

## **Abstract**

Arterial system is a system of vessels distributing blood. Ageing of arterial system leads to two distinct pathologies: atherosclerosis and arteriosclerosis - stiffening of arterial wall. These pathologies can coexist and interfere; however, they differ in their pathogenesis, location, scope and consequences. Progressive loss of elastic properties of large arteries is natural part of vascular ageing. It is directly responsible for several age dependent consequences, such as increase of central systolic pressure or prevalence of isolated systolic hypertension in the elderly. Clinically, central arteries stiffness manifests as aortic pulse wave velocity, which can be quantified, among other methods, using applanation tonometry. There is abundant evidence that aortic pulse wave velocity represents an independent predictor of cardiovascular mortality and morbidity. The most important mechanism in arterial stiffening is repeated mechanical damage which leads to fractures, fragmentation and thinning of elastin. Stiffening of large arteries can be accelerated by several other mechanisms, e.g. deposition of several substances (calcium, advanced glycation end-products, etc.), metabolic turnover of key elements of vascular extracellular matrix (collagen and elastin) or individual genetic susceptibility.

In this dissertation thesis, the main aim was to evaluate potential associations with increased aortic pulse wave velocity in a cross-sectional design. Moreover, potential predictors of accelerated arterial stiffening were assessed in a prospective study that lasted eight years. The main focus was on metabolic factors with emphasis on glucose metabolism. Secondary aim was to evaluate the effect of vitamin K and selected single nucleotide polymorphisms.

**Key words:** arterial stiffening, aortic pulse wave velocity, advanced glycation end-products, soluble receptor for advanced glycation end-products, vitamin K, genetic polymorphisms