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Journal Name:	Journal of Cancer and Tumor International	
Manuscript Number:	Ms_JCTI_31326	
Title of the Manuscript:	ANGIOTENSIN II TYPE 2 RECEPTOR: A NOVEL MODULATOR OF INFLAMMATION IN PANCREATIC DUCTAL ADENOCARCINOMA THROUGH REGULATION OF NF-KB ACTIVITY	
Type of the Article	Original Research Article	

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This journal's peer review policy states that **NO** manuscript should be rejected only on the basis of 'lack of Novelty', provided the manuscript is scientifically robust and technically sound.

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PART 1: Review Comments

	Reviewer's comment	Author's comment (if agreed with reviewer, correct the manuscript and highlight that part in the manuscript. It is mandatory that authors should write his/her feedback here)
Compulsory		
REVISION comments		
Minor REVISION comments	In this manuscript, the authors explored pro-inflammatory and pro-invasive roles of angiotensin II type 2 receptor (AT2R) in pancreatic ductal adenocarcinoma (PDAC) cells. The authors found that AT2R blockade reduced proliferation, migration and invasion of PDAC cells. In consequence, they speculated that one mechanism by which AT2R promote inflammation is through activation of constitutive and AngII-mediated NF-κB. Therefore, they suggested that AT2R-blockade could be a novel therapeutic strategy to target multiple pathways that mediate PDAC carcinogenesis. But several revisions would be considered especially in the discussion part. Finally, there are numerical errors in figures to be corrected with inconsistent use of words as follows. 1) Page 2, Line 29: 5 μm serial sections were 2) Page 3, Line 18: 2 x 10 ⁵ MiaPaCA-2 or AsPC-1 cells 3) Page 3, Line 22: using ImageJ software. 4) Page 3, Line 22: using ImageJ software. 5) Page 4, Line 2: using ImProm-II TM Reverse Transcription System_(Promega). 6) Page 4, Line 11 and Page 11, Line 6: abbreviation of NF-κB 7) Page 4, Line15: analyzed by Student-t-test. 8) Figure 3B, 5C, 6A, and 6B.	
Optional/General comments		

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