

NEUROGASTROENTEROLOGY

**TBI AFFECTS
INTESTINAL MOTILITY**

“Traumatic brain injury [TBI] affects nearly 1.5 million people each year in the USA, leading to an annual economic burden of US\$60 billion,” states Karen Uray. One secondary effect of TBI is feeding intolerance including oesophageal reflux and delayed gastric emptying. “Understanding the development of feeding intolerance is important in improving clinical outcomes in these patients,” Uray says. New findings by Uray and co-workers have shown that intestinal contractile activity is affected by TBI.

In this study, the researchers used a model of TBI in which rats received a controlled cortical impact injury. Rats were sacrificed 1, 3 and 7 days after TBI; intestinal smooth muscle was collected for measurement of contractile activity and cytokine levels. Ileum contractile activity decreased markedly at day 7 when compared with control (sham-operated) rats. Moreover, levels of proinflammatory cytokines (including IL-1 α , IL-1 β and IL-17) and NF- κ B activity were all higher in rats in the TBI group than in control rats.

“This study established that intestinal motility is significantly decreased after TBI, predominantly in the small intestine,” explains Uray. Of note, this model shows that a moderate TBI is sufficient to reduce intestinal motility. Professor John Furness, an expert in the field who was not involved in this study, notes that an alternative animal model, such as ferret, could be used to link motility changes to alterations in behaviour, as rats do not vomit nor show demonstratable links between nausea and feed intolerance.

“Decreased intestinal contractility seems to be a result of secondary systemic inflammation as shown by increased levels of inflammatory cytokines in intestinal smooth muscle, but more studies are needed to confirm the link between inflammation and dysmotility in TBI,” Uray adds. The mechanisms by which intestinal motility is decreased also remain unknown.

Current prokinetic drugs used to treat gastrointestinal motility in patients after TBI have limited effects or issues of drug resistance so further drugs are needed. “Understanding the mechanism by which intestinal contractile dysfunction occurs after trauma (such as TBI) is crucial in identifying new drug targets for the treatment of ileus,” concludes Uray.

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