Contribution ID: **1601** Type: **Oral**

Correlation analysis of cytoplasmic actin isoforms distribution in endothelial cells

Thursday 31 October 2024 15:35 (15 minutes)

Actin is one of the most abundant proteins in a living cell. Actin structures are found in all cells of a living organism and are involved in maintaining and changing the cell shape, exocytosis and endocytosis, cell adhesion to substratum and cell movement, and signal transduction. In mammals, β- and γ-actin are cytoplasmic actin isoforms in non-muscle cells. Despite minor differences in the amino acid sequence, β- and γ-actin localize in different cell structures and perform different functions. While cytoplasmic β-actin is involved in many intracellular processes including cell contraction, γ-actin is responsible for cell mobility and promotes tumor transformation. Numerous studies demonstrate that β- and γ-actin are spatially separated in the cytoplasm of fibroblasts and epithelial cells; this separation is functionally determined. The spatial location of β/γ-actin in endothelial cells is still a subject for discussion. Using super-resolution microscopy, we investigated the β/γ-actin colocalization in endothelial cells. For analysis, we used human pulmonary artery endothelial cells (HPAEC), primary cells isolated from the human pulmonary artery. Colocalization analysis of both wide-field and SIM images was performed using ImageJ software. To find out whether β- and γ-actin are colocalized in a given region, we used the Coloc2 plugin function of calculating the M1 and M2 Manders' coefficients (i.e., separately for two channels). We showed that β and γ-actin are partially colocalized in certain regions of the endothelial cytoplasm. In HPAEC, the β/γ-actin colocalization degree varies widely between different parts of the marginal regions and near the cell nucleus. In the basal cytoplasm, β-actin predominates, while the ratio of isoforms evens out as it moves to the apical cytoplasm. Thus, colocalization analysis suggests that β- and γ-actin are segregated in the endotheliocyte cytoplasm. The segregation is greatly enhanced during cell activation in the endothelial barrier dysfunction (pathological permeability disorder of the vascular permeability that occurs in a number of acute, life-threatening human states), modelling in vitro, and this data may demonstrate different roles of two cytoplasmic actin isoforms in the functional activity of endothelial cells.

Acknowledgments: The authors thank the Moscow University Development Program (MSU Development Program PNR 5.13) and Nikon Center of Excellence at A.N. Belozersky Institute of Physical and Chemical Biology for providing research infrastructure.

Primary author: SHAKHOV, Anton

Co-authors: CHURKINA, Aleksandra (A.N. Belozersky Institute Of Physico-Chemical Biology Lomonosov Moscow State University); ALIEVA, Irina (Cell Motility Lab, Belozersky Institute of Physico-Chemical Biology, Moscow State University)

Presenter: SHAKHOV, Anton

Session Classification: Life Science

Track Classification: Life Science