SUMMARY

Fresh liver was found to be a more potent source of the monkey anti-anemia factor than whole liver powder. Beef and pork livers had equal potency. Lyophilized liver retained all the active principle of fresh liver.

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DEMONSTRATION OF INFLUENZA VIRUS, TYPE B, IN A RECENT OUTBREAK OF UPPER RESPIRATORY INFECTION[1]

THERE are only a few published reports on the isolation of Type B influenza virus in this country.[2,3,4,5,6] We have recently (May, 1945) isolated a strain of influenza B virus from an outbreak of upper respiratory infection at Camp Atterbury, Indiana.

The outbreak was rather mild and well isolated in its case distribution within an area of the camp. The virus was detected in the second egg passage carried out by the method described by Hirst.[7] In making the passage the tracheas of injected embryos were ground and suspended in pooled allantoic fluid from the same eggs. The identity of the virus was established by means of the red blood cell agglutination-inhibition test,[8] using sera from chickens immunized against the Type A (PR8) and Type B (Lee) viruses.[9] The new strain was named "Saha." Table 1

TABLE 1

ANTIGENIC STUDY ON SAHA VIRUS RBC AGGLUTINATION-INHIBITION TESTS

Virus	Sera produced against:		
	Lee	Saha	PR8
Lee	800*	320	< 400†
Saha	160	1,280	< 400
PR8		<80	12,800

* Expressed as the reciprocal of the agglutination-inhibition titer.
† Titrated by Salk's method.

is representative of a number of tests on the antigenic nature of the Saha virus. It is apparent that the virus in question is antigenically related to but not identical with the Lee virus, and it has no common antigen with the Type A (PR8) virus.

To date the virus has been passaged nine times in chicken embryos by the allantoic route of inoculation. In line with the experience of other workers who have studied recently isolated strains of influenza B, we have found it somewhat difficult to maintain this virus in a limited series of mouse lung passages.

A study of acute and convalescent sera of patients was made by means of the red blood cell agglutination-inhibition test. All sera were examined by the Hirst method,[8] and some were also tested by the method described by Salk.[10] The paired serum specimens were received in three groups submitted at successive intervals after the onset of the epidemic. Although some paired specimens had relatively high titers against the PR8 virus, none showed an increase in antibody titer in comparative tests on acute and convalescent sera. In group I of 23 paired specimens, 12 (52 per cent.) had a significant increase in titer of four-fold or higher against the Lee virus and 17 (74 per cent.) showed a similar increase in titer against the Saha virus. On comparing the titers in group II of nine paired serum specimens, 8 (89 per cent.) had a significant increase in antibodies against both the Lee and Saha strains of virus. In group III of 15 paired specimens, 14 (93 per cent.) again demonstrated a significant rise in antibody titer against the two strains. These serological results furnish additional evidence that the outbreak was due to a Type B virus.

This localized epidemic in the late spring of this year (1945) may be of significance, especially in view of the reported slight increase in influenza in this country (*Public Health Reports*), which began in the week ending May 12th, compared with the incidence in the same week in 1944 and the median for 1940–1944. Although the outbreak referred to herein may remain an isolated episode, the possibility exists that it may represent the beginning of an epidemic wave of Type B etiology. The situation may parallel the experience of 1943, when an epidemic of influenza Type A was preceded by a localized outbreak of influenza in an Army camp, from which Salk, Menke and Francis[11] isolated a Type A virus.

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