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Equity Research
Initiation

NeOnc Technologies Holdings, Inc.

Buy

Blood-Brain Barrier Bypassing Platform for High-Grade Gliomas – Initiating Coverage with a Buy and $20 PT

Biotechnology	
NTHI – NASDAQ	May 18, 2026
Closing Price 5/15/26	**$5.50**
Rating:	Buy
12-Month Target Price:	$20.00
52-Week Range:	$3.20 - $12.99
Market Cap (M):	$139.1
Shares O/S (M):	25.3
Float:	42.3%
Avg. Daily Volume (000):	64.9
Debt (M):	$0.3
Dividend:	$0.00
Dividend Yield:	0.0%
Risk Profile:	Speculative
Fiscal Year End:	December

Total Expenses ('000)

	2025A	2026E	2027E
1Q	37,617	7,056A	4,141
2Q	5,708	3,062	4,321
3Q	6,935	3,317	4,681
4Q	8,021	3,445	4,862
CY	58,280	16,851	18,006

NeOnc went public via direct listing on 3/26/25.

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Summary

- NeOnc is developing therapies for CNS tumors that bypass the blood-brain barrier (BBB) with two shots on goal: NEO100 as a near-term value driver and NEO212, a significant, earlier-stage opportunity. P2a top-line data for NEO100 expected in 2H26, representing a key event for NeOnc. Updates around NEO212, which has completed a P1 in glioblastoma, are also expected over 2026. We believe on positive updates, NTHI shares should be positioned to rise in value.
- NEO100 is an intranasal perillyl alcohol (POH) formulation designed for direct nose-to-brain delivery, bypassing the BBB to enhance CNS exposure. A P2a in recurrent IDH1-mutant high-grade glioma is approaching top-line in 2H26. Prior P1/2 data supports safety and enhanced survival vs. historical benchmarks.
- NEO212 is a 'de-risked' oral bioconjugate of NEO100 and temozolomide (TMZ) for recurrent GBM. The combination is designed to enhance CNS penetration and address MGMT-driven resistance of TMZ (SOC chemo for GBM). Prior data suggests ~3X higher brain exposure vs. TMZ and MGMT degradation. Next step is Type B meeting to align on P2 design.
- **Financials.** We estimate NeOnc currently has ~$0.5M in cash, and has access to both a $75M ATM and a $10M line of credit, which could be used to extend cash runway, as well as other potential sources of capital. As a pre-revenue biotech company, we also factor in additional financings in our model. The key is getting to the next inflection point, which is the NEO100 data in 2H26.

Details

NEO100 – intranasal POH enabling direct CNS delivery in high-grade glioma

- Highly purified intranasal perillyl alcohol (POH), a naturally occurring monoterpene with antitumor activity, bypasses the BBB via olfactory and trigeminal pathways.
- Targeting recurrent IDH1-mutant Grade III-IV high-grade glioma, a setting with no SOC post-TMZ/radiation and poor outcomes (median OS ~6-15 months).
- P1 data demonstrated median OS ~15 months (vs. ~6-9 months historical), PFS-6 ~30-33% (vs. <10%-20%), and ~33% survival at 24 months.
- P2a (n=25) fully enrolled; interim data (n=18) show PFS-6 of 44% and 17% CR rate. Top-line data expected 2H26.

NEO212 — TMZ bioconjugate to enhance CNS delivery and overcome resistance in GBM

- Oral conjugate of temozolomide (TMZ) and NEO100 designed to enhance CNS penetration (~3X higher brain exposure vs. TMZ).
- Targets MGMT-mediated resistance via NEO100-driven MGMT degradation, with potential activity independent of MGMT status.
- Avoids toxic TMZ metabolites, supporting improved tolerability and minimal myelosuppression; also induces ER stress and apoptosis.
- P1 in recurrent GBM established MTD/RP2D with favorable safety/early efficacy, including a confirmed PR (~60% reduction) with >20-month durability; Type B meeting planned to align on P2 design.

Valuation. We model commercialization of NEO100 for recurrent IDH1-mutant high-grade glioma and NEO212 for recurrent GBM both in 2029 with 70% revenue risk adjustments. A 30% discount rate is then applied to our free cash flow, discounted EPS, and sum-of-the-parts models, which are equally weighted to derive a 12-month price target of $20.

CORPORATE PROFILE

NeOnc Technologies Holdings, Inc.
23975 Park Sorrento Suite 205
Calabasas, CA 91302
https://neonc.com/

Ownership summary:

Institutional Ownership:	8.4%
Insider Ownership*:	57.7%

*Founder: 17.4%
CEO: 14.3%
AFH Holding & Advisory, LLC: 14.7%

Balance Sheet Summary:
(as of 3/31/26) *

Cash:	$138.6K
Debt:	$0.3M
Shareholders' equity:	($13.2M)
Total assets:	$3.3M

*Subsequent to quarter-end the company raised $2.0M via equity financing. We estimate the company's current cash position to be ~$0.5M. The company has a $75M ATM and $10M line of credit.

Analysts Covering the Co.:
# of Buys: 1
# of Holds: 0
# of Sells: 0
(*Excluding Maxim Group LLC*)

Investor Relations:
James Carbonara
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Company Background. NeOnc Technologies Holdings, Inc. is a clinical-stage biotech developing therapies for CNS tumors designed to bypass the blood-brain barrier (BBB), a key limitation of current treatments, potentially positioning its candidates as differentiated options in high unmet-need settings. The company's lead programs include NEO100, an intranasally delivered purified form of perillyl alcohol (POH), and NEO212, a covalently conjugated molecule linking the chemotherapy temozolomide with POH, both of which are designed to enhance CNS penetration and therapeutic activity in malignant gliomas.

Senior Management:

Amir F. Heshmatpour, Chief Executive Officer – Amir Heshmatpour is the Executive Chairman and President of NeOnc Technologies Holdings, Inc., where he drives the company's mission to transform the treatment of brain cancers through breakthrough science and global partnerships. With over 25 years of executive and board leadership, he brings a proven track record of building companies, guiding capital markets strategies, and advancing life-changing innovations from concept to commercialization.

Thomas Chen, MD, Ph.D., Founder & CSO – Dr. Chen is the Founder and Chief Scientific Officer of NeOnc Technologies Holdings, Inc., where he leads the company's scientific strategy focused on advancing novel therapies for brain cancers. A board-certified neurosurgeon and Director of Surgical Neuro-Oncology at the Keck School of Medicine of USC, he brings deep expertise in glioma biology supported by an extensive publication record and leadership across national neurosurgery committees and academic journals.

Keithly A. Garnett, Chief Financial Officer – Keithly Garnett has served as Chief Financial Officer of NeOnc Technologies Holdings, Inc. since April 2023 and as a director since January 2023, where he oversees financial strategy, capital allocation, and corporate governance. He brings over 17 years of experience from Ernst & Young, where he specialized in transaction advisory, business valuation, and financial strategy for publicly traded companies, supporting capital markets execution and long-term growth planning.

Josh Neman, Ph.D., Chief Clinical Officer – Dr. Neman serves as Chief Clinical Officer of NeOnc Technologies Holdings, Inc., where he leads clinical development strategy. He serves as an Associate Professor of Neurological Surgery, Neuroscience, and Physiology at the Keck School of Medicine of USC, and held leadership roles including Program Chair of the Cancer Biology and Genomics PhD Program and Scientific Director at the USC Brain Tumor Center.

Additional Management details and Board of Directors details can be found on the company website and SEC filings.

INVESTMENT SUMMARY

Overview. NeOnc is a clinical-stage biotech developing therapies for brain tumors designed to bypass the blood-brain barrier (BBB), a key limitation of current treatments, potentially positioning its candidates as differentiated options in high unmet-need settings. The company's pipeline is centered on its lead asset NEO100, a highly purified, pharmaceutical-grade perillyl alcohol (POH) formulation delivered intranasally for gliomas*, enabling direct nose-to-brain transport. NEO100 has shown encouraging P1/P2 data and is currently being evaluated in a P2a study in recurrent IDH1-mutant high-grade glioma, with top-line data expected in 2H26. The second program, NEO212 is an oral bioconjugate of NEO100 and temozolomide (TMZ), the standard of care chemotherapy in glioblastoma (GBM). By linking TMZ with NEO100, NEO212 enhances stability and CNS exposure (~3x higher brain levels vs. TMZ) and actually degrades MGMT, a key driver of treatment failure with TMZ. The approach seems to be effective in TMZ-resistant patients, and independent of MGMT methylation status. Early P1 data in GBM* have been encouraging, and the company plans a Type B FDA meeting to align on a P2 design, which could potentially support a path toward accelerated approval.

*Glioma is a broad category of brain tumors arising from glial cells and can be low grade (slow growing, grade I, II) or high grade (fast growing, grade III, IV). Glioblastoma is a specific highly aggressive grade 4 glioma. So, all glioblastomas (GBM) are gliomas, not all gliomas are glioblastomas.

NEO100 is a novel intranasal therapy to enhance CNS delivery and drive anti-tumor activity in high-grade glioma. NEO100 is a highly purified, pharmaceutical-grade intranasal formulation of perillyl alcohol (POH). The program is differentiated by its intranasal, nose-to-brain delivery, bypassing the blood-brain barrier via olfactory and trigeminal pathways to improve CNS exposure. In addition, POH appears to transiently modulate tight junctions within the blood-brain barrier, facilitating drug penetration into the CNS without permanent disruption, which we view as a key component of its delivery advantage. Together, these mechanisms enable higher and more consistent drug levels in the brain. NEO100 is administered via a mobile nebulizer ~4X daily (~15-20 minutes per session), supporting compliance and chronic use, with a favorable safety profile to date and minimal systemic toxicity. In its target setting of IDH1-mutant (isocitrate dehydrogenase I) high-grade glioma, there is no SOC after TMZ and radiation and outcomes remain poor, with median overall survival of ~10-15 months (grade III) and ~6-9 months (grade IV). If NEO100 can meaningfully extend survival with a favorable safety profile, we view this as a significant potential value driver for NeOnc.

P2a ongoing in recurrent IDH1-mutant high-grade glioma, top-line data expected 2H26. The ongoing P2a study is a biomarker-enriched, open-label trial evaluating intranasal NEO100 in 25 patients with recurrent IDH1-mutant Grade III-IV glioma, a population selected based on P1 survival signals. While all gliomas ultimately have a poor prognosis, IDH1-mutant disease can be less aggressive, even at grade III-IV. An IDH inhibitor, vorasidenib (Voranigo), is a targeted therapy approved in 2024 for grade II gliomas following surgery (first-line) but there is nothing for grade III or grade IV recurrent disease. The P1 study included 12 patients with recurrent GBM treated with NEO100, which demonstrated a continued positive safety profile and drove median OS of ~15 months vs. (6-9 months historical), PFS-6 (6-months) of ~30-33% (vs. <10-20% historical), and ~33% of patients were alive at 24 months. While the totality of the data is compelling, there appeared to be more substantial benefit in patients (n=4) with IDH1-mutant disease. This enabled NeOnc to design a P2a study (n=25) that is biomarker-enriched for IDH1 mutation, which could increase the probability of success for NEO100 and inform the design of a registration study. Thus far, the interim P2a data (n=18) has demonstrated PFS-6 of 44% (vs 20-30% historical) and 17% complete response (CR) rate, with other patients achieving disease control. Further, in a pooled 25-patient cohort (P1/2a + compassionate use), NEO100 demonstrated a 24% radiographic remission rate (6/25; ~3x vs. ~8% historical salvage benchmarks) and 36% of patients alive ≥18 months (9/25). The key in glioma, regardless of grade and subtype is overall survival, and therefore, even disease control is meaningful as it could translate into longer survival times, just keeping the tumor "in-check". The P2a is fully enrolled with top-line data expected in 2H26.

NEO212 is a TMZ bioconjugate designed to overcome resistance and improve CNS delivery in GBM. As noted above, GBM is a grade IV, very aggressive glioma with standard of care not changing for decades; surgery followed by radiation and TMZ (the "Stupp regimen"). The usage of TMZ, at least in front-line therapy, is dependent on MGMT methylation status and has limitations due to severe myelosuppression. TMZ also does not get across the blood-brain barrier very well, but higher for longer doesn't work because of tox. Now, let's look at NEO212, which is an oral conjugate of TMZ and NEO100 designed to enhance brain penetration and address MGMT-driven resistance. There are several mechanisms that are important here for NEO212; getting ~3X more TMZ to the brain, which by default helps mitigate myelosuppression-related tox since the TMZ is getting to its target and the conjugate avoids generation of TMZ's toxic metabolite. As a result, more drug can get on target for longer, which should drive better outcomes, and so far this has been supported with clinical data. We also note the potential of just having more TMZ on its target could induce therapeutic benefit regardless of MGMT status. The NEO100 component actually degrades MGMT (demonstrated in preclinical work), which could be synergistic, making NEO212 a broad-spectrum glioblastoma therapeutic. Additionally, NEO100-driven ER stress also induces apoptosis. Preclinical data show greater anti-tumor activity vs. TMZ in MGMT-high and resistant models, alongside lower systemic toxicity and minimal myelosuppression.

NEO212 P1 data support differentiated safety and early efficacy, P2 design discussions upcoming. The Phase 1 dose-escalation study in recurrent GBM (n=14) evaluated oral NEO212, establishing an MTD of 810 mg and RP2D of 610 mg. NEO212 demonstrated a favorable safety profile across dose levels, with no meaningful myelosuppression, a key limitation of TMZ. While early and based on a small, heavily pretreated

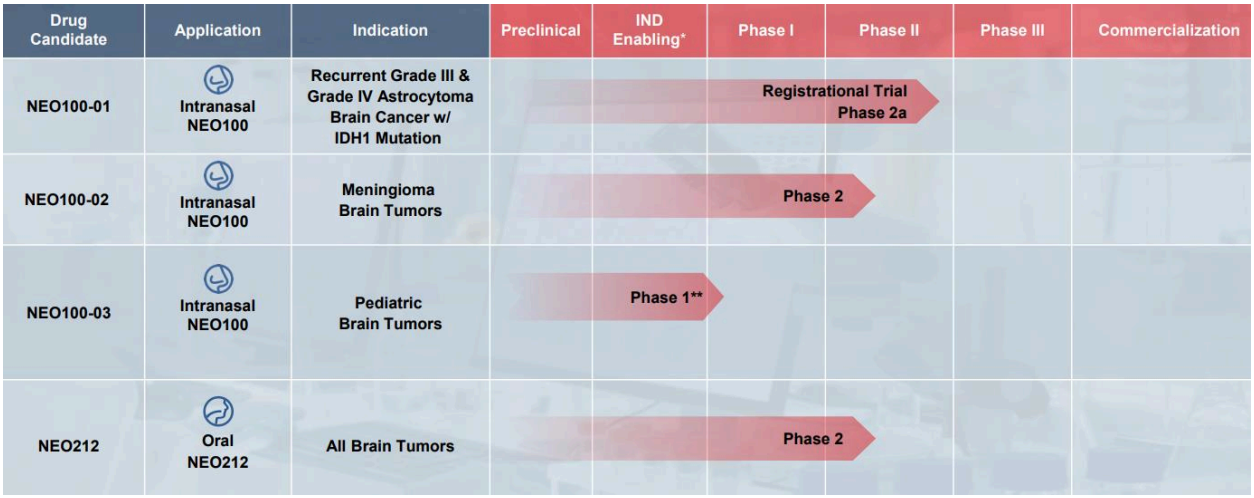
cohort, signs of antitumor activity were observed, including a confirmed partial response (~60% tumor reduction) with durable disease control extending >20 months despite prior TMZ failure, along with multiple cases of stable disease following progression on prior therapies. Several patients remained on treatment for >16 months, suggestive of durability. Taken together, these data, in our view, provide initial clinical validation of NEO212's potential to overcome MGMT-mediated resistance. The company plans a Type B meeting with FDA to align on a Phase 2 design, which could support a path toward accelerated approval.

Bottom line. NeOnc is an emerging neuro-oncology company with a differentiated pipeline driven by its NEO100 platform designed to bypass the blood-brain barrier, a key limitation that continues to limit efficacy for available therapies. The opportunity, to us, is compelling and anchored by NEO100 with a fully enrolled P2a study in recurrent IDH1-mutant high-grade glioma on track for top-line data in 2H26. This leads into a larger, earlier-stage opportunity in NEO212 as a partially de-risked "optimized" version of temozolomide (TMZ) for GBM, combining a validated backbone with ~3X higher brain exposure vs. TMZ, potential activity in MGMT-resistant disease, and decreased systemic side effects such as myelosuppression. P1 data supports initial efficacy, and an FDA Type B meeting is planned to align on a P2 design that could potentially support an accelerated approval path. We estimate NeOnc currently has ~$0.5M in cash but has access to both its $75M ATM and a $10M line of credit which could be used to extend runway, as well as other potential sources of capital. As a pre-revenue biotech company, we also factor in additional financings in our model. The next key event is P2a data for NEO100 in 2H26, and along with updates around NEO212, NTHI shares should be positioned to rise in value.

Finances.

- On 5/15/26, NeOnc filed its 10-Q and reported 1Q26 results with a net loss of ($8.8M) and ended the period with $138.6K in cash. Subsequent to quarter-end the company raised $2.0M via equity financing. We estimate the company currently has ~$0.5M in cash but it does have access to both its $75M ATM and a $10M line of credit which could be used to extend runway, as well as other potential sources of capital. As a pre-revenue biotech company, we also factor in additional financings in our model.
- On 4/20/26, NeOnc completed the fourth tranche closing under its multi-tranche PIPE, raising ~$2.0M through the issuance of ~0.28M shares at $7.20/share with five-year warrants ($9.00 exercise).
- On 3/20/26, NeOnc completed the third tranche closing under its multi-tranche PIPE, raising ~$1.0M through the issuance of ~0.14M shares at $7.20/share with five-year warrants ($9.00 exercise).
- On 2/25/26, NeOnc completed the second tranche closing under its multi-tranche PIPE, raising ~$1.45M through the issuance of ~0.20M shares at $7.20/share with five-year warrants ($9.00 exercise).
- On 1/29/26, NeOnc entered into and completed initial closings under its multi-tranche PIPE (aggregate commitment of up to ~$16.0M), raising ~$10.6M through the issuance of ~1.48M shares at $7.20/share with five-year warrants ($9.00 exercise).
- On 3/26/25, NeOnc began trading on the Nasdaq under the ticker NTHI via direct listing (no capital raised).

NeOnc pipeline.



Source: NeOnc presentation

NEO100, intranasal approach for recurrent IDH-mutant high-grade glioma

Recurrent IDH1-mutant high-grade glioma. Unlike glioblastoma (GBM), which most investors recognize as the most aggressive and common primary brain cancer, gliomas are a broad group of tumors arising from glial cells and include astrocytoma, oligodendroglioma, and GBM. GBM is now specifically defined as IDH-wildtype and typically develops rapidly with poor outcomes. In contrast, IDH1-mutant high-grade gliomas, grade 3 and 4 astrocytomas, represent a distinct subtype that usually develops more gradually from lower-grade tumors and follows a different clinical course. These tumors were previously referred to as "secondary GBM" but are now classified separately due to important biological differences. IDH1-mutant astrocytomas arise from astrocytes and are defined by mutations in the isocitrate dehydrogenase 1, IDH1, gene, most commonly R132H. This mutation occurs early in disease development and leads to accumulation of 2-hydroxyglutarate, 2-HG, which alters normal cellular function and drives tumor growth.[1] These tumors commonly harbor additional alterations in TP53 and ATRX, distinguishing them from GBM, which is instead associated with features such as EGFR amplification and chromosome 7 gain and chromosome 10 loss.[2] Clinically, IDH1-mutant astrocytomas span WHO grades 2 to 4, with grade 3, anaplastic astrocytoma, and grade 4 tumors representing the more aggressive forms of disease. Grade 4 classification may be based on histologic features such as necrosis or microvascular proliferation, or molecular findings such as CDKN2A/B homozygous deletion, even in the absence of classic histology.

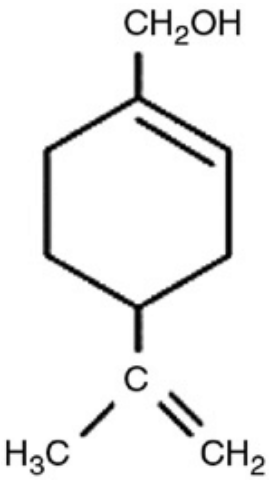
These tumors often progress from lower-grade to higher-grade disease over time, and many patients ultimately develop recurrent or treatment-resistant disease, representing a significant unmet medical need. The current standard of care (SOC) includes maximal safe surgical resection followed by radiotherapy with or without temozolomide, TMZ. While these treatments can delay progression, they are not curative, and most patients eventually experience tumor recurrence or malignant progression. In the recurrent setting, particularly after prior radiation and TMZ, treatment options are limited, typically involving clinical trials, off-label therapies, or re-irradiation in select patients. In terms of prognosis, IDH1-mutant astrocytomas have better outcomes than GBM, but survival declines with increasing grade and recurrence. Median overall survival from diagnosis is ~3-6+ years for grade 3 IDH-mutant astrocytoma and ~2-3+ years for grade 4, reflecting more aggressive higher-grade disease. After recurrence, median overall survival declines to ~10-15 months (grade 3) and ~6-9 months (grade 4), with progression-free survival of ~4-8 and ~3-6 months, respectively.[3] While IDH inhibitors have shown activity in earlier-stage disease, their impact in advanced grade 3 and 4 tumors have been limited. Overall, recurrent IDH1-mutant grade 3 and 4 astrocytomas represent a distinct, high-need subset of glioma, separate from GBM, characterized by progressive disease, limited treatment options at recurrence, and a significant unmet need. Across the US and EU, ~15K-8K patients are living with IDH1-mutant gliomas, with an estimated 35-45% either presenting with or eventually progressing to grade 3 or grade 4 disease over time.

Current treatment paradigm. The treatment paradigm for IDH1-mutant high-grade glioma (WHO grade 3 and grade 4 astrocytomas) differs between newly diagnosed and recurrent disease. Frontline management typically consists of maximal safe resection followed by radiotherapy and temozolomide (TMZ)-based chemotherapy. However, most patients ultimately experience disease progression. In the recurrent setting, there is no established standard of care, and treatment is highly individualized based on prior therapy, performance status, and tumor characteristics. Management in the recurrent setting typically involves multi-modality approaches, reflecting the limited efficacy and durability of available therapies, and includes:[4]

- **Repeat surgical resection.** Repeat craniotomy is considered in selected patients with accessible tumors and good performance status, primarily for cytoreduction and symptom relief (e.g., reducing mass effect or edema). It may also facilitate placement of local therapies such as Gliadel wafers or GammaTile.
- **Lomustine.** Lomustine is an oral alkylating agent that induces DNA cross-linking and tumor cell death. It is one of the most commonly used systemic therapies in recurrent glioma, either as monotherapy or in combination (e.g., with bevacizumab or temozolomide rechallenge), though clinical benefit is modest and limited by myelosuppression.
- **Bevacizumab.** Bevacizumab (Avastin) is an anti-VEGF monoclonal antibody that inhibits tumor angiogenesis. It is widely used to reduce edema and improve symptoms, often producing rapid radiographic responses; however, its impact on overall survival is limited, and its role is largely palliative.
- **Tumor treating fields (NovoCure/Optune).** Tumor treating fields are a non-invasive, device-based therapy developed by NovoCure (NVCR – NR), delivered via the wearable Optune system. The device generates low-intensity alternating electric fields via scalp electrodes that disrupt mitosis. In the recurrent setting, TTFields are used as an adjunct to systemic therapy, with variable adoption.
- **Gliadel wafers (carmustine implants).** Gliadel wafers are biodegradable polymer implants containing carmustine (BCNU), placed in the resection cavity to deliver localized chemotherapy. Their use is limited to patients undergoing surgery, with modest overall survival benefit (~1-2 months).
- **GammaTile (cesium-131 brachytherapy).** GammaTile is a collagen-based intracranial brachytherapy platform embedded with Cesium-131 seeds, placed intraoperatively following resection. It delivers localized radiation to the tumor bed over a short duration, aiming to improve local control while sparing surrounding tissue, and is available at select centers.

[1] *Raheme et al. Cancer Discov. 2023;13(10)*
[2] *Kapoor et al. Nature. 2012;483(7390)*
[3] *Rossi et al. Oncologist.2025;30(12)*
[4] *Pouyan et al. Mol Cancer. 2025;24(1)*

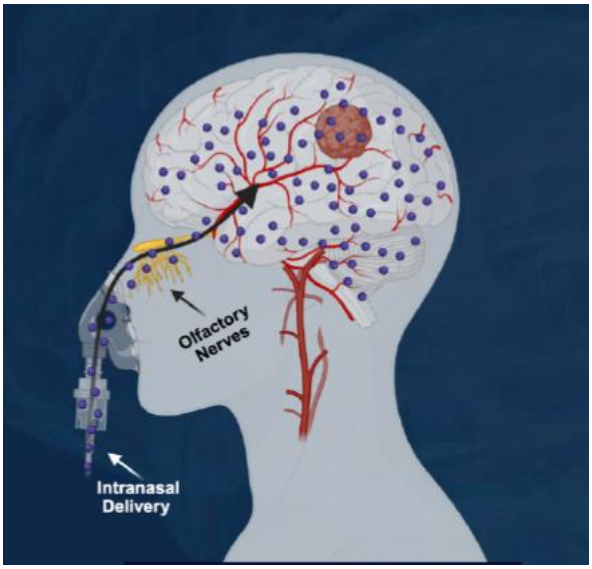
Perillyl alcohol (POH) is a naturally occurring monoterpene derived from limonene, a compound commonly found in the essential oils of citrus fruits such as oranges, lemons, and lavender. In oncology, POH has been investigated for its broad, multi-targeted anticancer activity, with preclinical studies indicating effects on key pathways involved in tumor cell proliferation, differentiation, and apoptosis. It has also been explored for its ability to interfere with post-translational modifications of signaling proteins (e.g., Ras), which are frequently dysregulated in cancer. Of particular interest is its capacity to penetrate the central nervous system, supporting research in brain tumors such as glioblastoma, as well as its use in alternative delivery methods (e.g., intranasal administration) to improve tolerability and drug exposure. Additionally, POH has shown potential to enhance the efficacy of conventional therapies, including chemotherapy and radiotherapy, suggesting a possible role as an adjuvant treatment.[5]



Source: Fonseca et al.

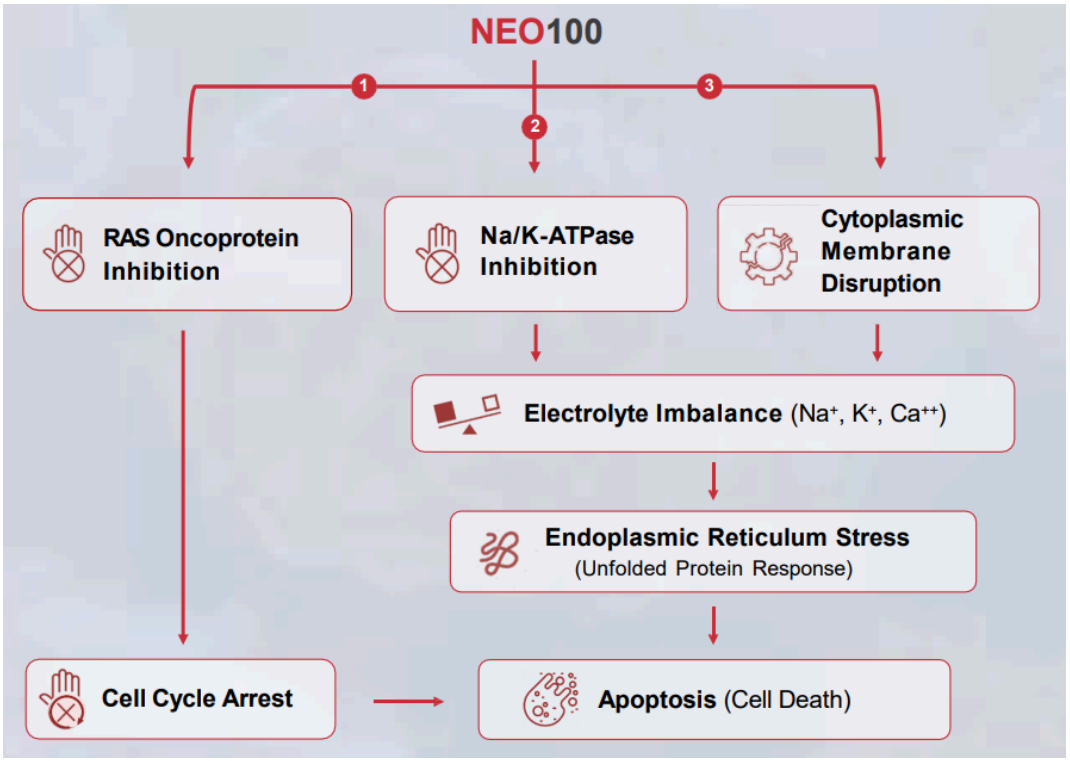
NE0100. NEO100 is a proprietary, highly purified, pharmaceutical-grade formulation of perillyl alcohol (POH) administered intranasally, a naturally occurring monoterpenoid with demonstrated broad anticancer activity across key hallmarks of tumor biology, including proliferation, survival signaling, and apoptosis resistance. The intranasal approach is designed to overcome limitations of oral POH (rapid metabolism, GI toxicity) by enabling direct nose-to-brain transport via olfactory and trigeminal pathways (cranial nerves I and V), bypassing the blood-brain barrier (BBB) and improving CNS exposure, which we view as a key differentiator. Of note, administration via a nebulizer (~4x daily, ~15-20 minutes) is non-invasive and can be performed at home or on-the-go, potentially supporting compliance and broader adoption, while also positioning NEO100 as a potential delivery enhancer for other CNS therapeutics. Mechanistically, POH exerts multimodal antitumor effects, including inhibition of Ras signaling, disruption of Na^+/K^+-ATPase, and induction of ER stress–mediated apoptosis. Clinically, a Phase 1 study in recurrent GBM has been completed with positive data, supporting advancement into an ongoing Phase 2a trial in IDH1-mutant recurrent high-grade glioma, with enrollment completed and top-line data expected in 2H26.

[5] *Sena et al. Curr Med Chem. 2024;31(42)*



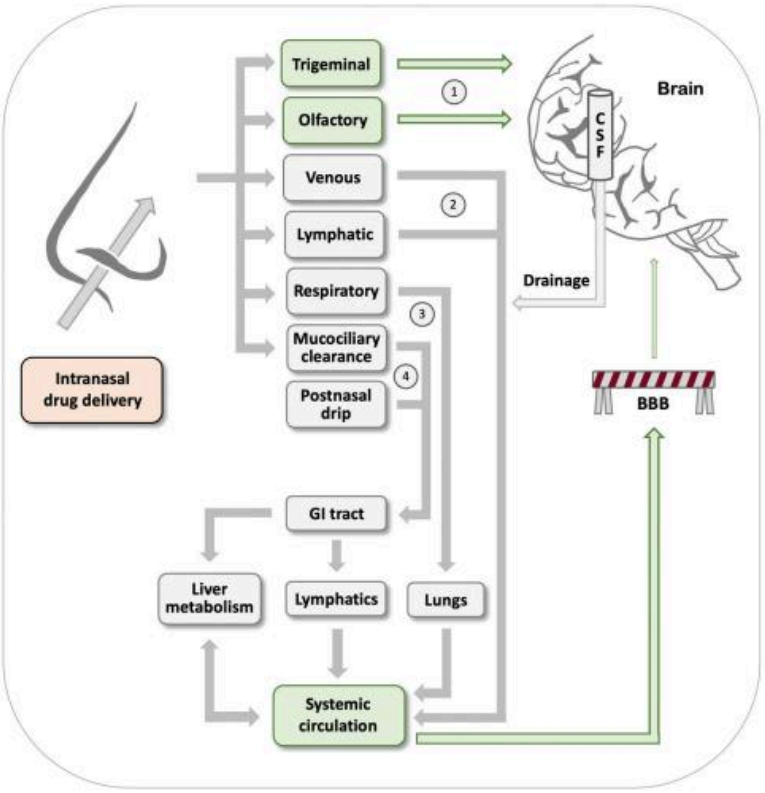
Source: NeOnc presentation

Mechanism of action (MOA). NEO100 drives anti-tumor activity via convergent cellular stress and apoptotic pathways. It inhibits RAS signaling to suppress proliferation and induce cell cycle arrest, while Na^+/K^+-ATPase inhibition and direct membrane disruption promote ionic imbalance (Na^+, K^+, Ca^{2+}) and loss of membrane integrity. These stresses trigger ER stress and the unfolded protein response, leading to apoptosis.



Source: NeOnc presentation

Intranasal delivery pathways to the brain. Following intranasal administration, drugs can access the brain through both direct and indirect routes. The most efficient pathway involves direct transport via the olfactory and trigeminal nerves, enabling brain delivery while bypassing the blood-brain barrier (BBB). Alternatively, drugs may first enter systemic circulation through nasal vasculature, pulmonary absorption, or gastrointestinal exposure due to mucociliary clearance. For these indirect routes, however, the BBB remains a significant barrier, limiting effective brain penetration for most therapies.

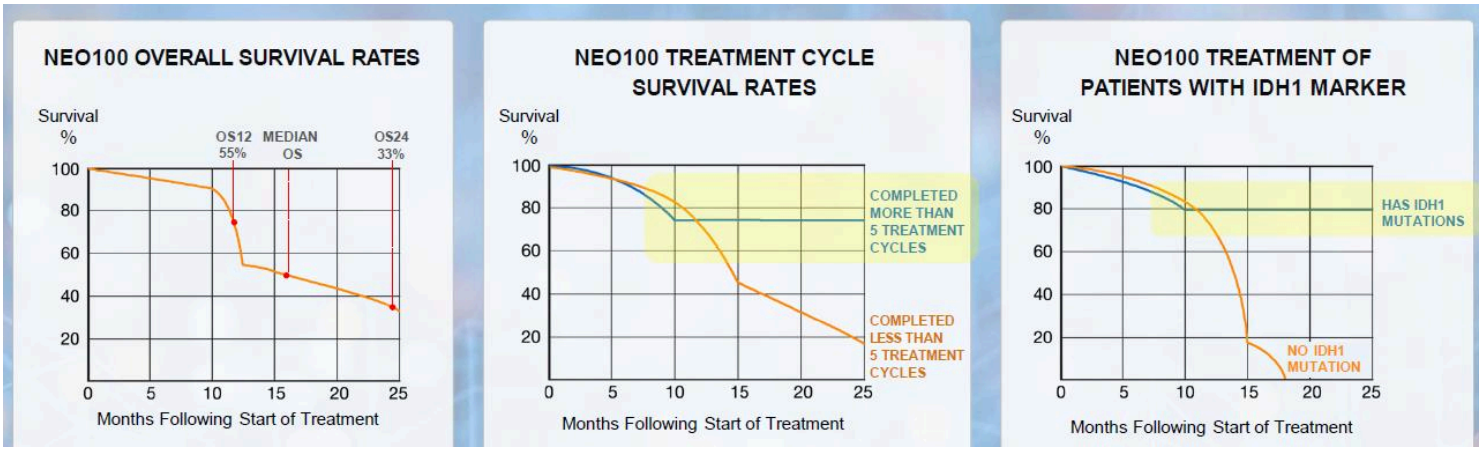


Source: Chen et al.

Clinical development

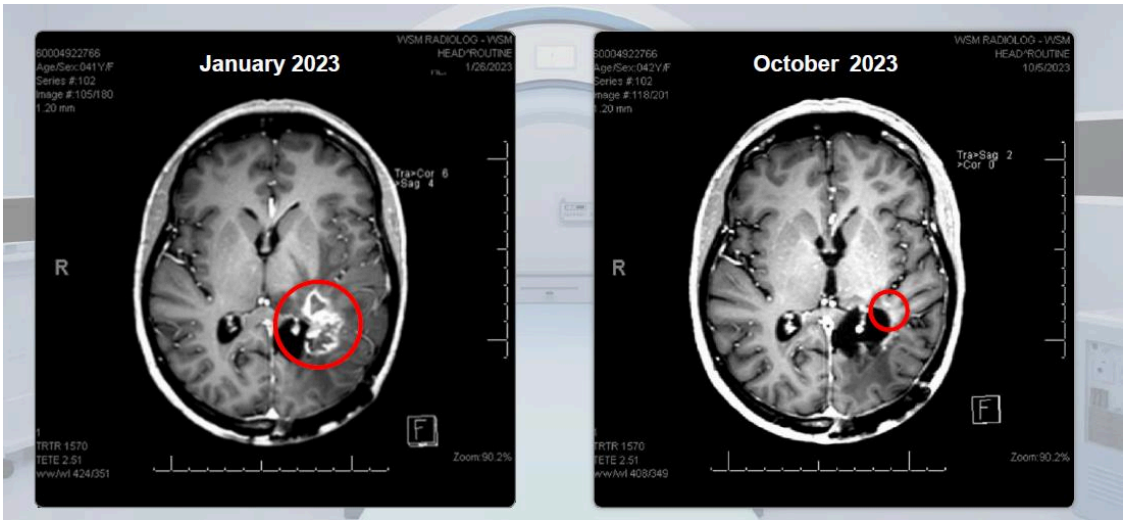
Phase 1 trial in recurrent glioblastoma (GBM). The P1 study of NEO100 was a multi-center, open-label, dose-escalation trial in patients with recurrent glioblastoma (GBM) following prior radiation and temozolomide. A total of 12 patients received intranasal NEO100 continuously until progression, at doses ranging from 384-1,152 mg/day (QID). The primary endpoint was safety and tolerability, including assessment of dose-limiting toxicities and treatment-related adverse events. Secondary endpoints included preliminary efficacy, specifically overall survival (OS), progression-free survival (PFS, including PFS-6), and duration of response, along with exploratory biomarker analyses (including IDH1 status). NEO100 was well tolerated across all dose levels, with no dose-limiting toxicities or severe adverse events reported. The study demonstrated a median OS of ~15 months, with OS-12 of ~55%, OS-24 of ~33%, and PFS-6 of ~30-33%, comparing favorably to historical outcomes in recurrent GBM (median OS ~6-9 months; PFS-6 typically <10-20%). Approximately one-third of patients survived >24 months, including a complete response at 2 years and a patient alive beyond 3 years. Exploratory analyses showed that long-term survival was concentrated in IDH1-mutant patients, informing the P2 (NEO100-01) trial design, which prospectively selects for IDH1-mutant Grade III-IV glioma patients to enhance the likelihood of demonstrating clinical benefit.

P1 NEO100 survival data. Phase 1 data for NEO100 demonstrated an encouraging survival signal in a heavily pretreated population, with ~55% overall survival (OS) at 12 months, ~33% at 24 months, and median overall survival (mOS) of ~15 months. Subgroup analyses indicate a treatment exposure-response relationship, with patients receiving >5 cycles demonstrating higher survival, and improved outcomes observed in IDH1-mutant vs. wildtype patients.



Source: NeOnc presentation

NEO100 demonstrates radiographic tumor reduction over time. Paired MRI images show a marked tumor reduction over ~9 months with NEO100, with clear shrinkage on the October 2023 scan vs. January 2023 baseline. While based on a single case, the imaging supports potential anti-tumor activity consistent with early clinical survival trends.



Source: NeOnc presentation

P1 NEO100 data vs. 2L recurrent GBM benchmarks. P1 NEO100 data show higher OS rates versus historical 2L recurrent GBM benchmarks (~55% at 12 months, ~33% at 24 months; ~15-month median OS) relative to outcomes reported for lomustine, regorafenib, and TTFields, though cross-trial comparisons should be interpreted with caution given differences in patient populations and early-phase sample size.

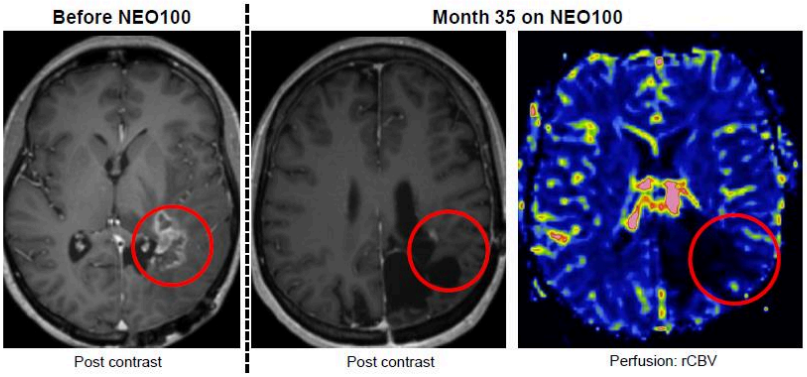
Therapy (Setting)	OS-12	OS-24	PFS-6	Median OS
NEO100 (intranasal POH) – Phase I (n=12)	**55%**	**33%****	**33%**	**15 mo**
Lomustine (CCNU) – control arm benchmark (REGOMA lomustine arm)	15%	NR	8%	5.6 mo
Regorafenib (Stivarga) – REGOMA	39%	NR	17%	7.4 mo
TTFields (monotherapy) – EF-11 (recurrent GBM)	~20%	~9%	Reported as endpoint (varies)	6.6 mo
Bev + Lomustine – EORTC 26101 (progressive GBM)	NR	NR	PFS improved	9.1 mo*
HDAC inhibitor (Vorinostat) – recurrent GBM Phase II	NR	NR	15%	5.7 mo

Source: NeOnc presentation

Ongoing P2a (NEO100-01) study in recurrent IDH1-mutant glioma, top-line data 2H26. The ongoing Phase 2a (NEO100-01) study is a biomarker-enriched, open-label trial evaluating intranasal NEO100 in patients with recurrent IDH1-mutant Grade III-IV gliomas, a population selected based on the survival signal observed in Phase 1 and representing a setting with limited treatment options and historically poor outcomes. The study is enrolling n=~25 patients across ~12 US centers, with patients receiving 1,152 mg/day (288 mg QID) via continuous intranasal dosing until progression. The primary endpoint is progression-free survival (PFS), with secondary endpoints including overall survival (OS), radiographic response, duration of response, and safety/tolerability. Initial data from 18 patients demonstrated a 6-month PFS (PFS-6) rate of 44% (8/18 with ≥6-month follow-up), comparing favorably to historical benchmarks of ~21-31%. Response outcomes included a complete response (CR) rate of 17% (3/18), with an additional 28% (5/18) achieving partial response or stable disease, while 50% (9/18) progressed. In a pooled 25-patient cohort (Phase 1/2a + compassionate use), intranasal NEO100 demonstrated a 24% radiographic remission rate (6/25; ~3x vs. ~8% historical salvage benchmarks), 44% PFS-6 (in evaluable patients), and 36% of patients alive ≥18 months (9/25), supporting the potential for durable benefit in a heavily pretreated population. Treatment was well tolerated, with no significant toxicity observed even with prolonged dosing, consistent with prior experience and supportive of chronic administration. The fully enrolled P2a (n=25) remains ongoing, with top-line data expected in 2H26, which we view as a key event for shares.

Interim P2a data. Interim data from the P2a in 18 patients demonstrated a 6-month progression-free survival (PFS-6) rate of 44%, comparing favorably to historical benchmarks of ~21–31%. Response outcomes included a complete response (CR) rate of 17%, with an additional 28% achieving partial response or stable disease, while 50% experienced disease progression. Representative imaging highlights radiographic tumor reduction and prolonged disease control in select patients, supporting the potential for durable responses in this setting.

Metric	NEO100-01	Historical IDH1 mut recurrent HGG[1,2]
6 months Progression Free Survival	44% (8/18) ≥6-month follow-up prior to data cutoff.	21-31% References: [1] Fanucci et al., 2023 [2] Walbert et al. 2010



CR: 17% 3/18
PR/stable: 28% (5/18)
Progression: 50% (9/18)

Source: NeOnc presentation

Next steps. The fully enrolled P2a (n=25) remains ongoing, with top-line data expected in 2H26, which we view as a key event for shares. If positive, the company plans to meet with FDA following the data to discuss next development steps.

P2 study NEO100 P2 ongoing in recurrent meningioma (NEO100-02). NEO100 is being evaluated in an ongoing Phase 2 study (NEO100-02) in patients with recurrent/malignant meningioma, with the trial initiated in July 2023. The study assesses safety, pharmacokinetics, and preliminary efficacy using continuous intranasal dosing designed to enable CNS delivery. Meningioma represents ~35% of primary brain tumors, with ~30-35% of patients experiencing recurrence following surgery and/or radiation, and no FDA-approved systemic therapies in the recurrent or radiation-refractory setting. Data from this study is expected in 2027.

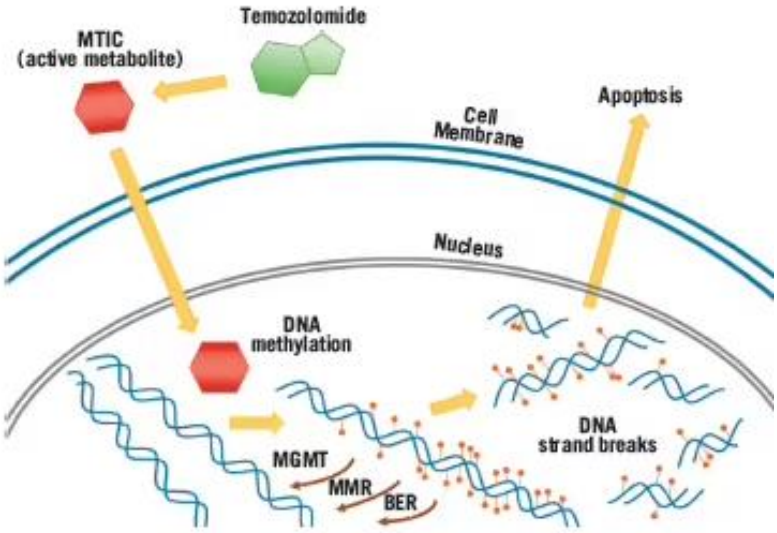
P1-ready NEO100 study in pediatric high-grade glioma (NEO100-03). NEO100-03 is advancing toward a Phase 1 study in pediatric high-grade glioma (including H3-altered/DIPG) following IND clearance from FDA. The planned study is expected to evaluate safety, dosing, pharmacokinetics, and CNS delivery via intranasal administration in pediatric patients. Pediatric high-grade gliomas account for ~15-20% of pediatric brain tumors, with median overall survival of less than 12 months. The program has received Rare Pediatric Disease designation, providing potential eligibility for a PRV. Enrollment is expected to start in 2Q26.

NEO212, TMZ bioconjugate targeting MGMT resistance and CNS delivery in GBM

Recurrent glioblastoma (GBM) represents a high-unmet-need oncology setting, with approximately 12,000-13,000 new cases diagnosed annually in the US, the majority of whom ultimately experience disease recurrence following first-line therapy. Standard initial treatment consists of maximal surgical resection followed by radiotherapy and temozolomide (TMZ), which modestly improves outcomes; however, median overall survival remains limited to approximately 14-16 months. In the recurrent setting, prognosis is particularly poor, with median survival typically in the range of 6-9 months.[6] There is no clearly established standard of care at recurrence, and treatment decisions are highly individualized, incorporating repeat surgical resection in select patients, re-irradiation strategies, and systemic therapies such as lomustine, bevacizumab, or TMZ rechallenge. These options generally provide limited durability of response, with bevacizumab offering improvements in progression-free survival and symptom control but no consistent overall survival benefit, and other agents, including regorafenib or combination regimens, delivering only incremental gains. A key challenge in this setting is resistance to alkylating agents, particularly in tumors with high expression of O6-methylguanine-DNA methyltransferase (MGMT), which repairs TMZ-induced DNA damage and reduces treatment efficacy; more broadly, resistance is driven by tumor heterogeneity, clonal evolution, and a restrictive tumor microenvironment, underscoring the urgent need for more effective and durable therapeutic options.[7]

Temozolomide (TMZ). Temozolomide (TMZ; Temodar) is an oral, second-generation alkylating agent that has formed the backbone of therapy for glioblastoma (GBM) since its approval in the mid-2000s and remains the standard of care in the front-line (1L) setting. It is administered following maximal surgical resection in combination with radiation therapy (the “Stupp regimen”), followed by adjuvant maintenance therapy, a protocol that demonstrated a survival benefit versus radiation alone and established TMZ as a foundational agent in GBM treatment. As a small, lipophilic prodrug, TMZ efficiently crosses the blood–brain barrier and undergoes spontaneous conversion at physiological pH to its active metabolite, MTIC (monomethyl triazeno imidazole carboxamide), which methylates DNA at the O6 and N7 positions of guanine, driving cytotoxicity primarily through O6-methylguanine–induced mismatch repair–mediated DNA damage and apoptosis.[8] Clinical response is strongly influenced by O6-methylguanine-DNA methyltransferase (MGMT), a DNA repair enzyme that reverses these lesions; epigenetic silencing via MGMT promoter methylation is associated with improved sensitivity and outcomes.

[6] *Davis et al. Clin J Oncol. 2016;20(5)*
[7] *Wang et al. Oncogene. 2023;42(27)*
[8] *Kumar et al. CNS Neurol. 2023;22(6)*



Source: Abrey et al.

Limitations of TMZ in GBM. Despite its widespread adoption, TMZ provides only modest and non-durable clinical benefit, with median overall survival (mOS) of ~14-16 months and progression-free survival of ~6-7 months in newly diagnosed GBM; approximately 70–80% of patients experience disease progression within 12 months. A primary limitation is resistance mediated by O6-methylguanine-DNA methyltransferase (MGMT), a DNA repair enzyme that reverses TMZ-induced cytotoxic lesions. While MGMT promoter methylation, present in ~35-45% of patients, is associated with improved response, TMZ is broadly used regardless of MGMT status, including in patients with unmethylated tumors who derive limited benefit.[9] In addition, systemic exposure to TMZ metabolites is associated with hematologic toxicity, including dose-limiting myelosuppression, which may constrain dosing independent of efficacy. In the recurrent setting, there is no clearly defined standard of care; available options such as TMZ rechallenge, lomustine, and bevacizumab offer variable and generally limited benefit, with mOS of ~6-9 months following progression. Combined, these limitations underscore the need for therapies that can overcome MGMT-driven resistance and improve outcomes in recurrent disease.[10]

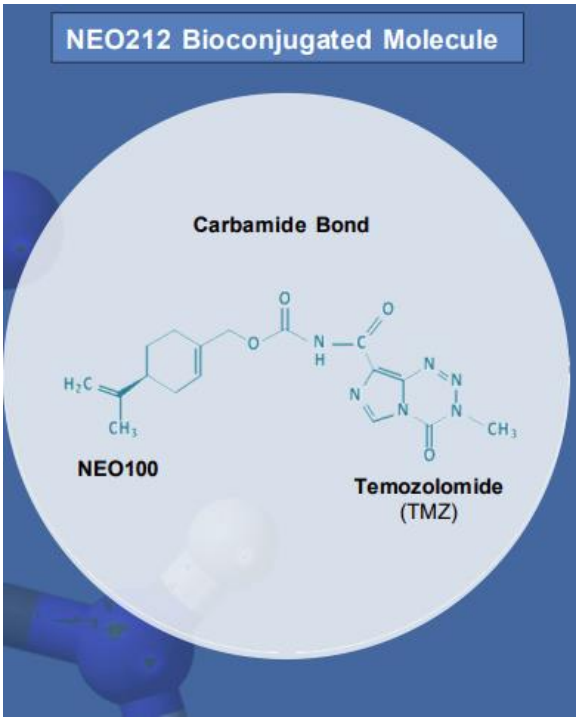
Limited benefit of current 2L therapies in recurrent GBM. As shown in the figure, second-line treatments for recurrent GBM, including CCNU, bevacizumab, combination therapy, and TTFields, provide limited benefit, with median overall survival of ~6-9 months and one-year survival rates below 40%. While bevacizumab may improve progression metrics, it does not consistently extend overall survival, and cytotoxic options like CCNU remain constrained by modest efficacy and tolerability. These benchmarks underscore the absence of an effective standard of care and the need for therapies that overcome resistance mechanisms, particularly MGMT-driven resistance, to achieve more durable outcomes.

Therapy	Median OS (mo)	OS-12 (%)	Key Trial Context
CCNU (lomustine) alone	5.6–8.6	~20–35%	REGOMA (~20%); EORTC 26101 (~30–35%)
Bevacizumab (Avastin) alone	~9.2	~38–43%	BRAIN Trial
CCNU + Bevacizumab*	~9.1	~35–40%	EORTC 26101
TTFields alone	~6.6	~20–25%	EF-11

Source: NeOnc presentation

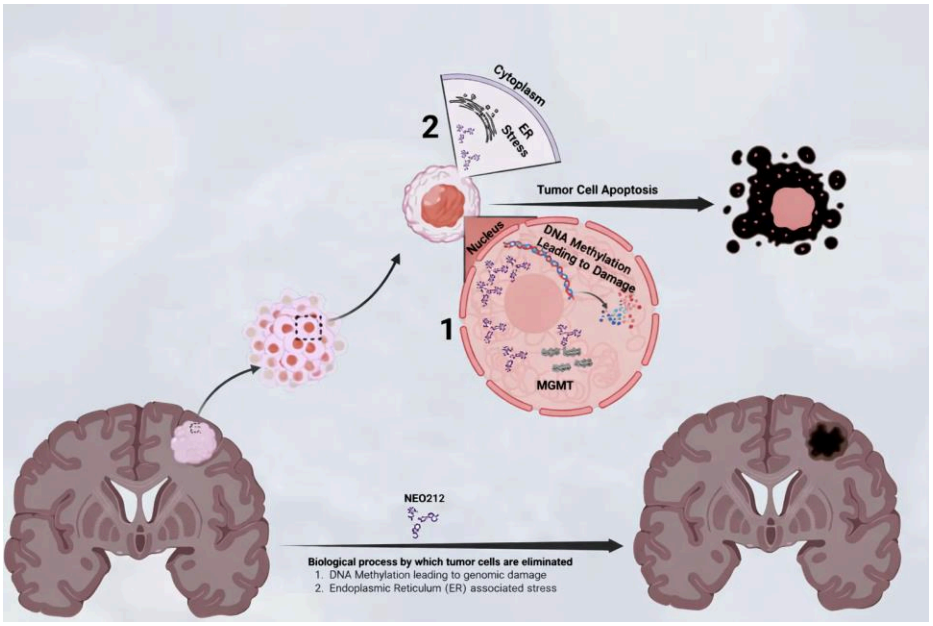
[9] *Lee et al. Genes Dis. 2016;3(3)*
[10] *Zhou et al. Cancer Biol. 2023;2(5)*

NEO212. NEO212 is an orally bioavailable bioconjugate of temozolomide (TMZ) and perillyl alcohol (POH; NEO100) designed to enhance central nervous system (CNS) delivery and overcome resistance in glioblastoma multiforme (GBM) and other brain malignancies. By covalently linking TMZ with POH, NEO212 preserves the validated TMZ backbone while improving pharmacokinetics, stability, and CNS exposure, with preclinical data demonstrating ~3X higher brain-to-serum ratios vs. TMZ, supporting enhanced BBB penetration and tumor targeting, which we view as a key differentiator. Mechanistically, NEO212 retains TMZ's cytotoxic activity via conversion to MTIC, inducing DNA damage and tumor cell death, while the POH component contributes complementary antitumor effects, including induction of ER stress and apoptosis, providing a dual mechanism within a single molecular entity. Importantly, NEO212 has demonstrated the ability to reduce and promote degradation of O6-methylguanine-DNA methyltransferase (MGMT), a key mediator of TMZ resistance, thereby potentially restoring sensitivity in MGMT-high or refractory tumors. In preclinical models, this has translated into greater antitumor activity vs. TMZ, including in resistant settings, alongside reduced systemic toxicity and less myelosuppression. Clinically, NEO212 has completed a P1 dose-escalation study, establishing a recommended P2 dose of 610 mg, with early signals of disease stabilization in heavily pretreated patients supporting continued development in recurrent GBM and brain metastases, particularly in post-TMZ and resistance-driven settings.



Source: NeOnc presentation

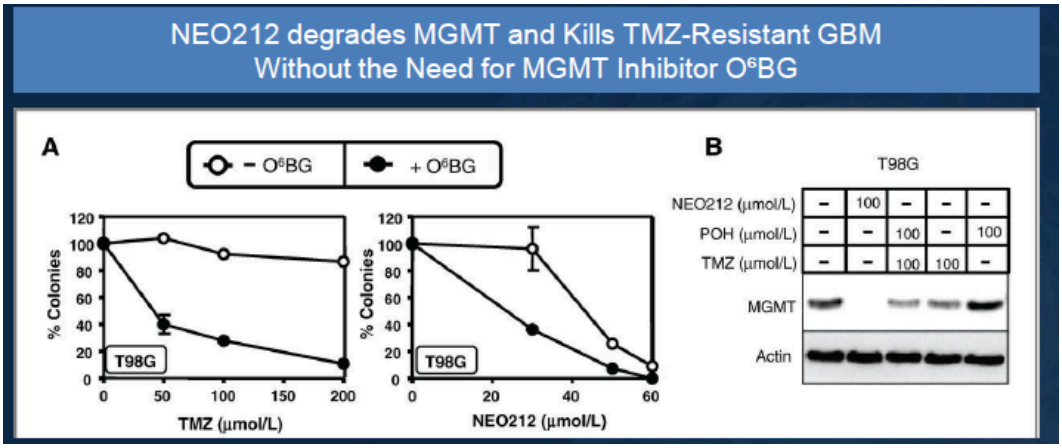
Mechanism of action (MOA). NEO212 enhances temozolomide (TMZ) anti-tumor activity through a dual mechanism driven in part by its NEO100 component. It increases intracellular TMZ delivery, leading to higher DNA methylation and greater genomic damage that can overwhelm MGMT-mediated repair. At the same time, it induces endoplasmic reticulum (ER) stress, disrupting protein folding and cellular homeostasis. Combined, these effects synergistically promote tumor cell apoptosis and improve therapeutic efficacy.



Source: NeOnc presentation

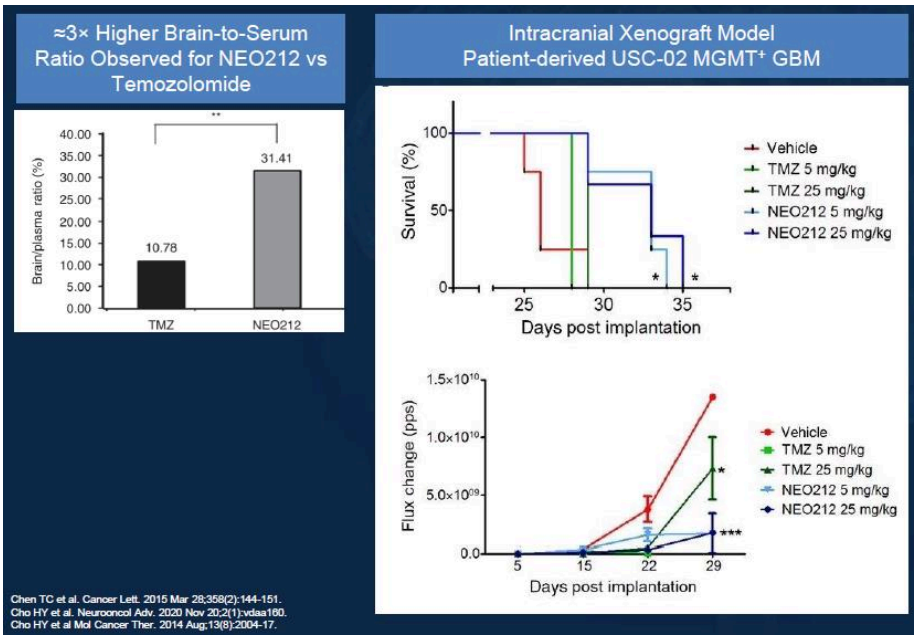
Preclinical data. Preclinical studies of NEO212 show a differentiated profile driven by enhanced CNS delivery, activity in resistant GBM models, and, critically, reduction of MGMT, a central driver of temozolomide (TMZ) resistance. In MGMT-expressing systems (e.g., T98G), NEO212 maintains cytotoxic activity and is associated with decreased MGMT protein levels, whereas TMZ does not alter MGMT and shows improved activity only with pharmacologic inhibition (O^6BG). Across multiple GBM models, including TMZ-resistant, MGMT-high, and MMR-deficient lines (U251TR, LN229, LN229TR2), NEO212 demonstrates greater dose-dependent cytotoxicity and colony suppression than TMZ, with limited effects in normal astrocytes. Following oral administration, pharmacokinetic analyses show higher brain penetration and intratumoral exposure, including increased TMZ and AIC metabolite levels and approximately 3X higher brain to serum ratios versus TMZ. In orthotopic, patient-derived $MGMT^+$ xenograft models, this translates into dose-dependent tumor growth inhibition and extended survival relative to TMZ, alongside higher accumulation of drug and metabolites in tumor-bearing brain tissue. These findings suggest NEO212 may improve CNS exposure while directly mitigating MGMT-driven resistance.

NEO212 degrades MGMT and bypasses TMZ resistance. NEO212 demonstrates direct modulation of MGMT, showing reduced MGMT protein levels in MGMT-expressing GBM cells (T98G) alongside sustained cytotoxic activity. Unlike temozolomide (TMZ), whose efficacy improves only with MGMT inhibition (O^6BG), NEO212 maintains dose-dependent cell killing without reliance on MGMT blockade, supporting a mechanism that may overcome a key driver of TMZ resistance.



Source: NeOnc presentation

NEO212 shows enhanced brain penetration and in vivo efficacy in TMZ-resistant GBM. Preclinical data show that NEO212 achieves ~3× higher brain-to-serum exposure than temozolomide (TMZ), supporting improved blood–brain barrier penetration and CNS drug delivery. In an orthotopic, patient-derived MGMT⁺ GBM xenograft model, this enhanced exposure translates into dose-dependent tumor growth inhibition and prolonged survival versus TMZ, with lower tumor burden observed across treatment groups. Together, these findings suggest improved pharmacokinetics may contribute to the observed in vivo efficacy in TMZ-resistant models.



Source: NeOnc presentation

Clinical development

P1 NEO212 trial design. The Phase 1 study of NEO212 was an open-label, dose-escalation trial primarily enrolling patients with recurrent glioblastoma (IDH-wildtype), with a smaller subset of patients with astrocytoma (IDH-mutant) and brain metastases included for broader CNS evaluation. Patients were treated with oral NEO212 once daily on Days 1-5 of a 28-day cycle across five dose levels (170, 220, 400, 610, and 810 mg/day), using a standard 2-3 patient per cohort design (total n=14). The population was predominantly GBM, with MGMT status distributed across methylated (n=7), unmethylated (n=2), equivocal (n=1), and non-applicable metastatic cases, allowing for early assessment across biologically relevant subgroups. The primary objectives were to evaluate safety and tolerability and to establish the maximum tolerated dose (MTD) and recommended Phase 2 dose (RP2D), with secondary endpoints including pharmacokinetics and preliminary anti-tumor activity. Dose escalation proceeded without dose-limiting toxicities, enabling evaluation through the highest planned dose (810 mg/day) and supporting advancement into Phase 2.

Subjects received NEO212 orally once daily on Days 1 through 5 of each 28-day treatment cycle.

	Tumor Type	MGMT Status	Total Subjects
Cohort 1 (170mg/day)	Squamous NSCLC-to-Brain Metastasis	N=1, Not applicable	3
	GBM *IDH1*-wildtype	N=2 Methylated	
Cohort 2 (220mg/day)	GBM *IDH1*-wildtype	N=2 Methylated, N=1 Unmethylated	3
Cohort 3 (400mg/day)	Esthesioneuroblastoma-to-Brain Metastasis	N=1, Not applicable	3
	Breast-to-Brain Metastasis	N=1, Not applicable	
	GBM *IDH1*-wildtype	N=1, Methylated	
Cohort 4 (610mg/day)	GBM *IDH1*-wildtype	N=1 Equivocal1, N=1 Unmethylated, N=1 Methylated	3
Cohort 5 (810mg/day)	GBM *IDH1*-wildtype	N=2 Methylated	2

Source: NeOnc presentation

P1 NEO212 trial data. Phase 1 dose-escalation data for NEO212 in recurrent GBM demonstrate a differentiated safety, PK, and early efficacy profile in a heavily pretreated population. Across five cohorts (170–810 mg QD, Days 1-5, 28-day cycle), the MTD was 810 mg and RP2D 610 mg. Notably, no clinically meaningful myelosuppression was observed; platelet, hemoglobin, WBC, and RBC levels remained stable with no dose-limiting cytopenias, contrasting with temozolomide; hepatic and renal markers (AST/ALT, creatinine) were similarly unremarkable. PK data showed low systemic metabolite exposure (mean peak AIC ~16.8 ng/mL at 610 mg), yet enhanced CNS delivery, with up to ~3.4x higher brain-adjusted exposure vs. temozolomide at 810 mg. Early efficacy signals, while based on a small cohort (n=14), were notable in this refractory setting: one confirmed partial response (~60% tumor reduction) with durable disease control exceeding 20 months post-recurrence despite prior temozolomide failure, alongside multiple cases of stable disease and measurable tumor shrinkage. Several patients remained on therapy for >16 months, suggesting potential durability of benefit. While limited by sample size, these data, in our view, provide initial clinical validation of NEO212's ability to overcome MGMT-mediated resistance and support continued development as a differentiated approach in recurrent GBM.

Low peripheral exposure of NEO212 metabolites supports a potentially differentiated PK profile. Peripheral blood levels of NEO212 metabolites (AIC and PA) remain low across all dose cohorts (170–810 mg QD), with modest increases and no clear dose-proportional accumulation. At the RP2D (610 mg), mean peak AIC (16.8 ng/mL) and PA (13.83 ng/mL) remain limited, while levels do not increase further at 810 mg, suggesting non-linear PK. Overall, these data are consistent with preferential CNS/tumor uptake and reduced peripheral metabolism, which may contribute to the favorable safety profile and potential differentiation from temozolomide.

	Cohort 1 (170 mg/QD)	Cohort 2 (220 mg/QD)	Cohort 3 (400 mg/QD)	Cohort 4 (610 mg/QD)	Cohort 5 (810 mg/QD)
N (evaluable)	3	3	3	3	2
Mean Peak AIC (ng/mL)	1.94	2.52	12.19	16.8	3.25
AIC Range (ng/mL)	0– 49.6	0 – 47.0	0 – 117.0	0 – 120.0	0– 36.0
Mean Peak PA (ng/mL)	0.17	0.07	2.04	13.83	6.96
PA Range (ng/mL)	0-27.7	0-12.5	0-21.2	0– 80.2	0– 43.6

Source: NeOnc presentation

NEO212 demonstrates higher brain-adjusted exposure vs. temozolomide. Dose-adjusted comparisons versus temozolomide (TMZ 200 mg/m²) show that NEO212 achieves increasing relative CNS exposure across dose levels, with brain-adjusted exposure reaching up to ~3.4x at the highest dose (810 mg). Notably, this occurs despite comparable or lower systemic exposure at lower and mid doses (e.g., 0.25–0.88x through 610 mg), suggesting a potentially more favorable CNS delivery profile. At the RP2D (610 mg), NEO212 achieves ~2.6x higher brain-adjusted exposure, supporting improved CNS penetration and tumor targeting.

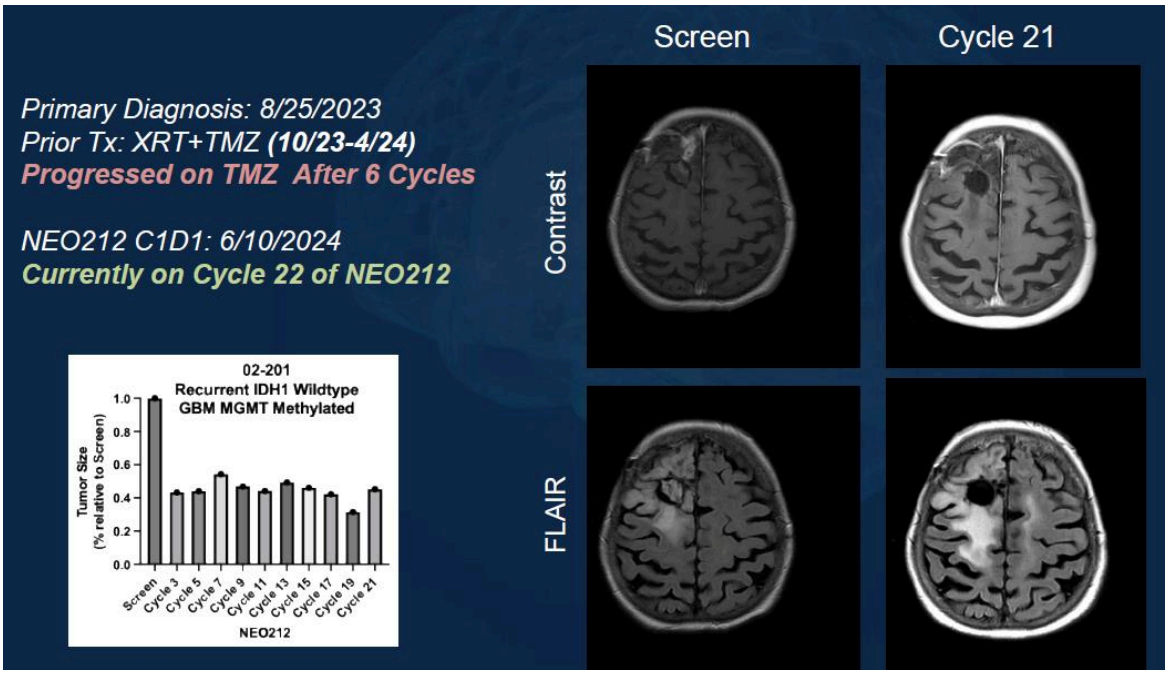
Adjusted for Body Weight, NEO212 Achieves Higher CNS Delivery vs. TMZ

Brain-adjusted exposure NEO212 vs TMZ 200 mg/m² (Cycle 2+)

NEO212 (mg/day)		NEO212 total over 5 days (mg)	NEO212 total 5-day dose (TMZ-equivalent, mg; conversion ratio = 0.521)	TMZ total over 5 days (mg)	Relative Exposure compared to TMZ	Brain-adjusted relative (3x BBB-permeable)
Cohort 1	170	850	443	1800	0.25	0.73
Cohort 2	220	1,100	573	1800	0.32	0.93
Cohort 3	400	2,000	1,042	1800	0.58	1.69
Cohort 4	610	3,050	1,589	1800	0.88	2.56
Cohort 5	810	4,050	2,110	1800	1.17	3.40

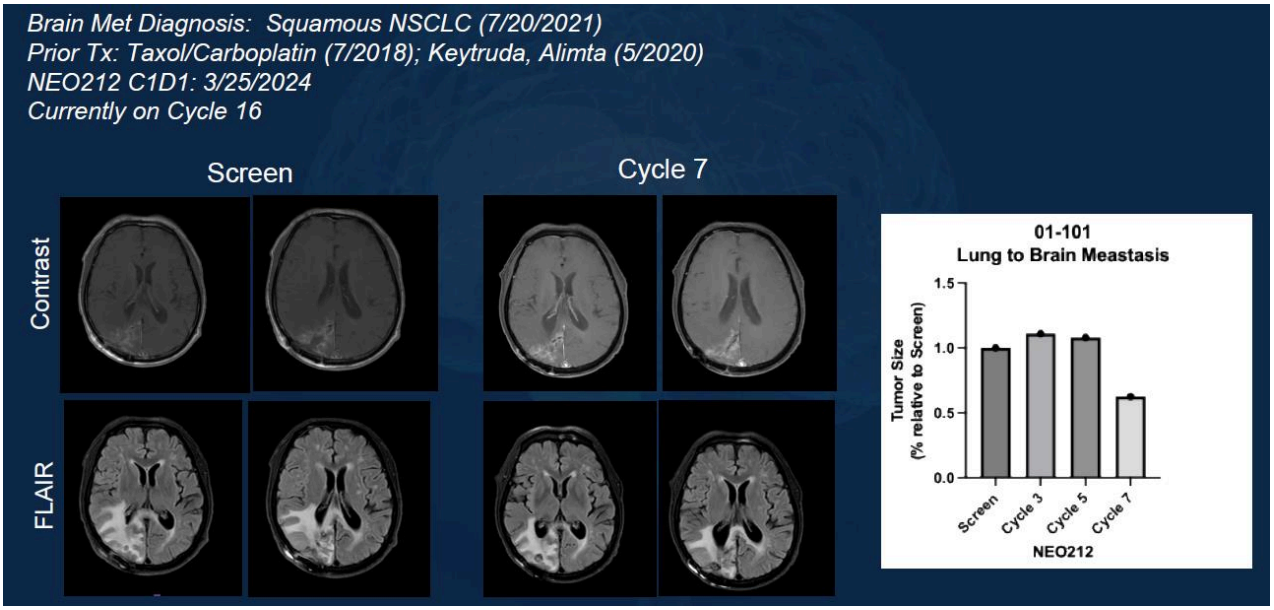
Source: NeOnc presentation

Partial response with prolonged disease control in P1 recurrent GBM patient. A confirmed partial response was observed in a recurrent IDH wild-type, MGMT-methylated GBM patient treated with NEO212 in the Phase 1 study, with ~60% tumor reduction from baseline and sustained disease control extending beyond 21 months post-recurrence despite prior progression on temozolomide. Longitudinal tumor measurements demonstrate consistent shrinkage and stabilization across treatment cycles, supported by radiographic improvement on MRI. While based on a single patient, this case highlights early evidence of durable clinical activity in a heavily pretreated setting.



Source: NeOnc presentation

Tumor reduction and disease stabilization in P1 lung-to-brain metastasis patient. In a heavily pretreated patient with brain metastases from squamous NSCLC (diagnosed ~55 months prior), NEO212 demonstrated evidence of tumor reduction and disease stabilization, with measurable decreases in lesion size by Cycle 7 following baseline assessment. This activity was observed after multiple prior lines of therapy, including chemotherapy and immunotherapy, with the patient remaining on treatment for at least 16 months. While based on a single case, these findings suggest potential activity of NEO212 in brain metastases beyond primary GBM.



Source: NeOnc presentation

Next steps. The company plans to engage with the FDA via a Type B End-of-Phase 1 meeting in the coming weeks to review safety, PK, preliminary efficacy, and RP2D selection, with the goal of aligning on a P2 study design for NEO212, including potential accelerated approval pathways, as the program advances into the next stage of development.

Intellectual property (IP).

Source: NeOnc presentation

Risks

The risk factors presented below could negatively impact the overall performance and prospects of NeOnc Technologies Holdings, Inc. and its stock price:

- **Clinical Risk:** Although NeOnc's pipeline is supported by early-stage clinical and preclinical data, its lead programs are being developed for primary and metastatic brain tumors, including high-grade gliomas such as glioblastoma (GBM), where historical clinical success rates have been low. These programs remain in early clinical development, and there can be no assurance that observed safety, tolerability, or preliminary efficacy signals will translate into meaningful clinical benefit in larger, controlled trials.

- **Capital Risk:** NeOnc has no approved products or product revenues and has incurred operating losses since inception and will require substantial additional capital to support ongoing clinical development, manufacturing scale-up, and potential pipeline expansion. There can be no assurance that NeOnc will be able to raise additional financing on favorable terms, or at all, and failure to secure adequate funding could delay development timelines, impact operations, or force the company to reduce or discontinue programs.

- **Regulatory Risk:** Regulatory authorities, including the FDA and international agencies, may assess the benefit-risk profile of NeOnc's product candidates differently than anticipated, which could result in clinical holds, requests for additional studies, delays in development timelines, or failure to obtain regulatory approval. The regulatory pathway for novel therapies targeting brain tumors is complex and uncertain, which may increase development costs, extend timelines, and materially impact the company's ability to commercialize its programs.

- **Commercial Risk:** Even if NeOnc achieves clinical and regulatory success, there can be no assurance of commercial viability, as the company has limited experience commercializing pharmaceutical products and may face challenges related to market adoption, pricing, reimbursement, and physician uptake. Competitive therapies and evolving standards of care in brain tumors could limit market penetration, which may negatively impact the company's ability to generate revenue and sustain long-term growth.

MODELING ASSUMPTIONS

1. We model commercialization of NEO100 in the US in 2029 for recurrent IDH1-mutant high-grade (III-IV) glioma.
2. We assume a US annual incidence of ~16,250 stage II-IV glioma patients, including ~15,238 recurrent patients following progression after surgery, radiation, and temozolomide, and ~9,263 patients with IDH1-mutant disease.
3. We assume US pricing of $175K per patient annually, benchmarked against current therapies in glioblastoma, including temozolomide and bevacizumab, with a 5% annual price increase.
4. We apply a 70% revenue-risk adjustment based on stage of development and clinical trial risk.

NEO100 recurrent IDH1-mutant high-grade (III-IV) glioma market model, US

NEO100, recurrent IDH1-mutant high-grade glioma (US)	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E	2034E	2035E	2036E
Stage III and IV glioma patients	16,250	16,819	17,407	18,017	18,647	19,300	19,975	20,675	21,398	22,147	22,922	23,725
% of patients failing TMZ (90%)	15,438	15,978	16,537	17,116	17,715	18,335	18,977	19,641	20,328	21,040	21,776	22,538
% of IDH1-mutant patients (60%)	9,263	9,587	9,922	10,269	10,629	11,001	11,386	11,784	12,197	12,624	13,066	13,523
Market Penetration					5.00%	10.00%	15.00%	25.00%	40.00%	50.00%	60.00%	60.00%
Total Patients Treated					531	1,100	1,708	2,946	4,879	6,312	7,839	8,114
Cost of Treatment					175,000	183,750	192,938	202,584	212,714	223,349	234,517	246,243
Increase in Cost					5%	5%	5%	5%	5%	5%	5%	5%
Total revenue ('000)					$ 93,003	$ 202,142	$ 329,517	$ 596,838	$ 1,037,782	$ 1,409,762	$ 1,838,471	$ 1,997,958
Risk adjustment					70%	70%	70%	70%	70%	70%	70%	70%
Total Revenue ('000)					**$ 27,901**	**$ 60,643**	**$ 98,855**	**$ 179,051**	**$ 311,335**	**$ 422,929**	**$ 551,541**	**$ 599,388**

Source: Maxim Group estimates

1. We model commercialization of NEO212 in the US in 2029 for recurrent glioblastoma (GBM) patients.
2. We assume US annual incidence of ~14,000 GBM patients, including ~13,300 recurrent glioblastoma patients following progression after surgery, radiation, and temozolomide.
3. We assume US pricing of $175K per patient annually, benchmarked against current therapies in glioblastoma, including temozolomide and bevacizumab, with a 5% annual price increase.
4. We apply a 70% revenue-risk adjustment based on stage of development and clinical trial risk.

NEO212 recurrent glioblastoma (GBM) market model, US

NEO212, recurrent glioblastoma (US)	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E	2034E	2035E	2036E
Incidence of glioblastoma	14,000	14,210	14,423	14,639	14,859	15,082	15,308	15,538	15,771	16,007	16,248	16,491
% of patients failing TMZ (90%)	13,300	13,500	13,702	13,908	14,116	14,328	14,543	14,761	14,982	15,207	15,435	15,667
MGMT unmethylated - poor/no response to TMZ (50%)	7,000	7,105	7,212	7,320	7,430	7,541	7,654	7,769	7,885	8,004	8,124	8,246
Market Penetration					5.00%	5.00%	15.00%	20.00%	30.00%	40.00%	50.00%	60.00%
Total Patients Treated					706	716	2,181	2,952	4,495	6,083	7,718	9,400
Cost of Treatment					175,000	183,750	192,938	202,584	212,714	223,349	234,517	246,243
Increase in Cost					5%	5%	5%	5%	5%	5%	5%	5%
Total revenue ('000)					$ 123,516	$ 131,637	$ 420,878	$ 598,067	$ 956,085	$ 1,358,597	$ 1,809,906	$ 2,314,688
Risk adjustment					70%	70%	70%	70%	70%	70%	70%	70%
Total Revenue ('000)					**$ 37,055**	**$ 39,491**	**$ 126,263**	**$ 179,420**	**$ 286,825**	**$ 407,579**	**$ 542,972**	**$ 694,406**

Source: Maxim Group estimates

VALUATION

We model NEO100 for recurrent IDH1-mutant high-grade glioma and NEO212 for recurrent glioblastoma (GBM), both in 2029. We apply 70% revenue risk adjustments based on the stage of development and clinical trial risk. A 30% discount is applied to the Free Cash Flow, Discounted EPS, and Sum-of-the-Parts Models, which are equally weighted to derive a 12-month price target of $20.

Free Cash Flow Model.

Average	$20

DCF Valuation Using FCF (mln):

	2025	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035	2036
units ('000)	**2025E**	**2026E**	**2027E**	**2028E**	**2029E**	**2030E**	**2031E**	**2032E**	**2033E**	**2034E**	**2035E**	**2036E**
EBIT	(62,146)	(16,851)	(18,006)	(24,921)	18,654	30,142	115,363	211,908	391,776	563,703	758,506	898,623
Tax Rate	0%	0%	0%	0%	0%	0%	0%	0%	0%	2%	5%	8%
EBIT (1-t)	(62,146)	(16,851)	(18,006)	(24,921)	18,654	30,142	115,363	211,908	391,776	552,429	720,580	826,733
CapEx	(500)	-	-	-	-	-	-	-	-	-	-	-
Depreciation	-	-	-	-	-	-	-	-	-	-	-	-
Change in NWC												
FCF	(62,646)	(16,851)	(18,006)	(24,921)	18,654	30,142	115,363	211,908	391,776	552,429	720,580	826,733
PV of FCF	(81,440)	(16,851)	(13,850)	(14,746)	8,491	10,553	31,071	43,902	62,436	67,722	67,950	59,970

Discount Rate	30%	
Long Term Growth Rate	2%	
Terminal Cash Flow 2036E	3,011,670	
Terminal Value YE2036E	218,461	
NPV (2026 - 2036)	570,556	
NPV-Debt		
Shares out ('000)	34,119	2036E
NPV Per Share	$18	

Source: Maxim Group estimates

Discounted-EPS Model.

Current Year	2026
Year of EPS	2036
Earnings Multiple	12
Discount Factor	30%
Selected Year EPS	24.23
NPV	$21

Source: Maxim Group estimates

	Discount Rate and Earnings Multiple Varies, Year is Constant						
	21.09	**5%**	**10%**	**15%**	**20%**	**25%**	**30%**
Earnings Multiple	**5**	74.38	46.71	29.95	19.57	13.01	8.79
	8	119.01	74.74	47.92	31.31	20.81	14.06
	10	148.76	93.42	59.90	39.13	26.02	17.58
	12	178.51	112.11	71.87	46.96	31.22	21.09
	15	223.14	140.13	89.84	58.70	39.03	26.37
	20	297.52	186.84	119.79	78.27	52.04	35.15
	25	371.90	233.55	149.74	97.84	65.05	43.94
	30	446.27	280.26	179.69	117.40	78.05	52.73

Sum-of-the-Parts Model.

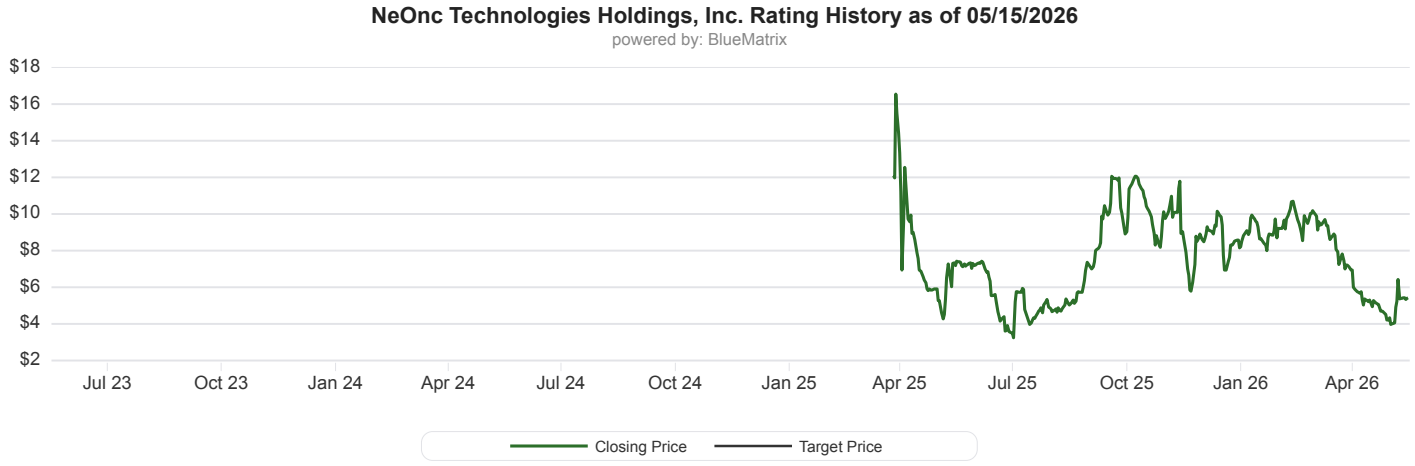
NeOnc Technologies Holdings, Inc.	LT Gr	Discount Rate	Yrs to Mkt	% Success	Peak Sales (MM's)	Term Value
NEO100, recurrent IDH1-mutant high-grade glioma (US)	5%	30%	3	50%	$599	$2,398
NPV						$10.22
NEO212, recurrent glioblastoma (US)	5%	30%	3	50%	$694	$2,778
NPV						$12
Net Margin						64%
MM Shrs OS (2036E)						34
Total						$22

Source: Maxim Group estimates

NeOnc Technologies Holdings, Inc. Income Statement ($000)																				
YE December 31	2025A	1Q26A	2Q26E	3Q26E	4Q26E	2026E	1Q27E	2Q27E	3Q27E	4Q27E	2027E	2028E	2029E	2030E	2031E	2032E	2033E	2034E	2035E	2036E
Revenue:																				
NEO100, recurrent IDH1-mutant high-grade glioma (US)													27,901	60,643	98,855	179,051	311,335	422,929	551,541	599,388
NEO212, recurrent glioblastoma (US)													37,055	39,491	126,263	179,420	286,825	407,579	542,972	694,406
Net revenue	-	-	-	-	-	-	-	-	-	-	-	-	64,956	100,134	225,118	358,472	598,160	830,508	1,094,513	1,293,794
Collaborative revenue:																				
Revenues	40																			
Other Income																				
Total Collaborative Revenue	**40**	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total Revenue	**40**	-	-	-	-	-	-	-	-	-	-	-	**64,956**	**100,134**	**225,118**	**358,472**	**598,160**	**830,508**	**1,094,513**	**1,293,794**
Gross Margins:																				
Cost of Goods Sold	-	-	-	-	-	-	-	-	-	-	-	-	12,991	20,027	45,024	71,694	119,632	166,102	218,903	258,759
%Gross Margin													80%	80%	80%	80%	80%	80%	80%	80%
Gross Profit	40	-	-	-	-	-	-	-	-	-	-	-	51,965	80,107	180,095	286,777	478,528	664,406	875,610	1,035,035
Operating Expenses:																				
Research and Development	3,638	1,286	1,135	1,230	1,277	4,730	1,741	1,816	1,968	2,043	7,568	11,351	17,027	25,541	28,095	30,904	33,994	37,394	41,133	45,247
%R&D																				
Selling, General and Administrative	7,299	1,677	1,927	2,088	2,168	8,029	2,401	2,505	2,714	2,818	10,438	13,569	16,283	24,425	36,637	43,965	52,758	63,309	75,971	91,166
%SG&A																				
Stock based compensation	35,555	2,732				2,732														
Advisory fees	11,788	1,360				1,360														
Total Operating Expense	**58,280**	**7,056**	**3,062**	**3,317**	**3,445**	**16,851**	**4,141**	**4,321**	**4,681**	**4,862**	**18,006**	**24,921**	**33,310**	**49,966**	**64,732**	**74,869**	**86,752**	**100,703**	**117,105**	**136,412**
Total Expenses	58,280	7,056	3,062	3,317	3,445	16,851	4,141	4,321	4,681	4,862	18,006	24,921	46,302	69,992	109,756	146,563	206,384	266,805	336,007	395,171
Operating Income (Loss)	(58,240)	(7,056)	(3,062)	(3,317)	(3,445)	(16,851)	(4,141)	(4,321)	(4,681)	(4,862)	(18,006)	(24,921)	18,654	30,142	115,363	211,908	391,776	563,703	758,506	898,623
	-																			
Interest income	328	5				5														
Amortization of debt issuance costs	(1,110)	(192)				(192)														
Interest expense	(2,505)	(983)				(983)														
Other income (expense)	71	(645)				(645)														
Gain (loss) on change in fair value of derivative liability	(690)	3				3														
Grant income		47				47														
	-					-														
Total Other Income	**(3,906)**	**(1,764)**	-	-	-	**(1,764)**	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Pretax Income	(62,146)	(8,820)	(3,062)	(3,317)	(3,445)	(18,616)	(4,141)	(4,321)	(4,681)	(4,862)	(18,006)	(24,921)	18,654	30,142	115,363	211,908	391,776	563,703	758,506	898,623
Taxes on income																		11,274	37,925	71,890
Tax Rate																		2%	5%	8%
GAAP Net Income (Loss)	(62,146)	(8,820)	(3,062)	(3,317)	(3,445)	(18,616)	(4,141)	(4,321)	(4,681)	(4,862)	(18,006)	(24,921)	18,654	30,142	115,363	211,908	391,776	552,429	720,580	826,733
Foreign currency translation loss																				
Total comprehensive loss	(62,146)	(8,820)	(3,062)	(3,317)	(3,445)	(18,616)	(4,141)	(4,321)	(4,681)	(4,862)	(18,006)	(24,921)	18,654	30,142	115,363	211,908	391,776	552,429	720,580	826,733
GAAP-EPS	(3.20)	(0.38)	(0.11)	(0.12)	(0.13)	(0.72)	(0.15)	(0.15)	(0.16)	(0.16)	(0.62)	(0.84)	0.58	0.90	3.45	6.31	11.62	16.32	21.20	24.23
GAAP-EPS (Dil)	(3.20)	(0.38)	(0.11)	(0.12)	(0.13)	(0.72)	(0.15)	(0.15)	(0.16)	(0.16)	(0.62)	(0.84)	0.58	0.90	3.45	6.31	11.62	16.32	21.20	24.23
Wgtd Avg Shrs (Bas) - '000s	19,399	23,293	26,817	26,844	26,870	25,956	26,897	29,424	29,454	29,483	28,815	29,557	32,303	33,310	33,443	33,577	33,712	33,847	33,982	34,119
Wgtd Avg Shrs (Dil) - '000s	19,399	23,293	26,817	26,844	26,870	25,956	26,897	29,424	29,454	29,483	28,815	29,557	32,303	33,310	33,443	33,577	33,712	33,847	33,982	34,119

Source: Company reports and Maxim Group estimates

DISCLOSURES



Maxim Group LLC Ratings Distribution			As of: 05/17/26
		% of Coverage Universe with Rating	% of Rating for which Firm Provided Banking Services in the Last 12 months
Buy	Fundamental metrics and/or identifiable catalysts exist such that we expect the stock to outperform its relevant index over the next 12 months.	**85%**	**51%**
Hold	Fundamental metrics are currently at, or approaching, industry averages. Therefore, we expect this stock to neither outperform nor underperform its relevant index over the next 12 months.	**15%**	**48%**
Sell	Fundamental metrics and/or identifiable catalysts exist such that we expect the stock to underperform its relevant index over the next 12 months.	**0%**	**0%**
	**See valuation section for company specific relevant indices*		

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The research analyst(s) primarily responsible for the preparation of this research report have received compensation based upon various factors, including the firm's total revenues, a portion of which is generated by investment banking activities.

Maxim Group makes a market in NeOnc Technologies Holdings, Inc.

Maxim Group expects to receive or intends to seek compensation for investment banking services from NeOnc Technologies Holdings, Inc. in the next 3 months.

NTHI: We use the BTK (ARCA Biotechnology Index) as the relevant index for NeOnc Technologies Holdings, Inc.

Valuation Methods
NTHI: We model commercialization of NEO100 in recurrent IDH1-mutant high-grade glioma and NEO212 in recurrent glioblastoma (GBM). We apply a revenue risk adjustment based primarily on stage of development and clinical trial risk. A discount rate is then applied to the free cash flow, discounted EPS, and sum-of-the-parts models, which are equally weighted to derive a 12-month price target.

Price Target and Investment Risks

NTHI: Aside from general market and other economic risks, risks particular to our price target and rating for NeOnc Technologies Holdings, Inc. include: (1) the regulatory and clinical risk associated with product development; (2) the rate and degree of progress of product development; (3) the rate of regulatory approval and timelines to potential commercialization of products; (4) the level of success achieved in clinical trials; (5) the requirements for marketing authorization from regulatory bodies in the United States and other countries; (6) the liquidity and market volatility of the company's equity securities; (7) regulatory and manufacturing requirements and uncertainties; (8) product and technology developments by competitors, potentially with more resources and commercial infrastructure; (9) inability, of product(s), if approved, to gain adequate market share and maintain adequate revenue growth; (10) the ability of the company to maintain its exchange listing; (11) NeOnc is a controlled company, with insiders controlling over 50% of the voting rights; (12) negative shareholders' equity poses a risk to NASDAQ Global Market listing compliance; while currently compliant, failure to maintain compliance could lead to delisting; (13) the ability to access capital to fund operations, if the company cannot secure sufficient capital, the company could cease operations.

RISK RATINGS

Risk ratings take into account both fundamental criteria and price volatility.

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