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Alzheimer Award

The Associate Editors of JAD selected the best article from Volume 5 as the recipient of the Alzheimer Medal. The 2004 winner of the Alzheimer Award, sponsored by Elan Pharmaceuticals, was Lester I. Binder for his article "Conformational changes and truncation of tau protein during tangle evolution in Alzheimer's disease" (*J Alzheimers Dis* **5**, 65–77, 2003).

2004 Awardee
Lester I. Binder, Ph.D.

Lester I. Binder received his Ph.D. from Yale University in Biology in 1978 where he worked on microtubule polarity demonstrating that these organelles added subunits from one end more readily than the other. This work represented some of the first papers on microtubule polarity. As a postdoctoral fellow at the University of Virginia, he turned his attention to the neuronal cytoskeleton, publishing the first papers on the subcellular localization of the microtubule-associated protein tau using the first monoclonal antibodies to tau (Tau-1, Tau-2, and Tau-5). This work led to independent collaborations with the Iqbal and Wood laboratories; these studies established Alzheimer disease neurofibrillary tangles as inclusions containing abnormally phosphorylated tau. Since that time, work from the Binder laboratory has focused on the formation of tau filaments, in vitro and neurofibrillary tangle "evolution" in situ during the course of Alzheimer disease.

Importance of published article

The work presented in the paper "Conformational changes and truncation of tau protein during tangle evolution in Alzheimer's disease" (JAD 5:65–77, 2003) was performed by a postdoctoral fellow in Dr. Binder's laboratory, Francisco Garcia-Sierra. Its main findings document seemingly linear alterations in NFTs that can be identified by two antibodies to different folded states of tau: Alz50 and Tau-66. The paper presents evidence indicating that the NFTs reactive with Alz50 predate Tau-66-postive tangles. These and other data strongly suggest a refolding of the tau molecule while in the polymeric inclusions during the course of Alzheimer disease that appear associated with both amino and carboxy truncation of tau.

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