It is probable that the hexose is actually galactose in view of the findings of Evans[2, 3] although there is some discrepancy concerning the ratio of hexose to hexosamine. This may probably be ascribed to the greater purity of our preparations.

A preparation made from non-pregnant mare serum by a similar procedure proved to contain equimolar amounts of mannose and galactose. In this case the carbohydrate moiety appears to be similar to that present in serum mucoid and in other serum glycoproteins.

It is interesting to note that the carbohydrate groups of the gonadotropins of pregnant mare serum and of urine of pregnancy in women are apparently similar and that they are different from those found in the pituitary gonadotropic hormones. It is difficult to believe that these results are purely fortuitous and that the presence of a different type of carbohydrate in the gonadotropins associated with pregnancy (gestation) is not a result of the marked metabolic changes occurring during pregnancy.

Summary. A qualitative as well as a quantitative study has been made of the hexose present in the gonadotropic hormones of the pituitary gland, pregnant mare serum and human pregnancy urine. The hormones obtained from the pituitary gland contain mannose and hexosamine in equimolar proportions. The gonadotropins of pregnant mare serum and human pregnancy urine appear to contain galactose rather than mannose. In these preparations the molar ratio of hexose to hexosamine is 2:1.

13460 P

Influence of Estrogen on Respiration of Rat Uterine Tissue.

WILLIAM R. CARROLL. (Introduced by F. L. Hisaw.)

From the Biological Laboratories, Harvard University, Cambridge, Massachusetts.

Although much is known about the morphological changes that take place in various tissues and organs due to the influence of the different sex hormones, little is known about true physiological effects, and still less about the actual biochemical action of the active substances. Hence an investigation has been undertaken to locate the mode and place of action in a physiological system of one of the female sex hormones, estradiol.

One of the physiological effects of estrogen, and one that lends itself to biochemical analysis, is the increase in oxygen consumption

of the uterus. There have been several investigations of the respiration of mouse and rat uteri,[1-5] and all of these agree that estrogen increases the oxygen consumption. Kerly,[4] and Büngler and Erhardt[2] showed that the anaerobic glycolysis of uterine tissue is increased by injection of estrogen, and the latter authors also found an increase in the aerobic glycolysis.

The technics used in the present study differ in several respects from those of previous studies. First, a pure estrogen was used. This was estradiol dipropionate, 50 μg of which is sufficient to produce a continuous estrous effect for at least a month.[6] Second, effects of a continuous treatment with estrogen were determined. Third, measurements were made on separate strips of endometrium and muscle. Fourth, dry weights were determined on other uterine tissue treated in the same way as that used for the respiration measurements. Summerson manometers were employed for the determination of oxygen consumption, aerobic glycolysis, and R. Q.

The changes in the oxygen consumption of uterine tissue with time after injection of estradiol dipropionate follow a peculiar curve. The normal value of the Q_{O_2} for uteri from castrated, uninjected animals is 3.9. There are only slight increases at 6 and 12 hours after injection, but at 24 hours the Q_{O_2} has reached 6.15. However, the peak, 8.0, is reached by 45 hours and is maintained until about 75 hours, when the Q_{O_2} falls off to a plateau of 6.6. This level is maintained for several weeks, or as long as the oestrous effect of the injection lasts. The values given for tissue after 24 hours' injection are for the endometrium. The Q_{O_2} values of muscle follow the same sort of curve but are slightly smaller: 6.65 at the peak, and 6.3 for the final plateau.

It may be significant that the values of the aerobic glycolysis ($Q_G^{O_2}$) reach their peak and then their final plateau much sooner than those of the Q_{O_2}. The normal value is 1.5; the peak comes at 24 hours and is 3.1 for the endometrium and 2.0 for muscle. The drop to the final plateau begins immediately and by 70 hours has reached 1.0 for endometrium and 0.5 for muscle.

R. Q. values increase from 0.89 in the normal untreated controls to 1.0 at 24 hours, but soon fall again to about 0.90 by 70 hours.

1 David, J. C., *J. Pharmacol.*, 1931, **43**, 1.

2 Büngeler, W., and Erhardt, K., *Klin. Wchnschr.*, 1931, **10**, 593.

3 Khayyal, M. A., and Scott, C. M., *Quart. J. Exp. Physiol.*, 1935, **25**, 77.

4 Kerly, M., *Biochem. J.*, 1940, **34**, 814.

5 MacLeod, J., and Reynolds, S. R. M., PROC. SOC. EXP. BIOL. AND MED., 1938, **37**, 666.

6 Miescher, K., Scholz, C., and Tschopp, E., *Biochem. J.*, 1938, **32**, 725.

Glucose has the expected effect of raising the R. Q. in all cases except those of uterine muscle from animals injected for more than 70 hours. In these cases it is lowered slightly.

A more noticeable influence of glucose is the inhibitory effect it has on the Q_{O_2} of both endometrium and muscle from the 24-hour injected animals. The inhibition averaged about 20%. After 24 hours, oxygen consumption was practically the same with and without glucose.

The values of the anaerobic glycolysis follow a pattern similar to those for the changes in oxygen consumption. The average value for uteri from untreated animals is 6.1 ($Q_G^{N_2}$), the peak at 60 hours is 11.5, and the plateau at 90 hours and beyond is about 9.25.

These results show the direction and magnitude of the effect which estrogen has on the respiratory metabolism of uterine tissue. A further analysis to find the point of action of estrogen in the respiratory cycle should be possible, perhaps with the aid of inhibitors of specific respiratory enzymes or enzyme systems.

13461 P

Encephalitis Virus "Antibody" in Sera of Experimentally Infected Animals by Agglutination of Virus-Coated Cells.

E. C. ROBERTS AND L. R. JONES.

From the Department of Bacteriology, St. Louis University School of Medicine.

Details of the technic involved in satisfactorily coating bacterial cells with encephalitis virus and the agglutination of such cells by the sera of encephalitis patients have been given in a previous report[1] wherein the test was designated as the bacterial agglutination (B.A.) method of detecting serum antibody. That encephalitis virus was actually adsorbed onto the bacteria was indicated by the infectivity of 'coated' cells for susceptible animals even after repeated washing to remove extraneous virus. The specific nature of the agglutination reaction with patients' serum was demonstrated by certain control tests wherein the reaction did not occur when non-coated cells or cells coated with normal mouse brain were used. Further, sera from cases of clinically recognized poliomyelitis were studied which did not agglutinate cells coated with encephalitis virus although they were

[1] Roberts, E. C., and Jones, L. R., PROC. SOC. EXP. BIOL. AND MED., 1941, 47, 75.