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ESPS PEER REVIEW REPORT

Name of journal: World Journal of Nephrology
ESPS manuscript NO: 12178
Title: Roles of (pro)renin receptor in the Kidney
Reviewer code: 02446027
Science editor: Fang-Fang Ji
Date sent for review: 2014-06-26 15:07
Date reviewed: 2014-07-08 12:19

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

COMMENTS TO AUTHORS

Overall/General Comments: This is a review regarding roles of prorenin receptor in the kidney. The topic is of considerable interest because PRR, a specific receptor for renin and prorenin, may play important pathophysiological roles in diabetic nephropathy. The present review has proposed the possible mechanism that PRR involved in the diabetic kidney may play a pathophysiological role in angiotensin I generation and renal fibrosis found in end-stage renal disease. Although I fully agree with the author's opinion, but there are several concerns about the manuscript which need to be improved. Therefore, I cannot recommend acceptance of the manuscript in its present form. Major Comments: 1. This paper does not have any discussion section, or summary section. I think adding a summary section to the paper will make the paper complete. 2. Conclusion section: this section should be redone to provide the expert perspective about PRR functions, and direct prorenin or renin signaling through PRR in diabetic nephropathy disorders. The authors should go beyond the conclusion and provide their interpretation of the data presented in the article and discuss the cellular interaction and important functions of PRR that are likely to be important in development of diabetic nephropathy. Minor Comments: Figures 1 and 2 are not clear. Please submit the clear original figures, not copies of the figures. There are too many abbreviations in this manuscript. The English as written is somewhat difficult and needs revision. Please go over the entire manuscript for this correction.

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ESPS PEER REVIEW REPORT

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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[ ] Grade A: Excellent	[Y] Grade A: Priority publishing	Google Search:	[Y] Accept
[Y] Grade B: Very good	[ ] Grade B: Minor language polishing	[ ] Existing	[ ] High priority for publication
[ ] 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	[ ] Existing	[ ] Minor revision
		[ ] No records	[ ] Major revision

COMMENTS TO AUTHORS

very useful and concrete, synoptic but well written review-will be helpful to scientists performing translational research in the area of kidney disease and diabetic nephropathy

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ESPS PEER REVIEW REPORT

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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	[ ] Existing	[ ] High priority for publication
[ ] Grade C: Good	[ ] Grade C: A great deal of language polishing	[ ] No records	
[Y] Grade D: Fair		BPG Search:	[ ] Rejection
[ ] Grade E: Poor	[ ] Grade D: Rejected	[ ] Existing	[ ] Minor revision
		[ ] No records	[Y] Major revision

COMMENTS TO AUTHORS

Dr. Oshima et al reviewed the role of (pro)renin receptor (PRR) in the kidney. Although authors reviewed well, several points should be addressed before publication. Especially, the section of "PRR in diabetic nephropathy" was logically inconsistent. 1. Authors claimed that transgenic rats overexpressing human PRR showed high PRR mRNA expression in tissue including kidney (page 6 line 18). However, this transgenic rats harboring human PRR under CAG promoter. I'm afraid that this sentence misinform about natural PRR expression, because PRR under CAG promoter is expected to be expressed ubiquitously In whole body. Especially, the activity of CAG promoter is very strong in podocyte. 2. From page 6 line 15 to page 7 line5, I think authors discussed about the effect of increased PRR activity in podocyte. However, several evidences are not the observation in mesangial cells or tubular cells. For example, authors claimed that PRR mRNA and protein up-regulation was observed in kidney, although this paper showed that PRR was up-regulated in mesangial cells and tubules, but not in podocyte. Authors should focus on PRR expression in podocyte in this section. 3. Authors claimed that this effect is thought to be independent of angiotensin II (page 6 line 23). But, in the next sentence, authors claimed that all RAS components are expressed in podocyte. Do authors think whether RAS is involved or independent in podocyte injury? 4. Authors claimed that PRR up-regulation in the kidney of DM rats was inhibited by ARB treatment (page 7 line 16). Authors also claimed that regression of nephropathy was observed in HRP treatment, but not ACEi treated DM rats. Authors should discuss why ACEi, which might also inhibit PRR

expression, was not effective in the regression of nephropathy.