IJAE
Vol. 118, n. 2 (Supplement): 82, 2013
ITALIAN JOURNAL OF ANATOMY AND EMBRYOLOGY

Analysis of the autophagic flux in astrocytes intoxicated by trimethyltin

Cinzia Fabrizi[1], Elena Pompili[1], Francesca Somma[1], Stefania De Vito[1], Angela Catizone[1], Paola Lenzi[2], Federica Fulceri[2], Francesco Fornai[2] and Lorenzo Fumagalli[1]

[1] Dipartimento di Scienze Anatomiche, Istologiche, Medico-Legali e dell'Apparato Locomotore, La Sapienza University, Rome, Italy
[2] Dipartimento di Ricerca Traslazionale e delle Nuove Tecnologie in Medicina e Chirurgia, University of Pisa, Pisa, Italy

Autophagy is an intracellular degradation process that controls the quality of the cytoplasm by eliminating protein aggregates and damaged organelles. In addition to its vital homeostatic role, this degradation pathway is involved in various human disorders, including neurodegenerative diseases. Our previous data show that in hippocampal and cortical neurons the neurotoxic compound trimethyltin (TMT) activates the autophagic pathway (Fabrizi et al., 2012).

Recently we extended our analysis to astrocytes, the main population of glia of the central nervous system. As already observed in neurons, in astrocytes autophagy is rapidly induced after TMT administration. LC3-II which is a distinctive marker of autophagy rapidly appeared in TMT-treated astrocytes but then it accumulates indicating a precocious block of the autophagic pathway. The inhibition of autophagy by 3-methyladenine at the level of the autophagosome formation partially rescues astrocytes from TMT-induced cell death. Interestingly, an impairment of autophagy was also observed by other authors following intoxication with arsenic and could represent a common feature of different environmental toxins.

References

[1] Fabrizi et al. (2012) Role of autophagy inhibitors and inducers in modulating the toxicity of trimethyltin in neuronal cell cultures. J Neural Transmission 119: 1295-305.

Key words
Autophagy, glia, environmental neurotoxins, LC3.