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Editorial – Short Notes

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Prion diseases developed usually after conformational changes of a native cellular protein resulting in its transformation to a pathogenic amyloid protein which accumulated intracellularly provoking cellular degeneration and death. The cellular prion protein (PrPc) and the pathogenic prion protein (PrPsc) possess the same amino acid composition, but vary in conformity. Therefore, infectivity gains and resistance to proteinase K are a consequence of conformational modification of PrPc by PrPsc. PrPc contain about 40% alfa-helix and less than 10% beta-sheet conformation where PrPsc contain about 50% as a beta-sheet [1].

Guanidine alone and guanidine containing molecules as streptomycin have been shown to interact with the prion protein leading to their aggregation though a hydrogen atom exchange between the guanidine group and the negatively charged amino acids on the prion peptide [2]. Also the infectivity of the PrPsc can be inactivated by adding guanidine alone [3] or streptomycin [4] without affecting the proteinase K resistance of the PrPsc indicating that infectivity and proteinase K resistance are dissociated. As streptomycin the guanidine-containing compounds based on a benzene organizing platform and displaying between 2 and 5 arms have shown the higher anti-bacterial activity against tuberculosis [5].

Inhibition of the proteinase K (PK) proteolytic activity was observed after addition of increasing volumes of different Calix[n] arene to either negative or positive mad cow disease brain suspensions before the addition of proteinase K [6]. After electrophoresis and immune staining, a profile similar to the non-proteinase K digested prion protein was observed even at higher PK doses indicating that the interaction of calix-arene with the positively charged amino acids lysine and arginine present on the prion protein render the negative and pathogenic prion proteins completely resistant to the proteolytic activity of the proteinase K. Therefore, it is recommended to do PK digestion before adding the Calix-arene.

Appling a unilateral electroconvulsive therapy (ECT) to the brain of an 82 elderly positively diagnosed Alzheimer disease woman had resulted in a complete clearance of the accumulated beta amyloid protein in that brain side compared to the non-treated brain region [7]. This result suggests that ECT might interfere with deposition *in vivo*.

Conclusion

The interaction of streptomycin with the negatively charged amino acids results in inactivation of the pathogenic prion infectivity and the interaction of Calix-arene with the positively charged amino acids on the prion peptide results in inactivation of proteolytic activity of the added proteinase K.

References

1. Prusiner SB (1982) Novel Proteinaceous Infectious Particles Cause Scrapie. Science 216: 136-144.
2. Moussa A, Coleman AW, Bencsik A, Leclere E, Perret F, et al. (2006) Use of streptomycin for precipitation and detection of proteinase K resistant prion protein (PrPsc) in biological samples. Chem Commun (Camb) 9: 973-975.
3. Gao JM, Zhou XB, Xiao XL, Zhang J, Chen L, et al. (2006) Influence of guanidine on proteinase K resistance in vitro and infectivity of scrapie prion Protein PrP(sc). Acta Virol 50: 25-32.
4. Moussa A (2014) Reduced accumulation of the prion protein (PrPsc) in mice spleen post inoculation with a mixture of the scrapie C506M3 strain and streptomycin. J Chromatograph Separat Techniq 5: 214-215.
5. Massima-Dibama H, Mourer M, Constant P, Daffé M, Regnouf-de-Vains JB (2015) Guanidinium compounds with sub-micromolar activities against Mycobacterium tuberculosis. Synthesis,characterization and biological evaluations. Bioorganic & Medicinal Chemistry 23: 5410-5418.
6. Coleman AW, Perret F, Cecillon S, Moussa A, Martin A, et al. (2007) Enhanced detection of the pathogenic prion protein by its supramolecular association with para-sulfonato-calix[n]arene derivatives. New J Chem 31: 711-717.
7. Vandenbulcke M, Bouckaert F, De Winter FL, Koole K, Adamczuk K, et al. (2015) Correspondence; Asymmetric Amyloid Deposition in the Brain Following Unilateral Electroconvulsive Therapy. J biopsych 09.016: e1-e3.

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