



ICC-04

The Fourth International
Conference on Cofactors

25th-28th August 2014
Parma, Italy

ICC-04

4th International Conference on Cofactors

25th-28th August 2014, Parma
(Casa della Musica) – Italy

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Official reference

Andrea Mozzarelli
Department of Pharmacy
University of Parma
43124 Parma, Italy
Phone number: 0039-0521-905138
Fax: 0039-0521905151

Welcome to the Fourth International Conference on Cofactors

Dear Participant,

we are happy to welcome you at the **Fourth International Conference on Cofactors** (ICC-04). The meeting combines the past 'Symposium on Vitamin B6, PQQ, Carbonyl Catalysis and Quinoproteins' and 'The Congress on Vitamins and Related Biofactors', also formerly known as 'Interdisciplinary Conference on Vitamins, Coenzymes, and Biofactors'. The conference includes sessions on pyridoxal 5'-phosphate-, NAD- and flavin-dependent enzymes, quinones and quinoproteins, heme-containing proteins and enzymes, biosynthesis of biofactors, biomedical aspects of vitamins and biofactors and special sessions dedicated to neurological disorders and drug development targeting cofactor-dependent enzymes.

We are eager to host an exciting program with world-class keynote speakers and a lot of space dedicated to talented young researchers and discussion. We are very pleased that Parma, Italy, has been selected to host the ICC-04. This choice probably is due to the reputation of Parma as a place of great music, beautiful historical buildings and ... excellent food. We tried to organize a program that is aimed to mix together science and local culture, in order to provide you with a terrific and unforgettable experience. We are confident that this international meeting will provide a unique opportunity to share experiences and interests, and to improve our knowledge through multidisciplinary contributions.

Enjoy the meeting!

President
Andrea Mozzarelli
University of Parma
Parma, Italy

Co-President
Loredano Pollegioni
University of Insubria
Varese, Italy

Programme at the glance

August 25th

14.00 – Registration

15.00 – *Welcome*

15.15 – Opening Lecture: Dan S. Tawfik

16.00 – Session I: Biotin, thiamine, pterin and SAM

18.30 – Get-together buffet

August 26th

8.30 – Registration

9.00 – Session II: Pyridoxal phosphate (first part)

10.30 – Coffee break

11.00 – Session III: Flavins

12.40 – Lunch and poster session

15.00 – Session IV: Fe/S (first part)

16.20 – Coffee break

16.50 – Session V: Heme

18.30 – Guided tour of Parma

August 27th

9.00 – Session VI: Biotechnological, biomedical and pharmaceutical applications of cofactors and cofactors-enzymes

10.30 – Coffee break

11.00 – Session VII: Pyridoxal phosphate (second part)

12.30 – Lunch and poster session

15.00 – Session VIII: Flavin and pyridoxal phosphate cofactors and pathologies

17.00 – Coffee break

17.30 – Session IX: Quinones

20.00 – Social dinner

August 28th

9.00 – Session X: Fe/S (second part)

10.30 – Coffee break

11.00 – Session XI: Pyridoxal phosphate (third part)

12.30 – Lunch and poster session

14.30 – Session XII: NAD

16.20 – Coffee break

16.50 – Session XIII: Metals

18.00 – Closing Talk: Marco Fraaije

18.30 – *Final remarks*

Lectures

ST24 The G308E Variant of the Apoptosis Inducing Factor, Responsible of a Rare Encephalopathy, is Hampered in NAD⁺/H binding

*Luca Sorrentino, Laura Rigamonti, Mirvan Krasniqi, Alessandra Calogero, Vittorio Pandini,
Maria Antonietta Vanoni, Alessandro Aliverti*

*University of Milan, Department of Biosciences, Milan, Italy
Email: luca.sorrentino@unimi.it*

The apoptosis inducing factor (AIF), a highly conserved mitochondrial flavoprotein, plays two opposite roles in eukaryotic cells: while in mitochondria, it is required for efficient oxidative phosphorylation (OXPHOS), but it triggers caspase-independent apoptosis when released into the cytosol (1). AIF undergoes dimerization upon reaction with NAD⁺/H, via formation of an unusually air-stable charge-transfer (CT) complex. This peculiar property of AIF is likely to be linked to its biological function.

Recently, the G308E mutation of human AIF was identified as the cause of severe neurodegeneration associated with OXPHOS defect (2). We introduced the equivalent amino acyl substitution in murine AIF (G307E) and characterized the resulting protein variant in vitro. This replacement dramatically decreases the rate of reaction between AIF and NAD⁺/H, but neither the rate of dissociation nor the O₂ reactivity of the resulting dimeric CT complex were affected. A detailed rapid-mixing and steady-state kinetic study of the reaction between AIF and NAD⁺/H allowed us to develop a two-step mechanism for CT complex formation. In addition, we found that FAD reduction induced a partial conformational reorganization of AIF, triggering dimerization which is therefore independent from ligand binding and CT complex formation. Our results shed new light on the mechanism of the possible redox-sensing role of AIF and show that the pathogenic G308E replacement may disrupt its functions, while in mitochondria, by specifically slowing down the formation of its dimeric CT complex.

1. Sevrioukova (2011) *Antioxid Redox Signal*, 14: 2545-2579
2. Berger et al (2011) *Mol Genet Metab*, 104: 517-520

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