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ESPS PEER-REVIEW REPORT

Name of journal: World Journal of Immunology
ESPS manuscript NO: 27281
Title: IgE regulates airway smooth muscle phenotype: Future perspectives in allergic asthma
Reviewer's code: 00502945
Reviewer's country: Argentina
Science editor: Xue-Mei Gong
Date sent for review: 2016-05-22 20:10
Date reviewed: 2016-07-15 09:21

CLASSIFICATION	LANGUAGE EVALUATION	SCIENTIFIC MISCONDUCT	CONCLUSION
[Y] Grade A: Excellent	[Y] Grade A: Priority publishing	Google Search:	[Y] Accept
[ ] Grade B: Very good	[ ] Grade B: Minor language polishing	[ ] The same title	[ ] High priority for publication
[ ] Grade C: Good	[ ] Grade C: A great deal of language polishing	[ ] Duplicate publication	[ ] Rejection
[ ] Grade D: Fair	[ ] Grade D: Rejected	[ ] Plagiarism	[ ] Minor revision
[ ] Grade E: Poor		[Y] No	[ ] Major revision
		BPG Search:	
		[ ] The same title	
		[ ] Duplicate publication	
		[ ] Plagiarism	
		[Y] No	

COMMENTS TO AUTHORS

The commentary manuscript of Redhu and Soussi Gounni focused on the effect of IgE on airway smooth muscle (ASM) cells and how anti-IgE therapy may prevent the pathogenic changes of airway smooth muscle in asthma. The subject is interesting and suggests that ASM cells are playing a direct role on the pathogenic mechanisms of asthma. The manuscript has been well written. The different gaps that require further investigation are presented in the manuscript. The figure and references are appropriate. Minor points - A list of abbreviations could be included. - In page 4, line 12: "chronic obstructive pulmonary disease (COPD)" should be added

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ESPS PEER-REVIEW REPORT

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CLASSIFICATION	LANGUAGE EVALUATION	SCIENTIFIC MISCONDUCT	CONCLUSION
[Y] Grade A: Excellent	[ ] Grade A: Priority publishing	Google Search:	[ ] Accept
[ ] Grade B: Very good	[Y] Grade B: Minor language polishing	[ ] The same title	[ ] High priority for publication
[ ] Grade C: Good	[ ] Grade C: A great deal of language polishing	[ ] Duplicate publication	[ ] Rejection
[ ] Grade D: Fair	[ ] Grade D: Rejected	[ ] Plagiarism	[Y] Minor revision
[ ] Grade E: Poor		[Y] No	[ ] Major revision
		BPG Search:	
		[ ] The same title	
		[ ] Duplicate publication	
		[ ] Plagiarism	
		[Y] No	

COMMENTS TO AUTHORS

This manuscript describes a possible role for ASM cells in pathogenic mechanisms of asthma. The subject is interesting and well written. References are appropriate and the figure is helpful. The involvement of IgE is presented discussed. In general, reading is clear. However, in some parts to many facts are presented without proper discussion. Thus the reader loses the authors' point. A better organization of ideas would support the authors' arguments. Some comments to improve the manuscript: The title concentrates on IgE, while the abstract centers the attention on the high affinity Fc epsilon receptor. Authors should decide where to put the readers attention and maintain a similar point of view. The manuscript contains too many abbreviations. This makes reading difficult. Some of them (COPD, and RMM for example) are not defined. The manuscript would be easier to read with much fewer abbreviations. Authors indicate that "This is even more interesting of a question since MAPK signaling, particularly p38, has already been in clinical trials of asthma [21]." The molecule p38 cannot be in clinical trials. This needs revising. Authors suggest that ASM cells are the source of cytokines and chemokines. Although, this may be the case, other cells (including mast cells)

can also contribute to the presence of cytokines, chemokines, and other mediators in ASM tissue. This should be properly discussed.

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ESPS PEER-REVIEW REPORT

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[ ] Grade B: Very good	[ ] Grade B: Minor language polishing	[ ] The same title	[ ] High priority for publication
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[ ] Grade D: Fair	[ ] Grade D: Rejected	[ ] Plagiarism	[ ] Minor revision
[ ] Grade E: Poor		[Y] No	[ ] Major revision
		BPG Search:	
		[ ] The same title	
		[ ] Duplicate publication	
		[ ] Plagiarism	
		[Y] No	

COMMENTS TO AUTHORS

This article is informative and well written. I have two comments to make The sentence, "This is even more interesting of a question since MAPK signaling, particularly p38, has already been in clinical trials of asthma [21]." Needs rephrasing - Do authors mean inhibitors in clinical trials? p38 cannot be in clinical trials. RBM – definition?

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CLASSIFICATION	LANGUAGE EVALUATION	SCIENTIFIC MISCONDUCT	CONCLUSION
[ ] Grade A: Excellent	[ ] Grade A: Priority publishing	Google Search:	[ ] Accept
[ ] Grade B: Very good	[Y] Grade B: Minor language polishing	[ ] The same title	[Y] High priority for publication
[Y] Grade C: Good	[ ] Grade C: A great deal of language polishing	[ ] Duplicate publication	[ ] Rejection
[ ] Grade D: Fair	[ ] Grade D: Rejected	[ ] Plagiarism	[ ] Minor revision
[ ] Grade E: Poor		[Y] No	[ ] Major revision
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		[ ] The same title	
		[ ] Duplicate publication	
		[ ] Plagiarism	
		[Y] No	

COMMENTS TO AUTHORS

Redhu and Gounni present a review on the role of IgE directly on airway smooth muscle. This is an interesting concept that supplements the more broadly known area of how IgE induces activation of mast cells and basophils. The anti-dogmatic focus of this review justifies the need for such a manuscript in the literature, as long as the conclusions are drawn in the context of the extensive mast cell literature. Some specific comments are below, in the order of appearance in the manuscript. 1. The second paragraph of the commentary (starts with "The allergic cascade is…") includes the statement, "The ongoing TSLP expression via the IgE/FceRI pathway may in fact explain the atopic or pro-allergic state…" It is unclear exactly to what the authors refer. TSLP can be made by multiple subtypes, including mast cells. Further, mast cells use similar signaling pathways as described for airway smooth muscle. Adding clarity to this sentence and the preceding sentences would add in the interpretation of the authors' conclusions. 2. The other major section of the manuscript that asserts airway smooth muscle function is near the end (paragraph begins with "Of note, mast cells are known to infiltrate…" The third points being used to argue that mast cells are

not involved do not support the conclusion. Inhibition of Syk would block mast cells and ASM. Even though no detectable contamination of mast cells was detected, the cytokines, chemokines, and other mediators released by mast cells can migrate substantial distance and penetrate the tissue. The fact that anti-IgE blocks functions, indicates that IgE is involved, but does not address the site of activity. 3. In many places in the manuscript, the reader is lost in a sea of facts that are not assembled into a cohesive argument. Better organization would support the authors' arguments.