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ESPS PEER-REVIEW REPORT

Name of journal: World Journal of Biological Chemistry
ESPS manuscript NO: 20242
Title: Connective tissue growth factor differentially binds to members of the cystine knot superfamily and potentiates platelet-derived growth factor-B signaling in rabbit corneal fibroblast cells
Reviewer's code: 01585205
Reviewer's country: China
Science editor: Yue-Li Tian
Date sent for review: 2015-05-30 16:54
Date reviewed: 2015-06-10 22:33

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

CTGF is known as an important growth factor in regulating diverse biological functions, including cell adhesion, migration, tissue wound repair, fibrotic disease cancers. In this paper, the author demonstrated that CTGF has different binding strengths to VEGF-A, PDGF-B,BMP-4, and TGF-β, which regulate these growth factors triggered downstream signaling pathway. The paper is well written, The data presented are clean with appropriate controls, the experiments are outlined clearly and in logical order. Additional comments as detailed below 1.PDGFR can bind with multiple tyrosine kinase through its SH2 and SH3 domain, the author proved that CTGF can influence PDGFR-mediated PI3K-AKT activation. Can CTGF also influence other PDGFR-mediated signalling pathway? 2.Why CTGF have no impact on PDGFR-triggered ERK activation?

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ESPS PEER-REVIEW REPORT

Name of journal: World Journal of Biological Chemistry
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[ ] Grade A: Excellent	[Y] Grade A: Priority publishing	Google Search:	[ ] Accept
[ ] Grade B: Very good	[ ] Grade B: Minor language polishing	[ ] The same title	[ ] 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	[Y] Minor revision
[ ] Grade E: Poor		[Y] No	[ ] Major revision
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		[ ] Duplicate publication	
		[ ] Plagiarism	
		[Y] No	

COMMENTS TO AUTHORS

The manuscript by Pi et al. compared binding of CTGF to several cycstine knot proteins including VEGF-A, PDGF-B, BMP4 and TGF-beta1. Binding data are consistent in two systems: yeast two hybrid and SPR. The binding to PDGF-B was shown to enhance its association with its receptor and the activation of downstream signaling. Some minor comments: The name PDGF-B was used in the yeast two hybrid study, but PDGF-BB was used in SPR and cell signaling assay. Are they referred to the same protein? Is there any reason that two different names should be used in the same paper?

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[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	[Y] Major revision
		BPG Search:	
		[ ] The same title	
		[ ] Duplicate publication	
		[ ] Plagiarism	
		[Y] No	

COMMENTS TO AUTHORS

The topic of this work is of high interest. The authors approach to the study of the interactions between CTGF and a number of its interactors using the LexA-based yeast two-hybrid system and SPR analysis, and tried to explain the results using some functional aspects. I suggest to accept with major revisions. Because there are some points unclear, in particular: A) the measured KD of 43 nM for the interaction between PDGF-B and CTGF by SPR analysis is not in agreement with the data obtained for LexA-based yeast two-hybrid experiments, infact TGF-β1 that in figure 1 show a better affinity toward CTGF, has a comparable KD between 30 and 60 nM. Please clarify this point. B) The amount of PDGF-B in the PDGF-B/PDGFRβ experiments in presence of CTGF is very low with respect to the amount of CTGF, how explain the authors the continued rise of the signal with the addition of CTGF without the reaching of a plateau? C) In the SPR analysis the author not exclude the possibility of a specific or nonspecific interaction of CTGF to the PDGF-B/PDGFRβ complex, probably new experiments should be carried out in order to demonstrate the obtained results. D)

Conclusions are very generic and lacking in the description of a clear mechanism of action, functional data do not support the other results so this part must be improved. Minor revision: 1) The authors forgot to indicate the figure 1A in the text. 2) In figure 2A there are 6 lines and 5 concentrations indicated (red line). 3) In figure 2B the concentrations of PDGF-B are different from the ones reported in the figure legend as well as in the text (the concentrations refer to the amount of PDGF-B in figure 3A) 4) Authors move from the use of nM (more precise) to the use of ng or ug/ml, this unit of measure avoid to compare the results with the previous experiments. I suggest to use only molar concentration in all paper for a better comparison of data. 5) Higher concentrations of CTGF are indicated to be used in materials for the PDGF-B/PDGFRβ experiments, but results are not showed. 6) It is unclear if the authors use PDGF-B and PDGF-BB to describe the use of domain, monomer or dimer of PDGF-B, please make order on this. 7) The controls in the interaction experiments between PDGF-B/PDGFRβ in presence of CTGF are important to demonstrate the absence of the direct interaction between PDGFRβ and CTGF, so please add as supplementary data. 8) Raw data for the elaborations in figure 3B referring to PDGF-B/PDGFRβ interaction in presence of increasing CTGF concerntrations, should be added as supplementary data.

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		[ ] Duplicate publication	
		[ ] Plagiarism	
		[Y] No	

COMMENTS TO AUTHORS

The manuscript is clear, well written and complete. The antagonism for the binding between CTGF and the receptors of several growth factors is clearly explained and shows the basis of CTGF action to regulate angiogenesis and fibrosis at specific moments of animal life, for example during embryo development