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### Bilirubin protection in Parkinson disease, the determinant role of Tnfa

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Following the increase in life expectancy, the prevalence of Parkinson's disease (PD) as the most common movement disorder is expected to rise. Despite the incredibly huge efforts in research, the current available treatments may only alleviate the symptoms, with none of them able to stop or slow down the disease evolution. Hence, disease-modifying treatment is still a paramount unmet medical need. Meanwhile, mildly elevated unconjugated bilirubin (UCB) levels, as seen in patients with Gilbert syndrome, have been shown to be protective against several extra CNS diseases, the effect being attributed to the well-known anti-oxidant capability of UCB, even higher than □-tocopherol. Because no data are so far available in the context of PD, we explored the neuroprotective effects of low concentrations of UCB in an ex vivo (organotypic brain cultures of substantia nigra - OBC-SN) model of the disease. We demonstrated that UCB fully protects dopaminergic neuron (DOPAn). Despite the modulation in redox sensors and glutathione in the PD model supported the alteration of the tissue redox balance, oxidative stress looks not to be the key player of DOPAn demise and UCB conferred protection. On the contrary, we strongly demonstrated the determinant role of TNF-α in DOPAn demise, and the normalizing effects of UCB in conferred protection. These findings might be of relevance in designing new therapeutic options.

#### **Biography**

Silvia Gazzin completed Degree in Biology, PhD in Molecular Medicine, one year visiting reseracher at the "blood-brain barrier unit" - INSERM, Lyon, France. Since 2004 involved in translational reserach (i.e. from the patient, through biomolecular laboratory approaches, back to the clinic) on the opposite roles of bilirubin on brain: toxic in severe neonatal hyperbilirubinemia, and protective in Gilbert. Since 2014 studying Parkinson disease. Since 2013 collaborating researches on juvenile MAFLD.