Effect of Acetylcholine and Eserine on the Spawning of *Hydractinia echinata*

It is usually considered that the responses of coelenterates are not mediated by acetylcholine[1], because: (1) there is no evidence of significant amounts of acetylcholine or choline esterase in their bodies; (2) neither these substances, nor atropine, nor curare, affects their neuromuscular activities[2]. However, I have recently found that acetylcholine (Roche) and physostigmine (eserine; B.D.H.) affect the spawning activity of *Hydractinia echinata*.

As previously reported[3,4], spawning is induced by a suitable periodicity of lighting, darkness conditioning the response which is triggered by light. When isolated gonophores are subjected to sea water containing either acetylcholine (concentration; 5×10^{-4}) or eserine (concentration; 10^{-4}) during the periods of darkness, discharge is inhibited in 50–80 per cent of the mature gonophores; but a number of treated gonophores, both male and female, spawn in the dark without light treatment. Concentrations of both substances above 10^{-3} inhibit spawning completely and those below 10^{-5} are ineffective.

As neither of the drugs acts when introduced at the beginning of illumination, or even 5 min. before the illumination is due to start, it is clear that the triggering process is insensitive to them.

Thus, they appear to act in two ways; in one, by interfering with the increase in photosensitivity during darkness, and in the other, by triggering off the final process of spawning. It is also worth noting that sensitivity to either acetylcholine or eserine coincides in time with sensitivity to calcium[4].

Whether a true cholinergic mechanism exists, and how it is related to calcium, remains to be discovered.

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Transmission of Passive Immunity in an Insectivore

The hypothesis[1] that antibodies are not transmitted through placentæ of the epitheliochorial and syndesmochorial types and are transmitted through hæmochorial and hæmoendothelial types was at one time widely accepted. More recently it has been demonstrated in the rabbit[2] and guinea pig[3] in which the placentæ are hæmochorial that the transmission of antibodies occurs exclusively via the yolk sac. In the rat transmission occurs by way of the yolk-sac endoderm and by way of the gut, and in this species some transmission across the hæmochorial placenta could not be excluded[4].

In the hedgehog the placenta is hæmochorial and in the young of animals immunized against *Brucella abortus*, specific agglutinins could not be detected in the sera before suckling. The females received immunizing injections before and during pregnancy, and the maternal antibody titres during pregnancy were of the order 1/640 to 1/1280. The sera of 10 young derived from 6 litters, which were removed from their mothers before suckling occurred gave negative results at dilutions of 1/10. In this species the yolk sac persists to term and its abembryonic wall remains intact[5], whereas in the rabbit, guinea pig and rat the yolk sac is of the inverted type in which the abembryonic bilaminar segment is broken down and the yolk-sac splanchnopleur is exposed to the uterine lumen.

The young of ruminants, horse and pig are born without antibodies, and in these species a rapid uptake of antibody occurs from the colostrum and milk during a 36-hr. period after birth. During this period the antibody titre of the serum of the young animal increases to become approximately equivalent to that of the maternal serum. The antibody of the colostrum in these animals attains titres which equal or exceed those of the maternal serum. In the hedgehog the antibody titre of the first milk closely approximates the maternal serum titre; but with suckling the titre declines so that in the nursing female six days after parturition it is about 25 per cent of the maternal serum titre. In this species there is an uptake of antibody from the milk by the gut; but even after several days the titre attained in the serum of the young hedgehog is only a small fraction of that in the maternal serum. The highest serum titre so far obtained in a young animal is 1/20, with partial agglutination at 1/40, at 6½ days of age, the titres of the maternal serum and milk being 1/640 and 1/160 respectively. In this representative of a primitive mammalian order transfer of anti-*Brucella* agglutinins does not occur prenatally, and the postnatal transmission is of a very low order when compared with other species in which the young obtain passive immunity after birth.

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Release of Histamine from Rat Mast Cells by Blood Treated with Dextran

Halpern[1] has shown that the injection of dextran into albino rats causes increased capillary permeability and shock. Coincident with the appearance of shock, there is a massive release of histamine into the blood stream of injected animals. The liberated histamine probably accounts for the greater part of the increased capillary permeability and shock resulting from the administration of dextran. It seemed of interest, therefore, to investigate the mechanism of the release of histamine. It was found that dextran reacts with a plasma protein to produce a substance which acts on mast cells to release histamine.

Peritoneal cavity cells, including mast cells, were obtained from the rat by a method previously described[2]. To detect mast cell disruption, one drop of the cell suspension was added to one drop of rat serum or serum fraction at room temperature, and the preparations were examined under the microscope. Histamine assays were performed by the method of Lowry *et al.*[3]

Satisfactory results were obtained only when the solutions had a *p*H of less than 7·5. For this reason blood was collected and centrifuged under paraffin.