Copper dysfunction in Alzheimer’s disease (AD): Alleles in ATP7B gene and increased levels of non-ceruloplasmin-copper raise the risk for AD

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A large number of studies indicate a specific role of copper in aging and in Alzheimer’s disease mechanisms. Specifically, it has been proposed that the hypermetallation of the Abeta peptide can be at the basis of redox cycles of oxidative stress, toxicity, Abeta oligomer formation and precipitation. We have contributed to this topic demonstrating in living AD patients that an increase in the serum copper fraction that does not bind to ceruloplasmin (Non-Cp-Cu), correlates with the AD typical deficits, cerebrospinal fluid (CSF) markers, and with a worse prognosis for AD. Furthermore, we have shown that loci of susceptibility for AD lie in genes pertinent to copper metabolism, in particular in the ATP7B gene, which codes for the ATP-pump controlling copper excretion through the bile and ceruloplasmin biosynthesis. Moreover, we have demonstrated that copper and non-Cp-cu levels are higher in AD patients vs. healthy controls by means of meta-analyses. Non-Cp-Cu helps in properly classifying subjects with Mild Cognitive Impairment (MCI) from healthy ones and provides prognostic information about the conversion to full AD.

Biography

Rosanna Squitti has completed her PhD at the age of 27 years from Sapienza University, Rome, IT and postdoctoral studies from the University of Liverpool, UK. She is the Director of the Laboratory of Neurobiology, at the Department of Neuroscience, AFaR - Osp. Fatebenefratelli, Rome. She has an h-index of 21 (Scopus) and published more than 75 papers in reputed journals and serving as an editorial board member of repute. She has an International patent: Colabufo, Squitti, Method and Kit for Determination of Free Copper in Serum, E.P.O.E. (RO/EP), PCT/EP2012/072063, 2012, and an Italian Patent: Colabufo, Squitti, BI4556R/RCGE/rfv, RM2013A000253, 2013.

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