



NANODIF

NEUTRON DIFFRACTION AND THE NANOSCALE

JULY 6-7, 2017 • DAEJEON, REPUBLIC OF KOREA



NANODIF 2017 IS A SATELLITE MEETING OF ICNS 2017, THE INTERNATIONAL CONFERENCE ON NEUTRON SCATTERING, DAEJEON, REPUBLIC OF KOREA

The aim of this cross-disciplinary meeting will be to review and discuss the state-of-the-art in neutron diffraction at the nanoscale and to bring together researchers from a variety of scientific disciplines including soft matter, materials science, and biology. Current and future developments in the field, covering in real space up to a few tens of nm will be presented to promote and adapt the use of neutrons to the needs of the community. The growing importance of instrumentation, sample environments and in-situ techniques will also be addressed in a dedicated session.



Organisation committee

Chris Garvey, ANSTO, Lucas Heights, Australia
Sung-Min Choi, KAIST, Daejeon, Republic of Korea
Viviana Cristiglio, ILL, Grenoble France
Bruno Demé, ILL, Grenoble, France
Tae-Hwan Kim, KAERI, Daejeon, Republic of Korea
Han Young Soo, KAERI, Daejeon, Republic of Korea

List of invited speakers

Anja Hörmann, Berlin, Germany
Anna Sokolova, Lucas Heights, Australia
Artem Feoktystov, Jülich, Germany
Leonie Van't Hag, Zurich, Switzerland
Liliana de Campo, Lucas Heights, Australia
Fred Heberle, Oak Ridge, USA
Wasim Abuillan, Tokyo, Japan
Norbert Kučerka, Dubna, Russia
Sung-Min Choi, Daejeon, Republic of Korea



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The interaction between amyloid-beta peptides and model membranes containing cholesterol and melatonin

Abstract

Alzheimer's disease (AD) is a devastating neurodegenerative disease caused by the formation of senile plaques, primarily consisting of amyloid-beta (Ab) peptides. The crucial role in this process is imparted by peptide-membrane interactions, changing the structural properties of membrane. These changes are known to be modulated also by membrane composition. In particular, cholesterol increases the order of lipid hydrocarbon chains and increases the stiffness of membrane. On the other hand, melatonin increases the fluidity of membrane. Our previous experiments [Drolle et al., BBA 2013] revealed the counteracting effect of melatonin to that of cholesterol in neat lipid membranes. We have extended our investigations recently by including transmembrane Ab peptide in these model membranes. Small angle neutron diffraction measured at four different contrast conditions was utilized for an unambiguous determination of structure in transversal direction. The obtained bilayer structure reflected the elevated amounts of cholesterol by its thickening, while the fluidizing effect of melatonin evoked the membrane thinning. Results of our experiments possibly confirm the melatonin's potential role in preventing the development of AD.

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Submitted by **Dr. KUCERKA, Norbert** on **Friday 21 April 2017**