HIGH SALT-INDUCED HUMAN VASCULAR ENDOTHELIAL AND SMOOTH MUSCLE REMODELING

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Salt sensitivity is a factor that contributes to an increase in blood pressure of at least 10%. It is a complex phenomenon whose cellular and molecular mechanisms remain controversial despite the various epidemiological studies and animal models developed. The dogma is that this salt sensitivity is primarily due to a renal defect in salt excretion. Recently, it has been suggested that an increase in plasma sodium of 2-4 mM induced by a high salt diet may lead to vascular dysfunction by directly exposing smooth muscle cells to extracellular sodium overload. The increased [Na+]e induces remodeling of human vascular endothelial and smooth muscle cells (hVSCs) and intracellular calcium homeostasis. Chronic increase in [Na+]e leads to a significant concentration-dependent increase in hVEChVSMC volume. This increase in cell volume is associated with an increase in the number of NTTs (nuclear T-tubules). By exposing the cells to 4 mM NaCl, there is an increase in the basal intracellular Ca^{2+} level of both hVECs and hVSMCs.Furthermore, salt-pre-sensitized cells are more sensitive to sodium than non-sensitized cells. The morphological and functional remodeling induced by the chronic increase of [Na+]e is irreversible in the short term. In conclusion, chronic exposure to extracellular sodium is associated with the remodeling of ionic and morphological homeostasis.

Keywords: salt sensitivity, hVSMC, sodium, calcium, NTT, hypertrophy, cell remodeling.

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