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ESPS Peer-review Report
Name of Journal: World Journal of Gastroenterology
Ms: 3866
Title: Interplay of autophagy and innate immunity in Crohn' disease: a key immunobiologic feature
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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[ ] Grade A (Excellent)	[Y] Grade A: Priority Publishing	Google Search:	[ ] Accept
[ ] Grade B (Very good)	[ ] Grade B: minor language polishing	[ ] Existed	[Y] High priority for publication
[Y] Grade C (Good)	[ ] Grade C: a great deal of language polishing	[ ] No records	
[ ] Grade D (Fair)		BPG Search:	[ ]Rejection
[ ] Grade E (Poor)	[ ] Grade D: rejected	[ ] Existed	[ ] Minor revision
		[ ] No records	[ ] Major revision

COMMENTS

COMMENTS TO AUTHORS:
This is a nice, complete and updated review about the role of autophagy and innate immunity in the pathogenesis of Crohn's disease (CD). Minor comments: the authors should pay be very careful when using definition of "gain of function" or "loss of function". E.g., frame-shift gene mutation NOD2 variant encodes a "loss of function" protein as concern NF-kB activation and a "gain of function" protein as concern the regulation of IL-10 transcription. In some cases, a semingly "gain of function" effect can result by complex compensatory mechanisms activating an inflammatory feedback. To stres this concept, the Authors may refer to the thypical pahotological lesion of CD, the granuloma. Indeed, granulomatous colitis can occur in a number of primary immune-deficiencies mainly affecting innate immunity: in these cases, the inflammatory phenotype can be view as the compensatory response to a primary deficiency that lead to dysbiosis and abnormal persistence of bacterial products in epithelial cells and/or phagocytes.