INTRODUCTION

The classical renin-angiotensin system **(RAS)** consists of renin, angiotensin-converting enzyme and angiotensinogen . ***(Hackenthal et al.,1990).***

Renin belongs to the family of aspartyl proteases and has only one known substrate, angiotensinogen, the precursor of all angiotensin peptides. ***(Morris , 1992).***

Angiotensinogen, is abundantly produced in the liver, but also has been identified in multiple tissues, including adipose tissue, heart, vasculature, brain and kidney. ***(Dickson and Sigmund, 2006).***

ACE likely represents the major, if not sole enzyme responsible for Ang II formation under normal physiological conditions in humans and other species. ***(Sadjadi et al., 2005a; Tokuyama et al., 2002).***

Angiotensin II receptors **AT1R** and **AT2R** have been identified as G Protein-coupled receptors which however do not share the same intracellular signaling pathways and spectrum of biologic activity. ***(Konishi et al., 1994).***

The circulating RAS is best known for its role as a regulator of blood pressure, and fluid and electrolyte homeostasis. ***(Timmermans et al .,1992).***

The first suggestion of a relatively independent human skeletal muscle ACE arose from vastus lateralis muscle biopsy specimens that demonstrated muscle ACE activity did not correlate with serum ACE. ***(Reneland and Lithell , 1994).***

The vascular endothelium accounts for much of the angiotensin II production and it is conceptually reasonable to consider skeletal muscle angiotensin II a product of the skeletal muscle vascular bed. ***(Ohishi et al., 1997; Phillips et al., 1993)*** .

It may be that a reduction in ACE has local muscle effects via the skeletal muscle RAS that increase muscle efficiency and contribute to the enhanced endurance associated with the I allele. ***(Kanazawa et al., 2002).***

The inverse relationship between blood pressure and muscle ACE levels indicate that muscle tissue ACE levels are influenced by haemodynamic factors***.(Reneland and Lithell , 1994).***

Animal studies have suggested that fibre composition of skeletal muscle may be linked to insulin resistance ***(Ueda et al., 1995).***

It becomes apparent that part of both the clinical state of CHF, with an upregulated RAS, and part of the mechanism of response to treatment is mediated by effects localized to skeletal muscle with the skeletal muscle RAS. ***(Schaufelberger et al., 1998).***

ACE inhibitors could work by preventing mitochondrial decline and improving endothelial function and muscle metabolism. So sarcopenia may be a potential therapeutic target for ACE inhibitors***.(Sumukadas et al., 2006) .***