THE SUPEROXIDE DISMUTASE AND CATALASE - LIKE ACTIVITIES OF ROSMARINIC ACID IN L-NAME INDUCED HYPERTENSIVE NEPHROPATHY

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Background Spontaneously hypertensive rats (SHR) chronically treated with an inhibitor of NO synthase, NG-nitro-L-arginine methyl ester (L-NAME), develop chronic kidney disease characterized by massive albuminuria, proteinuria, arteriolar fibrinoid necrosis and glomerular sclerosis. *Previously, we showed that rosmarinic acid (RA) supplementation could improve albuminuria in L-NAME/SHR model of hypertensive nephropathy* independently of NO bioavailability. Since albuminuria is associated with increased oxidative stress, we evaluated *whether chronic consumption of RA could affect kidney oxidative stress in L-NAME/SHRs by altering the antioxidant enzyme expression and activity.*

Experimental groups

Adult of SHR ~300 g

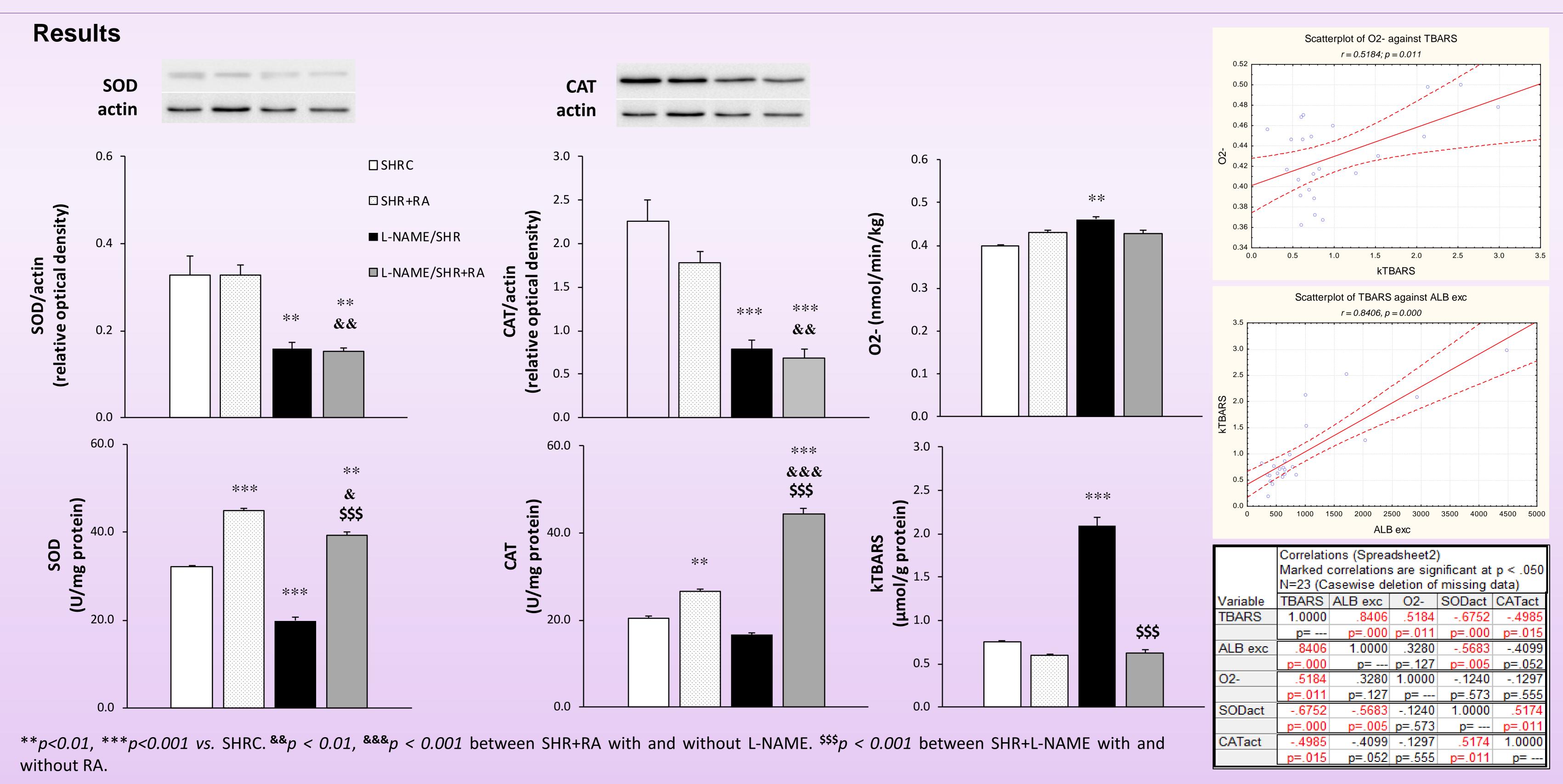
→ SHRC, tap water → SHR+RA, rosmarinic acid, 15mg/kg/day (RA dissolved in

1 ml 1% aqueous-ethanolic solvent) by gavage
→ L-NAME/SHR, 10 mg/kg/day of L-NAME in drinking water

L-NAME/SHR+RA, 10 mg/kg/day of L-NAME in drinking water + 15mg/kg/day of RA dissolved in 1 ml 1% aqueousethanolic solvent by gavage

Methods

- urine albumin (for determination of urinary albumin excretion - ALBexc), kidney superoxide anion (O₂⁻), kidney TBARS (kTBARS), superoxide dismutase (SOD) and catalase (CAT) activity were measured *spectrophotometrically*
- kidney SOD and CAT protein expressions were detected by western blot



Conclusion Our results indicate to a close link between oxidative stress and renal dysfunction in hypertensive nephropathy and

that the antioxidant efficacy of RA stems from its SOD and CAT mimetic activities.

4 weeks

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