AI-ASSISTED MEDICAL WRITING

Human vs. AI Medical Writing in Parkinson's Disease

A Physician-Led Quality and Safety Audit of an AI-Generated Evidence Review

Henrique C. Galvane, MD

Independent Portfolio Project

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DISCLAIMER

Independent portfolio project. The source article was generated by an LLM for evaluation. This document is educational and demonstrates medical-writing and editorial quality-assurance methods; it is not clinical guidance.

Executive Summary

Background

Generative AI can produce fluent medical prose, but fluency is not equivalent to evidence fidelity. This project tested whether an LLM could independently produce a current, citation-ready HCP review of disease-modifying therapy in early Parkinson's disease.

Objective

To identify factual, citation, currency, nuance, and safety problems in a frozen AI draft and demonstrate the editorial value added by physician-led source verification.

Methods

The five-page AI article was preserved without editing and decomposed into 40 high-impact, independently verifiable claim clusters. Claims were checked against primary publications, trial registries, peer-reviewed 2026 updates, and sponsor sources when peer-reviewed data were unavailable. Each claim received an evidence-status classification and editorial-severity assessment.

Key Findings

40	29	11	5
CLAIM CLUSTERS AUDITED	FULLY SUPPORTED	REQUIRED SUBSTANTIVE EDIT	CURRENCY/SOURCE FAILURES

Conclusion. The draft was directionally strong on historical trial evidence, but materially weaker on evidence currency, source hierarchy, and calibration of conclusions. Several of the most consequential problems occurred in the newest evidence - exactly where an apparently fluent review can be most misleading.

1. Audit Protocol & Methodology

Predefined framework used before judging the final draft

Source Document

- Title: *Current Evidence for Disease-Modifying Therapy in Early Parkinson's Disease*
- Intended audience: neurologists and healthcare professionals
- Declared evidence currency: September 2026
- Source length: 5 pages; 30-item reference list plus a verification note
- The source explicitly flagged several citations and current-trial statements for later verification; those flags were retained as audit findings rather than silently repaired.

Audit Unit

Claims were grouped into 40 high-impact clusters to avoid double-counting repeated or closely linked assertions. Each cluster contained one main scientific proposition that could reasonably be verified against an external source.

Evidence Status

SUPPORTED: materially accurate and appropriately represented. **PARTIALLY SUPPORTED:** direction correct, but qualification, population, endpoint, source hierarchy, or interpretation requires correction. **OVERSTATED:** wording exceeds what evidence establishes. **OUTDATED:** newer evidence materially changes representation. **UNSUPPORTED:** claim could not be supported. **UNVERIFIABLE:** insufficient information to confirm.

Editorial Domains

- Factual accuracy
- Citation validity and source hierarchy
- Evidence fidelity
- Clinical nuance and calibration
- Currency
- Hallucination / unsupported detail
- Potential patient-safety impact
- Scientific style and publication readiness

> **IMPORTANT DESIGN CHOICE**
>
> The model was not rewarded for "being cautious" if it delegated verification to the reader. Phrases such as "as I recall," "as I understand from sponsor reports," or "readers should consult its results" were treated as source-verification failures when definitive sources were already available.

2. Results

Audit Distribution

Classification	n / 40	Interpretation
Supported	29	Accurate and appropriately represented
Partially supported	6	Core direction correct; qualification or evidence fidelity needed
Overstated	1	Conclusion stronger than evidence supports
Outdated	1	Current evidence/status not incorporated
Unsupported	2	Claim not supported by located evidence
Unverifiable	1	Could not be substantiated from available primary/registry evidence

BOTTOM LINE

The AI draft was useful as a first-pass scaffold, not as a publication-ready medical review. Physician-led verification materially changed the interpretation of several late-stage programs and corrected source-selection failures that a surface-level edit would have missed.

3. High-Impact Findings

FINDING 01 - CURRENCY FAILURE: EXENATIDE-PD3

RAW AI CLAIM

The draft accurately summarized the reported 2025 phase 3 result and then treated the publication as settled evidence.

Audit result. By June 2026, The Lancet had issued an Expression of Concern for the Exenatide-PD3 article. A review explicitly claiming evidence current to September 2026 should have surfaced that notice. The numerical result can still be reported, but its evidentiary status requires qualification.

Classification: Partially supported / current-evidence omission. **Severity:** Major.

Editorial correction: Retain the published efficacy results, add the Expression of Concern, and avoid presenting the trial as fully settled without acknowledging the journal notice.

3. High-Impact Findings (continued)

FINDING 02 - SOURCE-VERIFICATION FAILURE: PADOVA

> **RAW AI WORDING**
> "As I recall, its primary endpoint of time to confirmed motor progression did not reach significance."

Audit result. The peer-reviewed PADOVA paper was already available in The Lancet in May 2026. The primary endpoint was not met: HR 0.84 (95% CI 0.69-1.01), p=0.066; median time to confirmed motor progression was 61.1 weeks with prasinezumab versus 49.7 weeks with placebo.

Classification: Direction supported; source handling unacceptable. **Severity:** Major.

Editorial correction: Replace memory-based language with verified numerical results and clearly separate the failed primary endpoint from exploratory signals.

FINDING 03 - NONEXISTENT RESULTS: SPARX3

> **RAW AI CLAIM**
> "A phase 3 successor (SPARX3) was designed to test this, and readers should consult its results."

Audit result. SPARX3 was an ongoing phase 3 exercise trial without posted definitive results in the audited evidence set. Directing the reader to "its results" falsely implies that results are available.

Classification: Unsupported / outdated status. **Severity:** Major.

Editorial correction: State that SPARX3 is ongoing and that phase 3 evidence is not yet available.

4. Evidence Fidelity & Calibration

FINDING 04 - MOVES-PD: PRIMARY PUBLICATION IGNORED

> **RAW AI WORDING**
> "Venglustat ... did not show benefit in GBA1-associated PD, as I understand from sponsor reports, which should be checked against the publication."

Audit result. MOVES-PD Part 2 had been peer-reviewed in The Lancet Neurology since 2023. In 221 participants, the between-group difference in MDS-UPDRS II+III change was 2.58 points (95% CI -1.10 to 6.27; p=0.17), with no beneficial treatment effect.

Classification: Supported conclusion / source hierarchy failure. **Severity:** Major.

Editorial correction: Use the peer-reviewed primary publication rather than a vague sponsor-based recollection.

FINDING 05 - PASADENA: IMAGING SIGNAL OVERCALLED

Audit result. The draft stated that PASADENA produced "signals in secondary motor measures and DaT-SPECT." The 2022 NEJM publication reported no substantial difference between prasinezumab and placebo in

dopamine-transporter SPECT, and most clinical secondary endpoints were similar. Exploratory motor analyses can be discussed, but the imaging wording should not imply a positive DaT-SPECT signal.

Classification: Partially supported. **Severity:** Moderate.

FINDING 06 - CLASS-LEVEL OVERSTATEMENT

> **RAW AI CONCLUSION**
> "Recent negative results for exenatide and BIIB122 rule out two mechanistically attractive strategies in unselected early PD."

Audit result. The statement is too broad. Exenatide-PD3 weakens the exenatide hypothesis but does not by itself invalidate the GLP-1 receptor agonist class, and the draft itself says so earlier. LUMA supports discontinuation of BIIB122 in idiopathic PD, while the genetically enriched BEACON program continues.

Classification: Overstated / internally inconsistent. **Severity:** Major.

Editorial correction: "These results weaken support for exenatide and for BIIB122 in unselected or idiopathic PD, but they do not establish failure of the broader GLP-1 or genetically targeted LRRK2 strategies."

FINDING 07 - CELL THERAPY CATEGORY ERROR

Audit result. The draft correctly noted that dopaminergic cell replacement is primarily restorative rather than a direct modifier of the underlying neurodegenerative process, but the conclusion then grouped cell-based approaches among the principal disease-modification prospects. Bemdaneprocel has entered randomized sham-controlled phase 3 testing; the Kyoto iPSC program cited in the same paragraph was a seven-patient phase I/II study, not the same development program.

Classification: Partially supported / conceptual inconsistency. **Severity:** Moderate.

5. Claim-Level Audit Matrix

Condensed view of all 40 audited claim clusters

Claim cluster	Status	Editorial note
No approved disease-modifying therapy for PD	Supported	Current guidelines/registries do not establish a neuroprotective therapy.
Three decades of trials were negative or uninterpretable	Partially supported	Directionally fair but compresses heterogeneous evidence into a sweeping statement.
Exenatide phase 3 was negative	Partially supported	Published result negative; June 2026 Expression of Concern omitted.
LUMA/BIIB122 was negative in early PD	Supported	Primary and secondary endpoints not met; idiopathic-PD program discontinued.
Anti-alpha-syn immunotherapy is the "main late-stage candidate"	Overstated	Editorial ranking is subjective; multiple late-stage strategies exist.
Prasinezumab has exploratory signals only	Supported	PADOVA missed primary endpoint; exploratory evidence supports phase 3.
Braak staging demonstrates spreading inclusions	Partially supported	Staging supports stereotyped distribution; direct propagation should be phrased as a hypothesis.
Motor signs emerge after substantial dopaminergic loss	Supported	Consistent with established PD pathophysiology.
SNCA genetics and SAA support alpha-syn biology	Supported	Appropriately framed as biological rationale.
GBA1 variants are the commonest genetic risk factor	Partially supported	Safer wording: among the most common/important genetic risk factors.
LRRK2 hyperactivity is a therapeutic target	Supported	Mechanistically supported; clinical efficacy remains unproven.
Mitochondrial/oxidative stress rationale	Supported	Historical disease-modification rationale accurately described.
GLP-1/incretin signaling is investigational rationale	Supported	Appropriate mechanistic framing.
Biological PD classifications have been proposed	Supported	SynNeurGe and NSD-ISS are research frameworks.
PPMI SAA showed high diagnostic performance	Supported	High overall performance, with important phenotype/genotype heterogeneity.
SAA frameworks are not routine clinical standards	Supported	Appropriate qualification.
Symptomatic confounding complicates DMT trials	Supported	Core trial-design issue.
MDS-UPDRS is a common progression endpoint	Supported	Appropriate.
PD heterogeneity can dilute subgroup effects	Supported	Plausible and widely accepted trial-design concern.
ELLDOPA showed discordant clinical/imaging signals	Supported	Accurate historical summary.

5. Claim-Level Audit Matrix (continued)

Claim cluster	Status	Editorial note
LEAP found no levodopa disease-modifying effect	Supported	Primary delayed-start conclusion supported.
PROUD did not support pramipexole disease modification	Supported	Supported by clinical and imaging outcomes.
DATATOP selegiline signal was symptomatically confounded	Supported	Appropriate historical interpretation.
ADAGIO 1 mg positive / 2 mg negative; no firm DMT claim	Supported	Accurate cautious interpretation.
CoQ10, creatine, pioglitazone, inosine, isradipine failed	Supported	Direction consistent with randomized trial evidence.
Deferiprone reduced iron but worsened clinical scores	Supported	Clinically important counterexample to biomarker target engagement.
SPARX phase 2 suggested possible benefit	Partially supported	Signal-generating only; not proof of slowing disease.
SPARX3 results are available to consult	Unsupported	Definitive results were not available in the audited evidence set.
LixiPark showed ~3-point placebo worsening and GI AEs	Supported	Consistent with 2024 NEJM report.
Exenatide-PD3 numerical summary	Supported	Numerical summary consistent with publication, separate from EoC issue.
Semaglutide is being studied in PD	Unsupported	No supporting PD trial identified in the searched evidence set.
Cinpanemab/SPARK was negative	Supported	No clinical or DaT-SPECT benefit versus placebo.
PASADENA missed primary endpoint	Supported	Correct.
PASADENA showed a DaT-SPECT signal	Partially supported	Primary publication reported no substantial imaging difference.
PADOVA primary endpoint was non-significant	Supported	HR 0.84; p=0.066; peer-reviewed by May 2026.
PARAISO phase 3 is recruiting ~900 participants	Supported	Roche materials/registry support ~900 global participants.
LUMA target engagement did not translate to benefit	Supported	Supported; idiopathic program discontinued.
MOVES-PD no benefit based on sponsor report	Outdated	Conclusion correct, but peer-reviewed 2023 publication should have been used.
ASPro-PD is an active phase 3 ambroxol program	Supported	Open to recruitment; 330 participants planned.
Cell replacement is disease-modifying	Unverifiable	Restorative efficacy and disease modification are conceptually distinct; long-term modification remains unproven.

6. Physician-Reviewed Article

Rewritten for accuracy, source hierarchy, calibration, and evidence currency

CURRENT EVIDENCE FOR DISEASE-MODIFYING THERAPY IN EARLY PARKINSON'S DISEASE

No pharmacological therapy has yet been established to slow the underlying progression of Parkinson's disease (PD) in routine clinical practice. Several symptomatic agents and investigational programs have produced signals compatible with disease modification, but delayed-start designs, biomarker discordance, small phase 2 studies, heterogeneous patient populations, and subsequent negative trials have repeatedly limited interpretation. As of September 2026, the field remains active, with major programs targeting alpha-synuclein, LRRK2, GBA1/lysosomal biology, incretin signaling, and cell replacement.

BIOLOGICAL RATIONALE AND TRIAL DESIGN

PD is characterized by progressive loss of nigrostriatal dopaminergic neurons and, in most sporadic cases, Lewy pathology rich in aggregated alpha-synuclein. Genetic and biomarker evidence has strengthened the biological rationale for targeting alpha-synuclein, lysosomal dysfunction, GBA1-related pathways, and LRRK2 kinase activity. Alpha-synuclein seed-amplification assays have shown high diagnostic performance in research cohorts, but assay performance varies by biological subtype and these frameworks remain primarily research tools rather than routine treatment-selection standards.

Demonstrating disease modification is difficult because symptomatic therapies can alter clinical scores, progression is slow and heterogeneous, and commonly used endpoints such as the MDS-UPDRS are influenced by medication state and measurement variability. Delayed-start and washout designs can reduce symptomatic confounding but do not eliminate all interpretive uncertainty. Biomarkers such as DaT-SPECT or biochemical target engagement may support mechanism but are not validated surrogates for clinically meaningful slowing of PD progression.

ESTABLISHED SYMPTOMATIC THERAPIES

Levodopa remains the most effective symptomatic therapy for motor symptoms but has not demonstrated a disease-modifying effect. ELLDOPA produced discordant clinical and imaging findings, while the later LEAP delayed-start trial found no significant difference supporting disease modification after 80 weeks. Pramipexole similarly failed to show a disease-modifying signal in PROUD. Selegiline delayed the need for levodopa in DATATOP, but a symptomatic effect could not be excluded. Rasagiline produced the best-known delayed-start signal in ADAGIO: the 1-mg dose met all prespecified delayed-start endpoints whereas the 2-mg dose did not, preventing a definitive disease-modification conclusion. These drugs should therefore be regarded as symptomatic treatments rather than proven neuroprotective therapies.

FAILED OR INCONCLUSIVE MECHANISTIC PROGRAMS

Multiple mechanistically attractive programs have failed to translate target engagement into clinical benefit. High-dose coenzyme Q10, creatine, pioglitazone, inosine and isradipine did not slow clinical progression in randomized trials. Deferiprone is a particularly important cautionary example: although nigral iron was reduced, participants receiving deferiprone had substantially worse clinical progression than those receiving placebo. These results illustrate why biomarker movement alone cannot establish disease modification.

GLP-1 RECEPTOR AGONISTS

GLP-1 receptor agonists remain an investigational class. A small phase 2 exenatide trial reported a persistent off-medication motor-score difference after treatment and washout, and LixiPark later found that motor scores remained relatively stable with lixisenatide while worsening by about three points with placebo over 12 months, although gastrointestinal adverse effects were frequent. The larger Exenatide-PD3 phase 3 trial did not demonstrate benefit on its primary or secondary endpoints. The Lancet subsequently issued an Expression of Concern for that publication in June 2026, which should be acknowledged when interpreting the result. The available evidence therefore does not justify prescribing GLP-1 receptor agonists specifically to modify PD progression outside research settings.

ALPHA-SYNUCLEIN IMMUNOTHERAPY

Cinpanemab failed to slow clinical progression or alter DaT-SPECT measures in the SPARK phase 2 trial. Prasinezumab has generated a more complex evidence pattern. PASADENA did not meet its primary MDS-UPDRS I-III endpoint and did not show a substantial difference in dopamine-transporter SPECT, although exploratory motor analyses suggested possible activity in faster-progressing subgroups. PADOVA enrolled 586 participants receiving stable symptomatic monotherapy and also did not meet its primary endpoint: the hazard ratio for confirmed motor progression was 0.84 (95% CI 0.69-1.01; p=0.066), with median time to progression of 61.1 weeks versus 49.7 weeks on placebo. Prespecified exploratory analyses nevertheless supported continued investigation. The phase 3 PARAISO study is now recruiting approximately 900 participants. Prasinezumab should therefore be described as promising but unproven, with phase 3 confirmation required.

LRRK2 AND GBA1 / LYSOSOMAL PROGRAMS

The LRRK2 inhibitor BIIB122 did not meet primary or secondary endpoints in the 648-participant LUMA phase 2b trial despite substantial peripheral target engagement. Development in idiopathic PD was discontinued, while the BEACON phase 2a program continues in carriers of pathogenic LRRK2 variants. These results weaken the case for BIIB122 in unselected PD but do not establish that genetically targeted LRRK2 inhibition is ineffective.

On the GBA1 axis, ambroxol has shown cerebrospinal-fluid penetration and biological target engagement in early studies. The phase 3 ASPro-PD trial is open to recruitment and plans to enroll 330 participants stratified by GBA1 status. In contrast, venglustat failed to provide clinical benefit in the peer-reviewed MOVES-PD Part 2 trial despite target engagement. The GBA1 pathway therefore remains biologically compelling, but clinical efficacy has not been established.

EXERCISE AND CELL REPLACEMENT

Exercise is recommended for symptomatic, functional and general health benefits, but a disease-modifying effect remains unproven. SPARX provided phase 2 signal-generating evidence for high-intensity treadmill exercise; the phase 3 SPARX3 program should be interpreted as ongoing unless definitive results are available.

Cell replacement should be distinguished from disease modification. Early trials of pluripotent-stem-cell-derived dopaminergic progenitors have demonstrated feasibility, graft-survival signals and acceptable early safety. Bemdaneprocel has entered a randomized, sham-surgery-controlled phase 3 trial. These approaches aim primarily to restore dopaminergic function rather than directly suppress the underlying disease process, although long-term clinical effects remain under investigation.

CLINICAL IMPLICATIONS

- No drug should currently be prescribed specifically with the claim that it slows PD progression.
- Symptomatic therapy should be selected according to established clinical indications, not inferred neuroprotection.
- Exercise should be encouraged for established functional and symptomatic benefits while disease-modification claims remain cautious.
- Genetic and biomarker testing may support counseling and trial eligibility, but currently does not identify an established disease-modifying treatment.
- For investigational therapies, clinicians should distinguish failed primary endpoints from exploratory subgroup or biomarker signals and should prioritize peer-reviewed primary sources over sponsor summaries whenever available.

CONCLUSION

Disease modification in Parkinson's disease remains an unmet therapeutic goal. Recent trials have narrowed several hypotheses but have not closed the field: exenatide and BIIB122 have produced important negative data, while prasinezumab, genetically enriched LRRK2 strategies, ambroxol and other biologically targeted approaches remain under study. The central editorial lesson is equally important: in a rapidly changing field, publication readiness depends not only on factual recall but on source verification, evidence calibration, and explicit handling of uncertainty.

7. Before / After Editorial Examples

RAW AI	"As I recall, [PADOVA's] primary endpoint ... did not reach significance."
PHYSICIAN-REVIEWED	"PADOVA did not meet its primary endpoint: confirmed motor progression was delayed numerically with prasinezumab versus placebo (HR 0.84; 95% CI 0.69-1.01; p=0.066), supporting continued investigation but not establishing efficacy."
WHY IT CHANGED	Replaced model recollection with a peer-reviewed primary source and numerical result.

RAW AI	"MOVES-PD ... did not show benefit ... as I understand from sponsor reports."
PHYSICIAN-REVIEWED	"In MOVES-PD Part 2, venglustat did not demonstrate clinical benefit versus placebo in GBA1-associated PD (between-group difference in MDS-UPDRS II+III change 2.58 points; 95% CI -1.10 to 6.27; p=0.17)."
WHY IT CHANGED	Upgraded source hierarchy from vague sponsor recollection to the 2023 primary publication.

RAW AI	"Readers should consult [SPARX3] results."
PHYSICIAN-REVIEWED	"SPARX3 is a phase 3 trial designed to test whether high-intensity exercise alters PD progression; definitive phase 3 results should not be inferred until reported."
WHY IT CHANGED	Removed a false implication that results were already available.

RAW AI	"Recent negative results ... rule out two mechanistically attractive strategies."
PHYSICIAN-REVIEWED	"The findings weaken support for exenatide and BIIB122 in unselected/idiopathic PD but do not establish failure of the broader GLP-1 or genetically targeted LRRK2 strategies."
WHY IT CHANGED	Calibrated a class-level conclusion to the actual population and intervention studied.

8. Limitations & Key Takeaways

Limitations

- The audit used 40 high-impact claim clusters rather than counting every sentence as a separate claim; this reduces double-counting but makes the denominator dependent on the predefined clustering method.
- Severity ratings reflect editorial and clinical relevance, not a validated regulatory scoring instrument.
- For the newest development programs, peer-reviewed publications were preferred; sponsor materials and trial registries were used only when they were the most current available sources.
- This project evaluates one frozen AI article and should not be interpreted as a general performance estimate for all models, prompts, or therapeutic areas.
- The reviewed article is a portfolio artifact and not a systematic review or treatment guideline.

What This Project Demonstrates

- Claim-level medical fact-checking and evidence verification
- Primary-source selection and citation-quality control
- Ability to distinguish primary endpoints from exploratory signals
- Calibration of medical claims to population, endpoint, and evidence strength
- Identification of outdated or unstable evidence in a rapidly moving field
- Physician-led editing for clinical nuance, safety, and publication readiness
- Use of AI as a drafting tool while retaining human accountability for scientific accuracy

PORTFOLIO TAKEAWAY

The differentiator is not that AI made errors. It is the ability to identify which errors matter, trace them to the evidence, correct the language proportionately, and produce a stronger final medical document.

9. Selected References

Primary and current sources used for the audit

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SOURCE NOTE

The original AI draft contained 30 references and a verification note acknowledging unresolved citation and status questions. This audit preserved those uncertainties as findings and replaced them, where possible, with peer-reviewed primary sources or current trial-registry information.