



Autophagy in the Nervous System

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Message from the Guest Editor

Autophagy is considered to be one of the main A β -eliminating pathways under normal conditions. The ubiquitin proteasomal system may be the primary mechanism to degrade endogenous tau, while aggregated tau does not undergo degradation by the proteasome. Autophagy is involved in the clearance of soluble and aggregated tau and NFT in the cell. Moreover, autophagy activation can reduce the secretion of tau and tau propagation. These facts imply that autophagy activation can be beneficial for AD via clearance of A β or tau. In fact, several therapeutic studies have been performed by modulating autophagy, including mTORC1-dependent and -independent autophagy inducers, other autophagy-inducing drugs, and gene therapy, including microRNA. However, it has been reported that A β can be produced in autophagy, and A β secretion can also be done by autophagy. This Special Issue will offer us precious information regarding autophagy and A β or tau, and autophagy and alpha synuclein, TDP43, and huntingtin. Additionally, it may shed light on whether autophagy activation is beneficial for AD and other neurodegenerative disorders causing dementia.





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Message from the Editor-in-Chief

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