

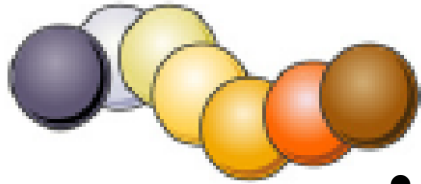
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Alamandine inhibits pathological retinal neovascularization by targeting the MrgD-mediated HIF-1 α /VEGF pathway

Key words: Alamandine; Pathological neovascularization; Retinopathy of prematurity (ROP); Oxygen-induced retinopathy (OIR); Mas-related G protein-coupled receptor D (MrgD); Vascular endothelial growth factor (VEGF)

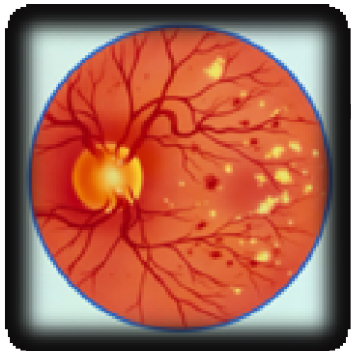
Research Summary

This article mainly focused on the key role of Alamandine in improving oxygen-induced retinopathy (OIR)-induced retinal neovascularization and the underlying molecular mechanisms:



Alamandine

- Alamandine attenuated hypoxia-induced pathological angiogenesis in vitro;
- Alamandine improved hypoxia-induced retinal vascular barrier dysfunction with restored glial cells network and alleviated inflammation response.



Alamandine inhibited retinal pathological neovascularization by inhibiting the HIF-1 α /VEGF pathway via MrgD ;

Retinal neovascularization

Innovation points

A series of comprehensive figures were generated to exhibit the protective effects of Alamandine on mitigating oxygen-induced retinopathy (OIR)-induced retinal neovascularization.

Figure 1-2 | Analysis of singlecell transcriptomic data in human and mice.

Figure 3 | LC–MS-mediated metabolomics analysis of OIR mouse model.

Figure 4 | Effects of Alamandine on VEGF-induced angiogenesis in HRMECs.

Figure 5 | Effects of Alamandine on hypoxia-induced HIF-1 α /VEGF pathway activation and apoptosis in HRMECs.

Figure 6 | Effects of Alamandine on pathological neovascularization in the retinas of OIR mouse.

Figure 7 | Effects of Alamandine on glial cells dysfunction and inflammation in the retinas of OIR mouse.

Figure 8 | Role of MrgD in the protective effects of Alamandine against VEGF-induced angiogenesis in vivo and in vitro.

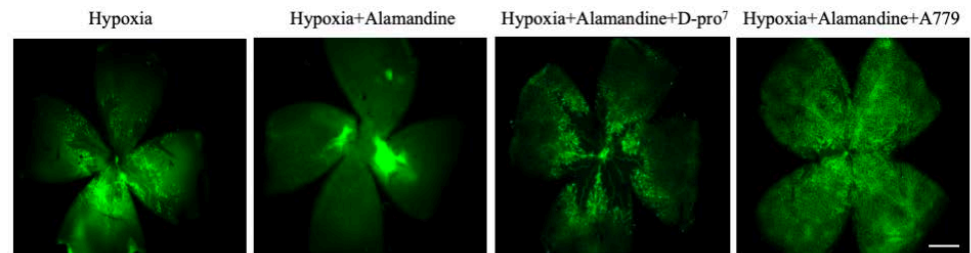


Figure 8