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Biomarker Detection for Neurodegenerative Diseases Using Streptomycin

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The prion protein structure contains a spherical core sequence with a generally highly negatively charged surface sequences and at least two positively charged pockets rich in lysine or arginine residues. Conformational changes in the cellular prion glycoprotein PrP^{c} leads to propagation of a beta-sheet conformed, detergent insoluble and Proteinase K (PK) resistant pathogenic protein, PrP^{sc}.

The accumulation of this PrP^{sc} in animals and man is associated with a wide range of Transmissible neurodegenerative Spongiform Encephalopathies (TSES), including scrapie in sheep, spongiform encephalopathy in cattle, chronic wasting disease in deer and Creuztfeldt-Jakob disease in humans (CJD). The recent discovery of a new variant of Creuztfeldt-Jakob disease transmissible by blood transfusion requires tests that allow detection of PrP^{sc} at very low concentrations in biological fluids.

Early laboratory diagnosis of diseases relies on the employed technique sensitivity for the detection of the corresponding disease biomarker. The use of streptomycin in the detection procedures of the pathogenic prion protein represents a new and attractive way to detect more PrP^{sc}, the best biomarker for the TSEs.

The addition of increasing quantities of streptomycin to constant amount of brain extracted and PK treated PrP^{sc}, followed by electrophoresis on 15% polyacrylamide gel, transfer and immunodetection revealed that the molecular mass increase for each of three PrP^{sc} bands was proportional to the quantity of the added streptomycin. When higher concentrations of streptomycin were used, it causes flocculation and aggregation of the PrP^{sc} which can then be separated by a low centrifugation step. Also the addition of increasing quantities of streptomycin to the Alzheimer peptide P53 revealed an increase of its molecular mass in the same manner as with PrP^{sc} [1,2].

The supposed mechanism of interactions between streptomycin and the prion proteins *in vitro* is suggesting occurring through hydrogen bound transfer between the 2 guanidine groups present on the streptomycin molecules and the amino acids of one or several prion proteins thus forming multimolecular aggregates. Analysis of molecules sharing common chemical functions with streptomycin and reproduced aggregation and precipitation of the PrP^{sc} ruled out the possibility of a shift-base reaction. Examples of such functional equivalence was shown also for Dihydro streptomycin, bis-3-aminopropylamine, guanidine hydrochloride and spermine tetrahydrochloride.

References

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