Parkinson's protein α-synuclein : membrane interactions and fibril structure

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Stellingen

behorende bij het proefschrift

Parkinson's Protein α-Synuclein: Membrane Interactions and Fibril Structure

1. The conical shape of Cardiolipin and not its charge is the decisive factor for α-Synuclein (αS) binding to the inner mitochondrial membrane. [Chapter 2]

2. Neither a model membrane with a charge density ρ = 1 nor one with a low charge density (ρ ≤ 0.3) is suitable to investigate the effect of phosphorylation on αS-membrane binding. [Chapter 3]

3. The biggest obstacle to study the intrinsic fold of αS in fibrils is the polymorphism of the fibrils. [Chapter 4 and 5]

4. A single technique is not sufficient to obtain a realistic picture of the fibril-fold of αS. [chapter 5]

5. The abundant occurrence in Lewy bodies of αS phosphorylated at position S129 may be a result of phosphorylation of aggregated αS at position S129 rather than phosphorylation of monomeric αS. [Oueslati A.J. Parkinsons Dis. 2016;6:39-51, Paleologou K. et al. J. Neurochem. 2010;30:3184-3198]

6. The approach using lipid nanodiscs to study membrane-protein interactions described by Schuler et al. can be applied to investigate membrane fusion in vitro. [Schuler M.A. et al. Methods Mol. Biol. 2013;974:415-433]

7. The sulfhydryl modification of the styrene-maleic acid copolymer (SMA-SH) described by Lindhoud et al. makes SMA-SH a potential target for spin labelling, which can be

used in the biophysical characterization of membrane proteins by EPR. [Lindhoud S. et al. Biomacromolecules 2016;17:1516-1522]

8. The O-GlcNAcylation (addition of a single monosaccharide N-acetyl-glucosamine) of αS surprisingly inhibits the phosphorylation at position S129 but not at position S87. [Marotta N.P. et al. Nat.Chem. 2015;7:913-920]

9. Ignorance creates an illusion of confidence that knowledge does not.

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