Eur Biophys J (2013) 42 (Suppl 1):S1–S236
DOI 10.1007/s00249-013-0917-x

9th European Biophysics Congress
Lisbon – Portugal – 13–17 July 2013

9th EBSA

European Biophysics Congress

July 13th – 17th 2013, Lisbon, Portugal

SPBf SOCIEDADE PORTUGUESA DE BIOFÍSICA

EBSA Biophysics in Europe

This supplement was not sponsored by outside commercial interests. It was funded entirely by the publisher.

Acknowledgements

Sponsors:

- Springer-Verlag
- JPK Instruments
- Avanti Polar Lipids Inc.
- Leica Microsystems
- Nanospot
- European Grid Infrastructure
- Bio-Logic
- PicoQuant GmbH
- Anton Paar GmbH
- K.I.T. Group GmbH
- Routledge Journals Taylor & Francis
- SARSTEDT
- Royal Society Publishing
- GE Healthcare
- NanoTemper Technologies
- Photometrics&Qimaging
- Luso–American Foundation
- Turismo de Lisboa

9th EBSA European Biophysics Congress Lisbon (Portugal), July 13th – 17th, 2013

Organized by The Portuguese Biophysical Society.

ORGANISING COMMITTEE

Chairpersons: Manuela M. Pereira (ITQB – UNL) and Manuel Prieto (IST – UTL)

SPBf Committee

Andreia S. Fernandes (IBMC – UP)
João Carlos Marcos (CQ – UM)
Lígia O. Martins (ITQB – UNL)
Armindo Salvador (CNC – UC)
Nuno C. Santos (IMM/FMUL – UL)
Cláudio M. Soares (ITQB – UNL)
Graça Soveral (FF – UL)
Marco Domingues (SPBf)

SCIENTIFIC COMMITTEE

José L. Carrascosa, Centro Nacional de Biotecnología, Madrid, Spain.
Ana Margarida Damas, University of Oporto, Oporto, Portugal.
Alberto Diaspro, The Italian Institute of Technology, Genoa, Italy.
Erick J. Dufourc, CBMN, University of Bordeaux, Bordeaux, France.
Yves Engelborghs, University of Leuven, Leuven, Belgium.
Michael A. Ferenczi, Imperial College London, London, United Kingdom.
Felix Goñi, University of the Basque Country, Bilbao, Spain.
Helmut Grubmüller, Max-Planck-Institut für biophysikalische Chemie, Göttingen, Germany.
J. Antoinette Killian, Utrecht University, Utrecht, The Netherlands.
Alexander Konstantinov, University of Moscow, Moscow, Russia.
László Mátyus, Medical and Health Science Center, Debrecen, Hungary.
Satyajit Mayor, National Centre for Biological Sciences, Bangalore, India.
Lennart Nilsson, Karolinska Institute, Stockholm, Sweden.
Manuela M. Pereira, New University of Lisbon, Oeiras, Portugal.
Manuel Prieto, Technical University of Lisbon, Lisbon, Portugal.
Catherine Royer, University of Montpellier, Montpellier, France.
Michael Savageau, University of California, Davis, USA.
Petra Schwille, Max Planck Institute of Biochemistry, Martinsried, Germany.
Ilpo Vattulainen, Aalto University School of Science and Technology, Finland.
Anthony Watts, University of Oxford, Oxford, United Kingdom.
Anthony J. Wilkinson, University of York, York, United Kingdom.

Abstracts

– Protein Folding, Assembly and Stability –

P-128

Studies on composition-dependent structure of neuronal and bio-inspired supramolecular assemblies using small angle x-ray scattering

S. Pregent, R. Beck

The Center for Nanoscience and Nanotechnology, Tel Aviv University, Israel

Understanding the nature of interactions between biological molecules enabling the assembly of supra-molecular structures is a central goal in biophysics. These structures are critical for a wide range of cellular functions, e.g. remodelling of the supra-molecular structure of cytoskeletal proteins complexes, which occurs during cell division, locomotion, intracellular trafficking and signal transduction.

The cytoskeleton comprises three negatively charged proteins, including filamentous-actin, intermediate filaments and microtubules. The supra-molecular assemblies of these cytoskeletal filaments are mediated, in vivo, by complex interactions between one or more types of cross-linking proteins and by unstructured regions radiating from the filament backbone.

The main objective of this project is to elucidate the nature of the structures and interactions in these supra-molecular assemblies. We aim to elucidate the dominant interactions between different subunits, their critical concentration for structure's integrity and the consequent structural alterations once this concentration is not met. We also aim at studying intermolecular interactions of interfilament proteins adsorbed onto various surfaces for new, highly functional, highly "tunable" smart materials and surfaces.

P-129

Characterization of HEWL fibrillation using the fluorescence properties of Alexa 488 and Nile Red

J. C. Ricardo[1], A. Fedorov[1], M. Prieto[1], A. Coutinho[2]

[1]CQFM/IN, IST, UTL, Lisbon, Portugal, [2]CQFM/IN, IST, UTL and DQB/FCUL, Lisbon, Portugal

Amyloid fibers contribute to the pathology of many diseases, including type II diabetes and Alzheimer's. Recent research has proposed that membranes containing acidic phospholipids can facilitate the initial steps of amyloid fibril assembly, by both amyloidogenic and non-amyloidogenic proteins. To elucidate this question, we have been focusing our studies on hen egg white lysozyme (HEWL) as a model non-amyloidogenic protein.[1] The conformational changes and fibrillation kinetics of HEWL in solution at pH 2.2 and 57 ^{0}C were monitored by following the variations in the fluorescence properties of both HEWL fluorescently-labeled with Alexa 488 (HEWL-A488) and using the extrinsic dyes Thioflavin T (ThT) and Nile Red (NR). The characteristic stages of HEWL fibrillation could be clearly identified by tracking the increase in HEWL-A488 fluorescence anisotropy over time. The general use of NR as an amyloidotropic dye was further confirmed by quantitatively measuring its binding to HEWL and insulin fibrils through fitting an associative model to their fluorescence anisotropy decays. Additional FRET studies are planned to clarify whether ThT and NR compete for the same binding site in HEWL fibrils. Supported by project PTDC/QUI-BIQ/099947/2008 FCT/Portugal.

[1]Melo *et al. J. Phys. Chem. B* **2013**, *117:*2906

P-130

Characterization and optimization of a novel protein refolding methodology

G. Roussel[1], S. L. Rouse[2], M. S. Sansom[2], E. A. Perpète[1], C. Michaux[1]

[1]Unit of Theoretical and Structural Physico-Chemistry, University of Namur, Belgium, [2]Structural Bioinformatics and Computational Biochemistry Unit, University of Oxford, United-Kingdom

Recently, a simple yet effective method to recover refolded and active proteins has been developed, based on the association of an anionic detergent (sodium dodecyl sulfate, SDS, known as denaturing agent) with an amphipathic diol solvent. Up to now, this cosolvent effect has been observed on both soluble and bacterial membrane proteins. None to say, it is crucial to have a clear picture of the physicochemical and molecular basis of these refolding processes. We have therefore used both experimental (intrinsic fluorescence and circular dichroïsm) and theoretical (coarse-grained and atomistic molecular dynamics) approaches to help understanding such intricate phenomena. We first start with the study of the detergent/cosolvent couple (by considering different alcohols molecules) and then investigate the protein/detergent/cosolvent complex (by using peptides as models for alpha- and beta- secondary structures). Such a study gives access to the nature and strength of the interactions between the molecules, and the system stability as a function of time. Original detergent/solvent pairs can be proposed in order to improve the efficiency of our refolding protocol, as well as to sharpen our understanding of its mechanism, paving the way to the full protein characterization.

P-131

Fibrillation of Human Serum Albumin at physiological pH is inhibited by oxidation

G. Sancataldo[1], V. Vetri[1], V. Foderà[2], V. Militello[1], L. Maurizio[1]

[1]Università di Palermo, Dipartimento di Fisica e Chimica, Palermo, [2]Department of Drug Design and Pharmacology, Faculty of Health and Medical Sciences, Copenhagen

Oxidative stress and amyloid fibrils formation have been suggested to underlie the loss of cellular function in pathologies like Parkinson's and Alzheimer's disease. The understanding of the temperate relationship between these two processes can be of great importance in revealing the molecular basis of neurodegeneration.

Here we show that oxidation of HSA induced by H_2O_2 inhibits amyloid fibrils formation. Structural properties and aggregation pathways of non-oxidized and oxidized HSA were studied by means UV-Vis absorption and fluorescence, CD, FTIR, light scattering and TEM. The mutual interactions of several mechanisms contribute to the formation of fibrils in HSA through a non-nucleated process being the aggregation pathway regulated by the balance between attractive and repulsive interactions in partially unfolded molecules. In the case of oxidized HSA, this equilibrium is altered by minute variations due to protein oxidation which causes changes in protein tertiary structure leading to protein compaction and resulting in increased stability and reduced association propensity of oxidized molecules. HSA forms thin and straight amyloid fibrils while Oxidized HSA forms curly and amorphous aggregates resulting from a slower process with different intermediate states.