

Anti-Diarrheal Activities of Hydromethanolic Crude Extract and Solvent Fractions of *Acacia seyal* (Fabaceae) Roots in Mice [Response to Letter]

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Dear editor

We used the traditional method of diarrheal induction (the castor oil-induced diarrheal model) to investigate the anti-diarrheal activities of hydromethanolic crude extract and solvent fractions of *Acacia seyal* (Fabaceae) roots in mice. Despite the fact that this is a conventional model, toxin damage to the tight junction, which alters the paracellular ion flux, could cause one of the types of diarrhea (secretory diarrhea). Generally, secretory diarrhea happens when there is an increase in the amount of fluid being pulled into the lumen of the bowel. Hence, the ability of the intestines to reabsorb is altered. This leads to an increase in the net secretion of ions (chloride or bicarbonate) and an inhibition of the net absorption of sodium and water.^{1,2} There could be several systems at work. Enteric pathogens, which include bacteria like *Escherichia coli*, *Salmonella*, *Shigella*, and others, may adhere to or infiltrate the epithelium during colonization and release enterotoxins or cytotoxins. They cause mucosal mast cells to degranulate and release histamine, which attracts inflammatory cells and helps to produce prostaglandins and platelet-activating factors, which in turn help to promote secretion. They also produce inflammatory mediators such as tumor necrosis factor and interleukin-6 by activating T cells and neutrophils.³⁻⁶ By modifying the intracellular Ca^{2+} -controlled ion transport pathways or cyclic adenosine monophosphate, cyclic guanosine monophosphate, or both, enterotoxins can also change how the second messenger system works. In people with cystic fibrosis, changes in these mediators result in Cl^- secretion via calcium-activated chloride channels, suppression of small intestine-coupled Na^+ - Cl^- transport, and transmembrane conductance regulator-mediated Cl^- secretion. An electric potential is created by the buildup of negatively charged chloride anions in the crypt, which draws sodium into the lumen and finally causes an uncontrolled secretion of sodium chloride and water.^{7,8}

Castor oil has a laxative action and causes mild diarrhea when taken orally. Ricinoleic acid, a hydroxylated fatty acid produced from castor oil by intestinal lipases in the upper section of the small intestine, mediates the effects of castor oil. Despite the widespread use of castor oil in both mainstream and alternative medicine, it is still unclear exactly how ricinoleic acid works at the molecular level. The capacity of the active metabolite of castor oil, ricinoleic acid, to cause diarrhea through mechanisms such as GI mucosal irritation, leading to the production of prostaglandin, which accelerates gastrointestinal motility and electrolyte secretion, may be the cause of the aforementioned symptoms.^{1,2,9,10} Therefore, because castor oil mimics enteric pathophysiological processes and makes it possible to observe quantifiable changes in the frequency of stools, enteropooling, and intestinal transit, the use of castor oil as a diarrheal disease inducer is feasible for all models (castor oil induced enteropooling, gastrointestinal motility, and castor oil induced diarrhoea), as mentioned in our study. However, as you pointed out, we did not address the quantity or level of secondary metabolites in each fraction or in the hydromethanolic crude extract because our study only performed qualitative phytochemical screening tests. Additionally, the study did not examine the interdependence of secondary metabolites. I am willing to conduct additional research if it would help to close these gaps and improve the data from our study.

Disclosure

The authors disclose no conflicts of interest in this communication.

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