

Probing domain-swapping of prion protein

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Transmissible spongiform encephalopathies, including bovine spongiform encephalopathy and human Creutzfeldt-Jakob disease, are fatal neurodegenerative disease. The protein-only hypothesis¹ holds that prion proteins are the infectious agents of transmissible spongiform encephalopathies. Two molecular forms of the prion protein, the infectious form (PrP^{Sc}) and the normal cellular form (PrP^C), are believed to differ only by their conformation. PrP^{Sc} has been found to differ from PrP^C by infectivity, an increased β -sheet content, an increased resistance to proteinase K and an oligomeric state rather than a monomeric state.

In the present study, we deal with prion's structure whether if prion-engaged fibers are domain swapped oligomer^{2,3}. By adopting several recently reported methods by which is PrP^C refolded into PrP^{Sc}-like form, we wanted to see if they are domain swapped. Some gel electrophoresis methods and electron paramagnetic resonance spectroscopy⁴ and fluorescence spectroscopy shows that some of these fibers are domain swapped oligomers.

References

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