

PEER-REVIEW REPORT

Name of journal: World Journal of Diabetes

Manuscript NO: 67684

Title: Profilin-1 involved in macroangiopathy induced by AGEs via VSMCs proliferation and proatherogenic mediators expression

Reviewer's code: 02630398

Position: Peer Reviewer

Academic degree: MD

Professional title: Doctor

Reviewer's Country/Territory: China

Author's Country/Territory: China

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Reviewer chosen by: AI Technique

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 Grade E: Do not publish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

SPECIFIC COMMENTS TO AUTHORS

Previous studies have shown that Diabetic macroangiopathy is the leading cause of morbidity and mortality in diabetic patients. After strict blood glucose and risk factors control, the incidence of diabetic vascular complications has not decreased. Based on previous studies on vascular lesions in patients with diabetes and AS, this study made bold assumptions: 1. The plasma levels of profilin-1 and RAGE in patients with coronary heart disease complicated with diabetes were significantly higher than those in patients with CAD alone. 2. AGEs can up-regulate the expression of profilin-1 in aorta or cultured VSMCs, causing vascular remodeling and VSMCs proliferation. 3. Profilin-1 may be involved in AGEs-induced inflammation and vascular remodeling through the JAK2/STAT3 pathway. Then, through clinical studies, cell experiments and animal experiments, the above hypothesis is verified, and the conclusion can properly summarize the data provided by this study. And it is expected to make profilin-1 a promising therapeutic target for the prevention of diabetes mellitus associated with vascular damage. The team's future research could be: 1. To develop a targeted drug against profilin-1, and further verify whether profilin-1 can truly become a new target for the prevention and treatment of diabetes secondary to AS with vascular injury. 2. Further study of profilin-1 on the exact molecular mechanism of AGE-induced vascular lesions.