



**ESPS PEER REVIEW REPORT**

**Name of journal:** World Journal of Biological Chemistry

**ESPS manuscript NO:** 11665

**Title:** Identification of host miRNAs that may limit human rhinovirus replication.

**Reviewer code:** 01805584

**Science editor:** Fang-Fang Ji

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CLASSIFICATION	LANGUAGE EVALUATION	RECOMMENDATION	CONCLUSION
<input type="checkbox"/> Grade A: Excellent	<input checked="" type="checkbox"/> Grade A: Priority publishing	Google Search:	<input type="checkbox"/> Accept
<input checked="" type="checkbox"/> Grade B: Very good	<input type="checkbox"/> Grade B: Minor language polishing	<input type="checkbox"/> Existing	<input checked="" 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"/> Existing	<input type="checkbox"/> Major revision
		<input type="checkbox"/> No records	

**COMMENTS TO AUTHORS**

This manuscript of ‘Identification of host miRNAs that may limit human rhinovirus replication’ investigates the role of miRNAs in the replication of Human Rhinovirus (HRV) in human bronchial epithelial cells. Some interesting results have been observed that both miR-128 and miR-155 target HRV-1B. Over-expression of miR-155 inhibits HRV-1B RNA accumulated in BEAS-2B cell line. In contrast, DICER knock-down and miR-155-specific anti-miR enhance the viral replication, as anticipated in-silico. These findings suggest a critical place of microRNA in the innate immune response to HRV infection, and may shed light on the novel strategy for antiviral treatment. Major comments 1. Multiple bioinformatic methods, such as TargetScan, miRbase, and miRanda, have already been developed to predict the targets of miRNAs. However, there is limited consistence between different algorithms. As compared to the single-algorithm based target prediction, which gives rise to significant bias, combination of multiple algorithms may serve as a better solution. Additionally, the exact target genes of miR-155, miR-128 have not been uncovered in the trascriptome of HRV-1B. Discussion about this problem is then suggested. Minor comments 1. There is only one dosage (30 nM) of anti-DICER siRNA used in the experiments. In order to confirm the efficiency of siRNA transfection, the percentage of cells positive for GFP (or other markers) should be evaluate. Similarly, authors should demonstrate the parameters of recombinant virus, such as TU, in both lentivirus-based and HRV-1B-based infection of BEAS-2B cell lines. 2. A comma may be needed in the Conclusion of Abstract, ‘our results suggest that pathological changes in microRNA expression, as



## BAISHIDENG PUBLISHING GROUP INC

8226 Regency Drive, Pleasanton, CA 94588, USA

Telephone: +1-925-223-8242

Fax: +1-925-223-8243

E-mail: [bpgoffice@wjgnet.com](mailto:bpgoffice@wjgnet.com)

<http://www.wjgnet.com>

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already reported for asthma or chronic obstructive pulmonary disease (COPD) have the potential to affect Rhinovirus replication and therefore may play a role in virus-induced exacerbations'. In addition, there seems to be some grammar mistakes in the text, such as 'the presence of secondary structures not accounted for by the prediction algorithm can be considered responsible for a lower than expected microRNA accessibility to the target site' (page 19).