He Jianwei, J Proteomics Bioinform 2017, 10:8(Suppl)
DOI: 10.4172/0974-276X-C1-0100

Structural insights into the mechanism of how polyphenols suppress amyloid fibrillation

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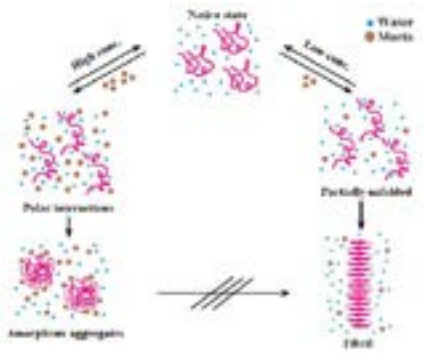
Polyphenols, especially natural flavanols, have received considerable public attention in China due to the positive association between food and traditional herbal consumption and beneficial health effects. Flavonoids has been demonstrated to be active inhibitors of fibrillation by amyloidogenic protein. We recently reported the inhibitory activity of Myricetin against HEWL fibril formation, in which Myricetin exhibited a stronger inhibition than the well-characterized polyphenol Quercetin. In contrast to our previous studies using other polyphenols, we find the generation of irregular structural aggregates formed by the binding of Morin to HEWL, which support a novel and distinctive model for how this small molecule inhibits amyloid formation. Moreover, we also demonstrated that EGCG was a potent inhibitor of amyloidogenic cystatin amyloid fibril formation *in vitro*. Through combining experimental and computational data, we could propose a mechanism by which EGCG inhibited the fibrillation of cystatin: EGCG appears to be a generic inhibitor of amyloid-fibril formation, although the mechanism by which it achieves such inhibition may be specific to the target fibril-forming polypeptide. In conclusion, our findings implicate the importance of diet and drink habits as playing a key role in guarding against amyloid fibril formation and promoting healthy aging.



Biography

He Jianwei is a Professor at School of Life Science, Liaoning University, China, and has received MS degree in Biochemistry in 2002, from Yamaguchi University, Japan. He completed his PhD in Bio resource in 2005 at Tottori University, Japan. His research interests include: 1) Using molecular dynamics and biochemical methods to study protein oligomerization progress and the importance of dimers and tetramers in the aetiology of amyloidotic diseases. 2) Mining, screening or designing of novel inhibitors of natural resources against protein misfolding and amyloid aggregation.

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