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ESPS Peer-review Report
Name of Journal: World Journal of Biological Chemistry
ESPS Manuscript NO: 7609
Title: The Mnk Kinase Pathway: Cellular Functions and Biological Outcomes
Reviewer code: 02567328
Science editor: Ling-Ling Wen
Date sent for review: 2013-11-26 23:14
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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[ ] Grade A (Excellent)	[Y] Grade A: Priority Publishing	Google Search:	[ ] Accept
[Y] Grade B (Very good)	[ ] Grade B: minor language polishing	[ ] Existed	[ ] High priority for publication
[ ] Grade C (Good)	[ ] Grade C: a great deal of language polishing	[ ] No records	[ ]Rejection
[ ] Grade D (Fair)		BPG Search:	[Y] Minor revision
[ ] Grade E (Poor)	[ ] Grade D: rejected	[ ] Existed	[ ] Major revision
		[ ] No records	

COMMENTS TO AUTHORS

In this review the authors have described the MnK kinase pathway focusing our attention on post transcriptional regulation, effectors, cellular functions and biological outcomes of these kinases. The authors have already published a review on role of MnK Kinases in cytokine signaling and regulation of cytokine responses and many papers on this subject. The review is well written and organized. The large number of references attests the detailed work carried out by the authors. There are some points should be clarified or modified: - In the paragraph "Post transcriptional regulation of Mnk kinases" line 4 the sentence "while engagement of the Erk MAPK pathway engagement is primarily mediated" should be changed in "while engagement of the Erk MAPK pathway is primarily mediated" - In the paragraph "Effectors of the Mnk kinases" the authors state: Notably, Mnk2 can exert kinase independent functions". Please clarify this point. - In the paragraph "eIF4E" the sentence "Based on X-ray crystallography data, Scheper et al. have speculated that the phosphate group on Ser 209 may negatively interact with the phosphate groups on the RNA backbone as well as the mRNA cap and have put forth a model in which Mnk mediated phosphorylation of eIF4E after the formation of the translation initiation complex leads to the release of eIF4E and thereby enables it to be recruited by other mRNAs [34].". The sentence is too long, please insert a dot. - In the paragraph "Sprouty 2" pag 9 line 6 substitute Sprouty2 with Spry2. - The paragraph "PSF2 is based only on one paper (Buxade et al 2008). - pag 18 line 5 substitute "Using an unbiased chromatin immune-precipitation sequencing approach to identify the transcriptional targets of YB-1; Astanehe et al. identified Mnk1 as a YB-1 transcriptional target [95] with "Using an unbiased chromatin immune-precipitation sequencing approach to identify the transcriptional targets of YB-1, Astanehe

et al. identified Mnk1 as a YB-1 transcriptional target [95]. (substitute semicolon ; with comma ,) - pag 21 substitute the sentences " Interestingly, while lack of the Mnk kinases does not inhibit Th1 and Th17 differentiation in vitro; immunization of mice with myelin oligodendrocyte glycoprotein peptide in complete Freund's adjuvant, an experimental model of autoimmune encephalomyelitis resulted in attenuated production of IFNγ and IL-17 by CD4 T cells and attenuated differentiation of Th1 and Th17 cells [112]." with "Interestingly, while lack of the Mnk kinases does not inhibit Th1 and Th17 differentiation in vitro, immunization of mice with myelin oligodendrocyte glycoprotein peptide in complete Freund's adjuvant, an experimental model of autoimmune encephalomyelitis, resulted in attenuated production of IFNγ and IL-17 by CD4 T cells and attenuated differentiation of Th1 and Th17 cells [112]. - In "Conclusions" substitute the sentence: "Multiple proteins such as those involved in mRNA translation (eIF4E, eIF4G), regulation of TNFα mRNA expression (hnRNPA1, PSF), regulation of platelet activity (cPLA2), regulation of receptor tyrosine kinase activity (Spry2) are regulated by the Mnk kinases" with "Multiple proteins such as those involved in mRNA translation (eIF4E, eIF4G), in TNFα mRNA expression (hnRNPA1, PSF), in platelet activity (cPLA2) and in modulation of receptor tyrosine kinase activity (Spry2) are regulated by the Mnk kinases". - Please check references since some references report the complete name of the journal and others the abbreviation (for example references 21, 30, 47, 48 etc)

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COMMENTS TO AUTHORS

In this review Joshi and Platanias extensively cover the various roles of the MNK kinases, MNK1 and MNK2, in a wide range of cellular and biological processes. Based on recent findings the authors highlight MNKs impact in translation and inflammation-induced malignancies and the need for uncovering the mechanisms that underlie these outcomes, along with the broad-spectrum potential of specific MNK inhibitors in clinic. In this reviewer's opinion, this review should be of sufficient interest to the readership of the World Journal of Biological Chemistry, as it highlights the increasing need to determine the full spectrum of the cellular and biological effects of MNKs. My specific comments are listed below: Major comments: - Additional figure(s) - As there is alternation throughout the text on distinct and opposing roles between MNK1 and MNK2, along with their isoforms, a figure detailing this, so that the reader can clearly see these differential effects would be beneficial. This may also be important as the review highlights unique substrates between the kinases. - Figures lacking detail - Although the figures provided visually summarize what is in the text, additional detail would be beneficial for the reader. For example, including whether functional/biological outcomes are MAPK-dependent/independent, cap-dependent/independent or induced/repressed by MNK1 and MNK2. Minor comments: - Explanation of some abbreviations (eg. ERK, MAPK) are missing

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COMMENTS TO AUTHORS

The manuscript entitled "The Mnk kinase pathway: cellular functions and biological outcomes" is a well written. The authors comprehensively discussed the cellular function and regulation of MnK kinase. This manuscript provided the up-to-date information in the field and discussed the effects of MnK kinase on tumorigenesis, drug resistance, cap-dependent translation, and inflammation. I have a couple of suggestions that may improve this manuscript. 1. A figure to illuminate the MnK isoforms and functional domains will greatly help readers to understand the complex processes. 2. If phosphorylation of eIF4E reduces affinity to 5′ m7G cap (stated on page 7), how does phosphorylation of eIF4E enhance translation of mRNAs with secondary structure at 5′UTR (stated on page 8) and promote lymphomagenesis by up-regulating mRNA translation of anti-apoptotic Mcl-1 (stated on page 15)? Please clarify it.