Summary and Conclusion
Ulcerative colitis is an inflammatory disease characterized by diffuse inflammation in the colon mucosa and sub-mucosal layers. The etiology of inflammatory bowel disease is not clearly understood. Dietary fatty acids are known to modulate the innate immune functions by a myriad of ways including alteration of lipid microenvironment in the membranes, apoptosis of immune cells, gene expression and eicosanoid synthesis. However, the efficiency of the alterations induced on the immune functions depends on the type of fatty acids.

Ulcerative colitis was induced by administering 5% (w/v) dextran sulfate sodium (DSS) in drinking water for 7 days in adult rats. Rats administered with DSS manifested adverse effects such as loss of body weight, severe diarrhea, rectal bleeding, mortality and shortening of colon length in rats. There was significant increase in colon myeloperoxidase activity, which was correlated to increased infiltration of neutrophils into the colon tissue. The colon antioxidant defense system such as catalase, superoxide dismutase, glutathione S-transferase enzyme activities and glutathione levels were reduced in colon. Histological examination of colon tissue sections of DSS-treated rats revealed that there was a denuded surface with severe ulceration, extensive loss of goblet cells, crypt distortion, edema and massive infiltration of inflammatory cells including neutrophils and lymphocytes. 5% DSS for 7 days successfully and consistently induced colitis with characteristic macroscopic, microscopic and biochemical alterations in rats.

The main objectives of the study was to assess the influence of dietary oils rich in saturated fatty acid, monounsaturated fatty acid, polyunsaturated fatty acid and long chain- polyunsaturated fatty acids (LC-PUFA) on inflammation in ulcerative colitis. We have selected medium chain triglycerides containing vegetable oil (coconut oil) as a source of saturated fatty acid, olive oil and rice bran oils as sources of monounsaturated fatty acids, sunflower oil as a source of n-6 PUFA, garden cress seed oil as a source of n-3 PUFA and fish oil rich in EPA and DHA as a source of LC-PUFA.

Dietary feeding of SFO rich in n-6 fatty acids manifested severe rectal bleeding, body weight loss, mortality, infiltration of neutrophils, depletion of colon antioxidant enzymes and elevation of inflammatory cytokines. n-6 PUFA (sunflower oil) aggravates colon inflammation in rats through elevation of linoleic and arachidonic acid levels in the body.
However, dietary feeding of rice bran oil rich in MUFA, PUFA and antioxidant oryzanol showed remarkably lower disease activity index, histological damage and inflammatory mediators in the colon tissue. DSS-induced depletion of colon antioxidant enzymes was remarkably prevented in the RBO fed group. Rice bran oil remarkably inhibited the TNF-α, IL-1β, nitric oxide, and myeloperoxidase activity in the colon and abrogated colitis. The minor components such as oryzanol and tocotrienol might be involved in the inhibition of disease activity in colitis; since, there was no significant reduction in linoleic and arachidonic acid levels in rats fed with rice bran oil. Similar results were observed in the GCO rich in n-3 PUFA however to a lesser extent compared to RBO group. GCO suppressed colon inflammation in rats through elevation of EPA and DHA, and also reduction in linoleic and arachidonic acid levels in the body.

Investigation on the influence of background dietary lipids on the beneficial effects of fish oil has shown that medium chain triglycerides (MCT) and monounsaturated fatty acids (MUFA) potentiate its beneficial effects such as hypolipidemic and anti-inflammatory effects by unequivocally elevating the EPA and DHA levels in the body than any of the lipids in the background diet. Subsequent studies on the individual and combined dietary intake of MCT or MUFA along with fish oil has shown that rats fed on high MCT exhibited exaggerated colon inflammation in active disease of colitis and also impaired the healing process of mucosa. Rats fed on MUFA has significantly lower disease activity index and protected the DSS-induced colon damage.

More importantly, fish oil remarkably inhibited the disease activity, inflammatory mediators and colon damage induced by DSS when fed along with MUFA. These effects were complemented by acceleration of mucosal healing after the course of colitis by TGF-β dependent mechanism.

Maternal supplementation of n-6 fatty acids during pregnancy and lactation elevates the serum triglycerides, cholesterol, leptin levels and adipose tissue growth in the weanling rats. In contrast, maternal dietary intake of high n-3 fatty acids counteracts the effects of n-6 fatty acids in the weanling pups. Maternal protein deficiency during pregnancy and lactation markedly affects the conversion of ALA to EPA and DHA and diminishes arachidonic acid (AA), EPA and DHA levels in the brain tissue of the weanling pups.
Maternal dietary intake of n-6 fatty acids during pregnancy and lactation aggravates colon inflammation in the neonates. Maternal and neonatal intake of balanced n-6/n-3 PUFA altered systemic ARA/DHA ratio and attenuated colon inflammation in the neonates.