

ESPS PEER REVIEW REPORT

Name of journal: World Journal of Gastroenterology

ESPS manuscript NO: 11826

Title: Dismicrobism in IBD and Colorectal cancer: changes in response of colocytes

Reviewer code: 00504462

Science editor: Yuan Qi

Date sent for review: 2014-06-10 20:20

Date reviewed: 2014-06-25 05:59

CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
<input type="checkbox"/> Grade A: Excellent	<input type="checkbox"/> Grade A: Priority publishing	Google Search:	<input type="checkbox"/> Accept
<input type="checkbox"/> Grade B: Very good	<input checked="" type="checkbox"/> Grade B: Minor language polishing	<input type="checkbox"/> Existing	<input type="checkbox"/> High priority for publication
<input checked="" type="checkbox"/> Grade C: Good	<input type="checkbox"/> Grade C: A great deal of language polishing	<input type="checkbox"/> No records	<input type="checkbox"/> Rejection
<input type="checkbox"/> Grade D: Fair	<input type="checkbox"/> Grade D: Rejected	<input type="checkbox"/> Existing	<input checked="" type="checkbox"/> Minor revision
<input type="checkbox"/> Grade E: Poor		<input type="checkbox"/> No records	<input type="checkbox"/> Major revision

COMMENTS TO AUTHORS

Dear Sir, Your manuscript is a really interesting review and the subject has the utmost importance nowadays, as dysbiosis has been related to diverse kind of diseases. However, as a review there are some concepts that you should define clearly in this kind of manuscript. For example, it would be useful if you could differentiate dysbiosis from dismicrobism. Even though they can be use synonymously, are there any differences in how the two terms are used? How can we diagnose eubiosis in the clinical practice? Are there any methods to obtain a quantitative and qualitative composition of a normal microbiota? Second, you elegantly reviewed the possible mechanism related to the inflammation, as well as the cancer formation with the IBD. However, are these changes related to the dysbiosis of the luminal or mucosal microbiota? Does it matter? How can changes in the luminal changes affect the inflammatory state of the mucosa and is this the only stimulus that matters in order to develop CRC? Is it necessary to have a change in the microbiota in order to have the inflammatory changes (changes in lymphocytes, MEP pathway, etc), or the modulation of the chaperoning system? Third, can we modify the natural history of the UC or the CRC with the only use of probiotics or omega 3 polyunsaturated fatty acids? Can there be other factors involved other than the microbiota? There are plenty of questions that are left unanswered with the current state of the manuscript, but I hope you can help us to clarify some of this points in order to publish it. Thank you for this opportunity. I hope to hear from you soon Sincerely

ESPS PEER REVIEW REPORT

Name of journal: World Journal of Gastroenterology

ESPS manuscript NO: 11826

Title: Dismicrobism in IBD and Colorectal cancer: changes in response of colocytes

Reviewer code: 02910903

Science editor: Yuan Qi

Date sent for review: 2014-06-10 20:20

Date reviewed: 2014-06-25 16:16

CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
<input type="checkbox"/> Grade A: Excellent	<input checked="" type="checkbox"/> Grade A: Priority publishing	Google Search:	<input type="checkbox"/> Accept
<input checked="" type="checkbox"/> Grade B: Very good	<input type="checkbox"/> Grade B: Minor language polishing	<input type="checkbox"/> Existing	<input type="checkbox"/> High priority for publication
<input type="checkbox"/> Grade C: Good	<input type="checkbox"/> Grade C: A great deal of language polishing	<input type="checkbox"/> No records	<input type="checkbox"/> Rejection
<input type="checkbox"/> Grade D: Fair	<input type="checkbox"/> Grade D: Rejected	<input type="checkbox"/> Existing	<input checked="" type="checkbox"/> Minor revision
<input type="checkbox"/> Grade E: Poor		<input type="checkbox"/> No records	<input type="checkbox"/> Major revision

COMMENTS TO AUTHORS

The alteration of the intestinal microbial flora is called dysbiosis. The dysbiosis is one of the primary causes for the activation of the mucosal intestinal lymphatic system called GALT (Gut Associated lymphoid tissue). The activation of Galt triggers an activation of biohumoral component , hence ,inflammation of the intestinal mucosa. This supports the mucosal inflammation typical of Crohn's disease and ulcerative colitis in the rectum, called IBD (intestinal bowel disease) The chronic mucosal inflammation is the substrate for the onset of colorectal cancer (CCR). Therefore, the maintenance of eubiosis is a protective factor for the onset of IBD and the JRC. This review is very clear, following a logic way in what may be the causes that lead to the formation of intestinal neoplasia. Therefore, the review deserves to be published with minor adjustments and revision of some english words.

ESPS PEER REVIEW REPORT

Name of journal: World Journal of Gastroenterology

ESPS manuscript NO: 11826

Title: Dismicrobism in IBD and Colorectal cancer: changes in response of colocytes

Reviewer code: 00680628

Science editor: Yuan Qi

Date sent for review: 2014-06-10 20:20

Date reviewed: 2014-06-28 22:10

CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
<input type="checkbox"/> Grade A: Excellent	<input type="checkbox"/> Grade A: Priority publishing	Google Search:	<input type="checkbox"/> Accept
<input type="checkbox"/> Grade B: Very good	<input checked="" type="checkbox"/> Grade B: Minor language polishing	<input type="checkbox"/> Existing	<input type="checkbox"/> High priority for publication
<input type="checkbox"/> Grade C: Good	<input type="checkbox"/> Grade C: A great deal of language polishing	<input type="checkbox"/> No records	<input type="checkbox"/> Rejection
<input checked="" type="checkbox"/> Grade D: Fair		BPG Search:	<input type="checkbox"/> Minor revision
<input type="checkbox"/> Grade E: Poor	<input type="checkbox"/> Grade D: Rejected	<input type="checkbox"/> Existing	<input checked="" type="checkbox"/> Major revision
		<input type="checkbox"/> No records	

COMMENTS TO AUTHORS

Major comments: Although the gut microbiota contributes to colon tumorigenesis, they are only part of environmental factors. Genetic alterations, inflammation, and involvement of nutrients, hormones and metabolic disorders also favor cancer genesis. The authors should address these points in the manuscript.

In the core tip, "The aim of this work is to focus on the molecular mechanisms that connect dysbiosis, IBD and CCR. Experimental studies are oriented towards the discovery of new probiotic-based therapies for the treatment and prevention of inflammatory and carcinogenetic processes." However, the contents of the abstract only mentioned about the relationship between intestinal microbiota and IBD. There is no molecular mechanism concerning interaction among intestinal microbiota, IBD and colorectal cancer. There is a growing body of evidence that the gut microbiota contributes to colon tumorigenesis. Clinical trials have shown that probiotics can inhibit the inflammatory process by enhancing host immune responses, altering the bacterial phylotypes in the colon and impacting the gut metabolome. They may also have anti-tumor properties through direct anti-proliferative activity on tumor cells. This manuscript should address this information. The depth of the manuscript is not enough. The evidence provided in the manuscript did not finish the core tip mentioned by the authors.

Minor comments: 1. There should be no references in the abstract section. 2. What's the meaning of the following sentence? "It is now established a close interaction between commensal bacterial flora and intestinal immune system are responsible for the onset and development of several diseases besides IBDs." (line 5-7, page 4) 3. Did the figures prepare



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