Protective effects of medium-chain triglycerides on the liver and gut in rats administered endotoxin.

**OBJECTIVE:** To determine if medium-chain triglycerides (MCTs) prevent organ injuries and mortality in rats administered endotoxin and to determine effects of MCT on the gut.

**SUMMARY BACKGROUND DATA:** Since dietary MCTs prevent alcohol-induced liver injury by inhibiting activation of Kupffer cells in the enteral feeding model, the authors hypothesized that MCT could prevent deleterious conditions in endotoxemia. **METHODS:** After a preliminary experiment determined the optimal dose of MCT, rats were given MCT (5 g/kg per day) or the same dose of corn oil by gavage daily for 1 week. Then, lipopolysaccharide (LPS) was administered intravenously and survival was assessed for the next 24 hours. For analysis of mechanisms, rats were killed 9 hours after LPS injection and serum and liver sections were collected. To investigate effects of MCT on the gut, pathologic change, permeability, and microflora were assessed. Kupffer cells isolated by collagenase digestion and differential centrifugation were used for endotoxin receptor CD14 immunoblotting, phagocytic index, and TNF-alpha production assay. **RESULTS:** All rats given corn oil died after LPS administration; however, this mortality was prevented by MCT in a dose-dependent manner. Rats given corn oil showed liver injury after LPS administration. In contrast, MCT prevented this pathologic change nearly completely. MCT blunted CD14 expression on the Kupffer cells and TNF-alpha production by isolated Kupffer cells; however, there were no differences in phagocytic index between the two groups. The length of the intestinal epithelium was increased in the MCT group compared to the corn oil group. Further, after LPS administration, increases in gut permeability and injury were prevented by MCT. Importantly, MCT also prevented hepatic energy charge and gut injuries in this condition. **CONCLUSIONS:** Enteral feeding using MCT could be a practical way of protecting the liver and intestine during endotoxemia.