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Precaution and Restorative Capability of Flavonoids in Peptic Ulcers

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DESCRIPTION

Peptic ulcer disease is a typical digestive problem affecting 20% of the world population. The treatment of peptic ulcers is still being tested due to the limited efficacy and severe symptoms of currently available drugs. Thus, the common mixtures, thanks to other healing, natural and safe properties, become wellknown potential competitors in the prevention and treatment of peptic ulcers. Flavonoids, the most abundant plant polyphenols, are protective against peptic ulcer disease in vivo and in vitro. In this examination, we summarized the ulcer control capabilities and systems, as well as the bioavailability, suitability and safety of flavonoid monomers in the gastrointestinal tract. Flavonoids exert cytoprotective and regenerative effects not only by enhancing protective factors, such as body fluids and prostaglandins, but also by protecting against harmful factors through anti-proliferative activity their oxidizing, mitigating and antibacterial properties. Although controlled clinical trials are limited at this time, flavonoids have been shown to be promising, preventable, and useful in the treatment of peptic ulcers. Peptic ulcer is described as mucosal rupture caused by erosion or leakage of pepsin in the gastrointestinal tract, especially the stomach and proximal duodenum. In addition, peptic ulcers penetrate through the mucosal layer causing mucosal ulcers, making the digestive system worse. Until the end of the twentieth century, the bacterium Helicobacter pylori (H. pylori) and the use of Nonsteroidal Anti-Inflammatory Drugs (NSAIDs) are considered major risk factors for peptic ulcer disease. Therefore, various circumstances, such as ischemia, Inflammatory Bowel Disease (IBD) and kidney disease, as well as unhappy lifestyles, including blood pressure, smoking, and excessive consumption of caffeine or alcohol, are also considered risk factors for gastric disease, ulcer disease. Recently, a high prevalence of 20% has been attributed to peptic ulcers, mainly seen in people between the ages of 30 and 60. Although the mortality rate from peptic ulcer disease is low, it is widespread and causes great suffering and inconvenience. Patients with peptic ulcer disease

commonly experience side effects such as epigastric pain, such as eating or biting, and common dyspeptic side effects such as swelling, nausea, bloating, and indigestion. On the other hand, some patients may experience confusion, such as death, perforation, and obstruction of the gastric tube. Of these, drainage is the most frequent drawback with expansion rate dependent on 15%, which can be dangerous. Perforations occur frequently and cause severe pain in the stomach area. In addition, the dilation and scarring cause the duodenum to contract, which can cause the gastric outlet to be checked. In these conditions, the patient may experience severe vomiting and even regurgitation. One tends to find that the complexity of this infection affects specifically the nature of the patient's life and furthermore makes the development of safe and viable drugs extremely fundamental. Flavonoids may also mediate the factor 2 (Nrf2)/ heme oxidase-1 (HO-1) pathway that binds to the erythrocyte atomic component 2 to act on oxidative stress. Nrf2 is an important registrar directing phase II detoxification and up-regulating HO-1 cytoskeletal enhancer quality. Upregulation of the HO-1 joint leads to increased accumulation of iron, bilirubin, and carbon monoxide, thereby reducing gastrointestinal cell reactivity to oxidative damage. Treatment of Int-407 cells with 100 µM catechin reduces ROS and lipid peroxidation, expands the migration of anticancer agent compounds, and upregulates the atom/cytosol ratio Nrf2 and HO-1 protein coupling in an accessory manner in a phase-contrast and ketoprofen-detecting aggregation patterns. Sprague-Dawley rodents pretreated with 35 mg/kg catechin for 21 days prior to ketoprofen also showed a decrease in gastric ulcer area.

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CONFLICT OF INTEREST

Authors declare no conflict of interest.

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