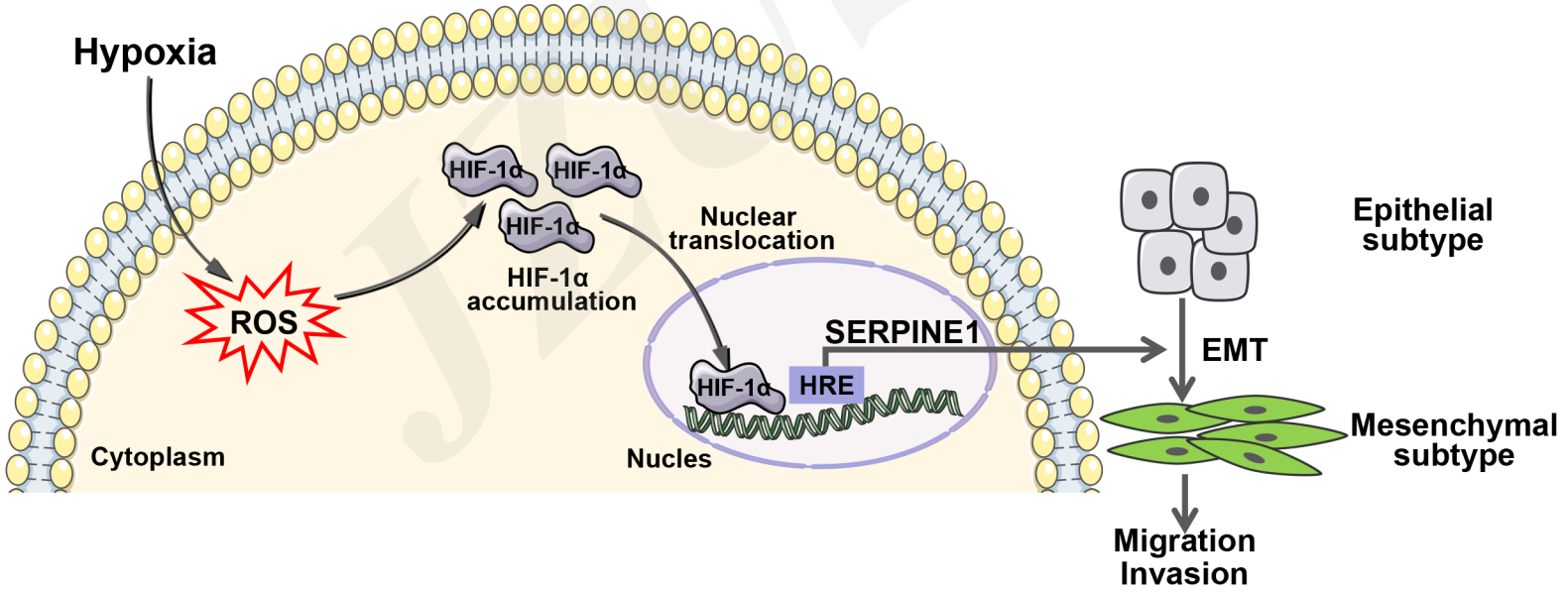
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Hypoxia-induced ROS aggravate tumor progression through HIF-1α-SERPINE1 signaling in glioblastoma

Key words: Glioblastoma, Hypoxia, ROS, HIF-1α, SERPINE1

Research Summary

Hypoxia promotes the generation of reactive oxygen species (ROS) in glioblastoma (GBM) cells, and elevated ROS contribute to HIF-1α accumulation and nuclear translocation. HIF-1α upregulates SERPINE1 by binding to the hypoxia response element (HRE) of SERPINE1 promoter, thereby promoting GBM migration and invasion through epithelial-mesenchymal transition (EMT). ROS may be potential therapeutic targets of GBM.



Innovation points

This study identified a feasible mechanism by which hypoxia contributes to GBM cell migration and invasion, and unraveled the vital roles of ROS-HIF-1α-SERPINE1 pathway in GBM adaptation to hypoxic stress. The main findings are as follows:

- **Hypoxia-induced ROS enhance GBM progression**
- **ROS accelerates hypoxia-promoted cell migration and invasion through activating HIF-1α in GBM**
- **HIF-1α up-regulates SERPINE1 transcription in hypoxia**
- **ROS-HIF-1α-SERPINE1 axis aggravates hypoxia-induced GBM progression**

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