LETTER

Suggestions for Enhancing the Spatiotemporal Characterization of Retinal Degeneration in the MNU-Induced RP Model [Letter]

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Dear editor

I recently read with great interest the article by Yan et al¹ titled "A Novelly-Spatiotemporal Characterization of the Disease Course in the MNU-Induced Retinitis Pigmentosa Model". This study offers valuable insights into retinal degeneration in the MNU-induced RP rat model, with a strong focus on spatiotemporal disease progression and microglial activation. The authors' use of advanced imaging techniques is commendable.

However, I would like to suggest a few areas for further consideration.

First, while the MNU-induced RP model is widely used, it does not fully replicate the genetic heterogeneity of human RP, which involves various genetic mutations.² The authors could benefit from discussing the model's limitations in representing the genetic diversity of RP. Incorporating insights from genetic RP models could enhance the clinical relevance of their findings.

Second, while the study highlights microglial activation, it would be valuable to explore the underlying molecular mechanisms in more detail. Investigating key inflammatory pathways (eg, NF-kB, JAK/STAT) and cytokine profiles could clarify the role of microglia in retinal degeneration.³ Functional experiments, such as microglial inhibition, could further elucidate their contributions to disease progression.

Lastly, the study focuses on short-term time points (1, 3, and 7 days), but RP is a progressive disease. Longer-term follow-up (eg, 14, 28 days) would provide a more complete picture of retinal degeneration over time. Additionally, examining dose-dependent effects of MNU would offer insights into the chronic nature of RP.

In conclusion, this study makes an important contribution to RP research, especially in terms of imaging and microglial activation. Addressing the points mentioned would strengthen the study's impact and clinical applicability.

Disclosure

The authors report no conflicts of interest in this communication.

References

1. Yan W, He Q, Long P, Zhang L, Wang H, Chen T. A novelly-spatiotemporal characterization of the disease course in the MNU-induced retinitis pigmentosa model. *J Inflamm Res.* 2024;17:9243–9254. doi:10.2147/JIR.S474102

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Shi E, Wang X, Jing H, et al. Synergistic effect of chitosan and beta-carotene in inhibiting MNU-induced retinitis pigmentosa. Int J Biol Macromol. 2024;268(Pt 2):131671. doi:10.1016/j.ijbiomac.2024.131671

Park SY, Bae YS, Ko MJ, Lee SJ, Choi YW. Comparison of anti-inflammatory potential of four different dibenzocyclooctadiene lignans in microglia; action via activation of PKA and Nrf-2 signaling and inhibition of MAPK/STAT/NF-kappaB pathways. *Mol Nutr Food Res.* 2014;58(4):738–748. doi:10.1002/mnfr.201300445

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