

The protein misfolding “mad cow disease” is still plaguing scientists

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“Mad cow disease” (Bovine Spongiform Encephalopathy) belongs to a contagious type of transmissible spongiform encephalopathy (TSE). Scientists believe it is caused by prions (the misfolding prion proteins) but they may have not yet solved the riddle of “mad cow disease”. This is due to a prion that is neither a virus, nor a bacteria nor any microorganism. So the disease cannot be caused by the vigilance of the organism immune system and it can freely spread from one species to another species. The humans exist the susceptibility of TSEs. For example, the human version of “mad cow disease” named Creutzfeldt-Jakob Disease (CJD) and variant CJD (vCJD) just happen randomly through infections of transplanted tissue or blood transfusions or consumption of infected beef products. Cats, mink, deer, elk, sheep and many animals are also susceptible to TSEs. However, rabbits, horses and dogs seem unaffected by prions. Scientists do not know the reason.

The prion protein is a naturally occurring protein *in vivo*. Its lesions in brain are not caused by the vigilance of the immune system. Recent studies have found that the lesions led astray as long as by contact with other normal prion proteins. The cells are arranged in accordance with the instruction of the gene and formed into proteins with different shapes and functions. But, like cardboard boxes, proteins need to be properly “folded” in order to ensure their normal work. When proteins are folded into the wrong shape, they do not work. Under normal circumstances, the cells will supervise these misfolded proteins and automatically decompose them. However, the supervision mechanism is not with 100% insurance. Scientists found that the rate of decomposition of Prions is not quick enough and these Prions accumulate and change the cellular metabolism and eventually kill the cell. This leads to the death of neurons in the brain. The dead neurons decompose and release more prion proteins into the biological mechanism to cause prion diseases. We have studied the protein structures and structural dynamics of human, mouse, deer, rabbit, horse and dog prion proteins to reveal the secret of the protein misfolding. Some drug targets for treating “mad cow” diseases are tried to seek.

Biography

Jiapu Zhang completed his Ph.D. degree from The University of Melbourne and The University of Ballarat, one Masters (in research) degree from The National University of Singapore, and one Masters (in research) degree and one Bachelor degree from China in 2004, 2000, 1996 and 1993 respectively. During 1996 to 1998, he lectured in Shandong University. Since 2005, he has worked in Australia's CSIRO (Commonwealth Scientific and Industrial Research Organisation), Ballarat University, Melbourne University, and other Australian universities as a research and teaching staff.

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