and acetate with time and thus promoting incorporation of carbon-14 into tissue constituents.

Muscular exercise and urethane administration may influence the rate of incorporation of carbon-14 into tissue constituents by acting on other metabolic steps than those mentioned above; such an influence is indicated by the observed increase following muscular exercise in the carbon-14 content of the fatty fractions of the muscles and in some experiments also in those of the intestinal mucosa. Nevertheless, the dilution effect is presumably to a large extent responsible for the changes observed in the incorporation of carbon-14 following muscular exercise and urethane administration.

The carbon-14 content of the average total tissue is found to be about twice as large after the lapse of 20 min. as after the lapse of 4·5 hr. This is mainly due to the fact that the proportion of non-metabolized acetate in the tissue is very much larger after 20 min. than after 4·5 hr., and also that the fatty components present, being renewed at a very rapid rate, are more active after 20 min. than after 4·5 hr. The probability of carbon-14 taken up by the protein being lost again within a few hours is small, and correspondingly the average tissue protein activity after the lapse of 20 min. is about one-third only of the value observed after the lapse of 4·5 hr.

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Hæmolysis by Newcastle Disease Virus

For a number of years it has been known in this laboratory that hæmolysis of moderate degree may occur when red cells are being treated with the viruses of mumps and Newcastle disease. This is not characteristic of any of the influenza viruses. The hæmolysis was of slight degree and apparently irregular in its occurrence. Following the appearance of the paper by Morgan, Enders and Wagley[1] on mumps virus hæmolysis, it was noted that when dilutions of Newcastle disease virus were made in phosphate solution instead of physiological saline, both fowl and human red cells were more extensively lysed. A comprehensive study of the phenomenon was afterwards begun.

The hæmolytic action of Newcastle disease virus appears to resemble in essential respects that shown by mumps virus, but differs greatly from any other type of hæmolytic effect. The most striking feature is the complete absence of hæmolysis of cells from which 'virus receptors' have been removed either by virus action or treatment with the cholera vibrio enzyme.

Hæmolysis is inhibited by low concentrations of calcium ions, and typical infected allantoic fluids have very little hæmolytic action when used undiluted unless a calcium-deionizing agent such as citrate is added. Most of the work has been done with preparations of virus eluted from red cells into saline lightly buffered at *p*H 7·35 with phosphate, or with virus precipitated by methanol in the cold according to the technique of Cox *et al.*[2] and dialysed against buffered saline.

The course of hæmolysis of washed fowl erythrocytes differs characteristically according to which of these preparations is used. At 37° the eluate shows a lag period of 30–40 min. before hæmolysis is detectable; with the methanol-precipitated virus the lag period ranges from 1½ to 4 min., according to the concentration of virus used. With methanol-precipitated virus in amount corresponding to about a hundred agglutinating doses and in a 2 per cent suspension of red cells, hæmolysis proceeds to about 90 per cent completion within 15 min. and then ceases. With progressively smaller concentrations of virus, the level at which hæmolysis virtually ceases falls. Over a fairly wide range, the level is approximately proportional to the logarithm of the virus concentration. Eluate virus gives similar results, but the time needed to reach the final level of hæmolysis is longer and the level itself considerably lower than with methanol-precipitated virus.

Hæmolytic activity is destroyed by heating in phosphate or phosphate saline to 54° C. for 30 min. without damage to hæmagglutinating capacity and with retention of infectivity for chick embryos. It is neutralizable by specific immune sera to titres corresponding closely to their anti-hæmagglutinin titres.

All the experimental results so far obtained are in accord with the view that the hæmolytic agent is the virus particle itself, and that hæmolysis cannot occur unless the virus particle is adsorbed to the cell surface by the well-known receptor mechanism. It is possible to prevent hæmolysis by Newcastle disease virus by prior treatment of the cells with the same virus under appropriate conditions, for example, in the presence of excess calcium ions. This makes it highly probable that there are at least two competitive types of interaction between virus and cell receptor, and that hæmolysis ceases when all remaining intact cells have lost their receptors as a result of the other (non-hæmolytic) reaction.

If the hæmolysis is due to an enzyme carried by the virus, it would appear that this enzyme is distinct from that responsible for the destruction of cell receptors which is present in all influenza viruses as well as in Newcastle disease virus. Hæmolytic action is lost at a temperature well below that which seriously reduces the enzymic action of Newcastle disease virus on ovomucin, and calcium ion inhibits hæmolysis while it favours slightly the receptor-destroying activity of the virus.

A full account of these experiments will be published elsewhere.

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