

Reactive oxygen species are usually assumed as dangerous, cytotoxic substances included in pathogenesis of variety of diseases due to their ability to damage biomolecules. However, ROS also play an indispensable role in many physiological processes. Low concentrations of ROS are involved in signal transduction as a part of pathways regulating protein phosphorylation, gene expression, NO availability or intracellular Ca^{2+} . They are important for cell cycle control and apoptosis. In the last years, the role of ROS in regulation of vascular tonus is intensively studied.

NAD(P)H oxidases are supposed the major source of ROS. These are enzymes similar to phagocyte NADPH oxidase, a key enzyme of phagocyte respiratory burst in immune response. We have studied NAD(P)H oxidase of vascular wall in normotensive and hypertensive rat. Activity of superoxide production by NAD(P)H oxidase differ between cells from normotensive and hypertensive rat. Control of NAD(P)H oxidase activity is also changed in hypertensive animals, at least with respect to the effect of angiotensin II and uric acid.

In our experimental work, we encountered unexpected properties of apocynin, a known inhibitor of phagocyte NADPH oxidase. We proved that apocynin can increase ROS production. The reason is that NAD(P)H oxidase is not inhibited directly by apocynin but rather by a metabolite of this compound. Apocynin is converted to the active inhibitor by action of hydrogen peroxide and a peroxidase.

We also performed pilot studies on the effect of uric acid on NAD(P)H oxidase activity. Uric acid in low concentrations decreases while in high concentrations increases superoxide production in vascular smooth muscle cells. This finding could explain current controversies between scavenging properties of uric acid and increased cardiovascular risk in hyperuricemia.