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**DEPARTMENT: ANATOMY**

**COURSE: CELLULAR BIOCHEMISTRY (BCH 308)**

 **ASSIGNMENT**

1. Describe in details the synthesis of two named neurotransmitters.

**SYNTHESIS OF GLUTAMATE**

Glutamate is a major constituent of a wide variety of proteins; consequently it is one of the most abundant amino acids in the human body. Under ordinary conditions enough is obtained from the diet that there is no need for any to be synthesized. Nevertheless, glutamate is formally classified as a non-essential amino acid, because it can be synthesized from alpha-Ketoglutaric acid, which is produced as part of the citric acid cycle by a series of reactions whose starting point is citrate.

Glutamate is synthesized in the central nervous system from glutamine as part of the glutamate–glutamine cycle by the enzyme glutaminase. This can occur in the presynaptic neuron or in neighboring glial cells. Glutamine is released by glial cells and, once within presynaptic terminals, is metabolized to glutamate by the mitochondrial enzyme glutaminase. Glutamate can also be synthesized by transamination of 2-oxoglutarate, an intermediate of the tricarboxylic acid (TCA) cycle. Hence, some of the glucose metabolized by neurons can also be used for glutamate synthesis.

**SYNTHESIS OF ACETYCHOLINE**

Acetylcholine is synthesized from acetyl coenzyme A and choline by the enzyme choline acetyltransferase. In the nervous system, this enzyme is thought to exist primarily in the nerve terminal cytoplasm. Coenzyme A is synthesized in mitochondria and accesses choline acetyltransferase following transport across the mitochondrial membrane into the cytoplasm. In addition to its synthesis in the liver, choline employed in acetylcholine production is derived from dietary sources. There is a carrier system in capillary endothelial cells that is responsible for transport of choline, in its free and phospholipid forms, into the brain. Hydrolysis of choline-containing phospholipids may also liberate choline that is used in acetylcholine synthesis. As choline acetyltransferase is not saturated by concentrations of acetyl coenzyme A and choline that are estimated to be present in the nerve terminal, it appears that the rate of acetylcholine synthesis is dependent on precursor availability. Enzyme activity is also regulated by product inhibition; by binding at an allosteric site on choline acetyltransferase, acetylcholine inhibits its activity. In addition, mass action and neuronal activity influence the rate of acetylcholine formation. Short-term regulation of enzyme activity is partly achieved by phosphorylation induced by protein kinases. There are no very specific and potent inhibitors of the enzyme and it should be noted that pharmacological blockade of this step (e.g. with naphthylvinylpyridine) in the life-cycle of acetylcholine produces a less profound effect on the transmitter than does inhibition of choline transport.