SAN2011

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L-Dopa can be added to the C-terminus of α-tubulin by tubulin tyrosine ligase, one of the enzymes involved in the postranslational cyclic tyrosination/detyrosination of tubulin. Now, we show that after its incorporation into tubulin present in soluble extracts from rat brain, L-Dopa cannot be released by tubulin carboxypeptidase, the other enzyme participating in the cycle. L-Dopa-tubulin can form microtubules as well as Tyr-tubulin. Amount of Dopa-tubulin in soluble extracts was inferred by analyzing the tyrosination state of tubulin by Western blots revealed with anti-total, Tyr-, Glu- and Δ2-tubulin. We suggest that during a prolonged administration of L-Dopa to Parkinson patients, L-Dopa-tubulin within cells gradually increases (and other tubulin species decrease), resulting in the formation of microtubules containing L-Dopa-tubulin that affect some of their properties, and this could be in some way responsible for the side effects of prolonged treatment with L-Dopa.

Cellular and Molecular Neurobiology
Poster Number 17 | **Session 2**

"Cholesterol modulates the rate and mechanism of acetylcholine receptor internalization"

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Stability of the nicotinic acetylcholine receptor (AChR) at the cell surface is key to the correct functioning of the cholinergic synapse. Cholesterol (Chol) is necessary for homeostasis of AChR levels at the plasmalemma and for ion translocation. Here we characterize the endocytic pathway followed by muscle-type AChR in Chol-depleted cells (Chol(-)). Under such conditions, the AChR is internalized by a ligand-, clathrin-, and dynamin-independent mechanism. Expression of a dominant negative form of the small GTPase Rac1, Rac1N17, abolishes receptor endocytosis. Unlike the endocytic pathway in control CHO cells, accelerated AChR internalization proceeds even upon disruption of the actin cytoskeleton. Under Chol(-) conditions, AChR internalization is furthermore found to require the activity of Arf6 and its effectors Rac1 and phospholipase D. The Arf6-dependent mechanism may constitute the default endocytic pathway followed by the AChR in the absence of external ligands, membrane Chol levels acting as a key homeostatic regulator of cell surface receptor levels.