
Abstract
Background
Copper deficiency affects the peripheral (PNS) and central (CNS) nervous systems and can lead to neurological deficits in humans. No studies have addressed whether copper deficiency affects the enteric nervous system (ENS). We hypothesized that ENS abnormalities impair intestinal function in copper deficiency.

Methods
We induced copper deficiency in rats by nutritional deprivation. Once hypocupremia was achieved, we euthanized the animals and harvested the small and large intestine. The longitudinal smooth muscle from the jejunum and colon was suspended in organ baths and contractility in response to electrical field stimulation (EFS) was assessed. Mucosa was also isolated from each region and placed into modified Using chambers to determine whether the copper deficiency leads to alterations in epithelial transport measured as a change in short circuit current across the mucosa in response to EFS.

Key Results
A copper deficient diet (CDD) in normal rats for 9 weeks was sufficient to produce hypocupremia. Colonic smooth muscle contractility was significantly decreased in response to EFS in rats fed a CDD compared with controls; however, jejunal smooth muscle contractility in response to EFS in rats fed a CDD rats resembled that observed in controls. No significant changes in secretory function were observed in either region in response to CDD.

Conclusions & Inferences
Dietary copper deficiency produces significant changes in the neural regulation of colonic smooth muscle contractility in a rodent model. Thus, along with CNS and PNS effects in humans, copper deficiency results in abnormal ENS regulation of intestinal function in rats.