

MODIFICATION OF GLOMERULAR ALBUMIN PERMEABILITY IN RAT ISOLATED GLOMERULUS BY RENIN-ANGIOTENSIN-ALDOSTERON BLOCKADE

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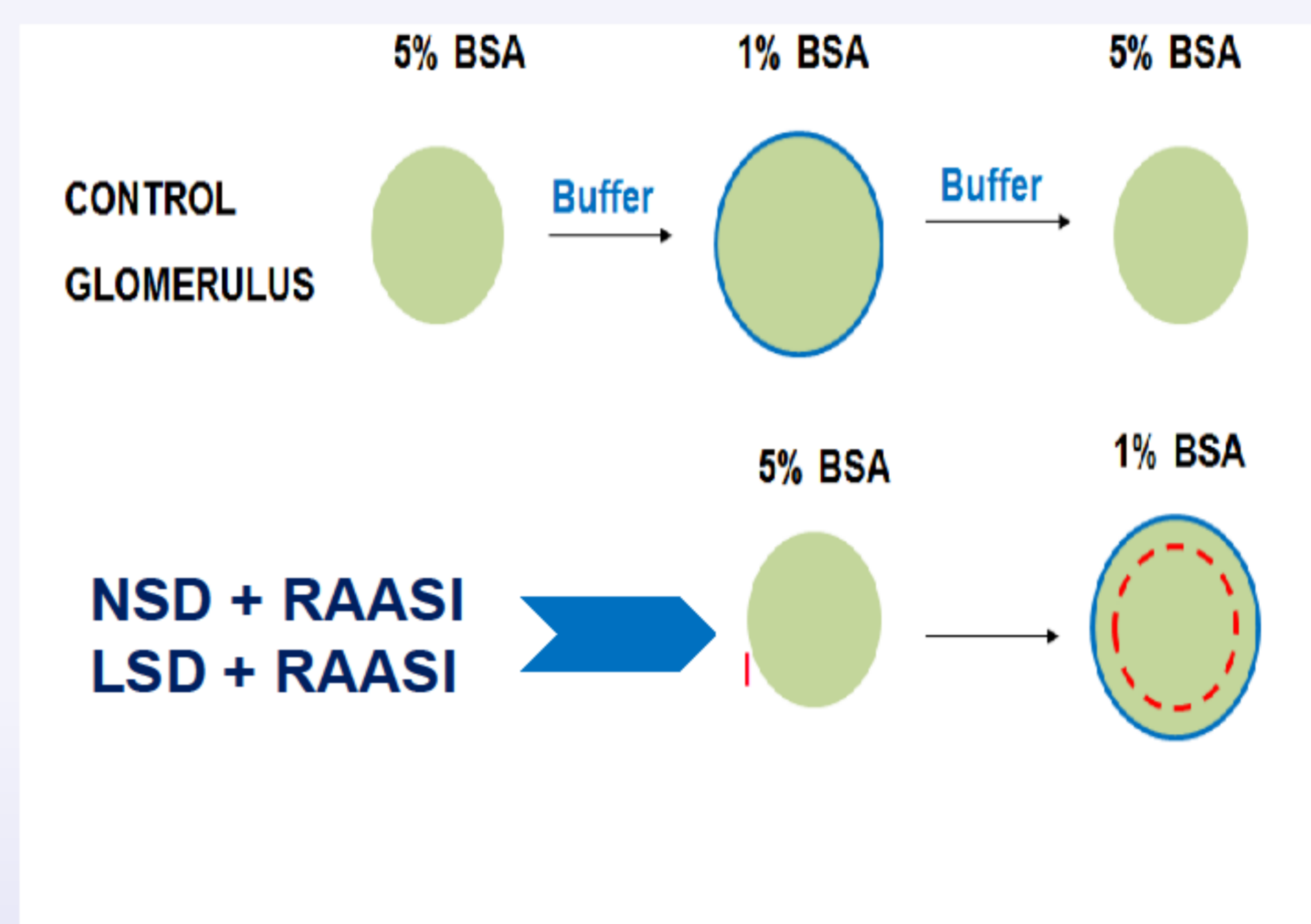
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Introduction and objectives

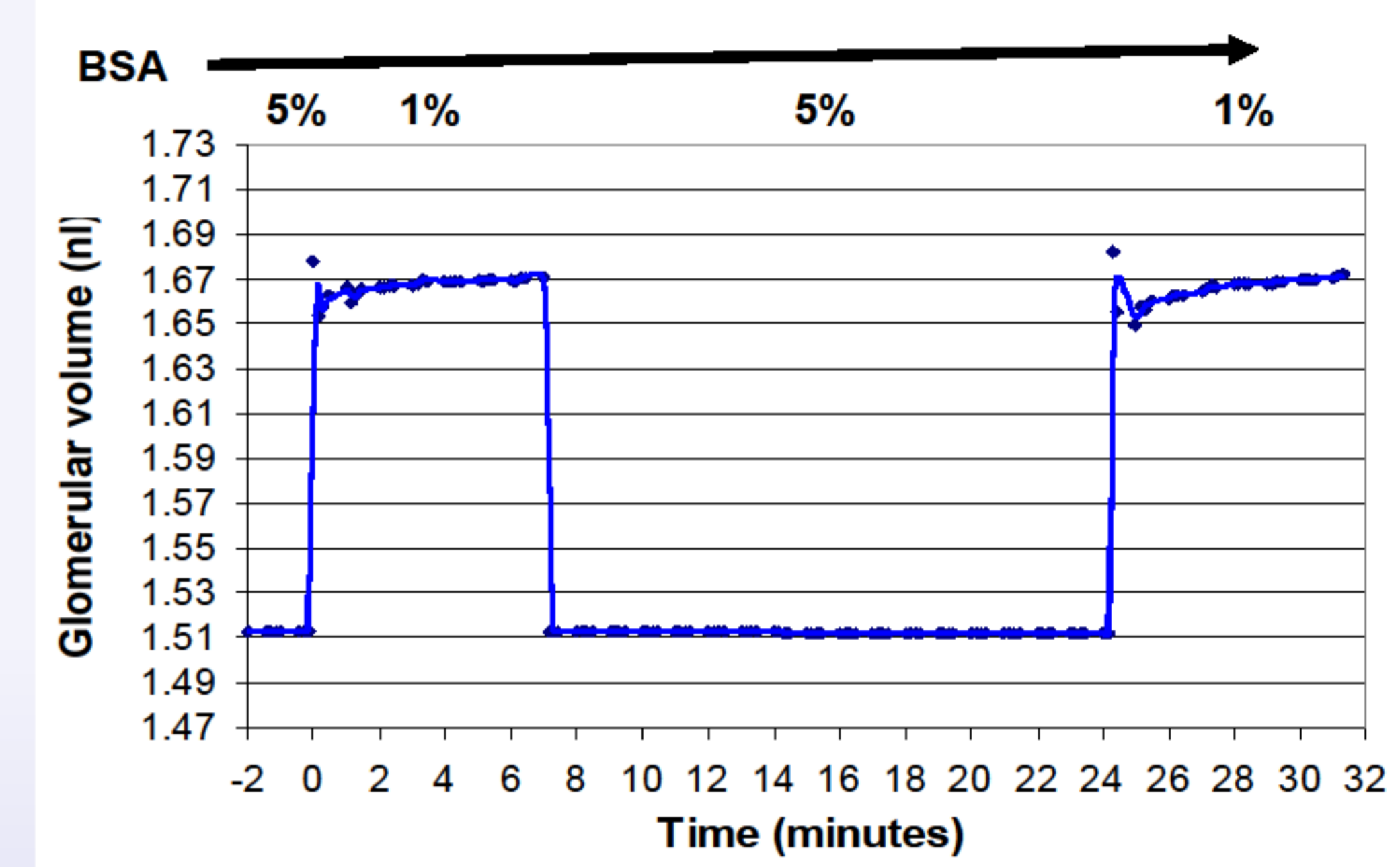
Glomerular filter consisting of endothelial cells, basement membrane and podocytes prevents plasma proteins e.g. albumin from entering the urinary space, an independent risk factor for the progression of renal failure. Activity of these cells is under control of renin-angiotensin-aldosteron system (RAAS), thus pharmacological inhibition of RAAS may affect the properties of glomerular filter.

Methods

Experiments were performed on Wistar rats maintained on normal/low sodium diet (NSD, LSD, 5 days) and RAAS inhibitors (RAASI, 7 days, p.o): aliskiren (4.3 mg/kg/24h), enalapril (0.14 mg/kg/24h), telmisartan (0.6 mg/kg/24 h) or eplerenone (0.36 mg/kg/24 h). Glomeruli were isolated and single affixed glomerulus was continuously observed with use of video-microscopy (Olympus, IX 51). Changes in the glomerular volume due to oncotic pressure gradient eliciting transglomerular fluid flux were used to calculate convectional albumin permeability (Palb). Nephritin and albumin in urine were measured by ELISA.



Measurement of changes of glomerular volume

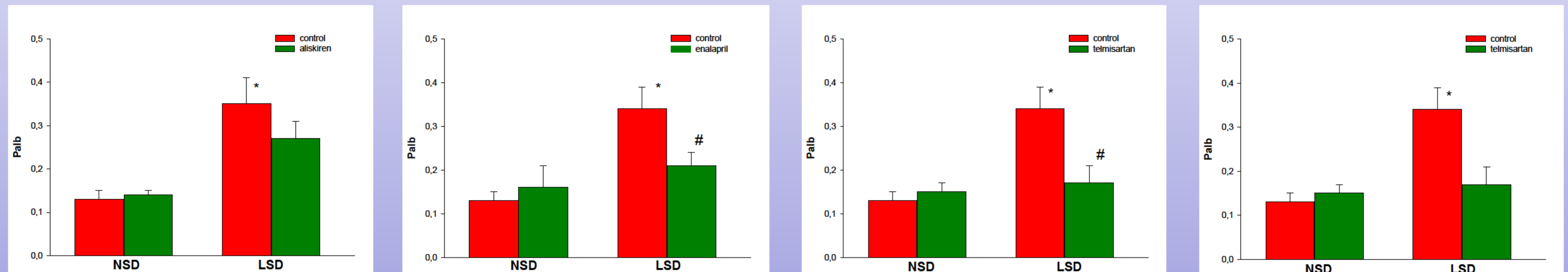


Representative recording of single glomerular volume changes in response to oncotic pressure medium

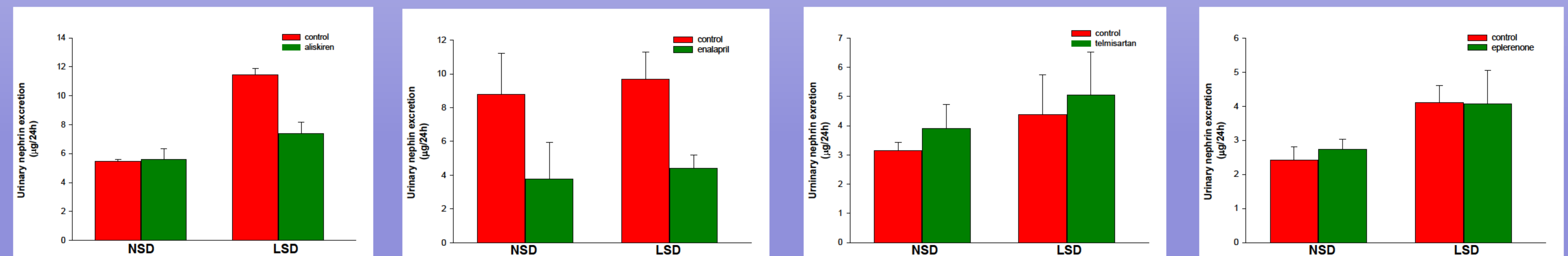
Results

Convectional albumin permeability

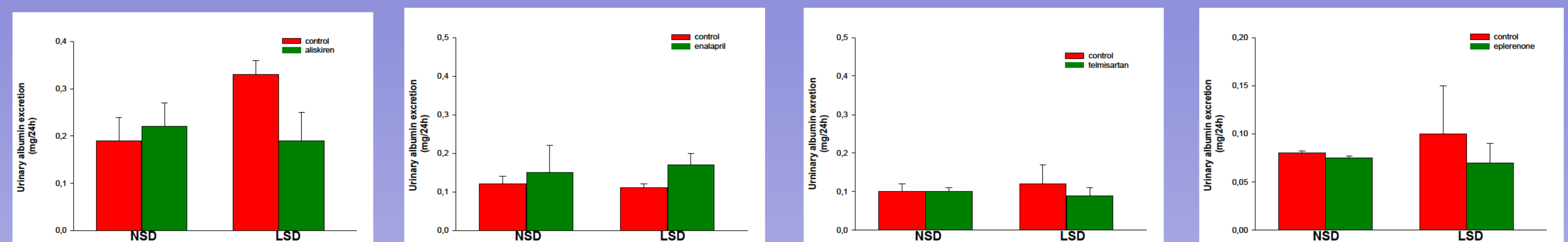
P<0.05, *vs control NSD, # vs control LSD



Nephritin excretion with urine



Albumin excretion with urine



RAAS blockade affects glomerular permeability for protein but its final urinary excretion may be modulated by postglomerular processes

