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EVALUATION OF ANTIULCER AND ANTIOXIDANT ACTIVITIES OF MURRYA KOENIGI AND JATROPHA CURCAS IN ALBINO RATS

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ABSTRACT

India has a wide variety of medicinal plants. Medicinal plants synthesize variety of phytochemicals that help to defend against various types of neoplasm. Due to environment pollution and feeding habits of people, risk of generation of Free radicals and reactive oxygen species is also increasing. These free radicals are responsible for generation of various ulcers, tumors and Neoplasms. The phytochemicals present in *M. koenigi* and *J.curcas* has antioxidant effects which may prevent against various Neoplasms. Use of plants as a source of medicine has been an ancient practice and is an important component of health system in India. General awareness, academic and

government interest in traditional medicine is growing rapidly due to increased side effects of adverse drug reactions and cost factors of modern system of medicine. The present study focuses on evaluation of anticancer and antioxidant activities of *Murrya koenigi* and *Jetropha curcas* plant extracts.

KEYWORDS: *Murrya koenigi* and *Jetropha curcas*.

INTRODUCTION

Cancer is also called Neoplasm and the growth of cancer is called Neoplasia. The term neoplasia means "new growth". Thus, the term Neoplasm may be defined as – 'A mass of tissue formed as a result of abnormal, excessive, uncontrolled, uncoordinated, autonomous proliferation of the cells.' Neoplasm may be Benign when they are slow growing and localized without causing much effect on the host or Malignant when they proliferate rapidly, spread throughout the body and eventually cause death of the host.

The common term used for the entire malignant tumor is Cancer. Hippocrates (460-377 BC), coined the term Karkinos for the cancer of breast. The word Cancer means Crab, thus reflecting the true characteristic of cancer, since it sticks to the affected part stubbornly like a crab.

TYPES OF PEPTIC ULCER

Duodenal Ulcers (DU), Gastric Ulcers (GU), Stress Ulcers (SU), NSAID Induced Ulcers 1.1. PEPTIC ULCERS AND HELICOBACTER PYLORI

H.pylori a gram negative bacteria that colonise the gastric mucosa and has been found in the cases of chronic gastritis and peptic ulcers. Its prolonged infection may lead to Lymphoma and Gastric carcinoma.

RESULT AND DISCUSSION

Table 1.2 Evaluation of antiulcer activity of *Murrya koenigi and Jetropha curcas* leaf extracts by Ethanol-inducd ulcer model.

Groups	SOD	CATALASE	GSH
Group-1			
Rat-1	1	1	1
Rat-2	1	1	1
Rat-3	1	1	1
Rat-4	1	1	1
Rat-5	1	1	1
Rat-6	1	1	1
Group-2			
Rat-1	3	2	3
Rat-2	3	3	3
Rat-3	2	3	3
Rat-4	3	3	3
Rat-5	3	2	3
Rat-6	3	3	3
Group-3			
Rat-1	2	2	2
Rat-2	2	2	0
Rat-3	2	1	2
Rat-4	1	0	1
Rat-5	2	1	2
Rat-6	1	2	0
Group-4			
Rat-1	1	1	1
Rat-2	1	1	0
Rat-3	2	1	1
Rat-4	1	1	0
Rat-5	1	1	0

Rat-6	0	0	0
Group-5			
Rat-1	0	1	1
Rat-2	0	0	1
Rat-3	1	0	2
Rat-4	0	1	1
Rat-5	0	1	1
Rat-6	0	1	1

TABLE 1.3 Evaluation of Antioxidant levels in Ethanol induced peptic ulcer -

Groups	Body Wt. (gm)	Treatment	Ulcer Index					Total Score	
			Normal Stomach	Red Coloration	Spot Ulceration	Hemorrhagic Streaks	Ulcer	Perforation	
1.	180	Normal Control	0	0.5	-	-	-	-	0.5
	175		0	0.5	-	-	-	-	0.5
	190		0	0.5	-	-	-	-	0.5
	205		0	-	-	-	-	-	0
	200		0	-	-	-	-	-	0
	195		0	0.5	-	-	-	-	0.5
	185		-	0.5	1.0	1.5	2.0	-	5.0
	180		-	0.5	1.0	1.5	2.0	-	5.0
	190	Control	-	0.5	1.0	1.5	2.0	-	5.0
2.	195		-	0.5	1.0	1.5	2.0	3.0	8.0
	198		-	0.5	1.0	1.5	2.0	-	5.0
	200		_	0.5	1.0	1.5	2.0	-	5.0
3.	180	Standard (20mg/kg)	-	0.5	1.0	-	-	-	1.5
	220		-	-	-	-	_	-	0
	191		-	0.5	1.0	-	-	-	1.5
	198		-	0.5	-	-	-	-	0.5
	205		-	0.5	-	-	-	-	0.5
	200		_	-	-	-	-	-	0
4.	220		-	0.5	1.0	1.5	-	-	3.0
	180		-	0.5	1.0	-	-	-	1.5
	190	Methanolic Extract of	-	0.5	1.0	-	-	-	1.5
	195	M.koenigi (100mg/kg)	-	0.5	-	-	-	-	0.5
	180		-	0.5	-	-	-	-	0.5
	210		-	0.5	-	-	_	-	0.5

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5.	220	Methanolic extract of J.curcas	-	0.5	-	-	-	-	0.5
	180		-	0.5	1.0	-	-	-	1.5
	190		-	0.5	1.0	1.5	-	-	3.0
	195		-	0.5	-	-	-	-	0.5
	180		-	0.5	1.0	-	-	-	1.5
	210		-	0.5	1.0	-	-	-	1.5

(key: 0=negligible level, 1=Mild level, 2=Moderate level, 3=High level)

4.1 DISCUSSION

Peptic Ulcer Disease (PUD) encompassing gastric and duodenal ulcer is the most prevalent chronic gastrointestinal disorder and is inflammatory in nature, Valle (2008). The pathophysiology of PUD involves an imbalance between offensive or injurious (acid, pepsin and *Helicobacter pylori*) and defensive mucosal factors (mucin, prostaglandin, bicarbonate, nitric oxide and growth factors), Wallace *et al.*, (2011).

Most widely used method for producing experimental gastric ulcers is Ethanol induced, as it is suitable for first line antiulcer screening, because the agents are retained in the stomach and may act by a variety of mechanisms, Vimlesh *et al.*, (2013). Reproducibility and high incidence of ulceration has been reported by this method. A major advantage of this method is that one can measure gastric secretory rate, percentage ulceration and ulcer severity in the same animal.

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Intragastric application of absolute ethanol is a reproducible method to produce gastric lesion in experimental animals, Robert *et al.*, (1979) and Szabo *et al.*, (1980). The protective effect against various irritants has been called cytoprotective activity, Robert (1979). The concept of gastric cytoprotection signifies protection against mucosal injury by a mechanism other than inhibition of acid secretion was introduced long ago, Rober *et al.*, (1979) and Vogel (2008). In the ethanol-induced gastric ulceration model, ethanol produces necrotic lesions by direct necrotizing action which in turn reduces defensive factors like the secretion of bicarbonate and production of mucus, Marhuenda *et al.*, (1993). Ethanol-induced gastric lesions impaired gastric defensive factors such as mucus and mucosa circulation, Ferreira *et al.*, (2008). Ethanol causes necrotic lesions of the gastric mucosa in a multifactorial way. It can reach the mucosa by disruption of the mucus-bicarbonate barrier and cause cell rupture in the wall of blood vessels. These effects are probably due to biological actions, such as of lipid peroxidation, formation of free radicals, intracellular oxidative stress, changes in permeability and depolarization of the mitochondrial membrane prior to cell death, Sannomiya *et al.*, (2005).

Oral administration of absolute ethanol is noxious to the stomach since it affects the gastric mucosa topically by disrupting its barrier and provoking pronounced microvascular changes within a few minutes after its application, Moleiro *et al.*, (2009). In addition, it produces linear hemorrhagic lesions, extensive submucosal edema, mucosal friability, inflammatory cells infiltration, and epithelial cell loss in the stomach, which are typical characteristics of alcohol injury, Jelski *et al.*, (2009). The pathogenesis of ethanol-induced gastric mucosal damage occurs directly and indirectly through various mediators such lipoxygenase, cytokines and oxygen-derived free radicals, Abdel-Salam *et al.*, (2001). Mucus secretion is regarded as a crucial defensive factor in the protection of the gastric mucosa from gastric lesions, Oluwole *et al.*, (2008). Thus in the present study, it could be assumed that the existence of the cytoprotective effect of compound is present in the methanol extract of the *Murrya koenigi and Jatropha curcas* leaves because it showed the significantly less ulcer index in the observation and high mucin concentration when the results were compared with the control group.

An antioxidant is any substance that retards or prevents deterioration, damage or destruction by oxidation, Dekkers *et al.*, (1996). A free radical is a compound with one or more unpaired electrons in their outer orbital, Jesberger *et al.*, (1991). The most dangerous free radicals are

the atomic and molecular varieties of oxygen which is known as Reactive Oxygen Species (ROS). While ROS are not technically free radicals, they are highly reactive with the molecules around them, Sharma *et al.*, (1996). To neutralize these free radicals antioxidants play an important role, Vani *et al.*, (1997). Antioxidant enzymes such as super oxide dismutase (SOD), Catalase and Glutathione peroxidases are known to attenuate the generation of ROS by removing potential oxidants or by transforming ROS and Reactive nitrogen species into stable compounds, Ashok, (2001).

The level of CAT, SOD and GSH was significantly decreased in diseased control group as compared to normal group. Administration of methanolic extract of both the plants had shown significant increased in the levels of CAT, SOD and GSH as compared to the diseased control animals, which suggests its efficacy in preventing free radical-induced damage.

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