Title:	Self-Assembly of Ovalbumin Amyloid Pores: Effects on Membrane Permeabilization, Dipole Potential, and Bilayer Fluidity
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Abstract:	Amyloid assembly is inherently a stochastic and a hierarchical process comprising the genesis of heterogeneous, transiently populated prefibrillar aggregates that are characterized to be non-native oligomeric conformers. These oligomers could be either off-pathway or on-pathway species en route to amyloid fibrils that are associated with a variety of neurodegenerative disorders, namely, Alzheimer's disease, Parkinson's disease, and prion disease, as well as in localized and systemic amyloidoses (type II diabetes and dialysis related, respectively). Morphological characterizations of these prefibrillar aggregates indicated that apparently the doughnut or annular structure is commonly shared among various prefibrillar species irrespective of the diverse native structures and aggregation mechanisms. In this work, we have elucidated the self-assembly mechanism of amyloid pore formation from ovalbumin using a range of biophysical techniques that shed light on the time-dependent protein structural changes as aggregation progressed. Additionally, on the basis of several pieces of evidence suggesting amyloid pore-mediated cytotoxicity, we have investigated the annular amyloid–membrane interaction using a comprehensive biophysical approach. The influences of annular pores on the intramembrane dipole potential and bilayer fluidity, as a consequence of membrane permeabilization, were examined in a protein concentration- and time-dependent manner that provided important insights into the pore–membrane interactions. Instantaneous membrane permeabilization kinetics suggested that plausibly a detergent-like carpet mechanism during membrane disruption was effective. Moreover, it was inferred that a loss of membrane integrity resulted in the generation of both disordered lipid and disoriented water dipoles that reside in the immediate vicinity of the membrane bilayer. These key findings may have implications in amyloid-pore-induced deleterious effects during amyloid–membrane interactions.
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