Title: **Prion-Like Neurodegenerative Diseases and Cancer: New Targets to Chemotherapy**

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Many neurodegenerative diseases have the common aspect of protein accumulation in the form of amyloid aggregates and fibrils. Key proteins involved in these diseases, such as Aβ, tau, α-synuclein, SOD1, and TDP43 can show a prion-like behavior. Recent studies have highlighted the participation of the prion protein in cancers and the prion-like behavior of proteins involved in cancer, which is the case of p53. p53 is a master regulatory protein that participates in cellular processes such as apoptosis, DNA repair and cell cycle control. The function of this tumor suppressor protein is lost in more than 50% of human cancers. Our studies have suggested that the formation of prion-like aggregates of mutant p53 is associated with loss-of-function, dominant-negative and gain-of-function (GoF) effects. Studies from our group have shown that p53 aggregation in a mixture of oligomers and fibrils that sequestrates the native protein into an inactive conformation, typical of a prionoid behavior. These aggregates are present in tissue biopsies of breast cancer especially in more aggressive ones. The prionoid properties of p53 aggregates are considered potential targets for drug development. (Supported by CNPq, FAPERJ, MS-DECIT, CAPES and FINEP).