May 24, 2018

Dear Editor,

Please find enclosed the edited manuscript in Word format (file name: 39768-Revised Manuscript.docx).

Title: Clinical correlation of B7-H3 and B3GALT4 with the prognosis of colorectal cancer

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The manuscript has been improved according to the suggestions of reviewers:
1. Reviewer's code: 00068625
i. The problem of too many acronyms has been improved so that the paper would be understood more easily.
ii. The quality of Figure 1 has been modified because we replaced the photos with high pixels.
2. Reviewer's code: 03001816
i. We constructed the plasmid vectors with green fluorescent protein (GFP) and neomycin resistant gene which overexpressed B7-H3 or knockdown of B7-H3. Then the mixture of plasmid and lipo2000 was added to the cell medium by lipofectin transfection. Finally, the stably transfected cell lines were generated through G418 sreening.
ii. There was a DNase step in the qRT-PCR experiment. We chose the reagent of reverse transcription from Vazyme (Nanjing , China), HiScript II 1st Strand cDNA Synthesis Kit (+gDNA wiper). In one step, gDNA wiper Mix treated total RNA in 42°C for 2min could eliminate the genomic contamination quickly and completely.
iii. Compared to wild-type cells, the stable overexpression of B7-H3 cells promoted proliferation, drug resistance, EMT, and inhibited apoptosis, vice versa. These results in our previous study had been published. Next, we intend to investigate other characteristics of cell physiology and specific regulatory mechanism to interpret the role of B7-H3 in colorectal cancer progression.
3. Reviewer's code: 00503405
i. We made the cell line study mainly for the primary validation of the regulatory relationship between B7-H3 and B3GALT4. In this study, we focused on the correlation of B7-H3 and B3GALT4 with the prognosis of colorectal cancer. We didn't provide the possible explanation of the positive correlation between B7-H3 and B3GALT4. In the subsequent research, we will consider to investigate which molecular mechanism involves in regulating the expression of B3GALT4 via B7-H3. Furthermore, the relationship of the immune function and glycosylation of tumor-associated protein in colorectal cancer should be discussed in our future work.
Then, format has been updated. References and typesetting were corrected.

Thank you again for publishing our manuscript in the *World Journal of Gastroenterology*.

Sincerely yours,

Ting Zhang

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