

ESPS Peer-review Report

Name of Journal: World Journal of Respiriology

Ms: 4329

Title: Von Hippel-Lindau protein and respiratory diseases

Reviewer code: 01235072

Science editor: Song, Xiu-Xia

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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 type="checkbox"/> Grade B: minor language polishing	<input type="checkbox"/> Existed	<input type="checkbox"/> High priority for publication
<input type="checkbox"/> Grade C (Good)	<input type="checkbox"/> Grade C: a great deal of language polishing	<input type="checkbox"/> No records	<input type="checkbox"/> Rejection
<input type="checkbox"/> Grade D (Fair)	<input type="checkbox"/> Grade D: rejected	BPG Search:	<input type="checkbox"/> Minor revision
<input type="checkbox"/> Grade E (Poor)		<input type="checkbox"/> Existed	<input type="checkbox"/> Major revision
		<input type="checkbox"/> No records	

COMMENTS TO AUTHORS

This review deals with the mechanisms underlining the pVHL-mediated pulmonary fibrosis. In cellular adaptation to hypoxia, hypoxia inducible factor (HIF) plays a key role. HIF is regulated by an E3 ligase von Hippel Lindau protein (pVHL). pVHL is overexpressed in lungs of patients with Idiopathic pulmonary fibrosis (IPF), which is a devastating disease with poor patient survival rates. Hallmarks of pulmonary fibrosis include proliferation of lung fibroblasts and exaggerated deposition of extracellular matrix (ECM) proteins, especially collagen and fibronectin. The proper assembly of fibronectin and collagen matrix requires the presence of pVHL, and loss of pVHL prevents fibroblast proliferation. This is a well-written paper that may only need small correction in grammar and brevity.