CONTINUING MEDICAL EDUCATION (CME)/MOC ACTIVITIES

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Instructions:

Category 1 credit can be earned by reading the relevant article and taking these CME examinations online at https://www.gastrojournal.org/cme/home. Answers to the questions are provided after taking the exams.

CME Exam 1: The Tight Junction Protein ZO-1 Is Dispensable for Barrier Function but Critical for Effective Mucosal Repair

Turner JR et al, Authors
Test ID No.: gastro00437 *Contact hours:* 1.0 *Expiration Date:* December 31, 2022

Question 1:

Which of the statements regarding intestinal epithelial ZO-1 is best?

a. Epithelial expression of the tight junction protein ZO-1 is increased in inflammatory bowel disease (IBD).
b. Epithelial ZO-1 deletion causes profound barrier defects and spontaneous disease in experimental animals.
c. Mucosal repair is accelerated in intestinal ZO-1 specific knockout mice.
d. Wnt—β-catenin signaling is enhanced in intestinal ZO-1 specific knockout mice.
e. ZO-1 associates with centrioles and directs mitotic spindle orientation.

Question 2:

In the study by Kuo et al, by which approaches did the authors conclude that ZO-1 expression is reduced in intestinal tissues from patients with IBD?

a. Analyze publicly available RNA microarray and RNAseq data.
b. Perform laser capture microdissection of intestinal biopsies.
c. Perform immunostains for ZO-1-associated proteins.
d. Perform in situ hybridization to detect ZO-1 mRNA transcripts.
e. Analyze tissue immunostains from mouse models of colitis.

Question 3:

What is the primary phenotype detected in intestinal epithelial-specific ZO-1 knockout mice?

a. Profound growth defects and failure to achieve normal body weight.
b. Sepsis and systemic immune activation.
c. Colorectal neoplasia.
d. Defective mucosal repair after injury.
e. Increased numbers of intraepithelial lymphocytes and lamina propria macrophages.

Question 4:

What are the principal mechanisms underlying the primary phenotype of intestinal epithelial-specific ZO-1 knockout mice?

a. Defective epithelial restitution and tight junction restoration.
b. Insufficient activation of epithelial proliferation and mitotic catastrophe owing to spindle misorientation.
c. Degradation of other tight junction proteins and up-regulation of Wnt—β-catenin signaling.
d. Goblet and Paneth cell loss.
e. Excessive mucosal immune activation and immune-mediated epithelial damage.

Question 5:

Which of the following is an important conclusion in the study by Kuo et al?

a. ZO-1 is essential for tight junction assembly.
b. DNA transfer technologies that increase intestinal epithelial ZO-1 expression protect mice from experimental colitis and may be a therapeutic approach in patients with IBD.
c. ZO-1 loss in IBD may contribute to failure of mucosal healing.
d. Enhanced mucus production may compensate for ZO-1 loss.
e. Intestinal epithelial ZO-1 loss prevents immune cell signaling to epithelial cells that activates epithelial proliferation.