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Education:

- Postdoc. 1987-1989, Mount Sinai School Medicine of CUNY
- Ph.D. 1985, New York University
- Postdoc 1985-1986, Population Council @ Rockefeller University
- B.Sc.1970, M.Sc. 1972, University of Lisbon, Portugal

Research Interest:

- **Role of the ubiquitin/proteasome pathway and inflammation in NEURODEGENERATION**

The research in our laboratory investigates mechanisms involved in neuronal degeneration. We specifically focus on mechanisms affected by impairment of the ubiquitin/proteasome pathway (UPP) and inflammation, as it is well established that most neurodegenerative disorders are characterized by two related pathological hallmarks:

(1) the accumulation of ubiquitinated proteins in disease specific neuronal inclusions, and

(2) signs of chronic neuroinflammation, such as gliosis, at the sites of neuronal damage.

Due to the widespread development of these two pathological hallmarks, the outcome of our research is clearly relevant to the prevention and/or treatment of most neurodegenerative disorders, such as Alzheimer's, Parkinson's and Huntington's diseases as well as amyotrophic lateral sclerosis.

One of the major roles of the UPP is to degrade ubiquitinated proteins, thus the neuronal accumulation of the latter is indicative of UPP malfunction. Notably, aging is associated with a decrease in proteasome activity. This supports the view that aging-dependent proteasome impairment is critical to the late onset of both familial and sporadic forms of neurodegenerative disorders. Furthermore, other conditions such as inflammation may affect proteasome activity and exacerbate the neurodegenerative process. In order to develop therapeutic strategies based on proteasome impairment it is critical to tease out which of the proteasome-affected mechanisms are involved in neurodegeneration.

To investigate the mechanisms affected by proteasome impairment we are using proteasome inhibitors and proteasome mutations that impair proteasome activity. Furthermore, we are investigating the effect of a product of inflammation, prostaglandin J2 (PGJ2), shown by our laboratory to be neurotoxic and to induce the accumulation of ubiquitinated proteins.

Publications

- See **Publications by Category**