

ESPS Peer-review Report

Name of Journal: World Journal of Clinical Oncology

Ms: 1329

Title: The role of E3 ubiquitin ligases in lung cancer

Reviewer code: 00227722

Science editor: l.jiang@wjgnet.com

Date sent for review: 2012-12-04 15:47

Date reviewed: 2012-12-24 00:30

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

COMMENTS

CONFIDENTIAL COMMENTS TO EDITOR:

The manuscript is suggested to be accepted for publication with minor revisions.

COMMENTS TO AUTHORS:

The manuscript by Snoek et al reviewed the E3 ubiquitin ligases that are deregulated in lung cancers. The authors made an in-depth discussion about the potential utility of them as anti-cancer targets. G. J. Peter's group has made considerable progress in pharmacological research of anticancer agents. In this reviewer they evaluate the E3 ubiquitin ligases that are related to lung cancer and their potency to function as drugable targets. The manuscript is well-organized, and has incorporated the lately findings and provides the comprehensive discussion. The manuscript is acceptable for publication in World Journal of Oncology with some minor revisions indicated below:

1. In Table 1, Siah2 is listed as E3 ubiquitin ligase of HIPK2 in regulation of both cell proliferation and apoptosis. However, when Siah2 was firstly mentioned as a regulator of cell proliferation (page 10), the function of HIPK2 was rarely discussed. Therefore, if the author could provide some background introduction of HIPK2 in context of cell proliferation, it would be better.
2. The full name for IKBKB is suggested to be inhibitor of kappa light polypeptide gene enhancer in B-cells, kinase beta (page 17).
3. The authors would be advised to editorial work of the manuscript, such as paragraph spaces and first line hanging (page 13 and 14).

ESPS Peer-review Report

Name of Journal: World Journal of Clinical Oncology

Ms: 1329

Title: The role of E3 ubiquitin ligases in lung cancer

Reviewer code: 00725035

Science editor: l.jiang@wjgnet.com

Date sent for review: 2012-12-04 15:47

Date reviewed: 2013-01-04 06:13

CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
<input type="checkbox"/> Grade A (Excellent)	<input type="checkbox"/> Grade A: Priority Publishing	Google Search:	<input type="checkbox"/> Accept
<input type="checkbox"/> Grade B (Very good)	<input checked="" type="checkbox"/> Grade B: minor language polishing	<input type="checkbox"/> Existed	<input type="checkbox"/> High priority for publication
<input checked="" type="checkbox"/> Grade C (Good)	<input type="checkbox"/> Grade C: a great deal of language polishing	<input checked="" type="checkbox"/> No records	<input type="checkbox"/> Rejection
<input type="checkbox"/> Grade D (Fair)		BPG Search:	<input type="checkbox"/> Minor revision
<input type="checkbox"/> Grade E (Poor)	<input type="checkbox"/> Grade D: rejected	<input type="checkbox"/> Existed	<input type="checkbox"/> Major revision
		<input checked="" type="checkbox"/> No records	

COMMENTS

CONFIDENTIAL COMMENTS TO EDITOR:

None

COMMENTS TO AUTHORS:

In their manuscript entitled "The role of E3 ubiquitin ligases in lung cancer" Snoek et al. provide a review of current literature on the role of E3 ubiquitin ligases in lung cancer and as a potential anti-cancer target. The investigators have previously published on mechanisms of bortezomib resistance, an FDA approved proteasome inhibitor used in the clinic. Specific E3 ubiquitin ligase inhibitors are being developed to circumvent the considerable toxicity associated with overall inhibition of proteasome-mediated degradation. Currently only one clinically tested drug, an MDM2 inhibitor, has resulted from these efforts. General comments: Do the authors think that inhibition of single E3 ubiquitin ligases may prove to be an effective treatment for lung cancer? Or do they expect it to be part of combination therapy, for instance with chemotherapy? The quality of the review will be enhanced by adding a review of the literature or at least a section on deubiquitinating enzymes as potential targets for anti-cancer therapy.