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Aβ-oligomer targeted therapy of Alzheimer's disease

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Alzheimer's disease (AD) is a progressive neurodegenerative disorder. Several lines of evidence suggest a central role of amyloid-β-peptide (Aβ) in the pathogenesis of AD. More than Aβ fibrils, small soluble Aβ oligomers are suspected to be the major toxic species responsible for disease development and progression. In any case, agents that interfere with oligomer and fibril formation may be valuable for therapy or prevention of AD. The present study reports on in vitro and in vivo properties of the D-enantiomeric amino acid peptide "D3". We show that next to plaque load and inflammation reduction, oral application of the peptide improved cognitive performance of AD transgenic mice. In addition, in vitro data elucidating the potential mechanism underlying the observed in vivo activity of D3 are provided. These data suggest that D3 precipitates toxic Aβ species and converts them into non-amyloidogenic, non-fibrilar and non-toxic aggregates without increasing the concentration of monomeric Aβ. Thus, D3 is a therapeutically active substance exerting an interesting and novel mechanism of action leading to even more potent substances, which will be presented as well.

Biography

Dieter Willbold has studied Biochemistry at the Universities of Tübingen (Germany), Bayreuth (Germany) and Boulder (Colorado, USA). He has completed his PhD at the University of Bayreuth and carried out postdoctoral studies in Bayreuth and Tel-Aviv. In 1998, he was heading his first own research group at the FLI in Jena. In 2001, he was appointed associate professor at the Heinrich-Heine-University in Düsseldorf, where he became full professor and director of the Institute of Physical Biology in 2004. In parallel and since 2005, he is director of the Institute of Complex Systems (ICS-6: Structural Biochemistry) at the Research Centre Jülich.

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