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Correlation analysis of cytoplasmic actin isoforms distribution in endothelial cells

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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 y-actin are cytoplasmic actin isoforms in non-muscle cells. Despite minor differences in the amino acid sequence, β - and y-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 βand 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.

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