Many neurodegeneratives diseases share the same hallmark, the accumulation of the aberrant proteines as intracellular inclusions. It was shown that the ubiquitin proteasome system (UPS) (Lowe and Al, 1988; Ciechanover and Brundin., 2003) plays an essential role in elimination of these abnormal proteines. The first indication of the implication of the UPS was demonstrated within patients suffering from the Alzheimer's disease. These patients present helicoid filaments per pairs (Mori and Ihara., 2003; Perry and Al, 1987). These markers were used as pathological signs of many diseases in humans (Alves-Rodrigues and Al, 1998). Several reports were published thereafter on the implication of the UPS in the neurodegeneratives diseases (Fergusson and Al, 1996).

The aberrant proteines are ubiquitinated and tranfered to the **26S Proteasome** complex This last is composed of a 20S core and at least one **19S** sub-complex, that is in charge for the recognition of the substrates marked with the molecules of ubiquitines. It vehicles thereafter substrates to the active sites of the proteasome (beta20S).

The present study is related to the immunoreactivities of the sub-units of 26S proteasome for the principal forms of tauo- and synucleinopathies. Several cerebral diseases were studied.

Many studies showed that the weakening of the function of the proteasome is associated with the cellular senescence. However, the data available are reduced in fragments and are contradictory (Bulteau, Petropoulos and coll, 2000; Reinheckel, Ultrich and coll, 2000; Keller and coll, 2000).

Six tauopathies were studied. All showed the immunoreaction of ATPase S6b in the hippocampus and the temporal cortex. For the Alzheimer and Down Syndrome patients, the pyramidal cells of the CA1 and CA3 of the hippocampus were the most positive zones. CA4, dentate gyrus and subiculum were less reactive with ATPase S6b. Of the two synucleinopathies studied (LBD and MSA), Lewy bodies were less immunoreactive as visualized in some brains of patients having Lewy Body Disease.

Our data suggest that the degree of weakening of the ubiquitin-proteasome system is much more dramatic in tauopathies (Keller and coll, 2000) than in the synucleinopathies.

The evidence accumulates more and more for a participation of the ubiquitin-proteasome system in the degradation of abnormal proteins in a variety of neurological disorders (Spillantini and Goedert, 1998; Fergusson and coll, 2000).