by adsorption on kaolin or any other adsorbent. Consequently we have recrystallized urease from 30% alcohol repeatedly in order to note whether it is possible to remove the sulfhydryl compound. After 3 recrystallizations the amount of color given by the nitroprusside test remains approximately the same as after one recrystallization. We believe, therefore, that the urease molecule itself contains sulfhydryl groups, or groups which give the nitroprusside test. These groups, if sulfhydryl, may account for the readiness with which urease is inactivated by silver, mercury and copper ions, and by quinone and other oxidizing agents, and why urease is protected by sulfhydryl compounds.

Finally, the dialysable substance in jack bean meal is not concerned with urease activity since urease which is freed from this material has high activity, and since addition of concentrated dialysate from jack bean meal exerts no favorable effect upon urease action.

We wish to thank Dr. D. B. Hand for his kindness in supplying us with pure glutathione.

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Transmission of Neurohumors in Animals by Other Means Than Blood and Lymph.

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It has been assumed that the contraction or expansion of dermal melanophores in fishes whereby these animals take on a light or a dark tint is dependent upon substances carried in the animal's blood and lymph (Parker[1]). Such an assumption appears to be justified by the fact that if a small amount of dissolved adrenalin is injected into the muscles of a dark chub, *Fundulus heteroclitus,* this fish within a quarter of an hour or less will become very light-colored. That the normal changes in tint shown by such a fish are brought about by other means than by dissolved substances carried in blood and lymph seems very probable from the following observations.

If a short transverse incision is made in the tail of a light-colored Fundulus, a dark stripe extending from the cut to the posterior edge

[1] Parker, G. H., Humoral agents in nervous activity with special reference to chromatophores. Cambridge, England, 1932, 79 pp.

of the tail will quickly appear. This stripe will gradually become lighter and lighter till in a few days it is no longer distinguishable from the rest of the tail. The transverse cut severs a number of radiating nerve fibers that pass out into the fish's tail and the dark stripe itself represents an area of denervation thus produced. That this stripe should be dark is due to the fact that the severance of the nerve fibers excites the discharge of an expanding neurohumor whereby the melanophores are made to enlarge and thus to darken the skin.

On placing a light-colored fish, whose stripe has become equally light-colored, on an illuminated dark background, the body of the fish will become fully dark in a little less than 2 hours. The stripe, however, will remain light-colored for some time, changing slowly to dark only in the course of a day to a day and a half. If now the completely dark-colored fish is put on a light background, the body of the fish will become fully light in from 4 to 5 hours, but the stripe will not lighten fully before somewhat over a day to over 2 days. Thus in both sets of changes the disappearance of the stripe is very slow as compared with the disappearance of the corresponding tint on the general surface of the fish. Thus in these changes the stripe lags well behind the general body.

The light coloration of the general surface of the fish is believed to be due to the discharge from a given set of nerve terminals of a neurohumor that induces a contraction of the melanophores and the dark coloration to the discharge from a second set of terminals of a neurohumor that will induce an expansion of the melanophores (Mills[2]). In the denervated stripe these neurohumors cannot of course be produced and the changes in such a stripe are believed to result from the transmission of the appropriate neurohumor from its region of production adjacent to the stripe into the stripe itself. This assumption is supported by the fact that when the stripes begin to disappear they start to fade first at their edges, after which the fading spreads to their interiors. Further it requires a stripe of 2 mm. width about twice as long to fade as one a single millimeter wide. For these reasons it is concluded that the fading of a stripe is due to an invasion by an appropriate neurohumor.

The rate of this invasion can be roughly measured and may be stated in averages to be for the dark-inducing neurohumor a millimeter in about 20 hours and for the light-inducing one a millimeter in about 26 hours. The periods of time implied in these rates are so

[2] Mills, S. M., *J. Exp. Zool.*, 1932, **64**, 231, 245.

long compared with the time it takes adrenalin to act, at most a quarter of an hour after injection, as to suggest at once an entirely different means of transmission in the two instances. It does not seem conceivable that the 2 neurohumors concerned with the color changes could be transmitted by blood and lymph whose flow, even at their slowest, would not be so retarded as is indicated by the rates given. Much more likely is it that the neurohumors are handed on from the adjacent innervated region into the denervated one through some other medium than blood and lymph and such a medium may well be the tissues themselves, whose cells next the nerve terminals of the innervated area may take up the neurohumor there produced and pass it on either through their cell sap or their outer lipoid coverings to other cells more nearly within the denervated stripe. Thus the neurohumor would make its way from the nerve terminals of the adjoining region to the denervated melanophores by steps that would of necessity be time-consuming and consequently consistent with the intervals observed as contrasted with what has been noted for transmission by blood and lymph. I, therefore, conclude that in Fundulus the neurohumors for the melanophores spread by tissue, probably cellular, means and hence with great slowness.

The explanation of neurohumoral spread just put forward is quite in accord with what must occur rather extensively in many of the lower animals as, for instance, in the coelenterates. In such animals as sea-anemones, coral polyps, hydroids, and the like the wall of the body, which may be relatively thick, is composed of 2 cellular layers, an inner entoderm and an outer ectoderm separated by a firm supporting lamella. Only the entoderm is next the digestive cavity and hence only its cells are in a position to absorb nourishment directly from the supply of digested food elaborated in that cavity. The ectoderm, on the other hand, a layer of great activity from the standpoint of both muscles and glands, can gain its nourishment only by having the digested materials passed on to it through the entoderm and the intervening supporting lamella. Such a transmission seems to be a necessary part in the economy of such creatures and must be accomplished by the tissues of these lowly animals in much the same way as has been suggested for the transmission of neurohumors in the tail of Fundulus. The coelenterates possess neither blood nor lymph and the nourishment of their parts not next the digestive cavity must of necessity depend upon tissue or cellular transmission. From an evolutionary standpoint this type of transmission unquestionably antedates that of blood and lymph and must be looked upon as the original method of transmitting materials, a method still of first importance among primitive types and retained to a significant extent even in the vertebrates.

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Effect of Dextrose-Alcohol Mixture upon Pulmonary Fat Embolism.

LOUIS G. HERRMANN. (Introduced by S. Bayne-Jones.)

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The therapy of post-traumatic pulmonary fat embolism has rereceived little attention. We have studied the effect of various organic and inorganic substances upon the fat globules which lodge in the capillaries of the lungs after the intravenous injection of a known quantity of sterile fat from an animal of the same species.

Twenty-four large Belgian hares and 4 dogs were used. Normal, healthy animals of the same litter were used in each series of experiments.

The sterile fat was warmed to 37°C., and injected into either the marginal vein of the ear of the hares, or into the saphenous vein of the dogs. The injections were carried out at the uniform speed of 1 cc. per minute and without undue force. Four animals were used in each series of experiments. Into each animal we injected 1 cc. of the warmed, sterile fat per kilo of body weight. One animal of each series was kept for the control. The other 3 animals were treated by an intravenous injection of 5 cc. per kilo of body weight of the following dextrose-alcohol mixture: Ethyl alcohol C. P. (96%) 3 parts; dextrose C. P. (25% solution in water) 7 parts.

Two of the remaining animals of each series received a second injection of the same quantity of this mixture at the end of 12 hours. Twenty-four hours after the injection of the fat, one of the last 2 animals was again treated by a third injection of the same quantity of the mixture intravenously. All of the animals of each series were killed by an intracardiac injection of sodium cyanide 48 hours after the fat was injected into the blood stream. Immediately after death the lungs of the animals were placed in a large quantity of 10% solution of formaldehyde in order to insure complete fixation.