# Introduction

The endothelium is the monolayer of endothelial cells lining the lumen of all blood vessels ***(Verma and Anderson, 2002).***

The endothelium is an active dynamic organ that fulfills many crucial roles. *First*, it is a physical barrier, ultra-structurally defining the components of the vessel wall and the contents of the vessel lumen. *Second*, this barrier affords movement of some small solutes (O2, glucose, etc.), in preference to large molecules (e.g., albumin), therefore, involved in tissue autoregulation .*Third*, the endothelium affords an antithrombotic environment and mediates vasoactivity. *Finally*, the endothelium mediates adherence of platelets and white blood cells to the vessel wall during injury and inflammation, respectively ***(Stevens T et al., 2001).***

Endothelial function may be tested by two approaches: *The first* is functional in nature while *the second*, approach rests on the measurement of the plasma concentration of specific biomarkers ***(Zoccali, 2006).***

The definition of endothelial cell dysfunction, its pathophysiology and therapy remain poorly defined. Based on the diversity of endothelial functions, it is logical to expect that the definition of the syndrome of endothelial cell dysfunction (ECD) should be broad enough to encompass disturbances in all its functions ***(Edmond et al., 2005).***

The pathophysiology of endothelial dysfunction is complex and involves multiple mechanisms. However, some of these seem to be common to most conditions : reduced nitric oxide (NO), elevated asymmetric dimethylarginine , oxidative excess, angII, OXLDL, hyperhomocysteinemia, diabetes, alterations in the local balance of

angiogenic and endothelial survival factors, the adipose tissue as a prime-time player in endothelial dysfunction and the endothelin system ***(Dierk H et al., 2004).***

The most common causes of CKD are atherogenic diseases (hypertension, dyslipidemia, and type 2 diabetes), diseases in which the underlying histologic alteration is commonly represented by nephroangiosclerosis ***(Go et al., 2004).***The most typical finding in nephroangiosclerosis is represented by intimal hyperplasia of medium and small renal arteries. Such an alteration is the expression of systemic endothelial damage being extended to the whole arterial system, from small vessels to the aorta.Therefore endothelial damage seems to be the basic anatomic alteration that eventually leads to disastrous vascular events in the kidney. The pathophysiology of endothelial dysfunction involves multiple mechanisms ***(Bonetti et al., 2003).***

Uremia is associated with increased vascular risk and premature death in both dialysis- and nondialysis-dependent patients and may be up to one hundred fold in certain patient groups. Uremic vascular disease is macro and microvascular, and is associated with reduced arterial compliance ***(Meuus et al., 2000).***

Emerging evidence suggests that endothelial dysfunction, oxidative stress, vascular calcification, and inflammation are strongly interrelated and together play a major role in the initiation and progression of vascular disease in CKD ***(Stenvinkel et al., 2008).***

Treatment of the underlying disease may restore endothelial function ***(Passauer et al., 2003).***

Other possible lines of therapy will be discussed through our work.