Editorial

this Special Issue of Neurobiology of Disease involving LKE-mediated neuroprotection in models of neurodegenerative disorders is provided by Dr. Hensley and colleagues (Hensley et al., 2015). These scientists show that LKE stimulates autophagy in RG2 glioma and SH-SY5Y neuroblastoma cells as evidenced by increased lipidation of microtubule-associated protein 1 light chain 3 (LC3) both in the absence and presence of bafilomycin-A1. These results discriminate between effects on autophagic flux versus blockage of autophagy clearance. LKE treatment caused changes in protein level or phosphorylation state of multiple autophagy pathway proteins including mTOR, p70S6K, unc-51-like-kinase-1, beclin-1, and LC3 in a manner essentially identical to effects observed after rapamycin treatment, the latter known to inhibit mTOR.

Drs. Barone and Butterfield review the potential involvement of heme oxygenase-1 [HO-1] in T2DM and AD (Barone and Butterfield, 2015). HO-1 and it partner, biliverdin reductase [BVR-A], lead to neuroprotective and antioxidant and anti-nitrosative bilirubin (Maines, 2010; Mancuso and Barone, 2009). Both enzymes are modified in brains of subjects with AD and MCI (Barone et al., 2014) and may contribute to the elevated oxidative stress in both these disorders (Butterfield et al., 2001; Butterfield et al., 2013; Nunomura et al., 2001). This review in this Special Issue compares and contrasts the beneficial aspects of stimulating HO-1 activity on the one hand and the recently posited notion of the potential involvement of HO-1 in insulin resistance, a prominent feature of AD as noted above, on the other hand. Much more research needs to be undertaken to determine whether such a scenario is applicable when HO-1 is oxidatively dysfunctional.

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