Neurodegenerative diseases, which affect approximately 44 million of people worldwide, are characterized by the progressive degeneration of the structure and function of neuronal cells. The prevalence of neurodenerative diseases is increasing worldwide due to extensions in lifespan and increased number of risk factors. In response to the need for new therapeutic strategies targeting neurodegenerative conditions we propose to evaluate a new pharmacological approach for the management of neurodegeneration, based on the utilizing of β-cyclodextrins for the improvement of the protective effect of 1,2,4-triazole derivatives. Our previous studies showed that 4-alkyl-5-substituted-1,2,4-triazole-3-thione derivatives were not only strong anticonvulsants acting on the voltage-gated Na^+ channels, but some of them exhibited additional antioxidant and reactive oxygen species (ROS) scavenging activities. Numerous studies confirmed that compounds combining these two features (i.e., Na+ channel block, antioxidant/anti-ROS activity) are characterized by superior neuroprotective potential over the molecules that exhibit only one of them. In turn, cyclodextrins, apart from their application as drug carriers, exhibit also their own neuroprotective effect observed in cell and rodent models of Alzheimer's disease, hypoxia-ischemia, stroke, Niemann-Pick disease. Having all these facts in mind, we assume that β-cyclodextrin-1,2,4-triazole inclusive complexes will combine the neuroprotective properties of their components and, additionally, will be characterized by beneficial physicochemical features (e.g., better solubility/permeability). This interdisciplinary project will include preparation and comprehensive assessment of neuroprotective potential of β-cyclodextrin-1,2,4-triazole inclusive complexes using numerous *in-vitro* and *in-vivo* methods enhanced by analytical imaging techniques. The investigated complexes will be tested in cell model of excitotoxicity, Alzheimer's and Parkinson's diseases. Moreover, their effect on ROS formation, apoptosis and inflammatory cytokines level in neuronal cells will be examined. Finally, two animal models of neurodegeneration will be used to comprehensively assess their neuroprotective activity. It is expected that the results of these studies enable us to verify the hypothesis that cyclodextrin-based carriers complexed with 1,2,4-triazole derivatives can effectively protect neurons from degeneration, cell death and physiological dysfunctions.