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Editorial Open Access

Clinical Chemistry

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Editorial

Clinical chemistry originated in the late 19th century for analyzing body fluids using simple chemical tests. A great progress was achieved later in the area of clinical pathology; with the development of another laboratory and biochemistry techniques such as chromatography, spectrophotometry, electrophoresis, immunoassay, molecular biology, proteomics and other separation techniques; and their use in fundamental and applied research. Today the obtained information with the application of these modern techniques progressively helped in resolving the recently developed medical life problems.

During this century, it was possible to explain the increasing number of diseases (Alzheimer and Parkinson diseases) where deposition of misfolded insoluble protein aggregates within cells of the central nervous system renders the protein dysfunctional.

Recently, the appearance in 1986 in great Brittan of the mad cow disease epidemic followed by the new variant of Creuztfeldt-Jacob disease in man was due to ingestion of infected cattle meet or by blood transfusion. These disorders are due to an infectious agent called prion protein (PrP^{sc}) originates through conformational changes of the normal cellular prion protein (PrP^{c}).

Adding streptomycin to proteinase K treated PrP^{sc} showed an increase of the molecular mass of the 3 bands proportional to the quantity to the added streptomycin. It induced flocculation and aggregation of the PrP^{sc} which can easily separate by low centrifugation step. The mechanism of interaction occurred through hydrogen bond transfer between the 2 guanidine groups present on streptomycin and the amino-acids of one or several prion proteins ruled the possibility of a schiff-base reaction.

The inoculation intraperitonially of a mixture of streptomycin and infected mouse brain homogenate of the scrapie strain C506M3 into C57BL6 mice showed 45 days post inoculation a decrease to even absence of accumulated prion infectivity marker PrP^{sc} in the mice spleen in comparison to controls. This result can be explained by changes of the surface electric charges due to the hydrogen bond transfer between the two guanidine groups present on streptomycin and the different amino acids on the PrP^{sc} peptides and thus probably affecting the stability and induced a drop in the PrP^{sc} infectivity. These results probably show that proteinase K resistance of the PrP^{sc} is not associated with infectivity.