Administrative Agencies: Recommendations of the Attorney-General's Committee. John F. Dulles, of the New York Bar; Walter Gellhorn, Columbia University; John Dickinson, University of Pennsylvania. Wilbert G. Katz, *chairman.*

Frontiers of Knowledge in the Geologic Sciences. Richard F. Flint, Yale University; A. I. Levorsen, American Association of Petroleum Geologists.

Cosmic Rays. Robert A. Millikan, California Institute of Technology; Bruno Rossi, Cornell University; William P. Jesse, Marcel Schein, Subrahmanyan Chandrasekhar and Ernest O. Wollan, University of Chicago.

The Place of Ethics in Social Science. Richard H. Tawney, University of London (tentative); Charles H. McIlwain, Harvard University; Jacques Maritain, Catholic Institute of Paris, Columbia University; Robert M. Hutchins, University of Chicago. John U. Nef, *chairman.*

In addition to the symposia, there will be the following lectures:

September 22. The Social Implications of Vitamins. Robert R. Williams, Bell Telephone Laboratories.

September 23. The Physiology of the Amino Acids. Donald D. Van Slyke, Rockefeller Institute for Medical Research.

September 24. Spinors and Projective Geometry. Oswald Veblen, Institute for Advanced Study. *Some Unsolved Problems of Theoretical Dynamics.* George D. Birkhoff, Harvard University. *Textile Research in the Interest of the Consumer.* Ruth O'Brien, U. S. Bureau of Home Economics.

September 25. The Historical Interpretation of Art and Literature. Halvan Koht, former Secretary of State of Norway. *Tuberculosis as the Chemist Sees It.* Florence B. Seibert. The Henry Phipps Institute. *Glaciation and Submarine Valleys.* Reginald A. Daly, Harvard University. *Advancing Frontiers of Nursing Education.* Isabel M. Stewart, Columbia University.

September 26. The Significance of Choline as a Dietary Factor. Charles H. Best, University of Toronto. *Virus Infection of the Mammalian Foetus.* Ernest W. Goodpasture, Vanderbilt University. *Nuclear Transformations.* Ernest O. Lawrence, University of California. *The Cosmical Abundance of the Elements,* Henry N. Russell, Princeton University.

The program of symposia and lectures will be followed immediately by an Academic Festival, September 27–29, the principal events of which will include an Alumni Assembly, a Service of Thanksgiving and Commemoration, a Reception of Delegates, a Festival Concert and a Convocation, at which honorary degrees will be conferred.

SPECIAL ARTICLES

THE AGGLUTINATION OF RED CELLS BY ALLANTOIC FLUID OF CHICK EMBRYOS INFECTED WITH INFLUENZA VIRUS

WHEN the allantoic fluid from chick embryos previously infected with strains of influenza A virus was being removed, it was noted that the red cells of the infected chick, coming from ruptured vessels, agglutinated in the allantoic fluid. Since red cells in the allantoic fluid of chick embryos inoculated with sterile materials did not agglutinate at all, it seemed that this agglutination phenomenon might be the result of infection with influenza virus in the chick.

To demonstrate the agglutination phenomenon in the infected chick embryos, the egg shell was opened over the air sac. The outer chorio-allantoic membrane was torn away, and several large blood vessels were purposely ruptured. Fifteen to 30 seconds were allowed for the embryo to bleed into the allantoic fluid before the contents of the allantoic sac were emptied into a petri dish. If the embryo had been infected with influenza virus, macroscopic agglutination of the red cells occurred within 15 to 30 seconds in the petri dish. If the agglutination did not appear promptly, it usually did not occur at all, and the differentiation between positive and negative eggs was easy. Virus titrations and serum neutralization tests were then set up in eggs, with this agglutination phenomenon as an index of infection. Egg-passage viruses and ferret sera were used in these tests. One tenth cc of the material was inoculated into the allantoic sac of 11-day old embryos which were then allowed to incubate for 2 days. The eggs were opened by the method described above, and positive and negative reactions were recorded. By using eggs in the same way that mice are used in serum titrations and virus titrations, it was found that serum neutralization tests and virus neutralization tests could be performed. The end points were as sharp as those obtained in the mouse test. The agglutination reaction worked equally well with strains of influenza A or B virus and with swine influenza virus. Cross neutralization tests were then set up with these viruses, which gave results consistent with the specificity of these strains as established in mice. A neutralization test with acute and convalescent serum from a case of influenza A demonstrated a rise in antibody titer in the convalescent serum which was consistent with the rise obtained in similar tests in mice.

Throat washings have been passed in eggs, and while this phase of the work is in a preliminary stage, we have so far isolated two strains of influenza A virus directly from throat washings and obtained the agglutination phenomenon in the chick embryo on the second passage. The virus from these throat washings was set up in a neutralization test with A and B antiserum in

eggs and its identity as A virus was established. In one case the total elapsed time from inoculation of the throat washings until the confirmation of the identity of the virus by specific serum was only 9 days. Whether B virus and other as yet undescribed viruses from influenza cases will behave similarly is now being determined.

At present neither the mechanism of the agglutination phenomenon nor its specificity for influenza virus infection is well understood. However, the following facts have emerged:

(a) When infected allantoic fluid, either fresh or stored at −72° C. (from which the red cells had been removed by low-speed centrifugation), was mixed in a test tube with washed normal adult chick red cells, an agglutination phenomenon occurred. This *in vitro* agglutination was somewhat slower than the one previously described. Here a positive agglutination reaction was usually visible in 5 to 20 minutes. The red cells sedimented rapidly and formed a characteristic, ragged, granular pattern on the bottom of the tube. If red cells were added in the test tube to allantoic fluid from uninfected chicks, only the slow sedimentation of red cells occurred with no aggregation, and in settling out the cells formed a sharp round disk in the bottom of the tube. If the allantoic fluid from chicks infected with PR8 virus was diluted before adding the red cells, agglutination was still visible in a concentration of 1:512 (final concentration of allantoic fluid).

(b) When normal chick embryo red cells were added in sufficient numbers to allantoic fluid and allowed to settle out, over 99 per cent. of the virus disappeared from the supernatant fluid.

(c) When the allantoic fluid was centrifuged (45 minutes, 11,500 r.p.m.) the "titer" of the supernatant in terms of agglutinating capacity dropped approximately four times. This fall in agglutinating titer was consistent with the expected drop in virus titer as determined by previous tests in mice with the same fluid which showed that 70 to 90 per cent. of the virus was sedimented.

(d) If, instead of infected allantoic fluid, the supernatant from centrifuged ground mouse lung infected with PR8 mouse passage virus was used, the added red cells agglutinated in a dilution as high as 1:5000 (final concentration of mouse lung).

(e) When influenza A ferret antiserum (PR8) in dilutions as high as 1:1024 was mixed with allantoic fluid infected with the homologous virus, the agglutination phenomenon was inhibited. The inhibition was specific, that is, influenza B ferret antiserum in dilutions as low as 1:8 failed to inhibit the agglutination of red cells by fluids containing influenza A virus.

(f) Such inhibition also occurred with human serum, and in Table I is a titration of acute and convalescent serum from a proven case of influenza A. Dilutions of serum were mixed with a constant amount of W.S. infected allantoic fluid; then a constant amount of chick embryo red cell suspension was added, and the agglutination was read in 1 hour. The change of titer of this agglutination-inhibiting substance following infection is obvious and appears to be of the same order of magnitude as the rise in the patient's neutralizing titer against PR8 virus as determined in the mouse.

TABLE I

COMPARISON BETWEEN IN VITRO INHIBITION OF RED CELL AGGLUTINATION AND MOUSE NEUTRALIZATION TESTS WITH ACUTE PHASE AND CONVALESCENT SERUM FROM A CASE OF INFLUENZA A

Serum	Constant amount of infected allantoic fluid tested against various dilutions of serum*		Results of neutralization test in mice against uniform dose of virus†	
	Dilution of serum	Agglutination after 1 hour	No. died	No. survived
Acute	1:8	++	4	0
	1:16	++++		
	1:32	++++	4	0
	1:64	++++		
Convalescent ...	1:8	−	0	4
	1:16	−		
	1:32	−	0	4
	1:64	−		
	1:128	−	0	4
	1:256	−		
	1:512	+	3	1
	1:1024	+++		
Saline control, no virus		−		
Virus control, no serum		++++		

* The W.S. strain of influenza virus was used for the infection of chick embryos.
† The PR8 strain of influenza virus was used for the protection test in mice.

Such an *in vitro* test as shown in Table I will be of great advantage over the mouse neutralization tests if it can be shown that it measures influenza neutralizing antibodies. Our results so far suggest that the amount of agglutination-inhibiting substances in sera parallels the neutralizing antibody titer more closely than the complement-fixing titer. Whether this *in vitro* test will be sensitive and specific enough to replace the mouse protection method for determining serum neutralizing antibodies, is a problem at present under investigation. A more complete report will be published at a later date.

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ON THE SPECIFIC ADSORPTION OF ALCOHOLS AT THE SALICYLALDEHYDE/WATER INTERFACE

DURING a recent investigation by one of us (D.F.C.) of the interfacial tensions between water and mixtures of isoamyl alcohol and salicylaldehyde, it was observed that, over a wide range of isoamyl