

# Proteinuria and Angiotensin Converting Enzyme (ACE) Gene Polymorphism

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## Abstract

**Introduction:** Although proteinuria is traditionally regarded as the result of glomerular damage, recent evidence suggests that proteinuria can directly cause renal damage through lysosomal injury, growth factor induced tubular fibrosis and transcriptase genes which trigger vasoactive and inflammatory renal damage. The renin angiotensin system which releases angiotensin, a most powerful vasoconstrictor and mediator of mesangial cell proliferation and matrix expansion, has been associated with progressive renal injury. This paper reviews the role of angiotensin converting enzyme (ACE) gene polymorphisms in the genesis of progressive renal failure, as well as their role in the response of proteinuria to ACE inhibitor (ACEI) or angiotensin receptor antagonist (ATRA) therapy. **Methodology:** The deletion/insertion polymorphism of the ACE gene has 3 genotypes DD, ID and II. Reported studies on the association of ACE gene polymorphisms with patients with IgA nephritis and progressive renal failure, including their response to ACEI or ATRA therapy, were reviewed. **Results:** Individuals with the DD genotypes have a higher risk of declining renal function with progressive IgA nephritis and more rapid deterioration to renal failure. Therapy with ACEI and or ATRA may be beneficial in patients with IgA nephritis with renal impairment and non-selective proteinuria as such patients may respond to therapy with improvement in protein selectivity, decrease in proteinuria and improvement in renal function. **Conclusion:** ACE genotypes appear to predict antiproteinuric response to ACEI therapy. ACEI/ATRA therapy probably modifies pore size distribution by reducing the radius of large non-selective pores, causing the shunt pathway to become less pronounced resulting in less leakage of protein into the urine.

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**Key words:** Genotypes, Glomerulonephritis, Protein selectivity, Renal injury, Therapy

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