became an intensive one when, during a study of the fusion of slime molds, certain plasmodia failed to fuse.

Two individual masses of slime mold protoplasm approaching each other will, under favorable conditions, meet and fuse. After uniting, extraordinarily large and well-developed "arteries" of active flow are established at the point of transfusion. The excessive development of the connecting "artery" and the abnormally rapid flow of protoplasm through it are due to the release of pressure in the region of the union. With the sudden release of the surface tension which opposes the outward flow, there is an onrush of protoplasm which augments the rate of streaming and produces over-development of the channel of flow. This phenomenon was the subject of my study. But often, while waiting for fusion, I waited in vain. Two approaching plasmodia would occasionally halt at a short distance from each other, and there remain. A zone of definite width was established and maintained between them.

Another form of the reaction of slime molds against the exotoxins of others of their kind is seen in the direction of movement or locomotion of plasmodia when several are in close proximity to each other. If the time is sufficient to permit waste products to form and collect between the plasmodia, the direction of movement of all of them is away from the center of the group, away from the center of maximum toxicity.

The pertinent facts may be stated as follows. When two slime molds rapidly approach each other and meet head-on, fusion always occurs and is immediate, provided the two plasmodia are of the same species and grown under similar conditions. But if the approach is gradual and there is thus time for the waste products of each plasmodium to collect at the approaching surfaces, then no fusion takes place. In short, no plasmodium will enter the zone of toxic waste products surrounding another plasmodium.

The toxic strip which separates two plasmodia is about 0.15 mm wide. The band is often of extraordinarily constant width which is maintained even when the plasmodia have irregular contours; the two boundaries may fit one into the other very much like two corrugated surfaces in which the crests of one fit into the troughs of the other.

The affinity and antagonism of cells for each other is a problem of great importance, applicable to a variety of situations throughout the living world. The failure of some sperm to accomplish the fertilization of eggs of the same species is due to lack of chemical affinity and this may be a question of exotoxins. Incompatibility due to the presence of exotoxins may also be the cause of the failure of conjugation between protozoa of the same species. "Mating types" in Paramecia may be determined by the reactions of one type to the exotoxins of the other type. The ingestion, or taking in of food, of one unicellular organism by another must in part be determined by the toxicity of the cell which serves as food, and this may be a matter of the environment of waste matter which surrounds the cell ingested. The engulfing of living cells by other cells assumes a special function in phagocytosis. The ingestion of some bacteria and not of others by the scavenger cells of the blood may be a question of the toxic effect of one cell upon another. All these examples, like that of the failure of two slime molds to fuse, may be determined by cellular exotoxins, by the secreted waste products of cells.

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CHOLINESTERASES

MENDEL and Rudney, in SCIENCE of January 14, 1944, again claimed priority for our earlier[1] discovery of two distinctive types of cholinesterase in the body. Their contention is apparently that since we failed to note a certain similarity of behavior between cholinesterases from blood cells and from serum we could not have been aware of their differences, which were in fact strikingly apparent in our comparisons of the two types of preparations. Even if one accepts their empirical test as definitive, their own data show only 2 per cent. of the activity of human serum to be due to cell type enzyme.

In the same note they quoted de Laubenfels as asserting that we had " 'thoroughly demonstrated' the existence of the true and pseudo-cholinesterase." This is inaccurate. De Laubenfels[2] correctly stated that we had proved the existence of two esterases capable of hydrolyzing acetylcholine, without implying that we had proffered a conclusion regarding their relative degree of specificity. Indeed, if we had, our reported evidence would have led us to the opposite conclusion from that of Mendel and Rudney.

We wish to record our support of de Laubenfels'[2] contention that "pseudo-cholinesterase" is an unfortunate name for an enzyme that has been so long and extensively studied under the name cholinesterase. In addition, current studies of serum cholinesterase in relation to disease, as in myasthenia gravis, as well as the findings of Glick[3] on the behavior of the enzyme of the cat superior cervical ganglion, make the acceptance of "pseudo-cholinesterase" as a suitable name for the serum enzyme seem inadvisable.

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[1] G. A. Alles and R. C. Hawes, *Jour. Biol. Chem.*, 133: 375, 1940.

[2] M. W. de Laubenfels, SCIENCE, 98: 450, 1943.

[3] D. Glick, *Jour. Gen. Physiol.*, 21: 431, 1938.