

Be Alert to Diabetes Nephropathy with Cognitive Dysfunction [Response to Letter]

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Dear editor

We have received a letter from Dr. Gan et al¹ about our recently published study.² And thank you for their comment on our study. Our article has predicted and preliminarily verified the targets and the important pathway of YQBS for the treatment of diabetic nephropathy with cognitive dysfunction based on network pharmacology and experimental validation. This laid the foundation for future clinical practice and experimental research, and also made progress in clarifying the mechanism of traditional Chinese medicine in treating inflammatory diabetic complications.

Firstly, we agree with the author's statement in question 1: there may be issues with incomplete database coverage. We also mentioned in the article that some targets of diabetic nephropathy and cognitive dysfunction may not be included in these public databases, but we selected as many authoritative databases as possible in our study to cover as many potential targets as possible. The databases we selected basically include some of the most commonly used databases in current network pharmacology research,^{3–5} which can meet the basic requirements of the study. Although Sprague Dawley rats can not completely simulate the complexity of humans, they have a high degree of homology with humans. And the method of constructing diabetic nephropathy animal model we used in the paper is very mature and is currently commonly used in many references,^{6–8} so it can meet the research needs. In addition, our research focuses on network pharmacology analysis and animal experiments. Although clinical trials have not been conducted, this study have laid the foundation and ideas for clinical research. In the future, we will continue to conduct clinical research to verify the clinical efficacy and potential mechanism of YQBS in treating diabetic nephropathy with cognitive dysfunction.

Secondly, due to the limitation of the experimental period, this study was unable to observe the long-term therapeutic effects of YQBS on diabetic nephropathy with cognitive dysfunction. But our experimental results showed that YQBS can improve blood glucose and urinary microalbumin in rats, which basically reflect the efficacy and safety of YQBS. Besides, previous study has showed that YQBS could ameliorate diabetes symptoms and complications in DM patients,⁹ which is mentioned in our article. In the experiment, we used sufficient amount of experimental animals, and adopted random group allocation and setted up control groups to reduce the influence of individual differences on the results. In addition, we tried to ensure consistency in external factors such as the environment to reduce research errors caused by non-experimental factors. Furthermore, we will design more comprehensive experiments in the future to verify the results of molecular docking. We agree that diabetic nephropathy and cognitive dysfunction involve multiple inflammatory factors. However, the network pharmacology results of this study indicated that TNF- α and IL-6 are the key factors in YQBS treatment of diabetic nephropathy with cognitive dysfunction, and these two important targets are included in the important pathway. Therefore, this study mainly explored these two targets and related pathways. At the same time, we also acknowledge that due to our limitations, some relevant inflammatory factors may have been overlooked. We will explore more relevant inflammatory factors and potential mechanisms in future researches.

Regarding question 3, we did not investigate the simultaneous use of multiple drugs in our experiment. In future studies, we will further improve the research design to explore the interactions between YQBS and other drugs, as well as the role and dosage of YQBS in clinical trials.

Finally, thank you very much for the suggestions and additions. We are willing to consider these reminders in our future research.

Disclosure

The authors report no conflicts of interest in this communication.

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