

## **Association of Polymorphic Antioxidant Enzymes with Dilated Cardiomyopathy**

**B. Ushasree<sup>1</sup>, S. Yasmeeen<sup>1</sup>, A. Venkateshwari<sup>2</sup>, C. Narsimhan<sup>3</sup>, R. K. Jain<sup>4</sup> and Pratibha Nallari<sup>1</sup>**

*1. Department of Genetics, Osmania University, Hyderabad 500 007, Andhra Pradesh, India*

*E-mail: ushasree\_b@hotmail.com*

*2. Institute of Genetics and Hospital for Genetic Disorders, Begumpet, Hyderabad 500 016, Andhra Pradesh, India*

*3. CARE Hospitals, Hyderabad, Andhra Pradesh, India*

*4. KIMS Hospitals, Hyderabad, Andhra Pradesh, India*

**KEYWORDS** Oxidative Stress. Superoxide Dismutase. Catalase. Alpha-1-Antitrypsin. Myocarditis. Electromorphs.

**ABSTRACT** Despite considerable public awareness and technological advances that foster early diagnosis and aggressive therapeutic interventions, heart failure, which results as a final outcome from an underlying cardiovascular disorder remains a critical and an unsolved problem. Among the various cardiomyopathies, Dilated cardiomyopathy with an obscure etiology is known to be the leading cause of heart failure and sudden cardiac death among young adults and children. Studies related to the molecular basis of the condition have implicated oxidative stress pathways, apart from primary disease causing sarcomeric, cytoskeletal and mitochondrial gene mutations in the disease onset. The present study aims to evaluate the role of oxidative stress markers in 97 DCM patients and 105 control individuals to identify specific electromorphic association of superoxide dismutase, catalase and alpha-1-antitrypsin with the disease. Our study has revealed an association of SODA2, catalase HPII and AAT 'M' and 'Z' alleles with DCM, thereby resulting in inefficient scavenging of the free radicals, which may confer decreased protection against oxidative stress induced tissue injury in the disease pathogenesis. The involvement of SOD and AAT in apoptotic pathway and as immunomodulators is also emphasized.