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PEER-REVIEW REPORT

Name of journal: World Journal of Gastroenterology

Manuscript NO: 52257

Title: Upregulation of MiR-34c after Silencing F-1 Inhibits Paclitaxel Combined with cisplatin Resistance in Gastric Cancer Cells

Reviewer's code: 02537403
Position: Editorial Board
Academic degree: PhD
Professional title: Senior Lecturer
Reviewer's country: Romania
Author's country: China
Reviewer chosen by: Artificial Intelligence Technique
Reviewer accepted review: 2019-11-09 19:47
Reviewer performed review: 2019-11-22 15:42
Review time: 12 Days and 19 Hours

SCIENTIFIC QUALITY	LANGUAGE QUALITY	CONCLUSION	PEER-REVIEWER STATEMENTS
[ ] Grade A: Excellent	[ ] Grade A: Priority publishing	[ ] Accept (High priority)	Peer-Review:
[Y] Grade B: Very good	[Y] Grade B: Minor language polishing	[Y] Accept (General priority)	[Y] Anonymous
[ ] Grade C: Good	[ ] Grade C: A great deal of language polishing	[ ] Minor revision	[ ] Onymous
[ ] Grade D: Fair	[ ] Grade D: Rejection	[ ] Major revision	Peer-reviewer's expertise on the topic of the manuscript:
[ ] Grade E: Do not publish		[ ] Rejection	[Y] Advanced
			[ ] General
			[ ] No expertise
			Conflicts-of-Interest:
			[ ] Yes
			[Y] No

SPECIFIC COMMENTS TO AUTHORS

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Cancer genomics is regulated by transcription factors, coding genes and miRNAs, which can influence the development of tumors. Studies have showed that the interaction between miRNAs and transcription factors can have an impact on cell proliferation and growth, and that E2F1 may promote tumorigenesis of GC. MiR-34c has low expression in many tumors, being associated with biological functions such as apoptosis and proliferation; E2F1 may mediate the transcriptional level of miR-34c. This study assessed the relationship between the molecular mechanisms of E2F1 and miR-34c, and their impact on GC. This research has a positive impact not only for understanding gastric cancer development, but also for discovering new treatment targets. The study was performed on paired gastric cancer tissues and adjacent normal tissues coming from 74 gastric cancer patients. MiR-34c and E2F1 were detected using qPCR and Western blot, and the drug resistance of gastric cancer cells to paclitaxel and cisplatin was induced by concentration gradient increasing method. Furthermore, E2F1 and miR-34c overexpression or low expression vectors were elaborated and transfected into drug-resistant gastric cancer cells. Moreover, drug resistance-related protein and apoptosis-related protein, cell apoptosis and cell cycle were detected. Study results confirmed that E2F1 could bind to the miR-34c promoter in a targeted way, therefore E2F1 could inhibit the expression of miR-34c at the transcriptional level, and might affect downstream genes and cell biological functions. Furthermore, by transfecting GC cells with miR-34c overexpressed/silenced expression vector, miR-34c can promote cell apoptosis, inhibit cell proliferation and combine cisplatin resistance with taxol; miR-34c exerts inhibitory effect on gastric cancer cells. E2F1 inhibits the expression of miR-34c, promoting cell proliferation and inhibiting cell apoptosis. Current study found that E2F1 may also regulate the expression of MRP and MDR-1, leading to the change of paclitaxel-cisplatin resistance in GC cells. MiR-34c seems to have an effect on drug resistance of GC cells, by mediating the expression of MAPT and other proteins. In

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conclusion, E2F1 inhibits miR-34c to promote the proliferation of gastric cancer and enhance the resistance to chemotherapy, while silencing E2F1 is favorable to improve the efficacy of chemotherapy in gastric cancer cells. The results of the study are important for clinical treatment – by regulating the expression level of E2F1/miR34c we will be able to improve the sensitivity of gastric cancer cells to paclitaxel combined with cisplatin and enhance the efficacy of chemotherapy.

INITIAL REVIEW OF THE MANUSCRIPT

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[ ] Plagiarism

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Reviewer's code: 00058340
Position: Editor-in-Chief
Academic degree: DSc, MD, PhD
Professional title: Professor
Reviewer's country: United States
Author's country: China
Reviewer chosen by: Jie Wang
Reviewer accepted review: 2019-11-22 15:40
Reviewer performed review: 2019-11-23 21:02
Review time: 1 Day and 5 Hours

SCIENTIFIC QUALITY	LANGUAGE QUALITY	CONCLUSION	PEER-REVIEWER STATEMENTS
[ ] Grade A: Excellent [ ] Grade B: Very good [Y] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish	[ ] Grade A: Priority publishing [ ] Grade B: Minor language polishing [Y] Grade C: A great deal of language polishing [ ] Grade D: Rejection	[ ] Accept (High priority) [ ] Accept (General priority) [ ] Minor revision [Y] Major revision [ ] Rejection	Peer-Review: [Y] Anonymous [ ] Onymous Peer-reviewer's expertise on the topic of the manuscript: [ ] Advanced [Y] General [ ] No expertise Conflicts-of-Interest: [ ] Yes [Y] No

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The authors examined expression of E2F1 and miR-34c in specimens of human gastric cancer and in adjacent normal tissue. They found that expression of E2F1 was increased, while miR-34c was decreased in gastric cancer. In separate, in vitro experiments they induced gastric cancer cells resistance to paclitaxel and cisplatin, and found that E2F1 expression increased, while miR-34c expression decreased. Further they found that both silencing E2F1 and over-expressing miR-34c could increase the sensitivity of drug-resistant gastric cancer cells to paclitaxel + cisplatin by promoting cell apoptosis and inhibiting cell proliferation. Comments: 1) The role of E2F1 and Mi-RNA-34 family in gastric cancer and drug resistance of gastric cancer are well recognized (see references below), therefore the authors should clearly spell out what is new in their paper comparing with the literature. 2) Numerous studies demonstrated that in significant proportion of gastric cancer H. Pylori genome is incorporated into cancer cells. The authors should at least elaborate on that and how this would change potential role of E2F1 and Mi-RNA-34 3) The paper would significantly benefit from including a diagram representing graphically representing the role of E2F1 and Mi-RNA-34 in gastric cancer and its resistance. 4) The paper requires extensive linguistic revisions. Wang AM at al. Yin Yang 1 is a target of microRNA-34 family and contributes to gastric carcinogenesis. Oncotarget. 2014 Jul 15;5(13):5002-16. Riquelme I et al. Emerging Role of miRNAs in the Drug Resistance of Gastric Cancer. Int J Mol Sci. 2016 Mar; 17(3): 424. PMID: 27011182

INITIAL REVIEW OF THE MANUSCRIPT

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