

Nephroprotective and antioxidant activities of *Tephrosia purpurea* L. on paracetamol and gentamicin induced albino rats

S. SATHYA AND P. VENKATALAKSHMI

Department of Biochemistry, S.T.E.T. Women's College, MANNARGUDI (T.N.) INDIA

E-mail: venkatalakshmisathish@gmail.com

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Nephroprotective and antioxidant activities of *Tephrosia purpurea* have been evaluated against paracetamol and gentamicin induced renal damage in male albino rats. Paracetamol (200mg/kg) and gentamicin (40mg/kg) induced renal damage was well manifested by significant increase in the levels of ALT, AST, ALP, urea, creatinine, sodium in serum. On the other hand, the levels of potassium, protein, albumin, enzymatic and non enzymatic antioxidants were lowered. The oral administration of varying doses of ethanolic extract of *Tephrosia purpurea* (5,10 and 15 mg/kg) for the period of 7 days reversed these altered parameters to normal levels indicating the antioxidative and nephroprotective efficacy of *Tephrosia purpurea* L. against paracetamol and gentamicin induced renal injury.

Key words : Acetaminophen, Carvedilol, Gentamicin and *Tephrosia purpurea*

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INTRODUCTION

Antioxidants are substances that markedly delay or prevent the oxidation of the substrate. Antioxidants may help the body to protect itself against various types of oxidative damages caused by reactive oxygen species, which are linked to a variety of diseases including cancer, diabetes, shock, arthritis, nephrotic syndrome and acceleration of the ageing process. Oxidation is a chemical reaction that transfers electrons from a substance to an oxidizing agent. Oxidation reactions can produce free radicals, which start chain reactions that damage cells. Free radicals may also be involved in a number of diseases and tissue injuries (Shahidi, 1997). Antioxidants terminate these chain reactions by removing free radical intermediates, and inhibit other oxidation reactions by being oxidized themselves. As a result, antioxidants are often reducing agents such as thiols, ascorbic acid or polyphenols.

Antioxidants may act by decreasing singlet oxygen concentration, intercepting singlet oxygen, preventing first chain initiation by scavenging initial radicals, binding metal ion catalysts, decomposing primary products to non-radical compounds, and chain breaking to prevent continued hydrogen abstraction from substrates. The hydroxyl

radicals derived from superoxide radicals and hydrogen peroxide is the most potent reactive oxygen radical which causes DNA damage (Gutteridge, 1984).

Nephrotoxicity can be defined as renal disease or dysfunction that arises as a direct or indirect result of exposure to medicines, and industrial or environmental chemicals. It is well established that toxic nephropathies are not restricted to a single type of renal injury. The renal response to injury is dynamic, and the kidney adapts to maintain homeostasis during the cascade of repair and recovery that follows the primary insult (Bach *et al.*, 1989). Depending on the type and frequency of the damage, and the region of the kidney that is damaged, the organ can respond by a recovery, a reduced functional reserve, or by a progressive degenerative change.

Gentamicin, an aminoglycoside class of bactericidal antibiotic, is effective against Gram-negative bacterial infections (Martinez-Salgado *et al.*, 2007). In spite of inducing nephrotoxicity, gentamicin is used clinically due to its wide spectrum of activities against Gram-negative bacterial infections caused by *Pseudomonas*, *Proteus*, and *Serratia* (Del Valle *et al.*, 1969; Miglioli *et al.*, 1999; Hendriks *et al.*, 2004). The gentamicin – induced nephrotoxicity occurs by selective