Abstracts submitted to The Biochemistry Global Summit (25th IUBMB Congress, 46th FEBS Congress and 15th PABMB Congress) from 9th to 14th July 2022 and accepted by the Congress Organizing Committee are published in this Supplement of FEBS Open Bio. Late-breaking abstracts are not included in this supplement. The abstracts are available as two PDF files: Talks (Plenary Lectures, Symposia and FEBS Special Sessions) and Posters.

About these abstracts
Abstracts submitted to the Congress are not peer-reviewed. In addition, abstracts are published as submitted and are not copyedited prior to publication. We are unable to make corrections of any kind to the abstracts once they are published.

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** DOIs are as follows:
Talks: 10.1002/2211-5463.13442
Posters: 10.1002/2211-5463.13440
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* The Abstract number begins with the letters PL, SS, S or ShT and can be found atop each abstract’s title in the PDF file.
S phase and melanocytes in the G1/G0 phase. Phototoxic reactions caused by Mel are associated with the induction of oxidative stress and can lead to severe damage to skin cells.

ShT-03.1-4
Phosphoregulation of the ATP synthase beta subunit contributes to mitochondrial respiration for G2/M progression
A.C. Leite1,2,3, T.S. Martins1,2,3, A. Campos1,2, V. Costa1,2,3, C. Pereira1,2

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Recently, we showed that the 2A-like protein phosphatase Sit4 promotes the dephosphorylation of ATP synthase catalytic beta subunit (Atp2 in yeast) at T124/T317. Phosphorylation at T124/T317 upregulates Atp2 levels, leading to an increase in ATP synthase activity and ATP production [1]. Using in silico analysis, four kinases were predicted as Atp2 regulators, namely Pkc1, Ipl1, Cdc5 and Hrr25. From these kinases, only Cdc5 overexpression increased Atp2 levels, suggesting Atp2 is regulated by Cdc5. As for the phosphatase Sit4, Cdc5 plays a prominent role in cell cycle regulation, suggesting that Atp2 phosphorylation may be cell cycle related. To investigate this hypothesis, we monitored Atp2 levels during cell cycle progression in synchronous cells. We found Atp2 levels vary during cell cycle phases, with an increase at G1 and at G2/M. Since the Atp2 phosphoproteins lie within prototypal APC/C recognition motifs, an ubiquitin ligase involved in cell cycle regulation, leading to the formation of a complex with APC/C. Since the Atp2 phosphosites lie within pro-apoptotic APC/C recognition motifs, an ubiquitin ligase involved in cell cycle regulation, leading to an increase in ATP synthase activity and ATP production [1]. In the absence of APC/C subunits Atp2 levels increased, but the mutation of the putative APC/C motifs in Atp2 or overexpressing APC/C do not influence Atp2 levels, indicating the stability of phosphorylated Atp2 is not the result of APC/C regulation. Since Atp2 phosphorylation promotes mitochondrial function, we monitored mitochondrial respiration during cell cycle phases. We found that preventing Atp2 phosphorylation, using an Atp2-T124A/T317A mutant, decreased the mitochondrial respiration peak at G2/M. In accordance, cell cycle progressed similarly in the Atp2-T124A/T317A mutant until G2/M, when the transition to G1 was delayed. Our results show that Atp2 phosphorylation is associated with cell cycle regulation, leading to increased mitochondrial respiratory activity that promotes cell cycle progression at G2/M. [1] Pereira C et al. (2018) Biochim Biophys Acta Bioenerg 1859, 591–601.

S-04.1–2
Septin filament assembly: The rules of the game
R. Garratt1, H. Muniz Pereira1, D.A. Leonardo1, H.V. Dias Rosal1, I.A. Cavin1, D. K.S.V. Castro1, A. Freitas Fernandes1, D. Cezar Mendonça1, S. Leite Guimarães1, R. Villares Portugal2, N. Fonseca Valadares1, A.P. Ulian da Araújo1, 1Institute of Physics of São Carlos, University of São Paulo, São Carlos, Brazil, 2Laboratório Nacional de Nanotecnologia, CNPEM, Campinas, Brazil, 3Department of Cellular Biology, University of Brasília, São Carlos, Brazil

Septins represent the fourth component of the cytoskeleton and their importance in a wide range of essential intracellular events, many of which involve membrane remodeling or diffusion barrier formation, have become increasingly apparent over recent years. Thirteen human septins assemble into a wide range of different heteropolimeric filaments which follow specific rules of assembly giving rise to a finite set of combinations. Heterologicome core particles first assemble into palindromic hexamers or octamers which subsequently polymerize end-to-end. For over a decade we have been attempting to understand the rules of assembly which guarantee how each individual subunit within a viable combination assumes its rightful position along the filament and how these subsequently unite into higher order structures which associate with membranes. We have used a “divide and conquer” approach by which we have dismantled the core particles into smaller assemblies and reduced individual monomers to isolated domains. By accumulating a large number of crystal and cryo-EM structures, several features are beginning to emerge, including the importance of 1) group-specific residues which guarantee that the correct interfaces are formed along a filament; 2) the metastable properties of the C-terminal domains allowing them to participate in both parallel and antiparallel coiled coils relevant to filament assembly and cross-bridging respectively; 3) the dynamics of a polybasic region important for forming electrostatic interactions with membranes and 4) the presence of a large internal cavity essential for allowing the relative movement of subunits along the filament. These phenomena will be described in an attempt to draw our current knowledge together into a coherent picture of filament assembly and relate it to function and malfunction, such as in the case of the off-target cleavage of SEPT2 by the Zika NS2B3 protease.

S-04.1–1
Chiral proofreading during protein biosynthesis and its evolutionary implications
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A major focus of our laboratory is on translation quality control with a special reference to chirality-based checkpoints in the cell. We earlier elucidated the ‘Chiral Proofreading’ mechanism,