***Field:* Chemistry/ Biochemistry**

Planning Group Members:
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Session Topic:
Chemistry of Neuro-Disorders: Their Mechanism, Diagnostics and Drug Development

The appearance of bovine spongiform encephalopathy (BSE) in the mid 80´s in Great Britain and the subsequent discovery of a new variant of Creutzfeld-Jacob (vCJD) disease in human prompted a lot of research on the biology of this group of diseases. These efforts eventually resulted in a revision of the paradigm that genetic material is required for the transmission of diseases. BSE and vCJD can be transmitted by a pure protein: the prion protein, which is a normal component of cells, can change the way how it is folded. As a result, aggregates (plaques) are formed in the brain, which eventually cause the fatal destruction of the brain structure. The misfolded protein can also initiate a chain reaction, which promotes misfolding of natural prion proteins. Thus, the disease is transmitted from one organism to the next one by the misfolded prion protein. However, many basic features of prion diseases are still unclear, particularly concerning the molecular mechanism of disease transmission and development. Therefore, the first two presentations of the session will report about latest findings on the molecular mechanism of the development and transmission of prion diseases, their therapy and diagnostics.

On the other hand, changes in protein folding seem to be a more common molecular mechanism leading also to other well known brain disorders such as Alzheimer´s disease. The misfolding of proteins results in cluster of proteins, so-called amyloid, forming microscopic fibril or plaques, which deposit in internal organs and interfere with normal function, sometimes lethally. In the case of Alzheimer's, these fibrils kill nerve cells in areas of the brain that are crucial for memory. However, in Alzheimer's disease, the cause of misfolding is not so obvious. A number of mutations are associated with rare forms of familial Alzheimer's, but not with most common cases (about 95 percent of the cases). Thus, it is critical to increase our understanding of mechanism of amyloid formation in order to devise therapeutic agents and cure the diseases. The second part of this session, two presentations will be given by two young scientists who have been making effort to understand the biochemical mechanism of Alzheimer´s disease.