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ESPS Peer-review Report
Name of Journal: World Journal of Biological Chemistry
ESPS Manuscript NO: 7365
Title: Ubiquitination and Destruction of KLF5 by FBW7
Reviewer code: 02446337
Science editor: Gou, Su-Xin
Date sent for review: 2013-11-16 15:32
Date reviewed: 2013-12-04 03:22

CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[ ] Grade A (Excellent)	[ ] Grade A: Priority Publishing	Google Search:	[ ] Accept
[Y] Grade B (Very good)	[Y] 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)	[ ] Grade D: rejected	BPG Search:	[Y] Minor revision
[ ] Grade E (Poor)		[ ] Existed	[ ] Major revision
		[ ] No records	

COMMENTS TO AUTHORS
Interesting Review. The translational aspects of KLF5 ubiquitination should be addressed, focusing on the role of KFL5 in cardiovascular disorders and angiogenesis A paragraph on cardiovascular disease should be added, quoting and briefly discussing the following articles on the importance of angiogenesis in cardiovascular disease: -Significance of the transcription factor KLF5 in cardiovascular remodeling. Manabe I. J Thromb Haemost. 2005 Aug;3(8):1569-76. -KLF5/BTEB2 is a target for angiotensin II signaling and an essential regulator of cardiovascular remodeling. Shindo T, Nat Med. 2002 Aug;8(8):856-63. -Evaluation of the anti-angiogenic properties of the new selective αVβ3 integrin antagonist RGDechiHCit. Iaccarino G. J Transl Med. 2011 Jan 13;9:7. -Flank Sequences of miR-145/143 and Their Aberrant Expression in Vascular Disease: Mechanism and Therapeutic Application. Liu X, J Am Heart Assoc. 2013 Oct 28;2(6):e000407. Epidemiology of Cardiovascular Disease in the 21st Century: Updated Numbers and Updated Facts G. Santulli JCvD 2013; 1(1): 1-2 I couldn't see Fig. 2 (I believe it is a Macro issue of Word).

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ESPS Peer-review Report
Name of Journal: World Journal of Biological Chemistry
ESPS Manuscript NO: 7365
Title: Ubiquitination and Destruction of KLF5 by FBW7
Reviewer code: 00227653
Science editor: Gou, Su-Xin
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Date reviewed: 2013-12-17 15:19

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	[ ] Existed	[ ] High priority for publication
[Y] Grade C (Good)	[ ] Grade C: a great deal of language polishing	[ ] No records	[ ]Rejection
[ ] Grade D (Fair)	[ ] Grade D: rejected	BPG Search:	[Y] Minor revision
[ ] Grade E (Poor)		[ ] Existed	[ ] Major revision
		[ ] No records	

COMMENTS TO AUTHORS

This manuscript summarizes the proteasomal degradation of KLF5 by FBW7, an E3 ubiquitin ligase. Authors describes interaction of KLF5 with FBW7 and poly-ubiquitination of KLF5 by FBW7. In addition, a specific phosphor-epitope, Cdc4 phospho-degron (CPD), of KLF5 which is required for GSK3β-mediated phosphorylation and interaction with FBW7 following phosphorylation. Furthermore, physiological consequences of FBW7-mediated KLF5 degradation in cancer cell proliferation are summarized in mouse model. The manuscript is well-written. However, I would like to comment minor points as below. Minor points 1. Page 2 (Abstract page), line 5: Post-translational 2. Page 2 (Abstract page), line 11: functional Cdc4 phospho-degrons (CPDs). 3. Page 2 (Abstract page), line 15: we summarize the progress of FBW7-mediated KLF5 ubiquitination and degradation 4. Page 3, line 20: spacing before reference (respectively [1]) 5. Page 3, line 24: delete etc 6. Page 3, line 32: post-translational 7. Page 3, line 33: Lys202 8. Page 4, line 45: one line spacing before subtitle 9. Page 5, line 56: SEL10 or AGO, delete FBW7 10. Page 5, line 57: delete SKP1 (SKP1 is redundantly noted) 11. Page 5, line 59: delete period after the word "Table" 12. Page 5, line 62: delete space between FBW and 7? 13. Page 6, line 72: FBW7-mediated 14. Page 6, lines 75-77: please describe this sentence in a different way 15. Page 6, line 78 subtitle: delete "both" 16. Page 6, line 84: abolished their interaction with FBW7 17. Page 6, line 84: WD40 repeats instead of W40 repeats 18. Page 6, line 85: please specify isoform of GSK3 19. Page 7, line 89 subtitle: spacing a line before subtitle 20. Page 7, line 98: Substitution instead of Mutation 21. Page 7, line 98: FBW7-mediated 22. Page 7, line 101: FBW7-deficient 23. Page 7, line 103: delete "both" 24. Page 7, line 105: FBW7-induced 25. Page 8, line 106: "Besides FBW7" instead of "Except for FBW7" 26. Page 8, line 111: "vice versa" instead of "vice verse" 27. Page 8, line 114 subtitle: spacing a line before subtitle 28. Page

8, line 117: Mutations of individual CPDs 29. Page 8, line 118: simultaneous mutations 30. Page 8, line 122: FBW7-mediated 31. Page 9, line 132: FBW7-mediated 32. Page 9, line 135: FBW7-mediated 33. Page 10, line 142 subtitle: FBW7-mediated 34. Page 10, line 146: KLF5 CPD mutant-mediated 35. Page 10, line 147: "KLF5 target genes" instead of "KLF5-transactivated genes" 36. Page 10, line 155: KLF5-dependent manner 37. Page 10, line 158: Spacing a line before subtitle 38. Page 11, line 160: occur instead of occurs 39. Page 11, line 163: A Fbw7 R482Q mutant mouse model was generated in Dr. Ian Tomlinson's laboratory 40. Page 11, line 167: particularly 41. Page 11, line 170: FBW7-mediated 42. Page 11, line 171: Dr. Shimano and colleagues show that 43. Page 11, lines 173-174: please correct this sentence 44. Please align phosphorylation sites of right column in the Table 1.

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ESPS Peer-review Report
Name of Journal: World Journal of Biological Chemistry
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Title: Ubiquitination and Destruction of KLF5 by FBW7
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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
[ ] Grade A (Excellent)	[Y] Grade A: Priority Publishing	Google Search:	[ ] Accept
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		[ ] No records	[ ] Major revision

COMMENTS TO AUTHORS

KLF5 is an important transcription factor involved in cell proliferation and tumorigenesis. In this review, Luan and Wang clearly showed the ubiquitination and destruction process of KLF5 by FBW7. The interaction between KLF5 and FBW7 is controlled by the three CPDs of KLF5, mutation of which interfere FBW7 function of KLF5 degradation. This review clearly and concisely shows the FBW7-KLF5 pathway including its importance.