Associazione di Biologia Cellulare e del Differenziamento

# Cell Stress: Survival and Apoptosis

**Organising Committee** 

Sergio Giannattasio CNR Institute of Biomembrane and Bioenergetics (Bari)

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## Programme & Abstracts

## Bari, 9-10 September 2016 http://CSSA2016.azuleon.org

#### Friday, 9 September

12:00	REGISTRATION AND WELCOME MINGLING COCKTAIL
14:00-14:10	Opening
14:10-15:10	DNA AND RNA STABILITY Chair: Alessandra Montecucco (Pavia)
14:10-14:30	Morena Catillo (Pavia) Splicing of transcripts for splicing factor SRSF1 is finely tuned in response to cell metabolism
14:30-14:50	<i>Cristina Mazzoni (Rome)</i> RNA oxidation and ageing in mRNA degradation mutants of <i>S. cerevisiae</i>
14:50-15:10	Annapina Russo (Naples) Regulatory role of rpL3 in cell response to nucleolar stress induced by Act D in tumor cells lacking functional p53
15:10-16:10	CELL DEATH AND METABOLISM Chair: Valter Longo (Los Angeles, CA, USA)
15:10-15:30	<i>Nicoletta Guaragnella (Bari)</i> Cell fate decision in yeast: within and between glucose sensing, Hog1 SAPK and mitochondrial retrograde pathways
15:30-15:50	<i>Enzo Martegani (Milan)</i> Accumulation of activated Ras in mitochondria and apoptosis
15:50-16:10	<i>Flavia Cuviello (Potenza)</i> Modulation of mitochondrial pyruvate carrier expression in HEK293 and HepG2 cells
16:10-16:30	Coffee break and poster viewing
16:30-17:30	<b>PLENARY LECTURE</b> <i>Valter Longo (Los Angeles, CA, USA)</i> Nutrient signaling, cellular protection and regeneration, and healthspan
17:30-19:00	Poster Session
19:00-20:00	HOST-PATHOGEN INTERACTION Chair: Cristina Mazzoni (Rome)
19:00-19:20	<i>Rosanna Salvia (Potenza)</i> The multifunctional polydnavirus ANK1 protein: new insights for apoptotis pathway
19:20-19:40	<i>Luisa Rubino (Bari)</i> Carnation Italian ringspot virus p36 expression enhances necrotic cell death in response to acetic acid in <i>Saccharomyces cerevisiae</i>

#### Regulatory role of rpL3 in cell response to nucleolar stress induced by Act D in tumor cells lacking functional p53

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Many chemotherapeutic drugs cause nucleolar stress and p53-independent pathways mediating the nucleolar stress response are emerging. Here, we demonstrate that ribosomal stress induced by Actinomycin D (Act D) is associated to the up-regulation of ribosomal protein L3 (rpL3) and its accumulation as ribosome-free form in lung and colon cancer cell lines devoid of p53. Free rpL3 regulates p21 expression at transcriptional and post-translational levels through a molecular mechanism involving extracellular-signal-regulated kinases1/2 (ERK1/2) and mouse double minute-2 homolog (MDM2). Our data reveal that rpL3 participates to cell response acting as a critical regulator of apoptosis and cell migration. It is noteworthy that silencing of rpL3 abolishes the cytotoxic effects of Act D suggesting that the loss of rpL3 makes chemotherapy drugs ineffective while rpL3 overexpression was associated to a strong increase of Act D chemotherapy depends on rpL3 status revealing new specific targets involved in the molecular pathways activated by Act D in cancers lacking of p53. Hence, the development of treatments aimed at upregulating rpL3 may be beneficial for the treatment of these cancers.