easily permeable substrate the inhibition of the enzyme will permit the attainment of a higher concentration, but only in the difficultly permeable muscles will there be an increased sensitivity. But this cannot explain why in muscle one enzyme inhibitor increases the sensitivity markedly, while another, even a stronger inhibitor, does not affect it. The acetylcholine sensitivity can apparently be influenced either by changes in the receptors or in the contractile elements themselves. The experiments with NaF, physostigmine or quinine indicate that the rise in sensitivity is determined by an enzyme-resistant choline-ester. It is further pointed out that it is difficult to understand why the increased sensitivity is caused by physostigmine during narcosis with chloralose or EtOH but not with ether.

S. MORGULIS (Chem. Abstr.).

Mechanism of the Biological Synthesis of Acetylcholine. (*Nature, vol. cxli, p.* 374, 1938.) *Mann, P. J. G., Tennenbaum, M., and Quastel, J. H.*

Stedman and Stedman (*C.A.*, xxxii, *p.* 2200) claim that $AcCH_2CO_2Na$ is involved in the formation of acetylcholine in the brain. It is here stated that more acetylcholine is formed at 18° without $AcCH_2CO_2Na$ than at 45° with it, using $CHCl_3$—extracted brain. It is suggested that the $AcCH_2CO_2Na$ assists in the breakdown of a complex precursor of acetylcholine, but that it is not directly involved in the synthesis of the latter. MARCELLE SCHUBERT (Chem. Abstr.).

The Mechanism of Acetylcholine Formation in the Brain in Vitro. (*Biochem. Journ., vol. xxxii, pp.* 243–61, 1938.) *Mann, P. J. G., Tennenbaum, M., and Quastel, J. H.*

Acetylcholine is present in fresh brain tissue in a combined form which is pharmacologically inactive. A method for the estimation of this precursor of acetylcholine is given. The breakdown of the precursor to give acetylcholine can be effected by shaking with chloroform or by treatment with acid. At room temperature pH 3·0 causes breakdown and at 37° a pH of 6·0–6·5 is sufficient. The complex is stable under neutral conditions at 0°, but less so at higher temperatures. It is synthetised in brain-tissue when the latter is allowed to respire in the presence of glucose, sodium lactate, or sodium pyruvate. No synthesis takes place under anaërobic conditions. The sodium salts of AcOH, succinic, aceto-acetic, -ketoglutaric, -glycerophosphoric and hexosediphosphoric acids cause no synthesis of the precursor, nor did *dl*-glyceraldehyde. Eserine had no effect on the synthesis or breakdown of the complex. The presence of choline or acetylcholine increases the rate of synthesis. The effect of acetylcholine is no greater than that of choline, and its effect is probably due to choline liberated by choline esterase. The effect of glucose, sodium lactate and sodium pyruvate on the synthesis is much greater than that due to acetylcholine. It is unlikely that acetylcholine is first produced and then converted to the precursor by an adsorption process. A provisional scheme for the synthesis of acetylcholine is given. E. W. SCOTT (Chem. Abstr.).

A Basis for the Acetylcholine Action of Choline Derivatives. (*Journ. Pharmacol., vol. lxii, pp.* 430–48, 1938.) *Renshaw, R. R., Green, D., and Ziff, M.*

The duration of activity of a large number of choline derivatives when injected intravenously in the cat was measured and found to be approximately the same for all compounds studied, while the relative rates of inactivation of the same compounds by whole blood varied widely. The duration of depressor activity of a blood-stable compound, ethoxycholine bromide, was prolonged by prior injection of eserine, and to the same extent as that of acetylcholine. A substance with the characteristics of acetylcholine was present in blood drawn from the heart of an animal undergoing infusion of ethoxycholine bromide. It is suggested that the vasodepressant action of the choline analogues mentioned above is due, at least in part, to the liberation of acetylcholine from an inactive complex by a process of cationic exchange adsorption. The possible fate of choline and its analogues injected into the blood-stream is discussed. L. E. GILSON (Chem. Abstr.).