Abstract

Vascular smooth muscle cells (VSMCs) express considerable phenotype plasticity. They are able to change their phenotype *in vivo* if necessary. It is important to know that during this phenotype switch the expression of transport proteins and channels is modified, which results in significant alteration of Ca^{2+} signaling in smooth muscle cells. In differentiated cells, which represent contractile phenotype, there are dominant rapid, transient events in intracellular Ca^{2+} concentration (Ca^{2+}i), while the resting cytosolic Ca^{2+}i concentration is low. In differentiated cells these Ca^{2+}i events are mainly caused by two components of the Ca^{2+} signalling pathways: 1) extracellular Ca^{2+} influx *via* L-type voltage-gated Ca^{2+} channels (L-type VGCC) in plasma membrane, and 2) depletion of intracellular Ca^{2+} stores *via* ryanodin receptors located on sarcoplasmic reticulum. Rapid Ca^{2+}i oscillations are quickly reduced by numerous Ca^{2+} ATPases of sarco/endoplasmic reticulum and plasma membrane. Proliferating vascular smooth muscle cells are characterized by a long-lasting Ca^{2+}i oscillations accompanied by sustained elevation of basal intracellular Ca^{2+} concentration. During phenotype switch from contractile phenotype to proliferative phenotype there is decreased Ca^{2+} ATPase activity, and store-operated Ca^{2+} entry is elevated. This is accompanied by the replacement of L-type voltage-gated Ca^{2+} channels with T-type voltage-gated Ca^{2+} channels. These changes are due to altered gene expression, which is dependent on transcription factors, mainly on CRE-binding protein and nuclear factor of activated T-lymphocytes (NFAT). Vascular smooth muscle cells of spontaneously hypertensive rats (SHR) have some characteristics similar to the proliferative phenotype. On the other hand SHR are characterized by important role of L-type VGCC with T-type VGCC, in vascular contraction which is typical for differentiated (contractile) VSMCs.