Abstract
Renal alterations related to hypertension are so intrinsic to the kidney that they can be transplanted with the organ. Normotensive recipients that receive a kidney transplant from a hypertensive offspring donor become hypertensive and vice versa. Oxidative stress (OX-ST) is stimulated when plasma levels of angiotensin II (Ang II) become inappropriate compared to total body sodium or vice versa. OX-ST potentiates the vasoconstrictor effects of Ang II decreasing nitric oxide, and/or stimulating vasoconstrictors such as isoprostanes, endothelins, etc. These effects are present in the so called "slow responses" to Ang II, where the prolonged infusion of sub-pressor doses of Ang II induces sodium retention and OX-ST, sensitizing the organism to vasoconstriction. These effects are mediated by a set of intracellular signals, the most important of which seems to be the Ang II induced activation of Src protein and epidermal growth factor. The production of superoxide induced by these factors could be sustained by an auto-catalytic reaction that accounts for vasoconstriction.

Keywords