ISSN: 2574-1241
DOI: 10.26717/BJSTR.2019.13.002464
Afonso Carlos Neves. Biomed J Sci & Tech Res

BIOMEDICAL Journal of Scientific & Technical Research

Opinion Open Access

Prion Paradigm: Understanding Neurodegenerative Disorders

Afonso Carlos Neves*

Neurology Department, Federal University of Sao Paulo, Brazil

Received: January 23, 2019; **Published:** January 31, 2019

***Corresponding author:** Afonso Carlos Neves, Neurology Department, Federal University of Sao Paulo, Brazil

Opinion

The concept of a protein that could work as an "infecting particle" was a revolutionary idea in Biology and Medicine and, as other paradigms when they arise, took time to be accepted by the scientific community . The international economic impact of Bovine Spongiform Encephalopathy and its linkage to a human epidemy, crossing the barrier among mammal species, drove special attention to the prion hypothesis. The "variant of Creutzfeldt Jakob Disease" (vCJD) characterized this new and mortal epidemy. In those circumstances, in 1997 Stanley B. Prusiner received the Nobel Prize for all his work that culminated with the proposition of the "prion", a name that he gave to the "infecting protein". Creutzfeldt Jakob Disease (CJD) was first attributed to a kind of "slow virus", a concept introduced by Sigurdsson in 1954 to some pathologic conditions with long latency period, also with progressive and fatal clinical feature [1].

The demonstration of the existence of that slow virus was pursued by several researchers, but without conclusive result . After his own experience, when he did medical residence, caring for a CJD patient, Prusiner started to study and research about the etiology of CJD. Opened to new findings that he was discovering, he needed to perseverate and struggle for his new ways to understand a revolutionary explanation for that etiopathogenic mechanism. Could be, the replication of a protein like a virus, a daring idea against a "biologic dogma"? The occurrence of the vCJD and the strong association with BSE confirmed the prion hypothesis consecrated by the Nobel Prize to Prusiner and a new scientific paradigm was established. So, the idea of contagion and infectious diseases were amplified beyond germs. After that, the continuing research about the prion mechanism in other diseases opened for the discovery of different forms of prion protein and its equivalent clinical manifestations. Under these conditions, we have now etiologic correlation of each specific protein complex to each neurodegenerative disease [2-4].

Even some other conditions have this hypothesis as, for example, type 2 diabetes. The transformation of a normal protein in a pathologic one can occur by genetic factors, sporadic mutation, or transmission . We need to think and teach more frequently that such diseases have a prion etiopathogenic mechanism, as Parkinson, or Huntington disease, for example. We have to engage in the new paradigm. One special aspect of all those conditions is Alzheimer disease. With the aging of the population, some degenerative disorders, as that one, became more frequent. In the United States, patients with Alzheimer, more than 65 years old, until the year 2050, are going from 5.2 million to 14 million. Each year, 500.000 new cases are expected. 200 billion dollars are spent annually to care for those condition in USA, including the caretakers.

About the treatment of Alzheimer, from 2002 to 2012, 413 clinical assays were performed with a tax of failure of 99,6%. The only success approved by the FDA was memantine and MDMA, that provide symptomatic treatment to moderate and severe cases. It is not yet established that Alzheimer could be transmissible, even with some suggestable experimental data with animals. So, we think that nowadays it is a moment to research about factors that could retard, or even stop the biological mechanisms that transform a normal prion protein in a pathologic one. We should know what biological factors could precipitate that transformation. But, at the same time, we cannot stop to care those people and advice to stimulate the brain with physical and mental exercises, together with a positive thinking under welfare conditions. We don't know yet if all these activities can influence that protein transformation. Those biological and complementary medical practices can integrate a new horizon for research on neurodegenerative disorders, specially Alzheimer disease.

References

1. Prusiner SB (2014) Madness and Memory-The Discovery of Prions-A New Biological Principle of Disease. Yale University Press, pp. 344.

2. Prusiner SB (1998) Prions. Proc Natl Acad Sci USA 95(23): 13363-13383.

3. DePaola D, Duarte F, Madi K (1990) Manual de Infecção Viral-Apresentação de Modelos Clínicos e Experimentais. Editora Médica e Científica Ltda.

4. Prusiner SB (2017) Prion Diseases. Cold Spring Harbor Laboratory Press.

ISSN: 2574-1241

DOI: 10.26717/BJSTR.2019.13.002464

Afonso Carlos Neves. Biomed J Sci & Tech Res