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Protein research could help in hunt for Alzheimer's and Parkinson's cures

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Research carried out at the University of Kent has the potential to influence the future search for treatment of neurodegenerative diseases that are linked to a family of protein molecules known as 'amyloid'.

The findings by a team of scientists led by Dr Wei-Feng Xue in the School of Biosciences could lead to a better understanding of the diseases, and suggest potential diagnostics and therapeutics strategies to combat amyloid-associated disease progression and their possible infectivity.

Currently, there is a gap in the knowledge of the factors that govern the infectious potential of amyloid in general. In an article published in the journal *eLife*, Dr Xue's team report on their investigations into why some forms of amyloid are highly infectious - the so-called prion form associated with BSE (Mad Cow Disease) and the human form, CJD (Creutzfeldt Jakob Disease) - while others are less infectious or even inert.

They discovered that the infectious potential of an amyloid is a complex biological property better described on a sliding scale rather than either one category (transmissible prions) or another (non-transmissible amyloid), as it is now.

Currently most amyloids apart from prions are viewed as non-transmissible between individuals meaning people would be unable to 'catch' Alzheimer's or Parkinson's simply by association. This research

sheds new light on why some amyloid forms can potentially spread between cells and tissues within the same individual, and some are considered prions that are transmissible from one individual to another.

The physical dimensions of amyloid aggregates control their infective potential as prion particles by Ricardo Marchante, David M. Beal, Nadejda Koloteva-Levine, Tracey J. Purton, Mick F. Tuite and Wei-Feng Xue is published in the journal *eLife*.

More information: *eLife*, DOI: 10.7554/eLife.27109.001