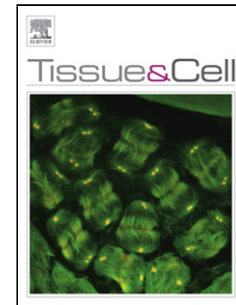


## Accepted Manuscript

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# GASTROPROTECTIVE AND MUCOSA HOMEOSTATIC ACTIVITIES OF COCONUT MILK AND WATER ON EXPERIMENTALLY INDUCED GASTROPATHIES IN MALE WISTAR RATS

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## ABSTRACT

In this biphasic study, 45 male wistar rats were divided into 9 groups. In Phase 1, Group 1 was treated with normal saline and served as the overall control, group 2 was treated with 95% Ethanol and represents the ulcer control, groups 3 and 4 received coconut water (Cw; 4ml/100g BWt) and milk (Cm; 4ml/100g BWt) for 4weeks while group 5 received Omeprazole (Omeprazole; 20mg/kg BWt) during terminal week. 95% Ethanol-induced ulceration followed the treatments in all except group 1. In the second phase, Group 1 was the overall control, group 2 served as ulcer control by receiving acetic acid only, group 3 received, coconut milk, and group 4 received omeprazole. Cm and omeprazole were administered post-ulcer induction for 3 and 6 days twice daily. Blood collection after 1hour was through cardiac puncture for haemocytometry, and gastric tissues harvested for histopathological investigations. Results showed significantly reduced ulcer score and gastric lesion index in Omeprazole, Cw and Cm groups compared to ulcer control. WBC, neutrophil, lymphocyte counts in Omeprazole, Cw and Cm groups were significantly reduced compared to ulcer and overall control groups. C-reactive protein was significantly reduced in Cm compared to control. Neutrophil Infiltration score reduced while mucus cell density increased significantly in Omeprazole; Cm compared to control. EGFR and CD 31 assessment revealed significantly higher expressions in coconut-milk group compared to the ulcer control. We conclude that the protective effects of coconut (water and milk) is expressed by inflammation suppression, upregulation of mucus cell population and catalyses mucosa homeostasis via angiogenesis and mucosal cell proliferation following mucosa. erosion.

Keyword: coconut, gastric ulcer, immunohistochemistry, inflammation.

## INTRODUCTION

Peptic ulcer disease is one of the most deleterious diseases known to man. According to the most recent world health ranking by the World Health Organisation, it solely accounts for about 262,911 deaths globally establishing it as the 43<sup>rd</sup> cause of death worldwide. It is also the leading cause of death in many African and Asian countries including Nigeria where it is rated 27<sup>th</sup> cause of death [1].

Peptic ulcer, also known as gastric ulcer, is often considered an alteration in the normal mucosa cyto-architecture outstretching the muscularis mucosa into the submucosa and sometimes deeper into the outermost serosa [2]. Gastric mucosa defence is established by an array of factors such as mucus, bicarbonate, micro-circulation and phospholipid barrier [3]. Gastric mucosal defence is supported by mechanisms activated both by the central nervous system and hormonal factors [4]. Gaseous mediators such as hydrogen sulphide (H<sub>2</sub>S), Carbon monoxide (CO) and nitric oxide (NO) also significantly contribute to gastric mucosal protection as they have been implicated in a number of gastroprotective activities [5,6,7]. Mucosa disruptive factors, on the other hand, predisposing the

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