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PEER-REVIEW REPORT

Name of journal: World Journal of Diabetes

Manuscript NO: 57891

Title: Role of ferroptosis in the process of diabetes-induced endothelial dysfunction

Reviewer's code: 03508914
Position: Peer Reviewer
Academic degree: PhD

Professional title: Professor

Reviewer's Country/Territory: China

Author's Country/Territory: China

Manuscript submission date: 2020-06-28

Reviewer chosen by: AI Technique

Reviewer accepted review: 2020-06-30 21:10

Reviewer performed review: 2020-07-01 01:37

Review time: 4 Hours

Scientific quality	[ ] Grade A: Excellent [ ] Grade B: Very good [Y] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish
Language quality	[ ] Grade A: Priority publishing [ ] Grade B: Minor language polishing [Y] Grade C: A great deal of language polishing [ ] Grade D: Rejection
Conclusion	[ ] Accept (High priority) [ ] Accept (General priority) [Y] Minor revision [ ] Major revision [ ] Rejection
Re-review	[Y] Yes [ ] No
Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous Conflicts-of-Interest: [ ] Yes [Y] No

SPECIFIC COMMENTS TO AUTHORS

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In the manuscript, the author found that HG and IL-1β induced ferroptosis in HUVECs and ferroptosis was involved in endothelial dysfunction via the p53-xCT-GSH. This is a novel opinion. However, there are many question to be resolved. 1. It is said that coronary artery atherosclerosis resulted from inflammation of local lesion, however, in this study, human umbilical vein endothelial cell was cultured in vitro, how to mimic the inflammatory environment which played an important role in the formation of atherosclerosis. 2. In previous studies, the injured endothelial cell caused by inflammation or other reasons might undergo necrosis or apoptosis; in this study, the author found that ferroptosis might involve in this process, so the author should which one play a more important role. 3. Cox-2 is pathological factor which is mainly secreted by inflammatory cells, does vein endothelial cell secret this inflammatory cytokines. In figure 1 and figure 2, the author detected the expression of Cox-2, FTH1 and GPX4, is there any connection between them? 4. In figure 2b, DFO could is a kind chelating agent of iron ion, how could it modulate the expression of GXP4? 5. In figure 3a, the mean of this set of experiments is difficult to understand, why the author set this experiment. 6. In previous researched, p53 was regarded as a protect factor for cell to defense oxidative stress, because p53 could work as a transcription factor to regulate the expression of some proteins of antioxidant in stress. However, in this paper, knockdown of p53 lead to the upregulation of Xct which played an antioxidant role, the conclusion is contradicted with most other conclusions, how to explain it. 7. In figure 3c, the expression of CD31, a marker of endothelial, was changed in different group, why? 8.

In the study, GXP4 was detected in many experiments, however the author just want to certify that p53-xCT-GSH axis played a role in ferroptosis of HUVECs, so why GXP4 was detected? 9. In figure 5a, Prussian Blue stain could show the iron ions in blue color, however, the lesion the author showed is brown, why? In addition, as the author said, the lesion is endothelial, however, the brown lesions are located at the tunica media. Another,

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the brown lesion looked like a nonspecific coloring; the author should supply more convincing data. 10. There are many grammatical errors, the language needs further polishing. For examples: (1) And then tested the cell viability (Lack of subject) ; (2) ferroptosis related marker should be ferroptosis-related marker.

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PEER-REVIEW REPORT

Name of journal: World Journal of Diabetes

Manuscript NO: 57891

Title: Role of ferroptosis in the process of diabetes-induced endothelial dysfunction

Reviewer's code: 03465463
Position: Peer Reviewer
Academic degree: FCPS, PhD

Professional title: Adjunct Professor, Professor

Reviewer's Country/Territory: Taiwan

Author's Country/Territory: China

Manuscript submission date: 2020-06-28

Reviewer chosen by: Ya-Juan Ma

Reviewer accepted review: 2020-09-30 10:03

Reviewer performed review: 2020-10-05 10:17

Review time: 5 Days

Scientific quality	[ ] Grade A: Excellent [ ] Grade B: Very good [Y] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish
Language quality	[ ] Grade A: Priority publishing [Y] Grade B: Minor language polishing [ ] Grade C: A great deal of language polishing [ ] Grade D: Rejection
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This submission demonstrated the role of ferroptosis in diabetes-induced endothelial dysfunction. It has novelty and the following concerns need to conduct. 1. Mediation of ferroptosis in endothelial dysfunction in peripheral diseases needs to introduce. 2. In the introduction, “an in-depth analysis of this topic” seems not suitable in scientific publication. 3. Promocell needs the source in detail because it supplied primary HUVECs. 4. Identification of ferroptosis was not showed in the methods. 5. HG (30 mM) used in current study needs the reference(s) to support, particularly the osmotic factor must rule out. 6. Images for ferroptosis, either in Figure 1 or Figure 2, failed to show in clear. 7. In Figure 5, legends remain unknown. Please revise it in detail. 8. In Figure 6, hyperglycemia seems better than DM and proposal model for working model in the wording. Additionally, action site of cytokine did not indicate. Why? 9. Data of PCR did not show. Why?