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The pathophysiological roles of peroxynitrite in salt-sensitive hypertension

Dewan Syed Abdul Majid
Tulane University School of Medicine, USA

Normally, superoxide (O^{2-}) remains at minimal levels in tissues as it reacts with nitric oxide (NO) to form peroxynitrite ($ONOO^-$). Although studies have demonstrated a reciprocal regulation of renal tubular sodium (Na+) reabsorption by NO and O^{2-}, the specific role for $ONOO^-$ in the regulation of renal sodium excretion is not yet clearly defined. It has been demonstrated that an interaction between NO and O^{2-} forming $ONOO^-$ plays an important reno-protective role in the kidney which helps to prevent excessive tubular Na+ reabsorption in conditions such as in elevated renin-angiotensin system. However, its regulation in various pathophysiological conditions, particularly in salt sensitive hypertension is not yet clarified. $ONOO^-$ formation is increased by Angiotensin II (AngII) as well as by High Salt (HS) intake as both of these stimulate both NO and O^{2-} production. However, conditions such as impairment in NOS activity, its pharmacological inhibition or gene deletion, reduces the formation of $ONOO^-$. Recent findings show that chronic AngII with HS intake result in aggravated hypertension and renal injury in endothelial NO synthase knockout mice (a model for minimal $ONOO^-$ formation) compared to those in wild-type mice that suggest a protective role for $ONOO^-$ in these adverse effects of AngII. This talk will present evidence from different studies in our laboratory and others implicating the functional roles of $ONOO^-$ in a coordinated regulation of kidney function, an imbalance of which could be involved in the patho-physiology of salt-sensitive hypertension.

majid@tulane.edu