

13-Apr-2017

World Journal of Gastroenterology

Dear Ya-Juan Ma Science editor

RE: Manuscript No. 33465

Manuscript entitled "Fibroblast-Derived CXCL12/SDF-1 α Promotes CXCL6 Secretion and Co-operatively Enhance Metastatic Potential Through PI3K/Akt/mTOR Pathway in Colon Cancer "

Thank you for your kind letter of 12-Apr-2017. Those comments are all valuable and very helpful for revising and improving our paper. We have studied comments carefully and have made correction. We hope that it is now suitable for publication in World Journal of Gastroenterology. I look forward to hearing from you again.

With best regards,

Yours sincerely,

Jiachi Ma, M.D., Ph.D.

Department of General Surgery, Gansu Provincial People's
Hospital, 204 Dong Gang West Road, Lanzhou 730000,
Gansu Province, People's Republic of China

Tel: +86-931-8281382

Fax: +86-931-8266957

E-mail: tsmjc@hotmail.com

Responds to the reviewer's comments

1. In Figure 1, there was no expression of CXCL12 (HT29 cells, etc.), but in Figure 2, the control level is similar (even less) with DLD cells. The discrepancy of the result to be explained.

Response: Thank you for kind suggestion. in Figure 2, the secreted level of CXCL6 by HT-29 (control) is $8.15 \pm 0.46 \text{ pg/ml/} 10^6 \text{ cells}$, but the secreted level of CXCL6 by DLD-1 (control) is $13.17 \pm 0.62 \text{ pg/ml/} 10^6 \text{ cells}$. The production of CXCL6 by DLD-1 is more high than HT-29. Because DLD-1 cells can secrete CXCL12.

2. To make conclusion of the signals for CXCL12 in the cells, the effect of the inhibitor against cell invasion or migration should be tested.

Response: Thank you for kind suggestion. According to your sincere advice, our team will mainly studied the antagonistic effect of PI3K/Akt/mTOR by inhibitor or antagonist on invasion or migration of colon cancer cell and HUVEC.

3. For minor, Page 5, line 8th form the bottom, the CXCL6 should be changed to CXCL12.

Response: Thank you for kind suggestion. I am very sorry for spelling errors. We have changed to "CXCL12".

With best regards,

Yours sincerely,

Ma Jiachi, M.D., Ph.D.

Department of General Surgery, Gansu Provincial People's

Hospital, 204 Dong Gang West Road, Lanzhou 730000,

Gansu Province, People's Republic of China

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Fax: +86-931-8266957

E-mail: tsmjc@hotmail.com