is therefore suggested that the medical examination and short-term observation of poliomyelitis contacts might provide a more realistic picture of familial and contact incidence and give further information on the transmission channels which result in clinical poliomyelitis in a proportion of those infected with the virus.

It was noticed that in some of the contacts, whether family or otherwise, there was an apparent similarity of the type of infection or the location of paralysis. The numbers dealt with here are too small to justify deductions, but it is suggested that this is an aspect for further inquiry in future investigations of family and contact cases.

I acknowledge with thanks the co-operation of Dr. A. B. Stewart, Divisional Medical Officer, L.C.C., in providing inoculation histories of poliomyelitis cases and information on antigens used, and also physician-superintendents and other hospital medical staff for kindly sending clinical information on Islington cases admitted.

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IMMUNITY TO OLD AND NEW STRAINS OF INFLUENZA A VIRUS

BY

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In the early days of influenza serology it seemed to be established that there were several immunologically distinct strains of influenza A, so that antiserum produced by infection with one strain showed a higher antibody level to that strain than to a heterologous strain (Francis and Magill, 1935; Bodily and Eaton, 1942; Hirst, 1942).

Since then, however, the following facts have emerged: Friedewald (1943), using complement-fixation tests, found that higher titres were sometimes given by convalescent sera against the PR8 strain of influenza virus—isolated in Puerto Rico in 1934 (Brown, 1936)—than against the strain of virus isolated from the patient's throat washings. Stuart-Harris and Miller (1947) also found higher serum antibody titres against PR8 than against the A strain causing the epidemic of that time (they used haemagglutination-inhibition tests). Morgan, Barnes, and Finland (1948) found that in convalescent serum a higher antibody level was present against PR8 than against the strain causing the epidemic concerned. The tests used in this instance included haemagglutination-inhibition, complement-fixation, and egg-neutralization techniques.

The following results show a similar tendency in the case of haemagglutination-inhibition and complement-fixation tests carried out in 50 normal sera against PR8 and BAR (Barclay) strains of influenza.

The time of the investigation was the first five months of 1950. Sera were obtained from blood transfusion donors at the Royal Infirmary, Edinburgh, and were taken nine months after the 1949 (March) epidemic of influenza A. The technique of the haemagglutination-inhibition tests was that of Salk (1944), except that a final concentration of 0.25% red-cell suspension instead of 0.125%, and calcium saline as diluent, were used. The latter consisted of 0.024% CaCl_2 in 0.85% NaCl . For the complement-fixation tests Hoyle's (1948) method was employed, except that the antigen used was infected allantoic fluid, prepared and harvested by the method of Francis and Salk (1942), instead of infected mouse-lung suspension.

Results

Tables I (Fig. 1) and II (Fig. 2) show the titre levels involved and Table III the distribution of sera giving different ratios.

FIG. 1.—Haemagglutination-inhibition tests: distribution of sera of different titres.

FIG. 2.—Complement-fixation tests: distribution of sera of different titres.

TABLE I.—Haemagglutination-inhibition Tests: Percentage Distribution of Sera of Different Titres

Titre:	<8	8	16	32	64	128	256	512	1,024	Total No. of Sera
Virus PR8 ..	2	0	2	4	27	11	35	18	0	55
Virus BAR ..	10	14	45	22	8	0	0	0	0	49

TABLE II.—Complement-fixation Tests: Percentage Distribution of Sera of Different Titres

Titre:	<2	2	4	8	16	Total No. of Sera
Virus PR8 ..	20	26	31	17	6	54
Virus BAR ..	62	38	0	0	0	45

TABLE III.—*Distribution of Sera Giving Different*Ratios: $\frac{\text{PR8 Titre}}{\text{BAR Titre}}$

Ratio: $\frac{\text{PR8 Titre}}{\text{BAR Titre}}$	1 or less	1	2	4	8	16	32	Over	Total No. Sera
Percentage of sera (using haemagglutination-inhibition tests) ..	2	2	11	19	23	23	8	1	47
Percentage of sera (using complement-fixation tests) ..	6	23	34	29	4	4	0	0	46

Note.—Titres are expressed as the reciprocal of the final dilution at the end-point. These figures are obtained using the assumption of a titre of 1 for any titre less than 2.

The average PR8 titre is 215 (H.I.T.) and 4.3 (C.F.T.), and the average BAR titre 20.0 (H.I.T.) and 1.4 (C.F.T.).

The ratio $\frac{\text{average PR8 titre}}{\text{average BAR titre}} = 10$, for haemagglutination-inhibition tests, and 4, for complement-fixation tests.

It is unlikely that these high PR8 antibody levels represent the result of contact with this virus at any period of more than a year or two ago, since it is known that, following the initial response to infection, specific antibody levels fall steadily over a period of one to two years by from 5 to 100 times in the absence of subsequent infection (cf. Stuart Harris *et al.*, 1938). It is possible that high PR8 titres might be maintained by the persistence of that strain in a form in which it cannot be isolated by present methods. Current strains of virus such as BAR may then represent variants of this latent PR8 form, which are capable of causing epidemics, capable of growing in eggs or infecting ferrets or mice, and therefore capable of being isolated.

Finally, and most probably, the present findings may be taken as falling into line with the work quoted earlier, especially that of Stuart-Harris and Miller (1947) and Morgan *et al.* (1948), leading to the conclusion that the PR8 antibody levels represent a non-specific response to infection or subinfection by a current virus strain, of which BAR is an example.

Summary

Haemagglutination-inhibition and complement-fixation tests were carried out on 50 normal serum samples, using influenza A strain PR8 (classical) and BAR (recently isolated), and the titres against these strains compared.

It was found that PR8 titres were on the average 10 times higher than titres against BAR virus by the haemagglutination-inhibition method, and four times higher by complement-fixation.

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PRESENCE OF CERTAIN SEROLOGICAL TYPES OF BACT. COLI IN THE HUMAN INTESTINE

BY

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Stevenson (1950) demonstrated the presence of *Bact. coli* D433 in the faeces of adults suffering from diarrhoea, but suggested that these organisms might be the normal inhabitants of the duodenum or jejunum and be carried down by increased intestinal activity due to other causes. This suggestion clearly required further study. Accordingly, during the period July 21 to October 30, 1950, swabs were taken from various parts of the alimentary tract of 51 cases (50 adults and a child of 7 months) examined *post mortem*, none of which had recently had diarrhoea or any treatment likely to interfere with the natural intestinal flora. These swabs were examined for the presence of *Bact. coli* of serological types D433, Aberdeen β , E893, E990, Vincent, and E611. Each swab was plated on half a Petri dish containing McConkey's agar medium and incubated aerobically at 37° C. for 18 to 24 hours. A comprehensive sample of the resulting growth was taken by sweeping a platinum loop across the surface of the medium, and this was first tested with a 1 in 500 solution of acriflavine in physiological saline.

Joan Taylor (personal communication) has found that some strains of *Bact. coli* other than D433 may be agglutinated on a slide by the specific sera prepared against *Bact. coli* D433, but most of these are also agglutinated by the acriflavine solution. On the other hand, none of the many strains so far examined that belong to the serological types which were the subject of this inquiry have ever been found to agglutinate in acriflavine. The acriflavine test is therefore very useful for preliminary "screening."

If no agglutination resulted in the acriflavine solution 12 individual colonies were tested with D433 K, Aberdeen β K, and a composite serum containing agglutinins to strains E893, E990, Vincent, and E611. If agglutination occurred in the acriflavine solution some four to six individual colonies were tested with the specific sera and a further sweep was subcultured on to plain nutrient agar. Those colonies which agglutinated in any of the specific sera were also subcultured to find if they should become acriflavine-negative. This routine was repeated on the following day, when only "acriflavine-negative" colonies were tested with the specific sera, at least 12 such colonies from each agar plate being tested before the swab was pronounced "negative." The slide-agglutination technique was used throughout.

The results are summarized in the Table.

The following is a list of the conditions from which the patients died: coronary thrombosis (6), carcinoma of the lung (5), congestive cardiac failure following hypertension (5), bronchopneumonia (5), haematemesis (3), carcinoma of stomach (3), senile myocardial degeneration (2), chronic nephritis (2), carcinoma of oesophagus (2); and one from each of the following: pyonephrosis, renal failure, carcinoma of breast, carcinoma