# Protective effect of Tea Seed (*Camellia oleifera* Abel.) Oil on Ketoprofen-induced Acute Gastrointestinal Ulcer in Rats

Gow-Chin Yen, Shu-Li Wu, Yu-Ting Cheng, Department of Food Science and Biotechnology, National Chung Hsing University, Taichung, Taiwan

Non-steroidal anti-inflammatory drugs (NSAIDs), including ketoprofen, are widely used to alleviate pain and inflammation in clinical medicine. Studies have indicated that gastrointestinal mucosal lesion is associated with mucosal lipid peroxidation and oxidative damage. Therefore, reducing oxidative stress may be an effective therapeutic strategy for preventing gastrointestinal ulcer. The oil of tea seed (*Camellia oleifera* Abel.) is used very common in China and Taiwan as cooking oil. Tea seed oil has also been reported with health effects for bowel, stomach, liver, and lung, etc., in traditional remedy. In the screening test of various commercial tea seed oils, we found that tea seed oil exhibited the potential antioxidant capacity and scavenging activity of reactive oxidative species (ROS), which may play an important role in gastrointestinal protection. We further evaluated the effects of tea seed oil on ketoprofen-induced acute gastrointestinal ulcer in rats. The data showed that treatment of Sprague-Dawley (SD) rats with tea seed oil (1 ml/kg/day) prior to the administration of ketoprofen (100 mg/kg/day) could inhibit inflammatory COX-2 and NF-κB mRNA expression, nitrite oxide (NO) and interleukin-6 (IL-6) production, and also decreased oxidative damage and reversed the impairment of the glutathione peroxidase (GPx), glutathione S-transferase (GST), superoxide dismutase (SOD) antioxidant system in the intestinal mucosa. Moreover, the pre-treatment of SD rats with tea seed oil (1 ml/kg/day) strongly inhibited the induction of gastrointestinal mucosa injuries by ketoprofen, as demonstrated by the histopathological staining of stomach and intestinal tissues. Our results support the possible use of tea seed oil as a dietary preventive agent against intestinal injuries from oxidative stress.