories, Lagos, Nigeria.

Treatment of Sera

Sera were heated at 56° C. for 30 min. before the test. In some experiments sera were treated with a crude filtrate of *Vibrio cholerae* (Burnet & Stone, 1947) prepared by N. V. Phillips-Roxane, Amsterdam and containing receptor-destroying enzyme (RDE).

Strains of virus

Shope's swine 15 (1931), WS (1933), PR8 (1934), FMI (1947) and A/Eng/1/53 viruses were used.

Antihaemagglutinin test

The technique recommended by the World Health Organization Expert Committee on Influenza (1953) was adopted.

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RESULTS

A comparison was first made of sera which had been inactivated by heat with the same samples treated with *V. cholerae* filtrate. It was found that treatment with *V. cholerae* filtrate did not significantly affect the antihaemagglutinin titre against swine, WS, FMI and A/Eng/1/53 viruses; there was, however, a big reduction in the antihaemagglutinin titre to PR8 virus and it appears that our strain of PR8 is very sensitive to normal inhibitors of agglutination present in human serum. The results with PR8 virus have not, therefore, been included in Figs. 1–4.

Antihaemagglutinin titres to swine, WS, FMI and A/Eng/1/53 were plotted against the age of the individual and the results are shown in Figs. 1–4. No serum pools were tested.

The results are in broad agreement with those found previously in the U.S.A. and England, but examination of individual Nigerian sera has revealed an earlier appearance of antibody to swine and WS viruses in some persons. Thus, Davenport *et al.* (1955) found that antibody to swine virus was first detectable at the age of 28 in Michigan sera and at 32 years in Sheffield sera, whereas in the Nigerian sera many individuals aged 20 or less showed low titres of antihaemagglutinin to swine virus, which were not affected by treatment with *V. cholerae* extract. A similar trend was noted for antibody to WS virus. The difference between our findings and those of other workers is probably due to the use of individual sera instead of pools rather than to a geographical difference, since individual sera from England tested in the course of trials of influenza vaccine have shown a similar antibody pattern to that found in the Nigerian sera.

DISCUSSION

The results illustrated in Figs. 1–4 are in broad agreement with findings in the U.S.A. and Europe. Thus in the sera of Nigerians up to 20 years of age antibodies to swine virus were usually low in titre or undetectable. Similarly, low titres to the WS virus were commoner in young adults than in older people. Presumably, therefore, viruses antigenically related to swine and WS viruses were current in Nigeria at about the same time as they were present elsewhere in the world. Antibodies to FMI virus were evenly distributed in the different age-groups, suggesting fairly widespread recent infection with viruses antigenically similar to FMI. Antibodies to A/Eng./1/53 virus were uniformly low in East Nigeria in 1952, suggesting that strains of this kind were not yet widely disseminated there. These findings point to the conclusion that over the last 30 years, at least for the influenza viruses investigated, antigenically similar strains were present in the U.S.A., Europe and a rather isolated community in East Nigeria at about the same time.

There is one finding, however, which contrasts with the findings of Davenport *et al.* (1955): these workers tested serum pools and found that antibody to swine virus was first detectable at the age of 28 or 32 and rapidly became maximal in titre. In the present study, we tested individual sera and found a number of young adults aged 10–20 who had low titres of antihaemagglutinin to swine viruses. These titres were not affected by treatment with *V. cholerae* extract, and a similar finding

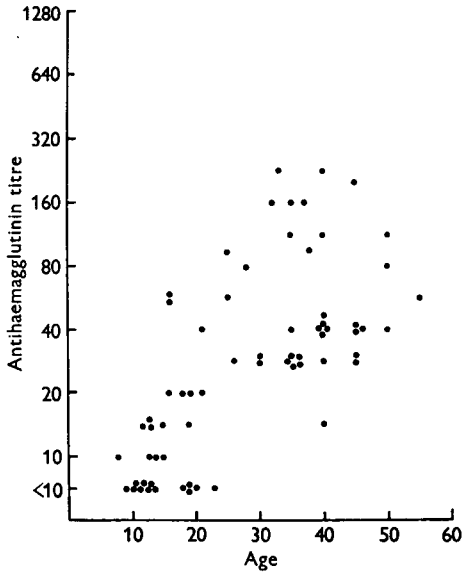

Fig. 1. Swine strain.

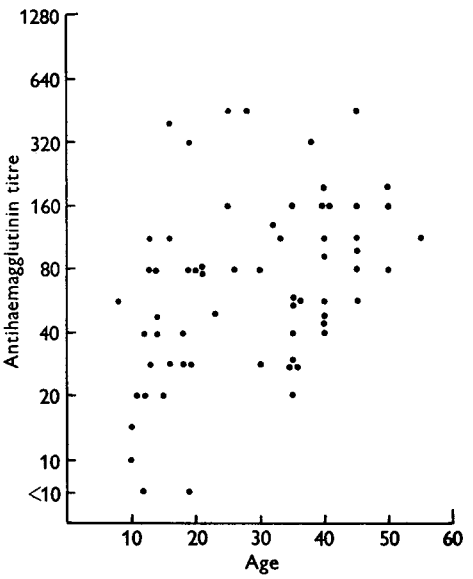

Fig. 2. WS strain.

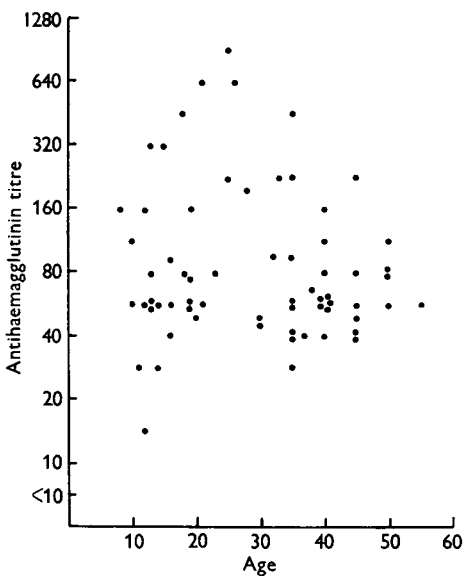

Fig. 3. FMI strain.

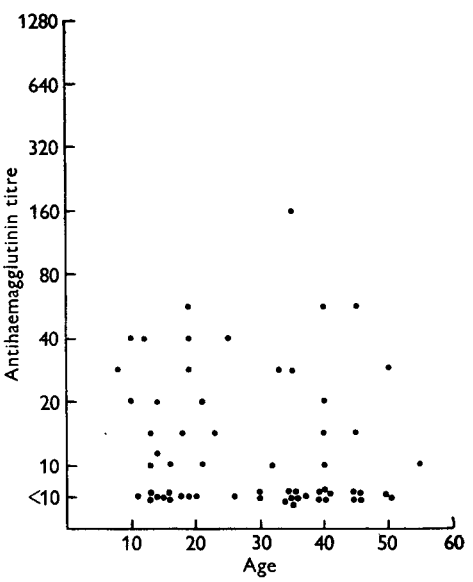

Fig. 4. A/Eng/1/53 strain.

Figs. 1–4. Antihaemagglutinin titres to four strains of influenza A virus in the sera of Nigerians of different ages. Each spot represents an individual serum titre.

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was made with sera from young adults in this country. The same trend was also noted in the Nigerian sera tested with WS virus. There are two possible explanations for these findings. First, we might assume that antibody to swine virus in individuals of 10–20 years of age is evidence that viruses of this antigenic character were present in East Nigeria and in this country until as short a time ago as 10–20 years, which is much more recent than has hitherto been assumed; a similar argument would apply to WS virus antibody in individuals of 10 years old, i.e. the antibody is accepted as evidence of past infection with these viruses. The second explanation is that these low titres of antibody are due to antibody to antigens of more recent viruses which share minor antigens with the swine virus. It is felt that the gradual increase in antibody titres to swine virus with age, shown in Fig. 1, strongly favours the second explanation, since if the first explanation were correct one would expect to find individuals aged 10–20 years showing either titres of antihaemagglutinin as high as those of adults over 30 years of age, or no detectable antibody. We conclude therefore that low titres of antibody to swine and WS viruses are not necessarily indicative of previous infection with these viruses and are more likely to be due to antibody to minor antigens of more recent strains.

SUMMARY

Sera of Nigerians aged between 10 and 60 years were tested individually for their antibody to five strains of influenza A virus. The results showed a similar pattern to those which other investigators have found with sera collected in the U.S.A. and in this country. However, antibody to the swine and WS strains was found in younger individuals than was reported previously; and the distribution of antibody titres suggests that this represents antibody to minor antigens of more recent strains, rather than persistence of swine and WS viruses.

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