Time-resolved GISAXS and SAXS Studies of Lipid Multibilayers with the Insertion of Amyloid Peptide

Tsang-Lang Lin (林滄浪)[1], Jhih-Min Lin (林智敏)[1], and U-Ser Jeng (鄭有舜)[2]

[1]Department of Engineering and System Science, Tsing Hua University, Hsinchu, Taiwan
[2]National Synchrotron Radiation Research Center, Hsinchu, Taiwan

The amyloid peptide (1-40) is one of the major components that form the Alzheimer's amyloid deposits. At higher concentrations amyloid peptide will self-assemble into fibril. The results of X-ray diffraction and NMR studies showed that amyloid fibril contain pleated β-sheet [1]. The presence of charged lipid vesicles could influence the transition between α-helix and β-sheet. Studies of the membrane insertion of amyloid showed that amyloid is surface active and can insert into lipid monolayers [2,3]. Interaction of amyloid peptide with lipid membrane can provide information about the mechanism of amyloid deposits. The interaction between peptide and lipid monolayer or bilayer is critical to the understanding of the formation of amyloid peptide deposits on the membrane. The study by C. Edge et al. [3] showed that the presence of amyloid in the subphase could induce morphology changes of the lipid monolayer.

We have studied the structural transition of 1,2-dipalmitoyl-sn-glycero-3-phosphocholine (DPPC) multibilayer with the insertion of amyloid peptide 1-40 by GISAXS due to the change in relatively humidity. We also studied the mixture of DPPC and amyloid peptide 1-40 in aqueous solution by SAXS. We mixed the DPPC and amyloid peptide in organic solvent at a 5 to 1 weight ratio, then cast it on silicon wafer to form the lipid multibilayer film. The sample was sealed in a sample chamber with temperature and humidity control. Time resolved GI-SAXS measurements were conducted during the swelling process when the relative humidity of the sample chamber was increased from 35%. The temperature was controlled at around 33 °C. As shown in Fig. 1, it was found that the thickness of the DPPC bilayer increased due to the swelling and the ripple phase appeared. When the DPPC bilayer started to absorb vapor, the original single Bragg diffraction peak turned into two with one extra peak at slightly small scattering vector. This extra peak should correspond to the swelled bilayers at top surface. The bilayers at lower part of the lipid film remained at the original less swelled state. This indicated that the structural transition was not completed and it would take about one hour to complete the swelling of the lipid film. As shown in Fig. 2, the multibilayer DPPC bilayer with the insertion of amyloid peptide 1-40 did not have the ripple phase. This can be attributed to the amyloid peptide inserted into the lipid bilayer. We also can find similar effect in the solution samples. We mixed the DPPC and amyloid peptide in organic solvent at a 10 to 1 weight ratio. In the Fig. 3, the pure lipid sample had sharp Bragg peak due to the lipid multilayer vesicles. When we added amyloid peptide 1-40 into it, the peak became broad and shifted to low Q. It meant that the amyloid peptide monomers were indeed inserted into lipid liposome bilayers.

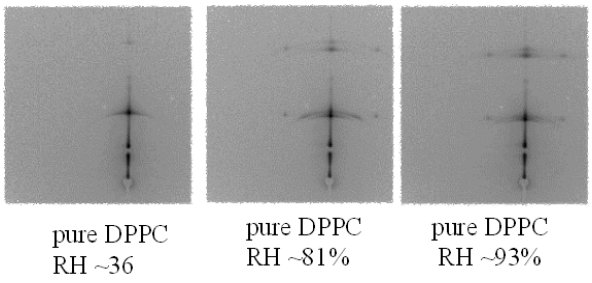

Figure 1. GISAXS of pure DPPC multibilayers on silicon wafer during the swelling process.

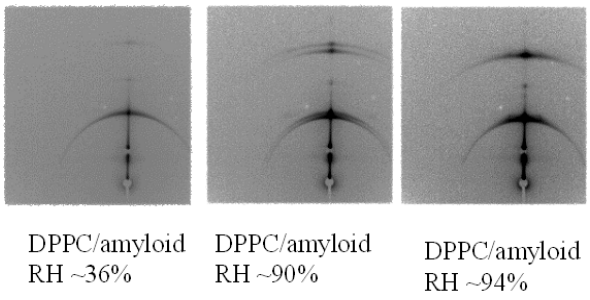

Figure 2. GISAXS of DPPC/amyloid multibilayers, at the weight ratio of 5 to 1, on silicon wafer during the swelling process.

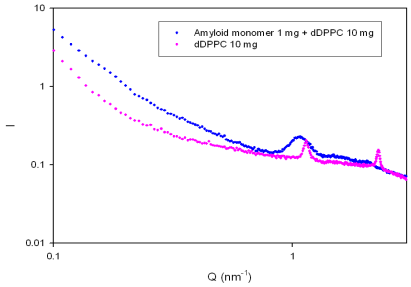

Figure 3. SAXS results of the mixed amyloid peptide 1-40 1 mg and dDPPC 10 mg in 0.5 ml D_2O and the pure dDPPC 10 mg at 0.5 ml D_2O.

References

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