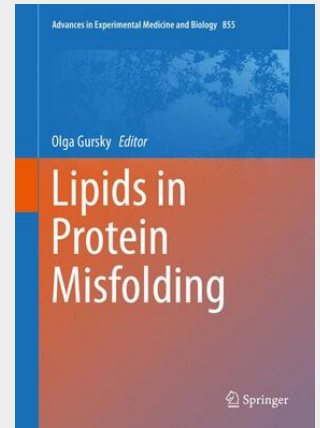


Lipids in Protein Misfolding

Protein conversion from a water-soluble native conformation to the insoluble aggregates and fibrils, which can deposit in amyloid plaques, underlies more than 20 human diseases, representing a major public health problem and a scientific challenge. Such a conversion is called protein misfolding. Protein misfolding can also involve errors in the topology of the folded proteins and their assembly in lipid membranes. Lipids are found in nearly all amyloid deposits in vivo, and can critically influence protein misfolding in vitro and in vivo in many different ways. This book focuses on recent advances in our understanding of the role of lipids in modulating the misfolding of various proteins. The main emphasis is on the basic biophysical studies that address molecular basis of protein misfolding and amyloid formation, and the role of lipids in this complex process.

This book addresses molecular mechanisms of protein misfolding and the role of lipids and related molecules in these complex processes. The focus is on the biophysical and structural studies of proteins that are involved in major human disorders such as Alzheimer's disease, systemic amyloidoses, diabetes II, inflammation and atherosclerosis. Misfolding often results from protein mutations or modifications. Misfolding of membrane proteins can cause topological changes that target the proteins for degradation. Misfolding of soluble globular proteins and peptides converts them into β -sheet-rich aggregates and amyloid fibrils. This process can disrupt the structural integrity of the lipid membranes and thereby contribute to amyloid toxicity. In turn, lipids and lipid-associated molecules such as apolipoproteins and heparan sulfate proteoglycans, which are ubiquitous constituents of amyloid plaques, can influence protein misfolding via diverse mechanisms that are addressed in this book. The book features chapters describing the role of lipids in the misfolding of a wide range of proteins, including small peptides, globular proteins, lipid surface-binding proteins, and integral membrane proteins. The role of individual lipid molecules, lipid surfaces, and the membrane field is addressed, including specific and non-specific interactions with protein oligomers and mature fibrils. Distinct effects of various lipids on the nucleation and growth of amyloid fibrils are discussed. Modern computational approaches to the analysis of amyloid formation are addressed. The book should be useful to experts in the field but is also accessible to novices.



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