From treatment survival to durable control.

Adult glioblastoma, IDH-wildtype. A public-level research framework connecting residual-cell biology, regrowth, exposure and neural function.

THE SCIENTIFIC QUESTION

Which surviving cells matter?

After a defined treatment background, which malignant populations cause or support later regrowth - and can a useful intervention change that contribution without unacceptable neural injury?

Published starting points

Cell-state diversity and plasticity make a single universal survivor population an assumption to test (Neftel et al., 2019). GPX4-related vulnerability has been shown in specific experimental glioma states (Banu et al., 2024).

Separate senolytic work motivates examining supporting-cell contributions (Tomimatsu et al., 2025). These external results arise in different models; their combination has not been validated.

The unresolved bridge

Treatment survival and marker expression do not establish causal regrowth control. A fair longitudinal comparison must separate direct founders, supporting cells and transient states.

EXTERNAL PUBLISHED MICROSCOPY

A state has a context.

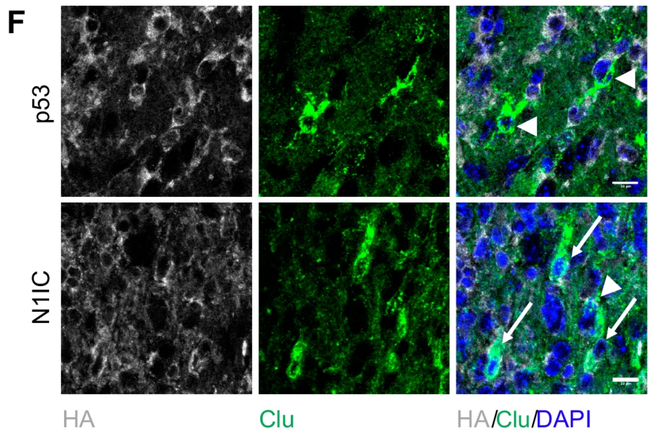


Figure 1 | Banu et al. (2024), Figure 1F. Representative immunofluorescence in engineered murine glioma models; external research, not a NEUVIOR experiment or human tissue image. Original Clu, HA and DAPI channels, labels and both 10 µm scale bars are retained.

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PROPOSED EVALUATION

Make the next experiment decisive.

Compare a defined treatment background with justified adjunct conditions and a combined approach only if the added step earns its complexity. Qualify the model and intended state before interpreting a negative result.

Follow malignant-cell identity and regrowth after withdrawal. Assess target mechanism using independent approaches, alongside measured exposure and neural function and recovery. Donors are biological units; repeated wells are nested observations.

What changes the decision?

Advance: reproducible, useful incremental control with an acceptable functional response. Revise: weak model retention, failed controls or imprecise estimates. Stop the tested route: no worthwhile added effect or persistent neural impairment at the required exposure.

Neuronal Gpx4-loss findings motivate delayed functional assessment; genetic deletion is not a candidate-specific drug-safety estimate (Hambright et al., 2017). Numerical thresholds, model access and study costs require specialist agreement.

Figure 2 | Proposed evidence relationships

Original NEUVIOR concept diagram; no measured values or validated surrogate.

Identity — Which population is present?

Contribution — Does it cause or support regrowth?

Durability — What remains after withdrawal?

Exposure + function — Is the effect compatible with recovery?

A common comparison links the questions; failure at one link can redirect the programme. Exposure, model validity and neural response must be interpreted together.

DOCUMENTED PROGRESS

Evidence synthesis, competing explanations and a decision-study framework are documented. The reviewed 8 October programme record reports no original NEUVIOR experimental dataset; the proposed comparison has not run. Working manuscript: not submitted or peer reviewed.

NEXT COLLABORATION

Start with a non-confidential capability enquiry. Agree the question, expertise, access and rights. A confidential briefing can follow by agreement where an NDA is appropriate. Public scientific context remains available without one.

GLIO | Sources and disclosure

Companion reference sheet to the research proposal poster. Prepared 13 October 2026. This is a proposed programme and selected-source synthesis, not original experimental research, a completed systematic review, clinical guidance or a journal publication.

Banu, M.A., Dovas, A., Argenziano, M.G. et al. (2024) A cell state-specific metabolic vulnerability to GPX4-dependent ferroptosis in glioblastoma. The EMBO Journal, 43, pp. 4492-4521.
https://doi.org/10.1038/s44318-024-00176-4

Tomimatsu, N. et al. (2025) Targeting cIAP2 in a novel senolytic strategy prevents glioblastoma recurrence after radiotherapy. EMBO Molecular Medicine, 17, article 6.
https://doi.org/10.1038/s44321-025-00201-x

Neftel, C. et al. (2019) An integrative model of cellular states, plasticity, and genetics for glioblastoma. Cell, 178(4), pp. 835-849.e21.
https://doi.org/10.1016/j.cell.2019.06.024

Hambright, W.S. et al. (2017) Ablation of ferroptosis regulator glutathione peroxidase 4 in forebrain neurons promotes cognitive impairment and neurodegeneration. Redox Biology, 12, pp. 8-17.
https://doi.org/10.1016/j.redox.2017.01.021

Figure attribution and integrity

Figure 1 reproduces the complete Figure 1F panel from Banu et al. (2024), © The Author(s), licensed under CC BY 4.0. Changes: panel F cropped from the complete figure and proportionally scaled; no recolouring, retouching, signal alteration or removal of panel labels or scale bars. The embedded image retains the decoded RGB pixels of the verified source crop. Figure 2 is an original conceptual evidence framework, not experimental data.

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Programme status is based on the accessible NEUVIOR research readiness and manuscript records dated 8 October 2026, read on 13 October. Scientific authorship, affiliations, contributions, conflicts and accountable review require confirmation before release. No third-party researcher or institution is represented as a NEUVIOR partner. Confidential materials, candidate identities, enabling experimental detail and private correspondence are excluded.

NEUVIOR | Research collaboration

https://www.neuvior.com/contact?interest=glioblastoma-research