Summary

This dissertation demonstrates not only the classic hemodynamic and metabolic cardiovascular (CV) risk factors affecting arterial rigidity but also the soluble receptor for advanced glycation end-products (sRAGE) was studied. The stiffening of central arteries may precede the onset of manifest atherosclerosis, thus being a potential target for early prevention of cardiovascular disease. The actual guidelines of Czech society for hypertension recommend the measurement of aortic pulse wave velocity (aPWV) in hypertensive patients. The AGEs/RAGE complex participates in various stages of atherosclerosis/arterial stiffening. The soluble form of RAGE probably acts as a decoy for RAGE ligands and it is a naturally occurring inhibitor of toxic impact caused by AGE-RAGE action. It is suggested to be a potential marker of further chronic disease.

This dissertation focuses on four publications investigating CV risk factors in two different population cohorts. We studied a random general population sample and offspring of patients with premature coronary heart disease sample compared to controls.

In the general population we demonstrated that aPWV increased linearly with the rising numbers of individual metabolic syndrome risk factors (*p* for trend 0,0001). The levels of sRAGE were significantly positively associated with HDL cholesterol and 25-OH-D3, while it was negatively correlated with age, body mass index, waist circumference, mean arterial pressure, triglycerides, glycemia and aPWV (*p* for all ≤0,0145*)*. Low sRAGE levels significantly predicted increased aPWV in hypertensive non-diabetic patients. Hypertensive patients with low sRAGE levels showed higher aPWV than those with high sRAGE (*p*=0,03), which was only when the patients were untreated with renin-angiotensin-aldosterone system blockers.

In the publications studying the offspring of patients with premature heart disease we demonstrated their higher total CV risk calculated by the SCORE system – the current risk related to the age of 40 years as well as to the age of 60 years (both *p*< 0,0001). The offspring cohort had also higher aPWV and lower sRAGE (both *p*≤0,009) than the control group.

The measurement of aortic stiffness contributes to identifying such individuals who are at low cardiovascular risk so far but have higher stiffness on the basis of their age. We would need any other markers for early identifying of subjects in increased cardiovascular risk. The axis AGE/RAGE seems to be a key player in the cardiovascular risk stratification and might be a potential target for further intervention as well.