

Passage Attribution for Spatial Cancer Reviews

Abstract

In immune-state and tumor evidence synthesis, a convincing output can still be invalid when source relevance confused with proof of a causal interaction. A bounded review of the literature is used to formulate, rather than claim to complete, an evaluation. The starting source uses a conceptual review proposing immunometabolic set points across immune and tissue compartments. Attribution asks what evidence influenced a statement and whether that evidence supports it. Those are different tests. Remove and replace passages while monitoring individual claims. Pair influence analysis with qualified support judgments, retaining failures in either dimension. The proposed comparison concerns glial niches, supporting sources, generated claims; it would test whether the specific failure scenario is detected without erasing legitimate uncertainty. An integrated account should preserve the distinction between tissue contexts, clinical endpoints, and mechanistic claims and any stronger downstream decision.

The interface under examination

Glial niches and generated claims occupy different positions in the evidence chain. Their relationship is the starting point for examining attribution fidelity. Consider the possibility of source relevance confused with proof of a causal interaction. This is a proposed stress scenario, not an observed event reported by the cited studies. Its purpose is to expose which evidence contract would be violated even if the final output looked plausible. For tissue contexts, clinical endpoints, and mechanistic claims, the relevant claim must identify its population or artifact class, its time window, and the action it is supposed to support. A failure at any of these levels may persist even after a local technical improvement. The analysis therefore treats published methods as bounded evidence and asks how their limitations interact with this particular decision. An operational analogy is useful only if it keeps those limitations visible.

Attribution asks what evidence influenced a statement and whether that evidence supports it. Those are different tests. A model may faithfully rely on an irrelevant passage, while a correct statement may receive a decorative citation unrelated to its generation. In this article, the relevant contrast is glial niches, supporting sources, generated claims. The proposed counterexample of source relevance confused with proof of a causal interaction puts that contrast under pressure. Remove and replace passages while monitoring individual claims. Pair influence analysis with qualified support judgments, retaining failures in either dimension. A favorable finding would show that the intended interpretation of generated claims remains defensible when glial niches is perturbed under the specified conditions. An unfavorable finding should narrow the claim rather than be discarded as an inconvenient case.

The strongest comparison would isolate the disputed transition in glial niches, supporting sources, generated claims. The proposed counterexample, source relevance confused with proof of a causal interaction, identifies what should not be accepted as equivalent evidence. The input record should retain enough information to decide which part of that scenario violates the intended contract. That includes the original source type, measurement or artifact status, and the reason the proposed interpretation exceeds its support. The evaluator would compare the system's response with a qualified judgment made without seeing that response. Disagreement should be classified rather than erased by rewriting the prompt. A model may faithfully rely on an irrelevant passage, while a correct statement may receive a decorative citation unrelated to its generation. A useful negative result would show that the interface cannot preserve this distinction under realistic use; a useful positive result would demonstrate preservation on independent cases rather than merely

on the example used to develop the rule. The primary source remains relevant through its particular contribution: The set-point framing asks whether durable remission requires changing a coupled metabolic state rather than only lowering an inflammatory marker. That contribution should be retained even if the integrated workflow fails. Conversely, successful integration would not remove the source's limitations or turn a proposed extension into an already observed result. This asymmetric interpretation protects both the literature and the user from an unjustified all-or-nothing verdict.

Checks for the proposed workflow

FAIR principles support reusable cohorts, signals, and analysis metadata (Wilkinson et al. 2016). Reusable evidence needs more than a downloadable file. Identifiers, metadata, provenance, and meaningful access conditions help another researcher determine what an artifact contains and whether it can answer a new question. Reuse does not mean universal permission or universal scientific comparability. The information needed to judge those boundaries should travel with the artifact rather than remain in private workflow knowledge. Within immune-state and tumor evidence synthesis, this distinction is relevant to attribution fidelity. The proposed failure scenario is source relevance confused with proof of a causal interaction; its evaluation must preserve the original source's methodological scope.

Causal inference requires a clearly specified intervention, time order, and exchangeability assumptions (Hernán and Robins 2020). A prediction can be accurate without identifying what an intervention would change. A useful causal analysis begins by defining the hypothetical comparison, eligibility, and time zero. Its assumptions concern the process generating exposure and observation, not merely the flexibility of a fitted model. In this review, causal vocabulary is appropriate only when that comparison can be specified and defended. The connection to passage attribution for spatial cancer reviews is a constraint on interpretation. It would be inappropriate to borrow the source's methodological authority as direct confirmation of the integrated domain claim.

Epidemiologic design makes confounding, selection, and measurement error part of interpretation (Rothman, Greenland, and Lash 2008). Confounding, selection, and measurement error are distinct problems and need not move an estimate in the same direction. An analysis should describe them before deciding which adjustment is adequate. A larger dataset can reduce random error while leaving systematic error almost untouched. Precision should therefore be interpreted alongside the quality of the design and the eligibility of its population. This is methodological support for examining attribution fidelity, not a claim that the source studied immune-state and tumor evidence synthesis. A separate application-specific test is required at the interface described in this review.

False-discovery control is central when many molecular features are screened (Benjamini and Hochberg 1995). Multiplicity arises from the family of questions actually searched. Controlling false discoveries within a named family does not automatically cover later tissue filters, alternative endpoints, or narrative choices. The proposed audit should retain exploratory branches so the family is visible. Independent confirmation then addresses a different question from whether a discovery threshold was correctly applied during screening. For the comparison involving glial niches, supporting sources, generated claims, the source supplies an analytical obligation. The review's proposed application remains a hypothesis, with the deliberately difficult scenario serving as a check on scope inflation.

Medical machine learning must fit data provenance, care pathways, and prospective evaluation (Rajkomar, Dean, and Kohane 2019). The clinical relevance of a model depends on how its output changes a care process. Development data can contain information that becomes available only after the intended decision. A review must identify that availability boundary before comparing algorithms. Prospective use

also changes who is measured and who is treated, so evaluation of a frozen historical dataset cannot establish the entire future workflow. This is methodological support for examining attribution fidelity, not a claim that the source studied immune-state and tumor evidence synthesis. A separate application-specific test is required at the interface described in this review.

Responsible health AI connects prediction with workflow, monitoring, and patient impact (Wiens et al. 2019). The point at which a model reaches practice is not the end of evaluation. New populations, changing measurements, and feedback can alter failure modes. A responsible deployment needs a route for recognizing deterioration and a person authorized to respond. Monitoring should be linked to feasible corrective actions; an alert that cannot change the workflow provides accountability only in a very limited sense. The connection to passage attribution for spatial cancer reviews is a constraint on interpretation. It would be inappropriate to borrow the source's methodological authority as direct confirmation of the integrated domain claim.

Calibration separates discrimination from confidence used in clinical escalation (Guo et al. 2017). Discrimination and calibration deserve separate evaluation. A score can order cases well yet systematically overstate its certainty. Aggregate calibration can also hide local miscalibration near the threshold where action changes. An audit should retain predicted probabilities and observed outcomes rather than report only correct classifications, and it should evaluate calibration on data not reused to choose the correction. This is methodological support for examining attribution fidelity, not a claim that the source studied immune-state and tumor evidence synthesis. A separate application-specific test is required at the interface described in this review.

Uncertainty can deteriorate under the site and cohort shifts common in biomedicine (Ovadia et al. 2019). Uncertainty is most valuable when a system moves beyond familiar conditions, yet those conditions can also weaken its uncertainty estimates. A warning mechanism must therefore be evaluated under shift rather than assumed to generalize because it works on ordinary held-out data. Different shifts can affect ranking, calibration, and error concentration differently. No single in-distribution curve demonstrates protection against them all. For the comparison involving glial niches, supporting sources, generated claims, the source supplies an analytical obligation. The review's proposed application remains a hypothesis, with the deliberately difficult scenario serving as a check on scope inflation.

Selective prediction formalizes abstention when evidence does not support action (Geifman and El-Yaniv 2017). Abstention creates a tradeoff between coverage and residual error. Comparing systems without matching coverage can make one look safer merely because it leaves more work unfinished. Deferred cases also consume human or computational resources. The evaluation should show the risk-coverage relation and account for the fate of excluded cases, rather than treating them as though they have ceased to matter. For the comparison involving glial niches, supporting sources, generated claims, the source supplies an analytical obligation. The review's proposed application remains a hypothesis, with the deliberately difficult scenario serving as a check on scope inflation.

Feature attribution can guide audit but does not establish biological causality (Lundberg and Lee 2017). Attribution decomposes a model output according to a defined explanatory convention. That decomposition can reveal dependence on suspicious features without establishing why the outcome occurs in the world. Reference choices and feature dependence can affect interpretation. A useful audit should compare explanations with known model behavior and should not transform a feature contribution into a treatment or mechanism claim. This is methodological support for examining attribution fidelity, not a claim that the source studied immune-state and tumor evidence synthesis. A separate application-specific test is required at the interface described in this review.

Evidence without scope inflation

Resetting Immunometabolic Set Points in Autoimmune Disease asks why autoimmune activity can persist or recur when conventional inflammation suppression does not durably change underlying cellular and tissue states (Xiao Yu et al. 2026e). Its methodological basis is a conceptual review proposing immunometabolic set points across immune and tissue compartments. The accessible evidence is specific: the review integrates cellular metabolism, nutrient/oxygen conditions, mitochondrial stress, and stromal metabolites as candidate mechanisms of persistence. The set-point framing asks whether durable remission requires changing a coupled metabolic state rather than only lowering an inflammatory marker. A unifying set point is a hypothesis, not an already validated clinical measurement or an established therapeutic protocol. For attribution fidelity, the important connection is the distinction between glial niches and generated claims. The source informs that distinction, but a stronger conclusion would require evidence at the point where one is used to stand for the other.

A useful boundary case is supplied by Machine Learning-Based Prediction of Early Visual Field Nonrecovery after Endoscopic Surgery for Nonfunctioning Pituitary Adenomas with Clinically Accessible Biomarkers: A Dual-Center Study, concerned with whether accessible preoperative biomarkers can predict early visual-field nonrecovery after surgery for nonfunctioning pituitary adenomas (Sunjiang Liu et al. 2026). Its methodological basis is a retrospective dual-center design with LASSO selection, multivariable logistic modeling, and a nomogram; training comprised 268 patients and validation 100. The accessible evidence is specific: the study includes 368 patients, defines early improvement at postoperative day seven, and reports age, vertical tumor diameter, and fasting glucose as predictors; reported C-indices are approximately 0.747 and 0.753. It develops an accessible early-risk tool with geographically separate validation instead of relying solely on model fit in development data. Day-seven nonrecovery is not permanent visual loss; retrospective selection, limited validation events, and unmeasured care differences constrain interpretation. For attribution fidelity, the important connection is the distinction between glial niches and generated claims. The source informs that distinction, but a stronger conclusion would require evidence at the point where one is used to stand for the other.

A useful boundary case is supplied by Immune Niches in Brain Tumors: Glial-Immune Cell Interactions and Spatial Microdomains, concerned with how spatial glial-immune interactions organize permissive and suppressive microdomains within brain tumors (Qian Guo et al. 2026). Its methodological basis is a review of astrocytes, microglia, oligodendrocyte-lineage cells, T cells, and myeloid cells across spatial tumor niches. The accessible evidence is specific: the abstract integrates cytokine, metabolic, hypoxic, and immunoediting mechanisms associated with immune suppression and exhaustion. It shifts attention from tumor-wide averages to interactions whose meaning depends on location and cell context. The review does not establish efficacy of a new intervention; co-location, state annotation, and functional interaction require different evidence. Reading the work through attribution fidelity changes the evaluation question: the issue is whether glial niches supports the intended interpretation of generated claims, rather than whether the reported method is impressive in its original task.

Splenic Extramedullary Granulopoiesis and Neutrophil States in Cancer Immunity: Systemic Immunosuppression and Therapeutic Resistance asks how splenic extramedullary granulopoiesis and neutrophil heterogeneity contribute to systemic cancer immunosuppression and resistance (Yidan Zhang et al. 2026). Its methodological basis is a review organized around stress-associated splenic blood-cell production and reprogrammed neutrophil states. The accessible evidence is specific: the official Cancer Bioscience recent-articles listing confirms the review's scope and emphasizes sustained stress and systemic reprogramming; no new patient cohort is inferred. The framing connects extra-tumoral immune production

with local cancer resistance questions. The accessible summary cannot establish specific quantitative contributions, universal cell-state functions, or clinical effects of proposed targeting strategies. The contribution becomes relevant to attribution fidelity when the workflow moves from glial niches to generated claims. That transition is the review's proposed analytical focus, not a new result attributed to the source.

A useful boundary case is supplied by *The Prediction of Extended Hospital Length of Stay in Patients after Endoscopic Endonasal Transsphenoidal Surgery for the Resection of Non-Functioning Pituitary Adenomas: A Dual-Center Retrospective Analysis*, concerned with which retrospective clinical features predict extended hospital stay after endoscopic endonasal surgery for nonfunctioning pituitary adenomas (Bibo Gao et al. 2026). Its methodological basis is a dual-center retrospective prediction analysis with a modeled extended-stay outcome and separate validation. The accessible evidence is specific: the accessible record describes a nomogram and a 100-patient validation sample; specific predictor effects and thresholds are not assumed. It treats hospitalization burden as an explicitly predicted outcome that can inform care-process evaluation. Length of stay includes organizational and discharge factors; it is not an interchangeable measure of tumor biology, surgical quality, or long-term recovery. The source supplies one part of the comparison involving glial niches, supporting sources, generated claims. Its evidence should retain its original unit and scope; the present synthesis proposes a connection without claiming that the paper directly tested this integrated workflow.

The relevant question in *AutoCrit: A Meta-Reasoning Framework for Self-Critique and Iterative Error Correction in LLM Chains-of-Thought* is whether structured self-critique can interrupt error propagation in language-model reasoning chains (Yinghao. “AutoCrit: A Meta-Reasoning Framework for Self-Critique Sang 2025). Its methodological basis is a reasoning agent, critique agent, and execution monitor provide iterative stepwise feedback and correction. The accessible evidence is specific: the abstract describes GSM8K, CSQA2, and ALFWorld evaluations, reported improvements over chain-of-thought baselines, and a fixed-point analysis of the correction process. It organizes critique as a monitored reasoning process rather than a single request to reconsider an answer. Shared model biases can correlate critic and reasoner errors, while extra iterations increase cost; benchmark gains do not establish clinical factuality. This source enters the present review as a methodological comparison for evidence processing, not as evidence about disease biology, patient outcomes, or clinical efficacy. The transferable question is whether glial niches remains correctly interpreted after a computational choice involving generated claims. Its task-specific validation cannot be borrowed to validate the clinical claim.

The sources should be compared through the roles they play, not merged into a single performance claim. *Resetting Immunometabolic Set Points in Autoimmune Disease* addresses why autoimmune activity can persist or recur when conventional inflammation suppression does not durably change underlying cellular and tissue states (Xiao Yu et al. 2026e). By contrast, *AutoCrit: A Meta-Reasoning Framework for Self-Critique and Iterative Error Correction in LLM Chains-of-Thought* addresses whether structured self-critique can interrupt error propagation in language-model reasoning chains (Yinghao. “AutoCrit: A Meta-Reasoning Framework for Self-Critique Sang 2025). That difference matters for attribution fidelity: the evidence concerning glial niches is not automatically evidence concerning generated claims. A successful software operation, a plausible mechanistic account, and a validated outcome are different achievements. Their integration may improve traceability, but it cannot create an unmeasured link. A coherent review should identify where the sources complement one another and where their scopes remain incompatible. Here the proposed failure scenario is source relevance confused with proof of a causal interaction. The analytical value of that scenario is that it can occur without requiring any cited paper to be wrong: an otherwise valid local finding can be misused at a later interface. Preventing that misuse requires evidence about the interface itself, not simply a larger collection of local results.

Research priorities and negative results

Remove and replace passages while monitoring individual claims. Pair influence analysis with qualified support judgments, retaining failures in either dimension. This would operationalize the concern with attribution fidelity rather than assume it has been resolved by one successful output. These procedures are proposed, not completed. The case set would include the scenario of source relevance confused with proof of a causal interaction, with its validity judged independently of the system's response. The relevant comparison axes are glial niches, supporting sources, generated claims.

An evidence-linking protocol would use a set of individually judged claims, eligible supporting passages, plausible but ineligible passages, and genuine counterevidence. The evaluation would retain rankings and intermediate interpretations. Source deletion and substitution would test whether support changes coherently. Fluency, citation presence, source influence, and actual support would be recorded separately, so an answer cannot receive full credit simply for sounding well documented.

For the immune review, proposed mechanisms should be tested in their stated compartments and disease settings. Spatial association, state annotation, and intervention response would be judged separately. A useful result could expose a missing bridge in the integrated account. It would not need to confirm every part of the preferred narrative to improve scientific understanding.

Interpretive boundaries

The cited sources still have design-specific limits even where their abstracts and methods are accessible. This review is a bounded conceptual synthesis rather than a systematic review with an exhaustive search protocol. It cannot establish the effectiveness of the proposed integrated workflow. Its specific contribution is to articulate how attribution fidelity changes the interpretation of glial niches, supporting sources, generated claims. Technical sources support computational comparisons only; they do not validate biological mechanisms or patient benefit. Reviews and correspondence retain their publication roles and are not counted as new experiments. Proposed stress cases are illustrative, not observed failures attributed to the papers. Future work should therefore test the weakest inference described here, preserve contrary results, and disclose the cost of checking rather than treating additional automation as intrinsically beneficial.

Conclusion

The counterexample of source relevance confused with proof of a causal interaction shows why local success and integrated validity must be evaluated separately. Remove and replace passages while monitoring individual claims. Pair influence analysis with qualified support judgments, retaining failures in either dimension. The remaining problem can be stated as a testable contrast involving glial niches, supporting sources, generated claims, with source relevance confused with proof of a causal interaction as a deliberately challenging alternative. The cited studies provide useful but different forms of support. Their integration should increase traceability and expose missing links, leaving clinical, mechanistic, and operational claims at the strength justified by their own evidence.

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