XV FISV CONGRESS
Sapienza University of Rome, Italy
September 18-21, 2018

Programme & Abstracts

AAI ABCD AGI SIB SIBBM SIBE SIBV SICA SIF SIGA SIMAG SIMGBM SIP MeT SIPaV

FISV - Federazione Italiana Scienze della Vita

XV FISV CONGRESS
Programme and Abstracts

Sapienza University of Rome, Italy
September 18 – 21, 2018

CONTENTS

Welcome Letter 3
Fisv Member Societies 4
Committees - Secretariat 5
Session Organisers 6

PROGRAMME 7

ABSTRACTS 26
The Embo Keynote Lecture 26

Plenary Lecture 27

Plenary Symposium 29
The Circadian Clock. A Living Organism's Best Friend in a Spinning World 29
Oxidative Stress, Protein Damage and Repair: Implications in Health and Disease 31
Life is Also a Matter of Taste and Smell 33
Farming for Pharming: Plants as Biofactories (in the Production of Vaccines, Antibiotics, Anticancer) 35
Regulatory Network Dynamics: from Interaction to Function 37

Parallel Symposia 39
Emergence and Spread of Archaic and Modern Humans: News from Bones and Genomes 39
Genetic and Epigenetic Mechanisms Regulating Transgenerational Inheritance 41
Proteins as Drug Target and Drugs, and Protein Degradation as a Therapeutic Strategy 43
Crossing Biological Barriers, in Health and Disease 45
RNA Biology 47
Inflammation and Disease 49
Carbon Cycle and Climate Change: the Future 51

Short Talks by Sponsors 53

Poster and Selected Short Talks 54
1 - Environmental Microbiology and Biotechnology 54
2 - Genomics, Proteomics and Systems Biology 65
3 - Chromosome Biology, Cell Division and Cell Cycle 73
4 - Transcriptional Mechanisms and Epigenetic Modifications 81
5 - Oncogenes and Tumor Suppressors 88
6 - Photosynthesis, Metabolism and Environmental Stress 98
7 - Genetics of Microorganisms 113
8 - DNA Replication, Repair and Recombination 119
9 - Non-coding RNA 130
10 - Plant Nutrition and Biofortification 138
11 - Cellular Stress, Apoptosis and Autophagy 143
12 - Development, Differentiation and Ageing 156
13 - Metabolism and its Regulation in Health and Diseases 163
14 - Human Genetics and Genomic Diversity 177
15 - Neurobiology and Neuropharmacology 185
16 - Immunology and Host-Pathogen Interaction 194
17 - Biotransformations 205
18 - Stem Cells, iPS, Cancer Stem Cells 210
19 - Nutrition Biochemistry 215
20 - Evolutionary Biology 225
21 - Glycoconjugates 229
22 - Plant Development and Disease 231

Author Index 242

COMMITTEES - SECRETARIAT

Scientific Committee

Gennaro Ciliberto (*FISV President*, Rome)
Giovanna Serino (*FISV Secretary*, Rome)

Antonio Antoccia, Rome
Stefano Biagioni, Rome
Alberto Boffi, Rome
Silvia Bonaccorsi, Rome
Bianca Colonna, Rome
Giulia De Lorenzo, Rome
Alessandra Di Masi, Rome
Gabriele Gentile, Rome
Rita Mancini, Rome
Giulia Piaggio, Rome
Isabella Saggio, Rome
Angela Santoni, Rome
Gianni Sava, Trieste
Eugenia Schininà, Rome
Maryanne Tafuri, Rome

Organising Secretariat

Marina Nobilio
Lucia Ugo
c/o Dipartimento di Biologia e Biotecnologie
"Charles Darwin"
Sapienza Università di Roma
Piazzale Aldo Moro, 5
00185 ROMA
congress2018@fisv.org

FISV 2018 Web Services

Azuleon sas

Elena Papinutto (fisv.2018@azuleon.org)

P3.1 - Satellite DNA is responsible for centromere clustering in mammals

Eleonora Cappelletti[1], I. Solovei[2], F.M. Piras[1], M. Corbo[1], S.G. Nergadze[1], E. Giulotto[1]
[1]Dept. Biology and Biotechnology "Lazzaro Spallanzani", University of Pavia, Pavia, Italy; [2]Biozentrum, Biology-II, Ludwig-Maximilian University of Munich, Munich, Germany

In the 3D architecture of mammalian nuclei, centromeres cluster at the nuclear and nucleoli periphery. It is a matter of debate whether centromere clustering depends on the presence of satellite repeats or on the centromeric function. We discovered that, in equid species (horse and donkey), several centromeres are satellite-free, whereas many satellite DNA loci are not centromeric (Wade et al Science 2009; Piras et al PLoS Genet 2010; Nergadze et al Genome Res 2018). Thus, Equids represent a unique model for investigating the basis of centromere clustering.

By studying their 3D distribution, we showed that centromeric and non-centromeric satellite DNA loci form clusters, indicating a tendency of satellite sequences to coalesce irrespectively of the centromeric function. On the other hand, satellite-less centromeres, although localizing mainly at nucleoli and nuclear periphery, do not associate to the satellite-based centromeres.

These observations are in agreement with the notion that in the mammalian nucleus, chromosomal loci tend to associate with each other according to their repeat enrichment as a result of mutual repeat recognition (Solovei et al Curr Opin Cell Biol 2016).

P3.2 - Two immortalized rat astrocyte cell lines as *in vitro* model for specific cell proliferation studies: cytogenetic and epigenomic characterization and diversification

Fabio Caradonna[1], I. Cruciata[1], C. M. Di Liegro[1], V. Vitale[1], R. Mormino[1], P. D'Oca[1], I. Di Liegro[2], G. Schiera[1]
[1]Department of Biological, Chemical and Pharmaceutical Sciences and Technologies, University of Palermo, Italy; [2]Department of Experimental Biomedicine and Clinical Neurosciences, University of Palermo, Italy

Here we report differences between: 1) a heterogeneous population of primary rat brain astrocytes (Primary), in culture since several years ago, and 2) a cloned cell line (Clone), obtained from the Primary cells. Both populations maintain astrocyte morphology but, according to cytogenetic and epigenomic characterization, differ for the chromosomal asset from rat normal cells (42 chromosomes): Primary cells show mostly a bimodal karyotype with 41 or 43 chromosomes, and Clone has a unique-modal karyotype of 43 chromosomes. Interestingly, we also found that both cell lines show genome-wide DNA hypomethylation, with Clone showing even more pronounced demethylation respect to Primary cells. These features, together with a faster doubling time, confer to Clone an altered proliferation control phenotype. Conversely, the Primary cell population is more similar to normal cells. Used together the two cell populations are a promising model to investigate in vitro modifications of genome, epigenome and others 'omics', mimicking tumor clonal evolution-derived heterogeneity, particularly useful in studies on CNS cancers, which derive mostly from glial cells.

P3.3 - The tumor suppressor p14^{ARF} hampers proliferation of aneuploid cells induced by CENP-E partial depletion

Danilo Cilluffo [1], L. Veneziano [1,], V. Barra [2], A. Di Leonardo[1]
[1]Dept of Biological, Chemical and Pharmaceutical Sciences and Technologies, University of Palermo, Italy; [2]Gustave Roussy, Dept of Genetic Stability and Oncogenesis, CNRS UMR8200, 94805 Villejuif, France

The Spindle Assembly Checkpoint (SAC) is a cellular surveillance mechanism that ensures