Redox Imbalance and Metabolic Defects in the Context of Brain Diseases

M. Perluigi[I]

[I]Sapienza University of Rome, Rome, Italy

Down Syndrome (DS) is the most common genetic disorder due to the abnormal triplication of chromosome 21 resulting in a variety of pathological phenotypes. Among these, individuals affected by DS show with ageing the accumulation of oxidative damage associated with defects of energy metabolism. DS is currently considered a human genetic model of early onset Alzheimer disease (AD). We hypothesize that redox dysregulation is closely linked to metabolic defects, including reduced glucose metabolism, energy production and aberrant insulin signaling. Further, loss of protein quality control, including proteasome and autophagy, contributed to the accumulation of oxidative damage and AD neuropathological hallmarks. The talk will discuss the role of trisomic genes which, directly and indirectly, contribute to the occurrence of an aberrant redox-phenotype and how it contributes to the impairment of several cellular functions ultimately resulting in Alzheimer-like neurodegeneration.