

1. Record Nr.	UNINA9910131028903321
Titolo	Lipids and cellular membranes in amyloid diseases [[electronic resource] /] / edited by Raz Jelinek
Pubbl/distr/stampa	Weinheim, : Wiley-VCH, 2011
ISBN	3-527-63433-9 1-283-14073-X 9786613140739 3-527-63434-7 3-527-63432-0
Descrizione fisica	1 online resource (298 p.)
Altri autori (Persone)	JelinekRaz
Disciplina	616.3995
Soggetti	Amyloid Amyloidosis Proteins - Metabolism - Disorders Electronic books.
Lingua di pubblicazione	Inglese
Formato	Materiale a stampa
Livello bibliografico	Monografia
Note generali	Description based upon print version of record.
Nota di bibliografia	Includes bibliographical references and index.
Nota di contenuto	Lipids and Cellular Membranes in Amyloid Diseases; Contents; Preface; List of Contributors; 1 Interactions of a-Synuclein with Lipids and Artificial Membranes Monitored by ESIPT Probes; 1.1 Introduction to Parkinson's Disease and a-Synuclein; 1.2 Structural Biology of a-Synuclein; 1.3 Methods for Studying AS-Lipid Interactions; 1.4 AS-Lipid Interactions; 1.5 Interactions of Monomeric AS with Artificial Membranes Monitored with ESIPT Probes; 1.5.1 Influence of Membrane Charge; 1.5.2 Influence of Membrane Curvature; 1.5.3 Influence of Membrane Phase; 1.5.4 Influence of Acyl Chains 1.5.5 Influence of Cholesterol 1.5.6 Binding Kinetics; 1.6 Aggregation of AS and the Effects of Fatty Acids Monitored with ESIPT Probes; 1.7 Concluding Remarks; References; 2 Structural and Functional Insights into a-Synuclein-Lipid Interactions; 2.1 Introduction; 2.2 Interaction of a-Synuclein with Model Membrane Systems; 2.2.1 Binding of a-Synuclein Species to Giant Unilamellar Vesicles; 2.2.2 Model Membrane Permeabilization by a-Synuclein Oligomers; 2.2.3 Structural Features of

a-Synuclein Oligomers; 2.3 Biological Significance; 2.3.1 Interaction Sites; 2.3.2 Membrane Penetration
References
3 Surfactants and Alcohols as Inducers of Protein Amyloid: Aggregation Chaperones or Membrane Simulators?; 3.1 Introduction; 3.2 Aggregation in the Presence of Surfactants; 3.2.1 General Aspects of Protein-Surfactant Interactions; 3.2.2 Effect of Surfactants on Protein Structure; 3.2.3 Stoichiometry of SDS Binding; 3.2.4 Aggregation of Proteins by SDS; 3.2.4.1 A β ; 3.2.4.2 β 2-Microglobulin and β 2-Glycoprotein I; 3.2.4.3 Tau Protein; 3.2.4.4 Prion Protein; 3.2.4.5 Acyl CoA Binding Protein (ACBP); 3.2.4.6 a-Synuclein (aSN)
3.3 Palimpsests of Future Functions: Cytotoxic Protein-Lipid Complexes
3.4 Aggregation in Fluorinated Organic Solvents; 3.4.1 Protein Examples; 3.4.1.1 Acyl Phosphatase; 3.4.1.2 β 2-Microglobulin; 3.4.1.3 a-Chymotrypsin; 3.4.1.4 Alteration of Fibril Structure by TFE; 3.4.1.5 Other Proteins; 3.5 From Mimetics to the Real Thing: Aggregation on Lipids; 3.5.1 Binding Surfaces and High Local Concentrations; 3.5.2 Conformational Changes Associated with Binding; 3.5.3 Chemical Variability of the Lipid Environment; 3.6 Summary; References
4 Interaction of hIAPP and Its Precursors with Model and Biological Membranes
4.1 Introduction; 4.2 Results; 4.2.1 The Conformations of Native proIAPP and hIAPP in Bulk Solution; 4.2.2 Fibrillation Kinetics and Conformational Changes of hIAPP and proIAPP in the Presence of Anionic Lipid Bilayers; 4.2.3 Effect of the Membrane-Mimicking Anionic Surfactant SDS on the Amyloidogenic Propensity of hIAPP and proIAPP; 4.2.4 hIAPP and proIAPP Aggregation and Fibrillation at Neutral Lipid Bilayers and Heterogeneous Model Raft Mixtures; 4.2.5 Comparison with Insulin-Membrane Interaction Studies
4.2.6 Cytotoxicity of hIAPP

Sommario/riassunto

Addressing one of the biggest riddles in current molecular cell biology, this ground-breaking monograph builds the case for the crucial involvement of lipids and membranes in the formation of amyloid deposits. Tying together recent knowledge from in vitro and in vivo studies, and built on a sound biophysical and biochemical foundation, this overview brings the reader up to date with current models of the interplay between membranes and amyloid formation. Required reading for any researcher interested in amyloid formation and amyloid toxicity, and possible avenues for the prevention or treatment.
