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PEER-REVIEW REPORT

Name of journal: *World Journal of Diabetes*

Manuscript NO: 99387

Title: Targeting neuronal PAS domain protein 2 and KN motif/ankyrin repeat domains 1: Advances in type 2 diabetes therapy

Provenance and peer review: Invited Manuscript; Externally peer reviewed

Peer-review model: Single blind

Reviewer's code: 05249683
Position: Editorial Board
Academic degree: BSc, MSc, PhD
Professional title: Professor
Reviewer's Country/Territory: Egypt
Author's Country/Territory: China

Manuscript submission date: 2024-07-21

Reviewer chosen by: AI Editor

Reviewer accepted review: 2024-07-23 08:32

Reviewer performed review: 2024-07-24 09:39

Review time: 1 Day and 1 Hour

Scientific quality	[ ] Grade A: Excellent [Y] Grade B: Very good [ ] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish
Novelty of this manuscript	[ ] Grade A: Excellent [Y] Grade B: Good [ ] Grade C: Fair [ ] Grade D: No novelty
Creativity or innovation of this manuscript	[ ] Grade A: Excellent [Y] Grade B: Good [ ] Grade C: Fair [ ] Grade D: No creativity or innovation

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Scientific significance of the conclusion in this manuscript	[Y] Grade A: Excellent [ ] Grade B: Good [ ] Grade C: Fair [ ] Grade D: No scientific significance
Language quality	[ ] Grade A: Priority publishing [Y] Grade B: Minor language polishing [ ] Grade C: A great deal of language polishing [ ] Grade D: Rejection
Conclusion	[ ] Accept (High priority) [ ] Accept (General priority) [ ] Minor revision [Y] 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

In this editorial article, the functions of NPAS2 and KANK1 in type 2 diabetes (T2D) were examined. Their potential as therapeutic targets and their role in β-cell dysfunction, along with the implications for novel therapeutic approaches, were highlighted. The study's understanding of cellular processes and gene regulation provides encouraging avenues for enhancing T2D management through focused therapies. I have the following comments for authors: 1) The topic is important in the research on diabetes; insert a figure that illustrates that. It is recommended that the figure also explain the role of Kanki and their relationship. 2) The language is fine with the exception of three notices: please remove one comma from the first page, line 7, the word new from the first line of the introduction, and replace "for" with "to" on line 7, page 5. The introduction is recommended to be changed as follows: The ongoing fight against type 2 diabetes (T2D) has recently gained momentum with discoveries about the genetic and molecular underpinnings of this widespread disease. A pivotal study by Yin et al. in the World Journal of Diabetes has highlighted the significance of the transcription factor NPAS2 (neuronal PAS domain protein 2) and its downstream target KANK1 (KN motif

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and ankyrin repeat domain 1) in the dysfunction of pancreatic β-cells, a key event in the development of T2D [1]. Since β-cell dysfunction is central to T2D pathogenesis, a deeper understanding of these regulatory mechanisms is crucial for advancing therapeutic approaches. NPAS2 is a component of the circadian clock system, influencing various physiological processes, including glucose metabolism [2]. Its role in β-cell function and insulin secretion has opened new avenues for diabetes research. The study by Yin et al. demonstrates how NPAS2 modulates KANK1 expression, thereby affecting the structural and functional integrity of β-cells. KANK1 plays a critical role in maintaining cell adhesion and cytoskeletal dynamics, which are essential for β-cell function [3]. The interaction between KANK1 and focal adhesion proteins influences the stability and organization of microtubules and actin filaments, thereby impacting insulin secretion [4]. Changes in KANK1 expression can cause disruption in these processes, which can result in β-cell malfunction and accelerate the development of type 2 diabetes. The purpose of this editorial is to place these findings within the larger framework of diabetes research. We can gain a better understanding of NPAS2 and KANK1's potential influence on upcoming T2D treatment approaches by investigating their complex pathways. Yin et al.'s work emphasises how crucial it is to focus on these molecular pathways to maintain β-cell function and enhance diabetic outcomes. The last part of conclusion is recommended to be changed as follows: Utilizing these molecular connections, new therapeutic approaches that specifically improve β-cell resilience can be created. These genetic and molecular interactions should be further investigated in the future, with an emphasis on how they might be altered to provide more precise and potent T2D treatments. As we learn more about the NPAS2-KANK1 pathway and its wider effects, the possibility of developing novel therapies that profoundly enhance the quality of life for people with type 2 diabetes grows. This strategy has the potential to not only maintain β-cell function but also to create all-encompassing plans for improved

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diabetes control [5, 6].

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RE-REVIEW REPORT OF REVISED MANUSCRIPT

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Reviewer's code: 05249683
Position: Editorial Board
Academic degree: BSc, MSc, PhD
Professional title: Professor
Reviewer's Country/Territory: Egypt
Author's Country/Territory: China

Manuscript submission date: 2024-07-21

Reviewer chosen by: Xin-Liang Qu

Reviewer accepted review: 2024-08-22 17:05

Reviewer performed review: 2024-08-22 17:19

Review time: 1 Hour

Scientific quality	[ ] Grade A: Excellent [Y] Grade B: Very good [ ] Grade C: Good [ ] Grade D: Fair [ ] Grade E: Do not publish
Novelty of this manuscript	[ ] Grade A: Excellent [Y] Grade B: Good [ ] Grade C: Fair [ ] Grade D: No novelty
Creativity or innovation of this manuscript	[ ] Grade A: Excellent [Y] Grade B: Good [ ] Grade C: Fair [ ] Grade D: No creativity or innovation

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Conclusion	[ ] Accept (High priority) [Y] Accept (General priority) [ ] Minor revision [ ] Major revision [ ] Rejection
Peer-reviewer statements	Peer-Review: [Y] Anonymous [ ] Onymous Conflicts-of-Interest: [ ] Yes [Y] No

SPECIFIC COMMENTS TO AUTHORS

No more compliments. I do not know why the system requires no less than 300 words for the comments. In this editorial article, the functions of NPAS2 and KANK1 in type 2 diabetes (T2D) were examined. Their potential as therapeutic targets and their role in β-cell dysfunction, along with the implications for novel therapeutic approaches, were highlighted. The study's understanding of cellular processes and gene regulation provides encouraging avenues for enhancing T2D management through focused therapies. I have the following comments for authors: 1) The topic is important in the research on diabetes; insert a figure that illustrates that. It is recommended that the figure also explain the role of Kanki and their relationship. 2) The language is fine with the exception of three notices: please remove one comma from the first page, line 7, the word new from the first line of the introduction, and replace "for" with "to" on line 7, page 5. The introduction is recommended to be changed as follows: The ongoing fight against type 2 diabetes (T2D) has recently gained momentum with discoveries about the genetic and molecular underpinnings of this widespread disease. A pivotal study by Yin et al. in the World Journal of Diabetes has highlighted the significance of the transcription factor NPAS2 (neuronal PAS domain protein 2) and its downstream target

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KANK1 (KN motif and ankyrin repeat domain 1) in the dysfunction of pancreatic β-cells, a key event in the development of T2D. Since β-cell dysfunction is central to T2D pathogenesis, a deeper understanding of these regulatory mechanisms is crucial for advancing therapeutic approaches. NPAS2 is a component of the circadian clock system, influencing various physiological processes, including glucose metabolism. Its role in β-cell function and insulin secretion has opened new avenues for diabetes research. The study by Yin et al. demonstrates how NPAS2 modulates KANK1 expression, thereby affecting the structural and functional integrity of β-cells.