

Instructions

The purpose of this form is to provide readers of your manuscript with information about your other interests that could influence how they receive and understand your work. The form is designed to be completed electronically and stored electronically. It contains programming that allows appropriate data display. Each author should submit a separate form and is responsible for the accuracy and completeness of the submitted information. The form is in six parts.

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Dai 1



Section 1. Identifying Inform	nation	
1. Given Name (First Name) Lan	2. Surname (Last Name) Dai	3. Date 02-December-2020
4. Are you the corresponding author?	✓ Yes No	
5. Manuscript Title Inhibition of sphingosine kinase 2 dow ovarian cancer	n-regulates ERK/c-Myc pathway and reduces cell pro	oliferation in human epithelial
6. Manuscript Identifying Number (if you ki ATM-20-6742	now it)	
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Section 4. Intellectual Prope	rty Patents & Copyrights	
	ned, pending or issued, broadly relevant to the wor	k? ☐ Yes ✓ No

Dai 2



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Wang 1



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1. Given Name (First Name) Wenjing	2. Surname (Last Name) Wang	3. Date 02-December-2020
4. Are you the corresponding author?	☐ Yes ✓ No	Corresponding Author's Name Wen Di; Lan Dai; Keqi Song
5. Manuscript Title Inhibition of sphingosine kinase 2 dow ovarian cancer	n-regulates ERK/c-Myc pat	hway and reduces cell proliferation in human epithelial
6. Manuscript Identifying Number (if you kn ATM-20-6742	now it)	_
Section 2. The Work Under C	onsideration for Public	cation
any aspect of the submitted work (including statistical analysis, etc.)? Are there any relevant conflicts of inter	g but not limited to grants, da	a third party (government, commercial, private foundation, etc.) for ata monitoring board, study design, manuscript preparation,
Section 3. Relevant financial	activities outside the s	submitted work.
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Do you have any patents, whether plan		

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Liu 1



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Song 1



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5. Manuscript TitleInhibition of sphingosine kinase 2 down ovarian cancer6. Manuscript Identifying Number (if you kr ATM-20-6742	n-regulates ERK/c-Myc pathway and reduces cell p	oroliferation in human epithelial
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Di 1



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