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ESPS Peer-review Report
Name of Journal: World Journal of Gastroenterology
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Title: Contribution of TLR signaling to the pathogenesis of colitis-associated cancer in inflammatory bowel disease
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Science editor: Ya-Juan Ma
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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[ ] Grade A (Excellent)	[ ] Grade A: Priority Publishing	Google Search:	[ ] Accept
[ ] Grade B (Very good)	[Y] Grade B: minor language polishing	[ ] Existed	[ ] High priority for publication
[Y] Grade C (Good)	[ ] Grade C: a great deal of language polishing	[ ] No records	[ ]Rejection
[ ] Grade D (Fair)	[ ] Grade D: rejected	BPG Search:	[Y] Minor revision
[ ] Grade E (Poor)		[ ] Existed	[ ] Major revision
		[ ] No records	

COMMENTS TO AUTHORS

This paper is a review on the contribution of Toll-like receptors (TLRs)-mediated immune responses to inflammation-related colonic carcinogenesis arising in inflammatory bowel disease. TLRs are expressed on the colonic and small intestinal epithelium are known to recognize pathogen-associated molecular patterns (PAMPs) and damage-associated molecular patterns (DAMPs) that initiate intracellular cell signaling that subsequently activates an inflammatory response and recruits inflammatory cells. Understanding the mechanisms through which TLRs on colon cancer cells and inflammatory cells regulate growth, survival, and metastatic progression can make them potential targets for colorectal cancer therapy. Comments 1. This review doesn't include some recent publications that address on the roles of TLR in bridging and in the regulation of innate and adaptive immune responses (de Kivit et al., 2014); in microbiota-associated gastrointestinal cancer metastasis focusing on TLR recognition of microbiota ligands, initiating inflammation, and promoting tumorigenesis (Santaolalla et al., 2011; Lu et al., 2013); in the maintenance and functioning of the epithelial barrier integrity in the gut regulating the MY88 adaptor protein and thereby a protective function in the control of intestinal inflammation and inflammation-associated cancer (Aviello et al., 2014); and in the activation of NF-κB signaling pathway, which transcriptionally controls a large set of target genes that play important roles in cell survival, inflammation, and immune responses (Tukhvatuline et al., 2013). 2. The signaling pathways utilized by various TLRs differ, which results in varied cellular responses. No attempt was made in this paper to delineate the expression of various TLRs in promotion of inflammation and development of colon cancer. This would help in

development of specific TLR agonists or antagonists useful for the prevention and treatment of IBD and colitis associated colon carcinoma. 3. In text, many words were wrongly spelt. For example, in page 2: intestinal homeostasis or intestinal hoemostasis?; in page 5: extracellular leucin-rich repeated sequences or extracellular leucin-reach repeated sequences? Please correct.