SECTION ON MICROBIOLOGY

MAY 19, 1948

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I. EXECUTIVE SESSION

a. Reading of the Minutes

b. Election of Section Officers
For Chairman—Gregory Schwartzman
For Secretary—Harry Most

c. Election of five members of the Advisory Board
Frank L. Horsfall, Jr. (1948-9)
Colin M. MacLeod (1948-50)
John G. Kidd (1948-51)
Rene J. Dubos (1948-52)
Ralph S. Muckenfuss (1948-53)

II. PAPERS OF THE EVENING

a. Studies on the mechanism of polysaccharide inhibition of virus multiplication
Harold S. Ginsberg (by invitation) and Frank L. Horsfall, Jr.
Hospital of the Rockefeller Institute

b. Stability of bacterial viruses in solutions of salts
Mark H. Adams, Ph.D. (by invitation)
New York University College of Medicine

c. Dextran-forming streptococci from the blood in subacute endocarditis and from the throats of healthy persons
Edward J. Hehre (by invitation)
Cornell University Medical College

d. Effect of nucleic acids and carbohydrates on the formation of streptolysin
Alan W. Bernheimer, Ph.D. (by invitation)
New York University College of Medicine

e. Treatment of amebic hepatitis with chloroquine
Neal J. Conan (by invitation)
New York University College of Medicine

Studies on the Mechanism of Polysaccharide Inhibition of Virus Multiplication

HAROLD S. GINSBERG (by invitation) and FRANK L. HORSFALL, JR.

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It was demonstrated recently[1, 2, 3] that type specific capsular polysaccharides of Friedländer bacilli alter the course of infection with PVM and mumps virus. Multiplication is inhibited when polysaccharide is injected as long as 4 days after inoculation of either virus in appropriate hosts. Polysaccharide does not inactivate or demonstrably alter the virus, nor is a virus-polysaccharide combination formed. Present evidence indicates that polysaccharide does not block virus "receptors" of host cells and even in the presence of large quantities of carbohydrate virus is adsorbed by host tissue thereby completing the first step in infection.

The capsular polysaccharide of Friedländer bacillus type B (Fr. B) does not inhibit multiplication of influenza A, influenza B and Newcastle disease viruses[2] which multiply rapidly. This suggested that the latter viruses may require different metabolic systems within the susceptible cells of appropriate hosts than do mumps virus and PVM which increase in concentration at a slower rate.

The viruses of influenza A, influenza B and Newcastle disease interfere with the multiplication of each other.[4, 5, 6] If these viruses did require host metabolic systems different than mumps virus and PVM for multiplication, it seemed possible that the former viruses would not interfere with the multiplication of the latter. Experiments were carried out to test this hypothesis.

Varying quantities of mumps virus were inoculated into the allantoic sac of chick embryos, and followed in 4 days by inoculation of varying quantities of the PR8 strain of influenza A or the Lee strain of influenza B virus. After a further period of 2 days, the allantoic fluids were removed, and their hemagglutination titers determined in the presence of a constant quantity of immune rabbit serum, either anti-mumps, anti-PR8 or anti-Lee. When quantities of influenza virus were employed, which were equal to or smaller than the quantity of mumps virus inoculated, both viruses multiplied simultaneously in the allantoic sac.

Due to the fact that the influenza virus strains employed kill chick embryos 3 to 4 days after inoculation, it was necessary to shorten the total period of incubation in order to use these influenza viruses as the first inoculum. Under these conditions, when as much as 10^6 E.I.D. of each virus was employed, simultaneous multiplication of influenza and mumps viruses was demonstrated.

Influenza A and B viruses mutually interfere in the mouse lung.[4] If the theory proposed above were correct, then PVM and influenza viruses should not interfere with the multiplication of each other in the mouse. The intranasal inoculation of PVM was followed in 4 days by the intranasal inoculation of a similar quantity of PR8 or Lee virus. After a further interval of 2 days the mouse lungs were removed, and appropriate procedures carried out to determine the presence of PVM and influenza viruses by the hemagglutination technique. In each instance there was simultaneous multiplication in the mouse lung of influenza viruses and PVM. In order to carry out experiments in reverse order, strains of influenza A and B viruses which had not been "adapted" to the mouse were employed.[7] When either the FM1 strain of influenza A virus or the B1103 strain of influenza B virus preceded the intranasal inoculation of PVM by 2 days, it also was found that influenza viruses did not interfere with the multiplication of PVM.

Due to the lack of a common host it was not possible to determine whether PVM and mumps virus mutually interfere, as theoretically they should. Present evidence suggests that mumps virus and PVM require different host metabolic systems for multiplication than the viruses of influenza A, influenza B, and Newcastle disease. This may afford an explanation for the fact that Fr. B inhibits the multiplication of mumps virus and PVM, but fails to alter the course of infection with the influenza-Newcastle group of viruses.

REFERENCES

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