

Study on humoral factor and intraluminal circumstances influencing salt absorption across the intestine

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Summary

The aim of this study was to investigate the effects of endothelins on net fluid and NaCl absorption across the jejunum. The jejunal fluid and NaCl absorption and mesenteric hemodynamics in jejunal loops were measured during infusion of saline, endothelin-1 or endothelin-3 into the superior mesenteric artery in dogs. The dogs were anesthetized with pentobarbital sodium (30 mg/kg i.v.). Polyethylene catheters were placed in the superior mesenteric arteries and portal vein for infusions and measuring arterial and portal venous pressure. Superior mesenteric arterial blood flow was continuously measured with an ultrasonic flow probe. A 30 cm long jejunal loop was made which was 10 cm from the duodenal fossa. Infusion of saline, endothelin-1, endothelin-3 or phenylephrine was initiated 10 min before pouring the test solution into the jejunal loop and lasted for 25 min. The net fluid (7.2 ± 0.9 ml, mean \pm SE, n=8), Na^+ (1.1 ± 0.1 meq), and Cl^- (1.1 ± 0.2 meq) absorption during saline infusion were not significantly different from those (7.0 ± 1.0 ml, 1.1 ± 0.1 and 1.1 ± 0.2 meq) during endothelin-1 infusion but significantly decreased to 4.8 ± 0.6 ml, 0.7 ± 0.1 and 0.7 ± 0.1 meq by endothelin-3 infusion. Infusion of endothelin-3 decreased the net fluid, Na^+ and Cl^- absorption; however, saline and endothelin-1 had no effect. Increases in portal venous pressure and vascular resistance and decrease in blood flow caused by endothelins infusion had no effects on the net jejunal absorption. To investigate the role of nitric oxide and soluble guanylate cyclase activation in the mechanisms underlying endothelin-3 induced decrease in the net fluid and electrolyte absorption, measurements were obtained in the presence of the nitric oxide synthesis inhibitor, nitro-L-arginine methyl ester or the soluble guanylate cyclase inhibitor, methylene blue. The endothelin-3 induced decrease in the net absorption was not influenced by the pretreatment with inhibitors. These results suggest that the endothelin-3 response was not mediated by nitric oxide or soluble guanylate cyclase.