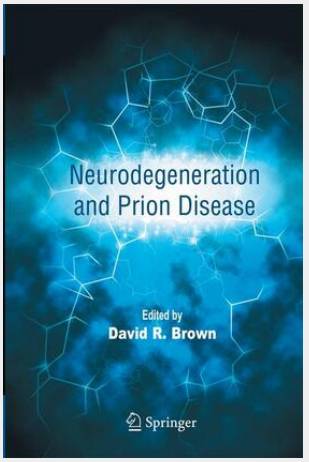
Brown

Neurodegeneration and Prion Disease



This is the first and only book on the subject of prions to cover the cause of cell death in the disease. It covers the full range of competing theories on the subject, from broad description and basic points up to the final details of the basic science.

David R. Brown Department of Biology and Biochemistry, University of Bath, Bath BA2 7AY, UK In 1982 Stanley Prusiner and colleagues puri?ed an abnormal protein from the brains of mice experimentally infected with a rare sheep dis- 1 ease called scrapie. This protein was called the prion protein. Earlier work had suggested that this diseases and others, loosely collected - gether as transmissible spongiform encephalopathies (TSEs), were not transmitted by conventional infectious agents. Prusiner suggested that 2 this new protein was the infectious agent in these diseases. Such a contentious suggestion lead to ferocious debate. Many researchers still maintained that there was no such thing as an infectious protein. - spite this, by 1990 most people accepted that the cause of the TSEs was the abnormal isoform of the prion protein his research group had id- ti?ed. The most convincing evidence for this had come from the work of Charles Weissmann, whose prion protein knockout mice could not be infected because they lacked expression of the protein that was now 3,4 forever linked to these disease. Since then it has become more widely accepted for these diseases to be termed prion diseases. In 1997 when 5 Stanley Prusiner won the Nobel Prize for his work on prion diseases. Even then, there was still an element of resistance in the scienti?c c- munity. It was considered that, in order the transmissible agent to truly be a protein only, the protein would have to be generated from a rec- binant source.

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