

ICMJE DISCLOSURE FORM

Date: Jun 24th, 2021

Your Name: Hui-Ting Fu

DouManuscript Title: Tumor necrosis factor- α promotes airway mucus hypersecretion by repressing miR-146a-5p and miR-134-5p levels in human airway epithelial cells

Manuscript number (if known): TCR-20-3375-R2

In the interest of transparency, we ask you to disclose all relationships/activities/interests listed below that are related to the content of your manuscript. "Related" means any relation with for-profit or not-for-profit third parties whose interests may be affected by the content of the manuscript. Disclosure represents a commitment to transparency and does not necessarily indicate a bias. If you are in doubt about whether to list a relationship/activity/interest, it is preferable that you do so.

The following questions apply to the author's relationships/activities/interests as they relate to the current manuscript only.

The author's relationships/activities/interests should be defined broadly. For example, if your manuscript pertains to the epidemiology of hypertension, you should declare all relationships with manufacturers of antihypertensive medication, even if that medication is not mentioned in the manuscript.

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Time frame: Since the initial planning of the work			
1	All support for the present manuscript (e.g., funding, provision of study materials, medical writing, article processing charges, etc.) No time limit for this item.	This work was supported by the National Natural Science Foundation of China (No. 81704025)	
Time frame: past 36 months			
2	Grants or contracts from any entity (if not indicated in item #1 above).	None	
3	Royalties or licenses	None	

4	Consulting fees	None	
5	Payment or honoraria for lectures, presentations, speakers bureaus, manuscript writing or educational events	None	
6	Payment for expert testimony	None	
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8	Patents planned, issued or pending	None	
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10	Leadership or fiduciary role in other board, society, committee or advocacy group, paid or unpaid	None	
11	Stock or stock options	None	
12	Receipt of equipment, materials, drugs, medical writing, gifts or other services	None	
13	Other financial or non-financial interests	None	

Please summarize the above conflict of interest in the following box:

Dr Huiting Fu reports the work was supported by the National Natural Science Foundation of China (No. 81704025).

Please place an "X" next to the following statement to indicate your agreement:

I certify that I have answered every question and have not altered the wording of any of the questions on this form.

ICMJE DISCLOSURE FORM

Date: Jul 7th, 2021

Your Name: Yan Zhang

DouManuscript Title: Tumor necrosis factor- α promotes airway mucus hypersecretion by repressing miR-146a-5p and miR-134-5p levels in human airway epithelial cells

Manuscript number (if known): TCR-20-3375-R2

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ICMJE DISCLOSURE FORM

Date: Jul 7th, 2021

Your Name: Ping Zhang

DouManuscript Title: Tumor necrosis factor- α promotes airway mucus hypersecretion by repressing miR-146a-5p and miR-134-5p levels in human airway epithelial cells

Manuscript number (if known): TCR-20-3375-R2

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ICMJE DISCLOSURE FORM

Date: Jul 7th, 2021

Your Name: Huan Wu

DouManuscript Title: Tumor necrosis factor- α promotes airway mucus hypersecretion by repressing miR-146a-5p and miR-134-5p levels in human airway epithelial cells

Manuscript number (if known): TCR-20-3375-R2

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ICMJE DISCLOSURE FORM

Date: Jul 7th, 2021

Your Name: Xuan-Qiu Sun

DouManuscript Title: Tumor necrosis factor- α promotes airway mucus hypersecretion by repressing miR-146a-5p and miR-134-5p levels in human airway epithelial cells

Manuscript number (if known): TCR-20-3375-R2

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ICMJE DISCLOSURE FORM

Date: Jul 7th, 2021

Your Name: Shu-Yang Shen

DouManuscript Title: Tumor necrosis factor- α promotes airway mucus hypersecretion by repressing miR-146a-5p and miR-134-5p levels in human airway epithelial cells

Manuscript number (if known): TCR-20-3375-R2

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ICMJE DISCLOSURE FORM

Date: Jul 7th, 2021

Your Name: Dan-Bo Dou

DouManuscript Title: Tumor necrosis factor- α promotes airway mucus hypersecretion by repressing miR-146a-5p and miR-134-5p levels in human airway epithelial cells

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