S30-3 Neurotoxicity of trace elements and the pathogenesis of neurodegenerative diseases

◯Masahiro KAWAHARA[1], Yutaka SADAKANE[1], Hironari KOYAMA[1]

[1]University of Kyushu Health and Welfare

Both of the deficiency and the excess of trace elements can cause several neuronal disorders. In particular, the link between aluminum (Al) and Alzheimer's disease (AD) has been argued for decades, and zinc (Zn) has been suspected to have a causal relationship delayed neuronal death after transient global ischemia. We have investigated characteristics and mechanisms of the neurotoxicity of Al and Zn using Ca^{2+} imaging, immunohistochemistry, RT-PCR, etc. We have found that Al caused the several abnormal changes similar to Alzheimer's pathology. Both of Al and Zn causes apoptotic neuronal death. Our results indicate that dyshomeostasis of Ca^{2+} may involve in the mechanism of Al- or Zn- induced neuronal death. Meanwhile, Zn inhibited the abnormal Ca2+ dyshomeostasis induced by Alzheimer's beta-amyloid protein, which is believed to play essential roles in the pathogenesis of AD. Therefore, Zn may play neuroprotective roles in the normal condition and neurodegenerative roles in the pathogenetic condition such as ischemia. Our results indicate the significance of trace elements in the brain functions and suggest their implication of the pathogenesis of senile type dementia such as Alzheimer's disease and vascular dementia.