BIOTECHNOLOGY EQUITY RESEARCH

NeOnc Technologies (NTHI, Buy, $15 PT)

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POHtentiating Better CNS Uptake to Improve Treatment of GBM and Aggressive Gliomas. Initiating at Buy with a $15 PT.

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WHAT YOU SHOULD KNOW: NeOnc is focused on improving the treatment of CNS tumors. The company is taking a multi-pronged approach by improving CNS delivery of agents with established antitumor activity such as temozolomide (TMZ) and also by developing novel agents active against hard-to-treat CNS tumors (including metastases). The key catalysts for the stock are incremental data sets supporting activity of the two lead drugs in difficult-to-treat patients and are expected later in 2026 and 2027. The key ingredient in NeOnc's efforts is the natural product Perillyl Alcohol (POH) that is itself active as an anti-cancer agent via multiple validated pathways (including the induction of ER-stress) and **also helps other agents cross the blood brain barrier (BBB) to reach the CNS**. The current lead products are NEO100 and NEO212, both of which have shown clinical activity against hard-to-treat CNS tumors like GBM and aggressive gliomas. NEO212 is the biggest driver of value and consists of TMZ, the workhorse drug for brain tumors, chemically conjugated to POH. The resultant molecule retains antitumor activity and is taken up into the brain at 3X the level of TMZ. Consistent with this more-is-better approach, the company recently reported antitumor effects in advanced GBM patients treated with NEO212 (patients that had failed TMZ). NEO100 is the most advanced candidate and is POH itself reformulated for nasal delivery in an effort to reduce systemic toxicities and has produced an ongoing human data set confirming activity. On the safety front, both drugs look remarkably safe for chemotherapies - a significant driver of uptake for these fragile patients. **Pipeline:** going forward, we expect the company to leverage the POH platform to develop novel therapies for CNS tumors that are broadly active against tumors, but unable to enter the CNS in unmodified form. These novel therapies have the potential to improve the treatment of primary brain tumors and also the much larger market of secondary brain tumors that start systemically but have escaped to the CNS.

- **NEO212 looks like a straightforward shot at a valuable drug.** NEO212 is a chemical fusion of POH to TMZ that retains TMZ's antitumor activity and is taken up into the CNS at about 3X the rate of TMZ (an approved drug for CNS tumors). In addition, as an alkylator (a broadly active class of antitumor agent) the drug is likely active against secondary tumors that have metastasized to the CNS. The math seems fairly simple, if TMZ is active in treating GBM and other CNS tumors, 3X the level of the TMZ warhead in the CNS should be more active. A recent P1 trial showed clear indications of antitumor effects in serious patients.
- **NEO100: two pipelines and a product.** NEO100 is interesting on its own - it's a very safe approach to treat aggressive gliomas with a 30-month PFS response seen in patients that would otherwise expect about one-third of that duration (see below). A final readout from an ongoing P2 trial is expected in 2H26.
- **The POH-conjugation pipeline.** The world of drug development for diseases inside the CNS is dominated by one key hurdle: getting the active therapeutic into the CNS. The rules for CNS entry are partially known (keep molecules small and a little greasy), but the implementation is tricky with almost 50% of compounds that enter human testing failing due to low CNS uptake. POH as a mixture (nasal delivery) or as a chemical conjugate could improve that situation and make NeOnc an attractive partner for CNS-targeting medicine, in our view.
- **Valuation:** We value NTHI using a DCF analysis (16% discount rate, 0.5% TG).

Company Data	
Closing Price	$4.95
Price Target	$15.00
Market Cap (M)	$117.84
Shares Out (M)	23.81
Avg Daily Vol-3 Months (M)	0.05

Revisions	Previous	Current
Rating	-	Buy
Price Target	-	$15.00
FY26E NI	-	(11.47)
FY27E NI	-	(12.04)
FY26E EPS	-	(0.53)
FY27E EPS	-	(0.53)

Net Income			
FY Dec	2025A	2026E	2027E
Q1	(38.00)	(2.95)	(3.09)
Q2	(5.68)	(2.29)	(2.40)
Q3	(8.62)	(1.98)	(2.08)
Q4	(9.85)	(4.26)	(4.47)
FY NI	**(62.15)**	**(11.47)**	**(12.04)**

EPS (GAAP)			
FY Dec	2025A	2026E	2027E
Q1	(2.10)	(0.14)	(0.14)
Q2	(0.30)	(0.11)	(0.11)
Q3	(0.45)	(0.09)	(0.09)
Q4	(0.46)	(0.19)	(0.19)
FY EPS	**(3.20)**	**(0.53)**	**(0.53)**
FY P/E	-	-	-

Source: FactSet, BTIG Estimates and Company Documents reported as $ currency.
FY = Fiscal Year **CY** = Calendar Year

Investment Thesis

NeOnc is focused on improving the treatment of brain tumors. The company is taking a multi-pronged approach by improving CNS delivery of agents that are known to be active against CNS tumors such as temozolomide (TMZ) and also by developing novel agents active against hard-to-treat CNS tumors (including metastases). Key catalysts for the stock are incremental data sets supporting activity of the two lead drugs in difficult-to-treat CNS tumor patients expected in later 2026 and 2027. The key ingredient in NeOnc's efforts is the natural product Perillyl Alcohol (POH) that is itself active as an anti-cancer agent via multiple validated pathways (including the induction of ER-stress) and **also helps other agents cross the blood brain barrier (BBB) to reach the CNS**.

Upcoming Catalysts

2H26 - P2 trial for NEO100 update.

MY26 - FDA agreement on P2/3 program to support accelerated approval of NEO212 in recurrent GBM.

2H26 - P2/3 trial of NEO212 starts in recurrent GBM and possibly metastatic brain tumors.

Base Case Assumptions: **$15 Price Target**

P2 trial for NEO100 updates in 2H26 to show continued signs of treatment effects in IDH1-mutated astrocytoma.

NEO-100 for recurrent gliomas - 40% PoS, 2030 market entry, 60% peak penetration in malignant gliomas in temozolomide resistant brain tumors with an IDH1 mutation, $15k per year

NEO-212 for glioblastoma - 50% PoS, 2030 market entry, 60% peak penetration in malignant glioblastomas in temozolomide resistant brain tumors, $15k per year

NEO-212 for 3L brain metastases- 30% PoS, 2031 market entry, 60% peak penetration in metastatic brain tumors from breast cancer, colorectal cancer, NSCLC, and melanoma, $15k per year

Upside Scenario

P2 trial for NEO100 updates in 2H26 to leave little doubt of the drug's effects in IDH1-mutated astrocytoma. Radiographic evidence in a few patients is very clearly supportive of treatment effects.

P2/3 program to support accelerated approval of NEO212 is approved by the FDA based on treatment effects in recurrent GBM.

The company signs a significant partnership for POH in or outside oncology.

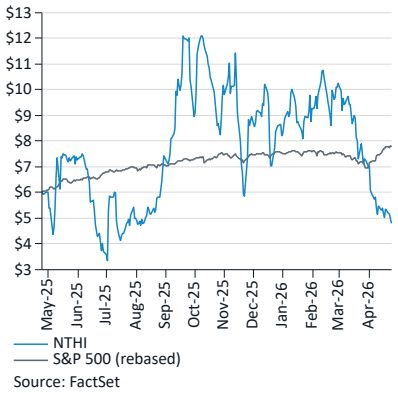
Progress on the POH-mixture side of the franchise.

Downside Scenario

Lack of execution on NEO212 trial planning.

No further signs of efficacy and/or safety issues emerging with ongoing P2 trial in NEO100.

Price Performance



Source: FactSet

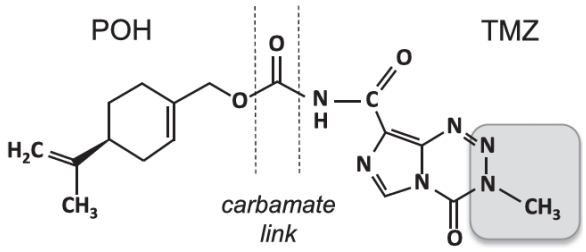
Company Description

NeOnc is focused on improving the treatment of brain tumors. The company is taking a multi-pronged approach by improving CNS delivery of agents that are known to be active against CNS tumors such as temozolomide (TMZ) and also by developing novel agents active against hard-to-treat CNS tumors (including metastases). Key catalysts for the stock are incremental data sets supporting activity of the two lead drugs in difficult-to-treat CNS tumor patients expected in later 2026 and 2027. The key ingredient in NeOnc's efforts is the natural product Perillyl Alcohol (POH) that is itself active as an anti-cancer agent via multiple validated pathways (including the induction of ER-stress) and also helps other agents cross the blood brain barrier (BBB) to reach the CNS.

Key Investment Considerations

NEO212 looks to be reduced-risk. NEO212 is a DNA alkylating agent related to temozolomide (TMZ). TMZ is a pro-drug of an active anti-tumor agent called AIC (4-amino-5-imidazole carboxamide) and the workhorse drug for treating serious brain tumors including GBM and aggressive gliomas. NEO212 is modified to improve on the modest BBB penetration of TMZ resulting in about 3X the concentration of the same active anti-tumor agent in the CNS (it's essentially a better AIC pro-drug). Importantly, the modifications that improve NEO212 relative to TMZ (Exhibit 1) don't hinder formation of the active agent or reduce the ability of delivered AIC to damage DNA in rapidly dividing tumor cells (the therapeutic mechanism of TMZ). As a result, NEO212 looks like an approach to **deliver more of an active anti-tumor drug to the CNS to treat CNS tumors**. This setup seems relatively low-risk.

Exhibit 1. NEO212 is a modified form of TMZ



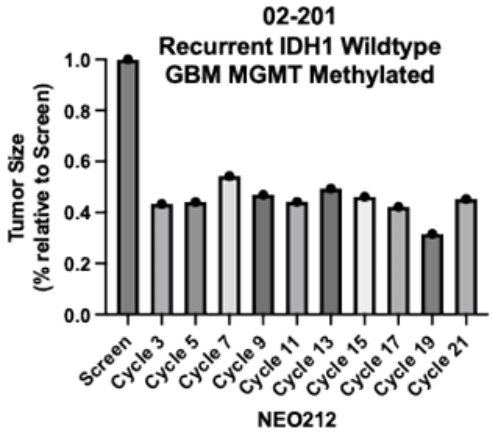
Source: NTHI company reports

Clinical data suggest activity. NEO212 is in the early stages of clinical testing, but we have already seen clear signs of anti-tumor activity in very advanced patients not expected to benefit from currently approved therapeutics. The recent P1 readout was principally designed to determine a go-forward dose for larger and potentially registrational trials – and the go-forward dose was successfully defined. The trial also confirmed the excellent safety of NEO212 where only the last cohort showed meaningful toxicity (defining the maximum tolerated dose). Two patients, that were both highly treatment-experienced, showed either tumor regression (Exhibit 2) or stable disease after periods of tumor growth on prior drug regimens. For P1 oncology trials, that type of result is about as good as it gets.

NEO212: more should be better. The ability of TMZ to effectively treat tumors like GBM is impaired by cells that acquire high levels of the DNA correcting enzyme O6-methylguanine-DNA methyltransferase (MGMT) which undoes the DNA modifications caused by TMZ and its active metabolite MTIC (the basis of anti-tumor activity in dividing tumor cells). The activity of the MGMT enzyme that corrects the anti-tumor effects of MTIC is termed a suicide reaction because MGMT is inactivated in the reaction. This type of resistance mechanism can, in principle, be overcome by either producing more MTIC (using up the enzyme) or stopping the replenishment of MGMT (less enzyme synthesis). Data to date confirm NEO212 works by both mechanisms and is active in TMZ resistant tumors. Preclinical studies demonstrate that NEO212 effectively promotes the degradation of MGMT, the crucial DNA repair enzyme that causes TMZ resistance. In addition, the

POH component of NEO212 causes ER-stress and reduces the replacement synthesis of MGMT. As a result, we see the chances that NEO212 is a better drug than TMZ at fighting aggressive brain tumors as relatively high.

Exhibit 2. NEO212: P1 readout suggested activity in difficult-to-treat patients.



Source: NTHI company reports

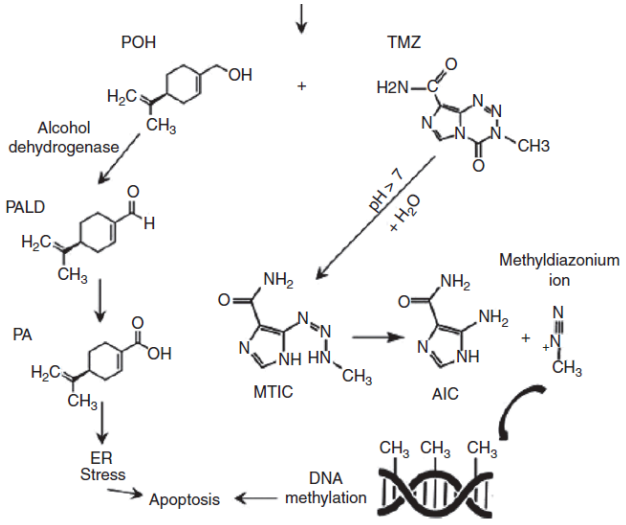
NEO212: mechanism also looks safer than TMZ. A key attribute of any anti-tumor drug is that it is safe to use and is more toxic to tumor cells than to healthy tissue. Much of this safety window (termed therapeutic margin) is based on the rapid division of tumor cells relative to normal mature human cells. TMZ has a therapeutic window, but the drug's dosing is constrained by its toxicity toward cells of the bone marrow (anemia and cytopenias). Relative to TMZ, NEO212 produces less MTIC in the blood where it is toxic and conversion to MTIC occurs principally after the drug has entered cells. This property probably explains the low levels of anemia seen with NEO212.

The other half of NEO212 may help fight tumors. NEO212 is ushered into the CNS via the POH side of the molecule. This uptake mediator also has anti-tumor activity and is being developed independently as NEO100 (see below). NEO100 has multiple anti-tumor activities including inhibition of mutant Ras oncoproteins and the development of endoplasmic reticulum (ER) stress in target cells. The former activity is intriguing as mutant Ras enzymes are a driver of many tumor types. Similarly, ER stress is a defined feature of tumor cells and is an apoptotic signal in most cells – so further driving this pathway should help tumor cells die. In addition, ER stress reduces protein expression in tumor cells resulting in less MGMT replenishment. This latter activity underscores the ability of NEO212 to function in tumor cells that are resistant to TMZ.

Secondary CNS tumors – a growth opportunity. NEO212 is an alkylating agent meaning it works by damaging DNA and forcing the cell to repair the damage before the next round of DNA replication or suffer the consequences of adding mutations to the genome. Because cancer is inherently a disease of too much and uncontrolled cell division – essentially all cancers are more sensitive to agents of this type. Some more than others, but most tumors have some susceptibility and a drug like NEO212 that produces higher levels of DNA damage (more MTIC in CNS and around tumor cells) and is safer to use (less MTIC in bone marrow) could be a very powerful addition to treating

secondary CNS tumors. As described below – this class of tumors is heterogeneous due to varied sites of origin (melanoma and NSCLC are common) and difficult to treat as few drugs can cross the BBB and reach a tumor once the neoplasm has spread to the CNS. As a result, NEO212 looks very promising in this large and poorly served treatment landscape and we believe any supportive data could be a nice positive for the NeOnc story.

Exhibit 3. The dual mechanism of NEO212



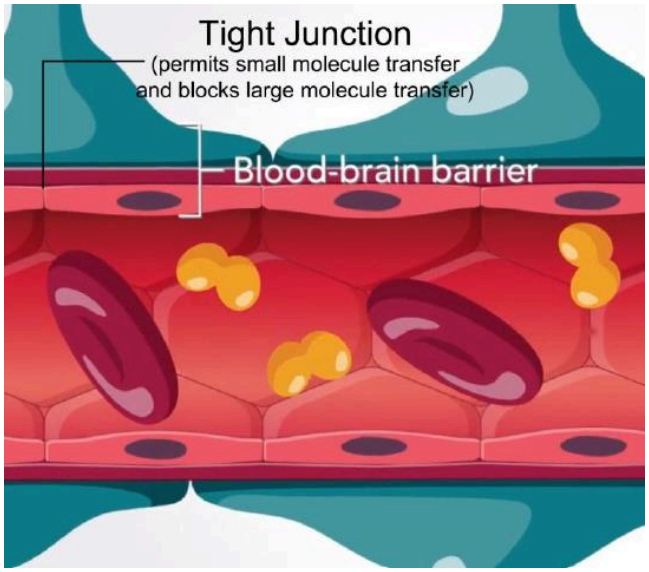
Source: NTHI company reports

NEO100 as a standalone drug. NEO100 is a cGMP-manufactured, highly purified (>99%) version of the natural monoterpene POH. As a therapeutic, POH has been tested as an oral molecule but has an unacceptable tolerability profile. To circumvent these issues, NEO100 is administered non-invasively via nebulizer and nasal mask four times daily to bypass the blood-brain barrier through direct nose-to-brain transport. At the cellular level, NEO100 operates through multiple convergent anti-tumor mechanisms, including ER stress induction, cell cycle arrest, Bax upregulation, and suppression of mTOR and Ras signaling. Importantly, the molecule retains cytotoxic activity independent of MGMT expression status, meaning it remains active in both TMZ-sensitive and TMZ-resistant gliomas. In the completed Phase I portion of an ongoing P1/2 trial (NCT02704858, n=12 recurrent GBM), NEO100 demonstrated an exceptional safety profile with no severe adverse events at any dose level. The initial efficacy readout was also interesting and compared favorably to current salvage options: PFS-6 of 33%, OS-12 of 55%, and a median OS of 15 months against a historical benchmark of 6–10 months. Intratumoral delivery was confirmed pharmacologically when both POH and its metabolite perillic acid were detected in resected brain tumor tissue.

Ongoing clinical trials of NEO100. With Phase I complete and a well-defined efficacy signal established in IDH1-mutant patients, NeOnc is actively advancing NEO100 through a Phase 2a clinical trial targeting patients with recurrent Grade III and Grade IV IDH1-mutant glioma — the subgroup in which all four patients surviving beyond 24 months were concentrated in Phase I. Holding both FDA Orphan Drug and Fast Track designation, NEO100 is currently enrolling a Phase 2a trial targeting IDH1-

mutant recurrent Grade III/IV glioma patients across up to 12 U.S. sites, with a Phase 2b/3 randomized study also in development. The Phase 2a trial is enrolling up to 30 patients, with all patients receiving 288 mg of NEO100 intranasally four times daily, equivalent to the highest and best-tolerated dose from Phase I. The trial is designed to generate the efficacy data necessary to pursue FDA approval in this defined IDH1-mutant population. Beyond the lead indication, NeOnc has initiated a second NEO100 program targeting meningioma (NEO100-02) and has commenced enrollment for a pediatric brain tumor application (NEO100-03), meaningfully expanding the long-term addressable indication set.

Exhibit 4. Crossing the BBB is a complex process which POH solves.



Source: NTHI company reports

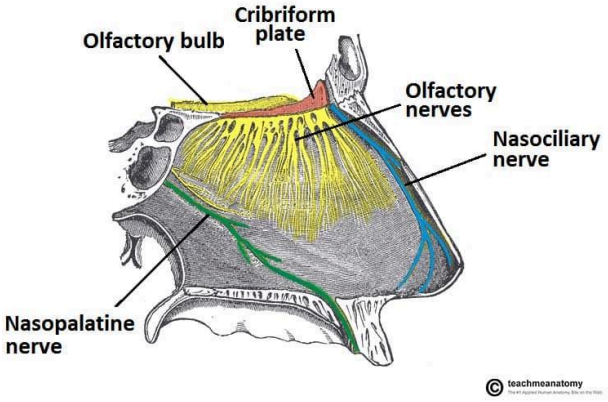
POH chaperones other molecules across the BBB. NeOnc is a small company and has plenty on its plate to develop NEO212 and NEO100 as quickly as possible. However, the company has already generated interesting data supporting the ability of POH to usher other chemotherapies into the CNS where they might be active. The mechanism is again only partially understood, but POH transiently disrupts biological membranes without causing permanent damage. The resulting delivery window allows other drugs that would normally be blocked by the tight BBB junctions between the epithelial cells that line the blood vessels in the CNS. To date, NeOnc has shown this effect with the powerful drug Velcade.

Risks to our investment thesis

Commercial and political risks. For the foreseeable future, novel oncology therapies are expected to be expensive and therapies for childhood diseases such as pediatric GBM to be particularly expensive. To date, these costs have been acceptable, both due to the overall price tag of the treatment (cost times number of treatments) and the "replacement of other care" (a fulltime caregiver) these therapies can replace. In addition, manufacturers generally argue that high drug prices are essential to reimburse for the significant development costs for therapies treating very sick patients. All this could easily change with a single executive order capping treatment prices.

Competitive risk. The biotech sector has long recognized the commercial potential of a powerful new drug to treat GBM. Efforts to date have been largely unsuccessful, but we certainly expect others to keep plugging away. This competition may become more intense as the rules for drugs that cross the BBB are better understood.

Exhibit 5. Nasal delivery to the brain may be uneven.



Source: NTHI company reports

Technical risk: distribution of olfactory transport. There is a chance that the ability of POH to deliver payload to the CNS might be limited to areas of the CNS near the end of the olfactory nerve highway. This idea might leave a drug like NEO212 or any POH-shuttled molecule active only in limited regions of the brain. This phenomenon, if observed, will make clinical trials more complex with some patients failing treatment due to the location of their tumors rather than to the activity of the drug. In other words, the approach may not work as well for posterior tumors or tumors deep in the brain.

Technical risk: complex dosing. Dose determination is always complex in drug development and NeOnc's drugs are unlikely to be an exception. This issue could be the greatest risk for pipeline molecules where the level of POH and active payload might both need to be optimized. In addition, dosing may be made more complex by the potentially uneven distribution of NeOnc's drugs delivered via nasal inhalation.

Mucosal safety with repeated dosing. The bar for accepting mild inconveniences, such as a runny nose, is likely high for later-stage brain cancer patients. However, if irritation gets too serious, NeOnc's approach might not be viable. Analogous to the oral mucositis that limits the dosing of

many cancer drugs, NeOnc's drugs can't be too painful to take over prolonged periods. This risk seems worth watching as NEO100's transient tight junction opening has been acceptable on the safety front to date. However, chronic and repeated disruption of nasal epithelial integrity could result in safety issues in some patients, and these effects might be much more intense in the case of other payloads.

Regulatory complexity. NeOnc's combination of novel nasal formulations with a delivery device always has the risk of attracting regulatory scrutiny. We expect the company will focus on well-characterized devices, but the history of drug-device combinations may give some investors pause.

NeOnc Pipeline and Upcoming Events

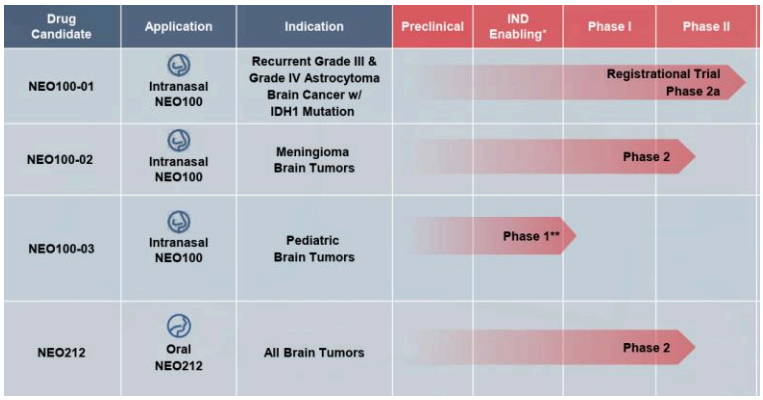
The NeOnc pipeline is based on the ability of Perillyl alcohol to move into the CNS and to deliver other molecules either as mixtures or as covalent conjugates (NEO212). Both lead molecules have shown anti-tumor activity in highly treatment-experienced patients that had exhausted their available treatment options. These clear signs of activity de-risk the story and make the key deliverable for the story to be to show these effects in more patients and to define optimal tumor types for upcoming trials.

Accelerated approval framework. Expected later in 2026 is an agreement with the FDA to define a P2 trial in GBM that could serve as the basis for approval. These types of P2/3 trials are never guaranteed to support early action, but agreement at least makes clear the types of trials that could provide compelling evidence of drug activity. Although not final, the company expects some type of basket trial with three arms each with about 20 patients. This basket trial would be used to identify the strongest signal.

- Arm 1 – 1st recurrence GBM patients (2nd line)
- Arm2 – 1L patients expected to be temozolomide resistant via the MGMT pathway (1st line in an underserved population).
- Arm 3 – Patients after Avastin failure (3rd line).

Based on the best signal (expected around YE26), the company expects to add another 120 patients to one arm in a randomized arm for accelerated approval. This first 140 or so patients would be the basis of a discussion with the FDA to agree on final sizing and the trial might then be further expanded for accelerated approval (around 200 total patients in the chosen arm). Going forward, we would expect the agency would require a second "confirmatory" P3 trial where the endpoint might be survival.

Exhibit 6. The NeOnc Pipeline



Source: NTHI company reports

NeOnc is running several small trials that could help de-risk the story.

NEO100-01 (IDH1-Mutant Glioma): Phase II clinical data expected 2H 2026. This trial has completed enrollment of 25 patients and will release data about five months after the enrollment was completed. This trial is large enough to give a meaningful treatment signal.

NEO100-02 is a smaller trial to test the drug's ability to fight tumors in deeper regions of the brain. We see this trial as very experimental and timelines are

broad with data in 2027. The trial had enrolled seven of a planned 30 patients at the end of 2025.

NEO100-03 (Pediatric pHGG): Enrollment expected to begin in Q1 2026 and six patients are expected to be enrolled. This trial is important as pediatric GBM tumors are a significant market. This drug will greatly test the safety properties of NEO100.

Valuation and Market Models

We value NTHI using a DCF analysis and assuming a 16% discount rate and a 0.5% terminal growth rate. Our DCF analysis captures revenue projections out to 2040 from the NEO100 and NEO212 programs.

The positive terminal growth rate reflects the company's potential to develop a broad pipeline based on POH's ability to get payloads into the CNS.

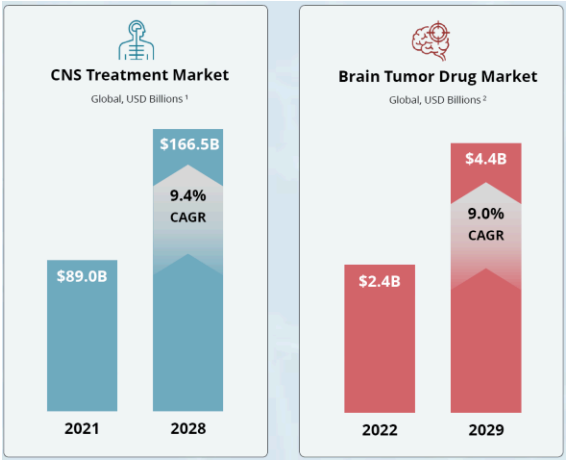
Exhibit 7. Comp Table: Oncology companies with P1 or P2 programs

Company Name	Ticker	Price	Market cap (M)	EV (M)	Shares out (M)	Shares out diluted (M)	BTIG Rating/TP
NeOnc Technologies Holdings	NTHI	$7.01	$180.20	$186.10	22.7	21.7	Buy, $15
Verastem Inc	VSTM	$5.30	$576.30	$447.60	87.8	69.3	Buy, $19
ImmunityBio	IBRX	$7.67	$8,986.00	$8,744	1,028.1	919.0	Buy, $13
Plus Therapeutics	PSTV	$0.16	$28.30	$17.90	171.6	77.8	Not Rated
Pyxis Oncology	PYXS	$1.46	$94.40	$26	62.8	62.1	Not Rated
Prelude Therapeutics	PRLD	$3.42	$291.10	$187.90	48.3	77.0	Not Rated
Celldex Therapeutics	CLDX	$31.72	$2,159.30	$1,645.10	66.6	66.4	Not Rated

Source: BTIG Research estimates, Factset

Note: IBRX and VSTM are covered by our colleague Jeet Mukherjee

Exhibit 8. Market opportunity



Source: NTHI company reports

Exhibit 9. NTHI Valuation

DCF Valuation		
Discount rate		16.0%
Terminal growth rate		0.5%
PV of FCF (MM)	$	336
Cash and equivalents 1Q27E (MM)	$	10
Debt 1Q27E (MM)	$	-
Market value (MM)	$	346
Shares outstanding 1Q27E (MM)		22
Price per share	$	15.40

Discount Rate \ Terminal Growth Rate	-1.0%	-0.5%	0.0%	0.5%	1.0%	1.5%	2.0%
14.5%	20.28	21.30	22.38	23.55	24.81	26.16	27.61
15.0%	17.64	18.53	19.49	20.51	21.60	22.78	24.05
15.5%	15.28	16.07	16.91	17.81	18.76	19.79	20.89
16.0%	13.17	13.87	14.61	15.40	16.24	17.14	18.10
16.5%	11.29	11.90	12.56	13.25	13.99	14.78	15.63
17.0%	9.60	10.15	10.72	11.34	11.99	12.69	13.43
17.5%	8.08	8.57	9.08	9.63	10.20	10.82	11.47

Source: BTIG Research estimates, NTHI company reports

NEO212 glioblastoma

- We assume a 50% probability of success.
- We estimate the current prevalence of primary brain tumors is approximately 108,810 patients in 2026 (Source: National Brain Tumor Society (NBTS) website).
- We assume that glioblastoma accounts for 49% of primary brain cancers.
- We assume that 50% of patients are ineligible for temozolomide and 90% of temozolomide-eligible patients either relapse or are refractory to the treatment.
- We assume market entry of NEO212 to be in 2030.
- We assume net prices per month to be $15,000 with six months of treatment per year, with an annual growth rate of 5%.
- We assume a peak market penetration of 60%.
- We assume a 30% royalty rate to NeOnc on ex-U.S. sales of NEO212.

Exhibit 10. NEO212 GBM Market Model

	2025	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035	2036	2037	2038	2039	2040
US Market																
Primary brain cancer Incidence		108,810	109,572	110,339	111,111	111,889	112,672	113,461	114,255	115,055	115,860	116,671	117,488	118,310	119,138	119,972
% growth		*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*
Addressable r/r Glioblastoma patients	-	13,062	13,153	13,245	13,338	13,431	13,525	13,620	13,715	13,811	13,908	14,005	14,103	14,202	14,302	14,402
% malignant	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*
% glioblastoma	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*
% ineligible for temozolomide	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*
% relapse/refractory to temozolomide	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*
Price per patient per month	-	-	-	-		$ 15,000	$ 15,750	$ 16,538	$ 17,364	$ 18,233	$ 19,144	$ 20,101	$ 21,107	$ 22,162	$ 23,270	$ 24,433
Treatment time (months)					6	6	6	6	6	6	6	6	6	6	6	6
% growth	-	-	-	-	-	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*
% penetration	-	-	-	-	*0%*	*12%*	*18%*	*30%*	*45%*	*51%*	*57%*	*60%*	*60%*	*60%*	*60%*	*60%*
					0	0.2	0.3	0.5	0.75	0.85	0.95	1	1	1	1	1
Gross-to-net adjustment					*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*
Glioblastoma Revenue (MM) (U.S.)	**-**	**-**	**-**	**-**	**$ -**	**$ 131**	**$ 207**	**$ 365**	**$ 579**	**$ 694**	**$ 820**	**$ 912**	**$ 964**	**$ 1,020**	**$ 1,078**	**$ 1,140**
EU Market																
Primary brain cancer Incidence	-	245,553	246,290	247,029	247,770	248,513	249,258	250,006	250,756	251,509	252,263	253,020	253,779	254,540	255,304	256,070
% growth		*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*
Addressable r/r Glioblastoma patients	-	29,477	29,565	29,654	29,743	29,832	29,921	30,011	30,101	30,192	30,282	30,373	30,464	30,556	30,647	29,121
% malignant	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*
% glioblastoma	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*	*49%*
% ineligible for temozolomide	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	
% relapse/refractory	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*
Price per patient per month	-	-	-	-	-	-	$ 11,250	$ 11,813	$ 12,403	$ 13,023	$ 13,674	$ 14,358	$ 15,076	$ 15,830	$ 16,621	$ 17,452
Treatment time (months)						6	6	6	6	6	6	6	6	6	6	6
% growth	-	-	-	-	-	-	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*
% penetration	-	-	-	-	-	*0%*	*6%*	*9%*	*15%*	*23%*	*26%*	*29%*	*30%*	*30%*	*30%*	*30%*
Gross-to-net adjustment						*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*
Glioblastoma Revenue (MM) (EU)	**-**	**-**	**-**	**-**	**-**	**$ -**	**$ 33**	**$ 52**	**$ 91**	**$ 143**	**$ 171**	**$ 201**	**$ 223**	**$ 235**	**$ 248**	**$ 247**
% Royalty to Nuvation						*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*
Total	-	-	-	-	-	131	240	417	669	837	991	1,114	1,188	1,255	1,326	1,387

Source: BTIG Research estimates

NEO212 secondary CNS tumors

- We assume a 30% probability of success.
- We assume market entry of NEO212 in brain metastases to be in 2031.
- We assume that the majority of tumor types that metastasize to the brain include breast cancer (in 40% of cases), NSCLC (18%), colorectal cancer (2%), and melanoma (7%). We are using death rates as a proxy for 3L+ patients.
- We assume market entry of NEO212 for secondary CNS tumors to be in 2031.
- We assume net prices per month to be $15,000 with six months of treatment per year, with an annual growth rate of 5%.
- We assume a peak market penetration of 60%.
- We assume a 30% royalty rate to NeOnc on ex-U.S. sales of NEO212.

Exhibit 11. NEO212 secondary CNS tumors model

	2025	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035	2036	2037	2038	2039	2040	2041	2042	2043	2044	2045
US Market																					
Breast cancer (ER- Her2+/TNBC) 3L incidence	12,651	12,740	12,829	12,919	13,009	13,100	13,192	13,284	13,377	13,471	13,565	13,660	13,756	13,852	13,949	14,046	14,145	14,244	14,344	14,444	14,545
Colorectal cancer 3L+ incidence	53,200	53,572	53,947	54,325	54,705	55,088	55,474	55,862	56,253	56,647	57,044	57,443	57,845	58,250	58,658	59,068	59,482	59,898	60,317	60,740	61,165
NSCLC 3L+ incidence	111,969	112,753	113,542	114,337	115,137	115,943	116,755	117,572	118,395	119,224	120,058	120,899	121,745	122,597	123,455	124,320	125,190	126,066	126,949	127,837	128,732
Melanoma 3L+ incidence	6,850	6,898	6,946	6,995	7,044	7,093	7,143	7,193	7,243	7,294	7,345	7,396	7,448	7,500	7,553	7,606	7,659	7,712	7,766	7,821	7,876
% growth		*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*
Breast cancer 3L+ patients with brain metastases	5,060	5,096	5,131	5,167	5,204	5,240	5,277	5,314	5,351	5,388	5,426	5,464	5,502	5,541	5,580	5,619	5,658	5,698	5,737	5,778	5,818
% brain metastases+	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*
Colorectal cancer 3L+ patients with brain metastases	798	804	809	815	821	826	832	838	844	850	856	862	868	874	880	886	892	898	905	911	917
% brain metastases+	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*
NSCLC 3L+ patients with brain metastases	20,154	20,296	20,438	20,581	20,725	20,870	21,016	21,163	21,311	21,460	21,611	21,762	21,914	22,068	22,222	22,378	22,534	22,692	22,851	23,011	23,172
% brain metastases+	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*
Melanoma 3L+ patients with brain metastases	480	483	486	490	493	497	500	503	507	511	514	518	521	525	529	532	536	540	544	547	551
% brain metastases+	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*
Addressable 3L+ brain metastases patients	26,492	26,678	26,865	27,053	27,242	27,433	27,625	27,818	28,013	28,209	28,406	28,605	28,805	29,007	29,210	29,415	29,620	29,828	30,037	30,247	30,459
	100%	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*
Price per patient per month	-	-	-	-	-	-	$ 15,000	$ 15,750	$ 16,538	$ 17,364	$ 18,233	$ 19,144	$ 20,101	$ 21,107	$ 22,162	$ 23,270	$ 24,433	$ 25,655	$ 26,938	$ 28,285	$ 29,699
Treatment time (months)							6	6	6	6	6	6	6	6	6	6	6	6	6	6	6
% growth	-	-	-	-	-	-	-	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*
% penetration	-	-	-	-	-	-	*12%*	*18%*	*30%*	*45%*	*51%*	*57%*	*60%*	*61%*	*61%*	*62%*	*62%*	*63%*	*64%*	*64%*	*32%*
							0.2	0.3	0.5	0.75	0.85	0.95	1	1.01	1.0201	1.030301	1.040351	1.0504513	1.0605516	1.0706519	1.0807522
Gross-to-net adjustment							*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*
3L+ Brain Metastases (MM) (U.S.)	-	-	-	-	-	-	**$ 269**	**$ 426**	**$ 750**	**$ 1,190**	**$ 1,426**	**$ 1,686**	**$ 1,876**	**$ 2,003**	**$ 2,140**	**$ 2,285**	**$ 2,439**	**$ 2,604**	**$ 2,780**	**$ 2,968**	**$ 1,569**
EU Market																					
Breast cancer (ER- Her2+/TNBC) 3L inicidence	28,550	28,635	28,721	28,807	28,894	28,981	29,067	29,155	29,242	29,330	29,418	29,506	29,595	29,683	29,772	29,862	29,951	30,041	30,131	30,222	30,312
Colorectal cancer 3L+ incidence	120,057	120,417	120,779	121,141	121,504	121,869	122,234	122,601	122,969	123,338	123,708	124,079	124,451	124,825	125,199	125,575	125,951	126,329	126,708	127,088	127,470
NSCLC 3L+ incidence	252,682	253,440	254,200	254,963	255,728	256,495	257,264	258,036	258,810	259,587	260,366	261,147	261,930	262,716	263,504	264,295	265,087	265,883	266,680	267,480	268,283
Melanoma 3L+ incidence	15,458	15,505	15,551	15,598	15,645	15,692	15,739	15,786	15,833	15,881	15,929	15,976	16,024	16,072	16,121	16,169	16,217	16,266	16,315	16,364	16,413
% growth		*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*
Breast cancer 3L+ patients with brain metastases	11,420	11,454	11,488	11,523	11,558	11,592	11,627	11,662	11,697	11,732	11,767	11,802	11,838	11,873	11,909	11,945	11,981	12,016	12,053	12,089	12,125
% brain metastases+	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*	*40%*
Colorectal cancer 3L+ patients with brain metastases	1,801	1,806	1,812	1,817	1,823	1,828	1,834	1,839	1,845	1,850	1,856	1,861	1,867	1,872	1,878	1,884	1,889	1,895	1,901	1,906	1,912
% brain metastases+	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*	*2%*
NSCLC 3L+ patients with brain metastases	45,483	45,619	45,756	45,893	46,031	46,169	46,308	46,447	46,586	46,726	46,866	47,006	47,147	47,289	47,431	47,573	47,716	47,859	48,002	48,146	48,291
% brain metastases+	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*	*18%*
Melanoma 3L+ patients with brain metastases	1,082	1,085	1,089	1,092	1,095	1,098	1,102	1,105	1,108	1,112	1,115	1,118	1,122	1,125	1,128	1,132	1,135	1,139	1,142	1,145	1,149
% brain metastases+	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*	*7%*
Addressable 3L+ brain metastases patients	59,786	59,965	60,145	60,325	60,506	60,688	60,870	61,052	61,236	61,419	61,604	61,788	61,974	62,160	62,346	62,533	62,721	62,909	63,098	63,287	63,477
	100%	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*	*100%*
Price per patient per month	-	-	-	-	-	-	-	$ 11,250	$ 11,813	$ 12,403	$ 13,023	$ 13,674	$ 14,358	$ 15,076	$ 15,830	$ 16,621	$ 17,452	$ 18,325	$ 19,241	$ 20,203	$ 21,214
Treatment time (months)								6	6	6	6	6	6	6	6	6	6	6	6	6	6
% growth	-	-	-	-	-	-	-	-	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*
% penetration	-	-	-	-	-	-	-	*6%*	*9%*	*15%*	*23%*	*26%*	*29%*	*30%*	*30%*	*31%*	*31%*	*31%*	*32%*	*32%*	*16%*
Gross-to-net adjustment								*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*
3L+ Brain Metastases (MM) (EU)	-	-	-	-	-	-	-	**$ 67**	**$ 105**	**$ 185**	**$ 292**	**$ 349**	**$ 411**	**$ 455**	**$ 484**	**$ 515**	**$ 548**	**$ 583**	**$ 620**	**$ 659**	**$ 347**
% Royalty to NeOnc								*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*
Total 3L+ Brain Metastases (MM)	-	-	-	-	-	-	**$ 269**	**$ 493**	**$ 856**	**$ 1,375**	**$ 1,719**	**$ 2,035**	**$ 2,287**	**$ 2,459**	**$ 2,624**	**$ 2,800**	**$ 2,988**	**$ 3,187**	**$ 3,400**	**$ 3,627**	**$ 1,916**

Source: BTIG Research estimates

NEO100 recurrent gliomas

- We assume a 40% probability of success.
- We estimate the current prevalence of primary brain tumors is approximately 108,810 patients in 2026 (Source: NBTS, as above).
- We assume net prices per month to be $15,000 with six months of treatment per year, with an annual growth rate of 5%.
- We assume that 50% of patients are ineligible for temozolomide and 90% of temozolomide eligible patients either relapse or are refractory to the treatment.
- We assume market entry of NEO100 to be in 2030.
- We assume a peak market penetration of 60%.
- We assume a 30% royalty rate to NeOnc on ex-U.S. sales of NEO100.

Exhibit 12. NEO100 Glioma Market Model

	2025	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035	2036	2037	2038	2039	2040
US Market																
Primary brain cancer Incidence		108,810	109,572	110,339	111,111	111,889	112,672	113,461	114,255	115,055	115,860	116,671	117,488	118,310	119,138	119,972
% growth		*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*	*0.7%*
Addressable Stage III and IV Glioma patients	-	16,126	16,239	16,352	16,467	16,582	16,698	16,815	16,933	17,051	17,170	17,291	17,412	17,534	17,656	17,780
% malignant	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*
% ineligible for temozolomide	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*
% relapse/refractory to temozolomide	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*
% IDH1 mutant	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*
Price per patient per month	-	-	-	-		$ 15,000	$ 15,750	$ 16,538	$ 17,364	$ 18,233	$ 19,144	$ 20,101	$ 21,107	$ 22,162	$ 23,270	$ 24,433
Treatment time (months)						6	6	6	6	6	6	6	6	6	6	6
% growth	-	-	-	-	-	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*
% penetration	-	-	-	-	*0%*	*5%*	*15%*	*25%*	*40%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*
Gross-to-net adjustment					*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*
Recurrent Glioma Revenue (MM) (U.S.)	-	-	-	-	$ -	$ 67	$ 213	$ 375	$ 635	$ 1,007	$ 1,065	$ 1,126	$ 1,191	$ 1,259	$ 1,331	$ 1,408
EU Market																
Primary brain cancer Incidence		245,553	246,290	247,029	247,770	248,513	249,258	250,006	250,756	251,509	252,263	253,020	253,779	254,540	255,304	256,070
% growth		*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*	*0.3%*
Addressable r/r Glioblastoma patients		30,645	30,737	30,829	30,922	31,014	31,107	31,201	31,294	31,388	31,482	31,577	31,672	31,767	31,862	31,958
% malignant	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*	*26%*
% ineligible for temozolomide	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*	*50%*
% relapse/refractory to temozolomide	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*	*90%*
% IDH1 mutant	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*	*60%*
Price per patient per month	-	-	-	-	-	-	$ 11,813	$ 12,403	$ 13,023	$ 13,674	$ 14,358	$ 15,076	$ 15,830	$ 16,621	$ 17,452	$ 18,325
Treatment time (months)						-	6	6	6	6	6	6	6	6	6	6
% growth	-	-	-	-	-	-	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*	*5%*
% penetration	-	-	-	-	-	*0%*	*3%*	*8%*	*13%*	*20%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*
Gross-to-net adjustment						*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*	*10%*
Recurrent Glioma Revenue (MM) (EU)	-	-	-	-	-	$ -	$ 15	$ 47	$ 83	$ 139	$ 220	$ 231	$ 244	$ 257	$ 270	$ 285
% Royalty to Nuvation						*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*	*30%*
Total	0	0	0	0	0	67	228	422	718	1146	1285	1357	1434	1516	1601	1692

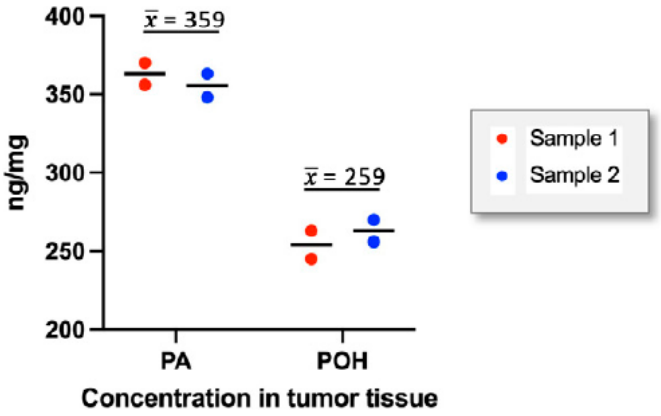
Source: BTIG Research estimates

NTHI Secret Sauce – Crossing the BBB

Probably the biggest reason that brain tumors are so difficult to treat is not that they are inherently drug-resistant or aggressive. Rather, the key complexity is that most drugs can't reach them when delivered systemically. This problem is very much by design as the brain is surrounded by a structure termed the blood-brain barrier (BBB) that reduces communication between the systemic tissue and the CNS by blocking the leakage of all but a select few molecules out of the blood vessels that traverse the CNS (BBB structure reviewed below). In addition, direct delivery into the brain (circumventing the BBB), although possible, is extremely involved and often too toxic in fragile patients.

NeOnc is overwhelmingly focused on solving this BBB issue based on the remarkable properties of POH. First, POH has the magic properties that allow it to cross the BBB alone – where it has anti-tumor effects (NEO100). Second, POH potentiates the crossing of other molecules when the mixture is delivered intranasally (multiple examples are described in company reports). Third, covalent linkage of POH to active cancer drugs can lead to the uptake of the combination molecule. If the combination molecules retain the anti-cancer activity of the original moiety, the result is a risk-reduced candidate to be an improved anti-CNS tumor agent (shown for NEO212).

Exhibit 13. Measurement of POH and PA levels in postsurgical tumor tissue



Source: Schonthal AH, et al. J Neurosurg Case Lessons. 2022;4(8): CASE22215.

Taking on the road not travelled. Perillyl alcohol (POH) is a naturally occurring molecule with documented ability to achieve CNS levels via nasal delivery that are not achievable via systemic administration. The exact mechanism that allows POH to cross the BBB and enters the CNS is not defined (and may be a combination of pathways), but the molecule is small (~152 Da) and lipophilic – two properties common to molecules that can make their way into the CNS. In addition, relative to systemic oral dosing, nasal delivery avoids the first-pass hepatic metabolism and eliminates a major obstacle to CNS bioavailability seen with oral dosing. The data are also clear surrounding POH's ability to escort other molecules into the CNS as the approach has been validated with several passenger chemotherapies and is being tested with multiple other payloads including ASOs and anti-

infective drugs. These latter indications probably require much lower levels of transport to show activity on the other side of the BBB than we would expect for POH.

Pathway #1 - Through the BBB. Molecular data are available in support of the pathway where POH's structure allows it to perturb lipid membranes and cross the BBB directly. Molecularly, POH intercalates into phospholipid bilayers, increasing membrane fluidity - much like the natural function of cholesterol. This disruption likely creates a permeability window. In addition, POH has demonstrated an ability to transiently modulate the tight junction proteins in nasal epithelium without causing lasting damage.

Pathway #2 - Around the BBB. Alternately, POH may go around the BBB via the nerves in the nasal cavity (Exhibit 14). This pathway is well known as a path for other molecules to enter the CNS and some details are described below.

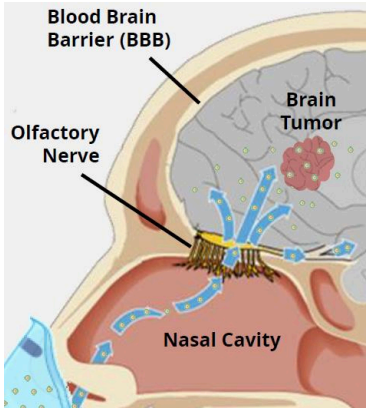
The highways from the nose to the brain.

The **olfactory and trigeminal nerves** provide a direct anatomical route from the nasal mucosa to the CNS, bypassing the BBB entirely. This is the central rationale for intranasal POH delivery in neuro-oncology.

- **Olfactory nerves** — inhaled molecules can be taken up by olfactory receptor neurons in the upper nasal epithelium and transported along olfactory nerve axons through the cribriform plate directly into the olfactory bulb and then deeper CNS structures.
- **Trigeminal nerves** — branches of the trigeminal nerve innervate the nasal mucosa and provide an additional conduit to the brainstem and other CNS regions.

This nose-to-brain transport allows drugs to reach CNS tissue at concentrations that would be unachievable with systemic dosing, while minimizing systemic exposure and toxicity.

Exhibit 14. Olfactory nerve is a multi-lane highway



Source: NTHI company reports

Keep things in the CNS. Another way to increase CNS exposure is to stop the active elimination of drugs from the CNS. These elimination "pumps" are well known in cancer where numerous anti-tumor agents are made ineffective due to the tumor's increasing ability to pump the agent back out of the cell after entry (a common form of drug resistance). The responsible

"pumps" are called P-glycoproteins (P-gps). Important to the NeOnc story, POH has been shown to inhibit P-gp's pump activity. This effect would help drugs shuttled into the CNS via the POH molecule to remain in the CNS and may contribute to the efficacy of both NEO100 and NEO212.

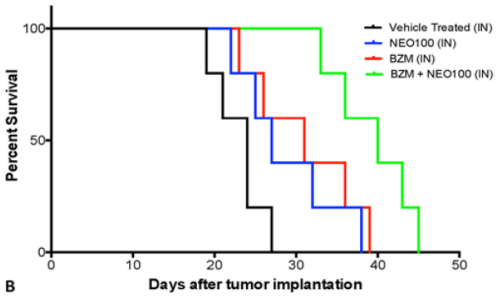
A low bar: other approaches to CNS delivery. The basic approach of disrupting the BBB to allow the passage of drugs into the CNS is not new. For example, there are methods to temporarily disrupt the BBB and allow therapeutic agents into the brain. These approaches can be dangerous and involve the injection of hyperosmotic solutions (mannitol or arabinose) or chemical disruptors (bradykinins and certain reactive lipids called leukotrienes). These somewhat brute force approaches can lead to changes in cerebral fluid balance leading to vasogenic brain edema, focal seizures, and transient neurological deficits.

As an alternative to BBB opening it is possible to circumvent the BBB by delivering a drug via interventional catheters directly into the cerebrospinal fluid (CSF). This procedure also can involve complications, including mistargeting, drug backflush, the need to replace catheters, intraoperative complications, and infections.

NEO100 as a CNS Drug Delivery Platform

Beyond its standalone therapeutic activity, NEO100's most transformative long-term potential may lie in its capacity to serve as a CNS drug delivery platform — enabling brain-targeted delivery of agents that are otherwise entirely excluded by the blood-brain barrier. A particularly compelling set of preclinical experiments demonstrated NEO100's utility as a CNS drug delivery platform. Bortezomib (BZM) — a proteasome inhibitor with established in vitro GBM activity but essentially no CNS penetration via systemic delivery — was selected as a proof-of-concept co-delivery agent. Following IV administration, BZM brain concentrations were more than 100-fold lower than serum concentrations, confirming near-complete BBB exclusion from the systemic route, with a brain/serum ratio of only approximately 1%.

Exhibit 15. POH potentiates the crossing the BBB by BZM.



Source: NeOnc company reports

In contrast, intranasal co-delivery of BZM with NEO100 in rodent orthotopic glioma models produced markedly different results. BZM was detectable in both tumor tissue and normal brain immediately after intranasal delivery and at all subsequent time points up to 60 minutes. The presence of NEO100 in the formulation created a 10-fold increase in BZM CSF concentrations without any corresponding increase in serum BZM levels — directly implicating nose-to-brain transport rather than systemic uptake as the

operative mechanism. This distinction is critical: the preservation of equivalent serum BZM levels irrespective of NEO100 presence effectively rules out a systemic pharmacokinetic explanation for the brain concentration enhancement and instead points to direct olfactory and trigeminal nerve-mediated transport as the primary driver. Survival outcomes reflected this pharmacological advantage. Intranasal BZM combined with NEO100 significantly extended survival over vehicle, BZM alone, and NEO100 alone in U251 orthotopic mouse models, while IV BZM with or without NEO100 produced no survival benefit at all. Two mechanistic models are under investigation: additive cytotoxic combination effects of BZM and NEO100, or NEO100 acting as an amphipathic co-transport carrier facilitating BZM brain entry. Regardless of the precise mechanism, we believe these findings establish a compelling preclinical framework for NEO100 as a CNS delivery platform extending well beyond its standalone therapeutic activity.

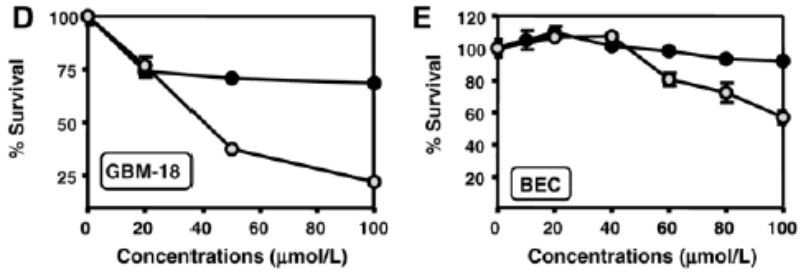
Clinical Programs:

NeOnc has two programs in the clinic. We see NEO212 as the more immediate driver of value as the oral delivery is more straightforward and the active agent is well understood (TMZ). NEO100 is more novel, but the molecule's ability to shuttle other agents into the CNS is a source of significant potential upside for the NeOnc story, in our view.

NEO212 Recent P2a Readout Confirms Activity

NEO212 looks like a low-risk project. NEO212 is a DNA alkylating agent related to temozolomide (TMZ), the workhorse drug for treating serious brain tumors including GBM and aggressive gliomas. NEO212 is modified to improve on the modest BBB penetration of TMZ resulting in about **3X the concentration of active anti-tumor agent in the CNS** (both drugs are pro-drugs of the active agent AIC). Importantly, the modifications that stabilize NEO212 relative to TMZ don't reduce the ability of the drug to generate DNA lesions in target cells (that cancer cells need to correct to survive). As a result, NEO212 looks like an approach to deliver more of an active anti-tumor drug to the CNS to treat CNS tumors. This setup seems relatively low-risk to us.

Exhibit 16. NEO212 consistently looks more active than TMZ.



Source: NeOnc company reports

Potential pivotal trial in sight. Probably the key readout from the P1 trial is that the company has established an MTD dose and is ready to start P2. The trial details are being worked out, but the trial would likely focus

on MGMT-unmethylated and TMZ-refractory patients. This is the unmet need group expected to lead to accelerated approval if results are compelling. In addition, management has suggested patients that have discontinued TMZ due to hematologic toxicity would be included. This group is a first step in establishing NEO212 in earlier lines. The trial would likely be head-to-head with TMZ.

NEO212 is a cleavable complex of POH and TMZ. NEO212 was generated via covalent conjugation of Perillyl alcohol (POH) to TMZ (Exhibit 1). Oral TMZ is the current chemotherapeutic standard of care for the treatment of patients with newly diagnosed glioblastoma (GBM), where it is administered concomitantly with radiotherapy and subsequently as monotherapy during the adjuvant phase.

TMZ is a pro-drug in that it doesn't kill tumors until it undergoes spontaneous hydrolysis at physiological pH into its active metabolite, 5-(3-methyl triazen-1-yl) imidazole-4-carboxamide (MTIC) (Exhibit 3). This intermediate is then rapidly degraded into a methyldiazonium ion - the highly reactive species that methylates DNA. The reaction also generates the metabolite AIC. This metabolite is longer-lived and its detection has been used to explain the excellent safety profile of NEO212 relative to TMZ (less bone marrow toxicity. The methyldiazonium ion is highly reactive and can interact with DNA at several spots but O6-methylguanine (O6MeG) lesion is the most cytotoxic lesion and activity at that site is likely the source of the drug's therapeutic effects.

Recent P2a readout.

NEO212: P1 readout strongly suggested activity in difficult-to-treat patients. The recent P1 readout was principally designed to determine a go-forward dose for larger and potentially registrational trials. The trial also confirmed the excellent safety of NEO212 where only the last cohort showed meaningful toxicity (defining the maximum tolerated dose). Two patients, that were both highly treatment-experienced showed either tumor regressions or stable disease after periods of tumor growth on prior drug regimens. For P1 oncology trials, that type of result is about as good as it gets.

Exhibit 17. Patients in P1 trial of NEO212

	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 company reports

Efficacy signals. The P1 trial of NEO212 achieved its intended goal of defining a maximum tolerated dose after two patients in the 810 mg dose experienced a DLT (drug-related toxicity). The trial was too small to provide estimates of response rates, and the treatment experience of the patients

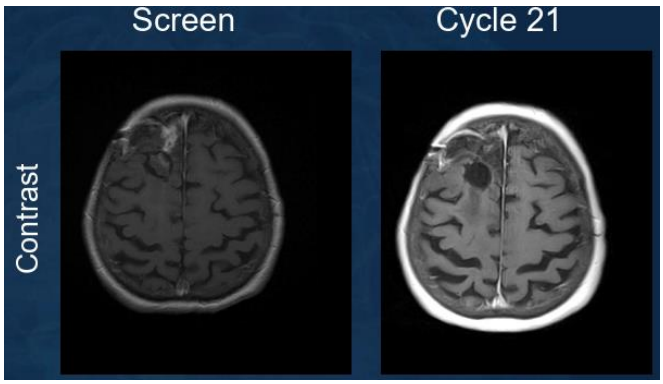
makes any determination of this type a rough approximation. However, two patients showed clear signs of anti-tumor activity while on the drug and are summarized below.

Patient 1 – rapid and sustained tumor response

The most striking patient in the P1 trial was a GBM patient with an MGMT methylated tumor. The patient had received standard therapy of surgery, radiation and TMZ. The patient progressed after six cycles of TMZ suggesting an aggressive tumor.

After progressing, the patient was enrolled in the NEO212 P1 trial. The response to the drug was quite remarkable with the patient showing rapid tumor reduction (Exhibit 18 and 19) that was stable for 22 cycles.

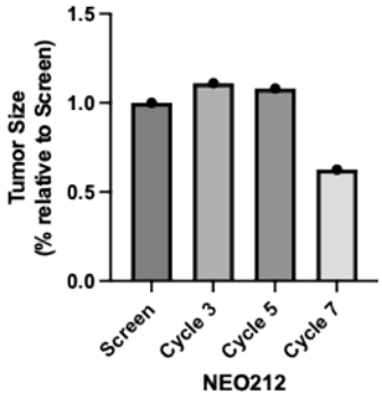
Exhibit 18. Patient with a significant response



Source: NeOnc company reports

Patient 2. A second patient of note had experienced metastases of an NSCLC tumor to brain. This tumor was very aggressive and had been surgically resected three times (highly unusual). After treatment began with NEO212 the patient had experienced stable disease with some small signs of tumor regression (Exhibit 19). Although not a true "response", stable disease was still considered impressive as tumors at this stage generally don't stop growing on their own.

Exhibit 19. Patient with stable disease

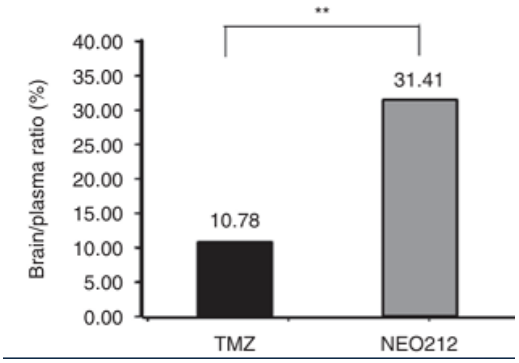


Source: NeOnc company reports

Preclinical background for NEO212.

With tumor responses in patients, preclinical data is less interesting, but the basic properties of NEO212 are worth some review. The POH domain leads to significantly better uptake of NEO212 relative to TMZ (Exhibit 20). This increase is likely important as TMZ is often considered "underdosed" due to the bone marrow toxicity seen with the drug. As a result, most early-line GBM patients get some response to the drug, but almost all recur. Whether NEO212 treatment will improve this initial response rate and duration of responses needs to be shown but is consistent with the "more of a good thing" properties of the drug and we believe represents significant upside to the NeOnc story.

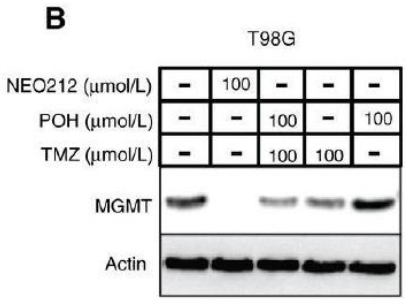
Exhibit 20. NEO212 reaches the CNS at 3X higher levels than TMZ.



Source: NeOnc company reports

Resistance-a-go-go. A second interesting property of NEO212 is the drug's ability to cause the degradation of the MGMT enzyme that is the basis of TMZ resistance (Exhibit 21). This ability to drive degradation probably follows directly from the better crossing of the BBB relative to TMZ. Each time a molecule of TMZ is destroyed, the MGMT enzyme is turned over – more TMZ leads to less MGMT until the enzyme disappears.

Exhibit 21. NEO212 makes MGMT disappear – but it's not magic.

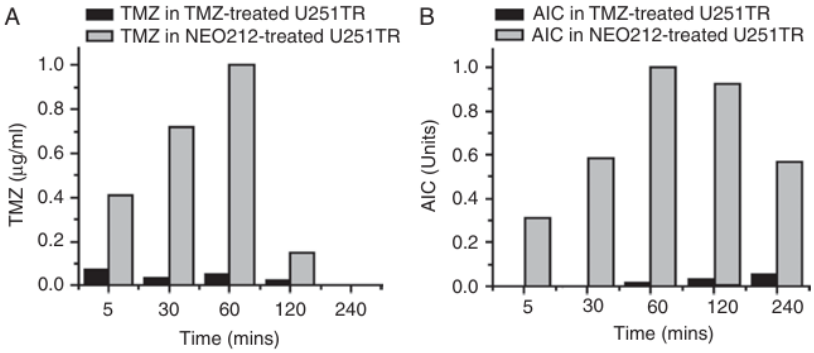


Source: NeOnc company reports

NEO212: mechanism also looks safer than TMZ. A key attribute of any anti-tumor drug is that it is safe to use and is more toxic to cells than to healthy tissue. Much of this safety window (termed therapeutic margin) is based on the rapid division of tumor cells relative to normal mature human cells and

this more rapid division makes tumor cells more susceptible to the drug. TMZ has a therapeutic window, but the drug's dosing is constrained by its toxicity toward cells of the bone marrow (anemia and cytopenias). Relative to TMZ, NEO212 produces less AIC in the blood where it is toxic and conversion to AIC occurs principally after the drug has entered cells. This property probably explains the low levels of anemia seen with NEO212.

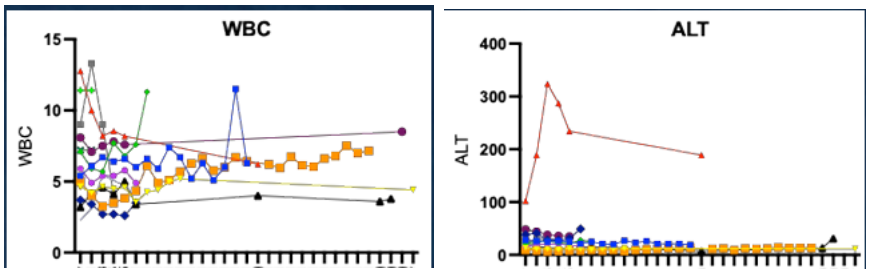
Exhibit 22. Relative exposure of non-tumor cells is much lower for NEO212



Source: NeOnc company reports

Detailed safety readouts. Lab data are always a starting point for defining the safety of a drug. In the recent P1 readout for NEO212, NeOnc released extensive patient-level safety data for the drug. As seen in Exhibit 23, the drug resulted in essentially no anemias (WBCs shown but many tested) and liver enzymes were not elevated (ALT shown). Much more safety data needs to be generated before a drug can be approved but given the safety of both POH and TMZ, the safety of NEO212 isn't a huge surprise.

Exhibit 23. Heme and liver safety: a lot of nothing to report.



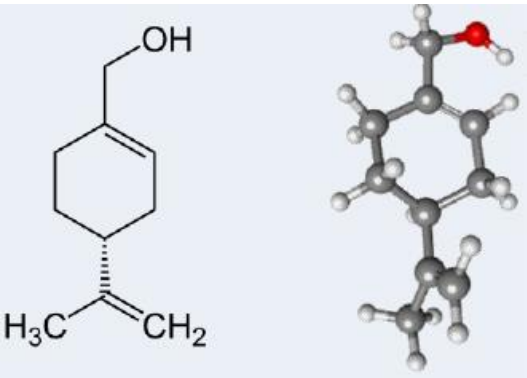
Source: NeOnc company reports

NEO100: Two Pipelines and a Product

The somewhat playful description "pipeline in a product" is probably overused in the biotechnology sector. **Undeterred**, we argue below that NeOnc has already established POH as a potential product and provided support for two independent approaches where POH could serve as a key ingredient of a pipeline. We fully understand NeOnc can't hope to develop all these potential products alone – but as POC is established for some of the more advanced candidates, we expect partners might appear to take on alternative applications. These applications might be in areas outside oncology as drug delivery to the CNS is a challenge throughout neurodegeneration and neuropsychiatry.

A product. Neo100 is cGMP-grade POH and underpins the NeOnc efforts to date. The drug has stand-alone anti-tumor activity but at concentrations that made oral dosing untenable. NeOnc has taken some clues from some small clinical studies of POH (Brazilian study, see below) and is now developing a nasal formulation it has named NEO100.

Exhibit 24. NEO100 has the right stuff to sneak past the BBB.



Source: NeOnc company reports

A pipeline. In addition to its drug-like activity in astrocytomas, NEO100 can usher other molecules across the BBB after covalent conjugation. The lead program from this effort is NEO212 which we see as the major value driver for the story. Management has commented that the carbamide linkage in NEO212 is a relatively simple chemistry and can likely be adapted to other payloads – an effort we see as the company's lowest-risk pipeline. In summary: if the drug-POH fusion gets across the BBB in NHPs and the drug-POH molecule is active in anti-tumor assays in cells – there is a good chance it's an active therapeutic.

Another pipeline. Less clear cut, but perhaps even more powerful is the ability of POH to help other molecules cross the BBB when co-formulated (mixed, not chemically bonded). This approach has been validated for a number of conventional chemotherapies and seems very straightforward to at least try with other payloads. We expect success will be very dependent on payload, but the potential opportunities, including beyond cancer, seem very large to us.

Proposed Mechanisms in GBM Specifically

Beyond its general antitumor mechanisms, POH may have relevance in GBM through:

- **Ras pathway inhibition** — Ras signaling is constitutively active in many GBMs through EGFR amplification, PTEN loss, and other upstream alterations
- **Autophagy induction** — POH induces autophagic cell death in glioma cell lines, which may be particularly relevant given GBM's relative apoptosis resistance
- **Differentiation induction** — some evidence of pro-differentiation effects on glioma stem cells
- **Sensitization to temozolomide** — preclinical data suggest POH may enhance TMZ sensitivity, supporting combination approaches
- Mechanism studies in humans (rather than cell lines) are limited

NEO100 Clinical Evidence

Brazil Phase I/II Studies The foundational clinical proof-of-concept for intranasal POH delivery came from prospective studies conducted in Brazil. An important study enrolled 198 patients with recurrent malignant glioma (including GBM) that had previously failed standard therapy. A drug-formulated form of POH was administered intranasally four times daily.

The studies demonstrated durable tumor responses and a favorable long-term safety profile. On the safety front, adverse events were limited to occasional nasal soreness (n=5) and rare epistaxis (n=2) at higher doses. No systemic, hematological, or gastrointestinal toxicities were observed even in patients treated continuously for more than four years. Prognostic factors influencing response included peritumoral edema and tumor location. These results provided the clinical rationale for the U.S. NEO100 program.

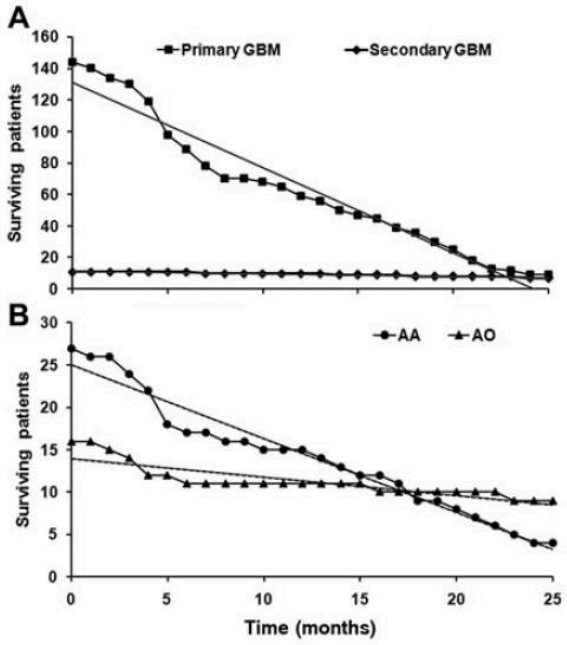
U.S. Phase I Trial (NCT02704858). The completed Phase I component of an ongoing Phase I/IIa study enrolled 12 patients with recurrent GBM at four U.S. centers (Cleveland Clinic, University of Washington, University of Wisconsin, USC) who had progressed after standard chemoradiation with TMZ. Four escalating dose cohorts received 384, 576, 768, or 1,152 mg/day, administered via nebulizer four times daily, with response assessed by RANO criteria every two months.

Safety profile was exceptional. No severe adverse events were reported at any dose level, and no dose-limiting toxicities were identified — a stark contrast to the bone marrow suppression associated with lomustine or the hemorrhage risk with bevacizumab.

Efficacy: Results compared favorably against historical benchmarks for this population:

PFS-6: 33%; OS-12: 55%; Median OS: 15 months (vs. a generally expected 6–10 months); OS-24: 33% (four of 12 patients)

Exhibit 25. Overall survival of glioma patients included in Phase I/II

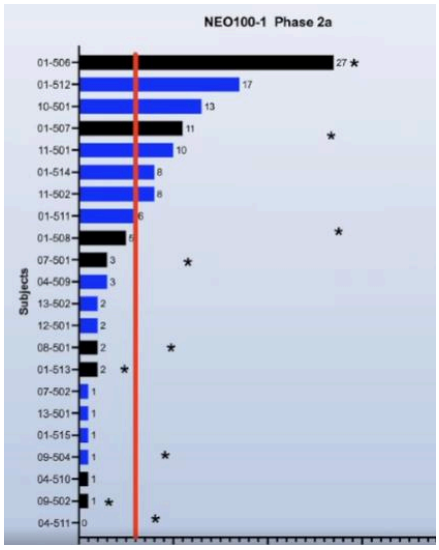


Source: NeOnc company reports

The most compelling efficacy signal emerged from the IDH1-mutant subgroup: all four patients surviving beyond 24 months harbored IDH1 mutations, while IDH1 wild-type patients showed no survival beyond 18 months. Patients completing more than five treatment cycles showed substantially better outcomes. This IDH1-mutant enrichment signal now underpins the ongoing Phase 2a trial design.

Ongoing NEO100-1 Trial This trial of 25 IDH1-mutant Grade 3/4 astrocytoma patients is fully enrolled and the next big readout for the stock. Interim data were released in November 2025. The topline result is interesting with eight of 18 patients that were treated for six months showing a PFS of six months or more (18 of 24 were evaluable). Partial results of this type are not definitive, but the calculated PFS6 of 44% is around twice the expected rate of 21% to 31%.

Exhibit 26. Swimmers plot from NEO100-1 trial.

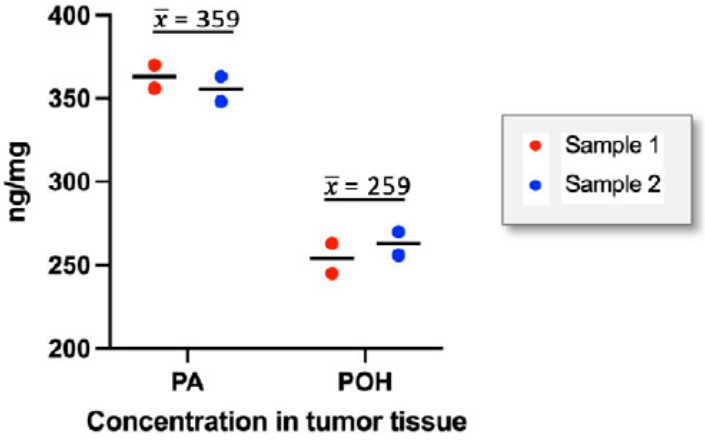


Source: NeOnc company reports

Case Report Evidence of Brain Delivery. In a pivotal case report, a patient with recurrent grade IV IDH-mutant glioma received IN NEO100 for more than three years (35 cycles). Upon a second recurrence requiring repeat surgery, a final dose was administered shortly before resection, and subsequent HPLC analysis of the **resected tumor tissue confirmed the presence of both POH and its primary metabolite perillic acid (PA)**. This represented the first-ever direct demonstration that intranasal NEO100 delivers active drug to intracranial tumor tissue in a human patient — a key piece of pharmacological validation that directly supports the clinical mechanistic model.

Anti-tumor response. The company has highlighted the effects of NEO100 in a 39-year-old woman initially diagnosed with low-grade glioma and progressed to recurrent grade IV IDH1-mutant, MGMT-methylated glioblastoma. Following surgical resection and standard-of-care treatment, the patient received daily intranasal NEO100 therapy. Over the subsequent three-plus years, serial MRI scans obtained at 1, 2, and 3 years demonstrated disease stability. This three-year stoppage of tumor progression is remarkable given historical median overall survival for recurrent GBM patients with IDH1 mutations of < 10 months post-progression. The patient remained alive 54 months after recurrence, having experienced more than 36 consecutive months without disease progression while on NEO100.

Exhibit 27. Measurement of POH and PA levels in postsurgical tumor tissue.



Source: Schonthal AH, et al. J Neurosurg Case Lessons. 2022;4(8): CASE22215.

Oral POH is active although probably unusable. The anti-tumor activity of POH was demonstrated in several phase I/II clinical trials, where it was administered orally. The drug was generally viewed as active only at relatively high concentrations and even at those concentrations, the activity seemed marginal. In addition, the continuous daily regimen required to deliver POH was considered hard to tolerate and oral POH was dropped at a relatively early stage of development. As an alternative, an intranasal formulation of POH was developed.

NEO100 Preclinical Evidence – Multi-Pronged Anti-Tumor Activity

NEO100 demonstrated cytotoxic activity across a broad panel of human glioma cell lines, including U251, U87, LN229, and A172, as well as primary patient-derived GBM specimens. Critically, activity was maintained in established TMZ-resistant derivatives of these lines, including U251TR and LN229TR2, directly relevant to the recurrent disease setting where TMZ resistance is near-universal. At the mechanistic level, in vitro work established that NEO100-induced cytotoxicity operates through several convergent pathways. This combination of cytostatic, cytotoxic, and anti-angiogenic mechanisms **may help explain the durable responses observed in a subset of clinical patients** — an effect that would be unlikely from a single-mechanism agent.

- NEO100 activates ER stress through elevated expression of GRP78, ATF3, and CHOP, driving the unfolded protein response and ultimately apoptosis.
- Simultaneously, it inhibits G1 cell cycle progression, upregulates the pro-apoptotic protein Bax, and arrests the mTOR and Ras signaling cascades.
- Additional mechanistic work identified inhibition of Na/K-ATPase — disrupting cytoplasmic membrane electrolyte balance — as a further contributor to NEO100's cytotoxic activity.

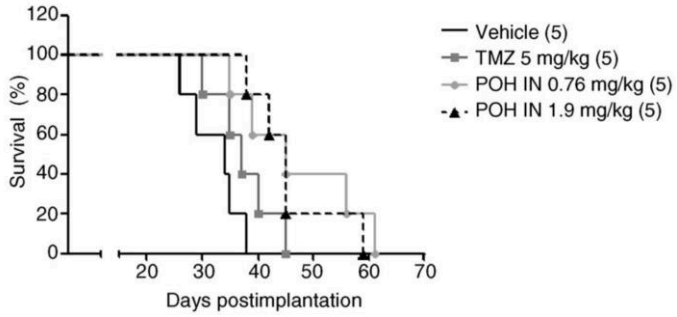
Importantly, this cytotoxic activity was demonstrated to be independent of MGMT expression status, meaning NEO100 retains potency in both TMZ-sensitive and TMZ-resistant glioma cells regardless of the primary mechanism driving TMZ resistance.

Animal models. The development of drugs for GBM and glioma is aided by many animal models. However, many of these animals have relatively leaky BBBs – so the mode is most valid if the drug is shown independently to cross the BBB.

- In orthotopic rodent glioma models, intranasal NEO100 demonstrated significant anti-tumor activity. Mice bearing intracranially xenografted GBM cells receiving NEO100 at 0.76 or 1.9 mg/kg intranasally on alternating nostrils every other day survived significantly longer than vehicle-treated controls, with no apparent pathological abnormalities observed in histopathological analysis of normal tissues.
- In TMZ-resistant models specifically, intranasal NEO100 reduced tumor growth and extended survival, validating its activity in the setting most clinically relevant to the intended patient population.

As seen in cellular assays above, the mechanisms of *in vivo* activity appear to be multifactorial, encompassing ER stress-mediated tumor cytotoxicity, decreased tumor angiogenesis consistent with the *in vitro* anti-VEGF findings, and reduced tumor invasive capacity.

Exhibit 28. Intranasal POH prolongs survival in orthotopic mouse model



Source: Cho HY, et al. Mol Cancer Ther. 2012 Nov;11(11):2462-72.

Background GBM and aggressive gliomas.

Glioblastoma (GBM) is the diagnosis given for WHO Grade 4 astrocytoma and is the most common and most aggressive primary brain tumor found in adults. The diagnosis has evolved with time and once originally based on histology (how the cells look and stain) is increasingly based on molecular markers such as IDH-wildtype status (in adults), mutations in the TERT promoter, EGFR gene amplification or chromosomal changes. IDH-wildtype GBM is the canonical form, representing ~90% of cases. This form occurs primarily in older adults (median age ~65) and arises de novo – not from a lower-grade precursor. This conventional GBM is the worst case and carried a median survival of ~15 months in clinical trials using the standard treatment. Importantly, clinical trials tend to have a selection bias for patients healthy enough to participate in the trial and the prognosis of GBM in the real world is probably even worse.

Aggressive gliomas are a family of brain tumors that are best considered as "just short" of GBM in the severity of their diagnoses. The best characterized form is probably IDH-mutant Grade 4 astrocytoma. These tumors were once called "secondary GBM," but are now classified separately due to a better prognosis despite being equally aggressive histologically (very odd-looking cells but better genetic markers). IDH-mutant Grade 4 Astrocytoma was previously called "secondary GBM" but is now classified separately under the 2021 WHO scheme. It tends to affect younger patients, arises from lower-grade gliomas, and has meaningfully better outcomes (median survival 3–4 years). Despite identical histology, it is no longer called GBM in the current classification.

GBM Molecular Subtypes

Several molecular markers define subgroups with clinical relevance, and these classifications have been more important in treatment strategies since the 2021 WHO Classification of CNS Tumors. This classification formalized these molecular distinctions, moving away from histological grading toward an integrated molecular diagnosis — so a tumor that *looks* like GBM under a microscope may now be classified differently based on its molecular profile. All that being said, with TMZ as the main drug doctors can reach for, all the molecular dissections awaits better therapies.

- MGMT promoter methylation — present in ~40–45% of GBMs. This is a key biomarker and predicts better response to temozolomide and improved survival.
- EGFR amplification / EGFRvIII mutation. This mutation is found in ~40–50% of GBMs and as expected for any mutated growth factor pathway is associated with aggressive behavior but might also allow targeting with small molecule inhibitors that cross the BBB.
- TERT promoter mutation. This mutation is very common and is used to confirm an IDH-wildtype genotype.
- PTEN loss, CDKN2A/B deletion, chromosome 7 gain and chromosome 10 loss are all frequent mutations that reinforce GBM diagnosis and IDH-wildtype tumors – a poor prognosis.

Secondary brain tumors

In addition to primary brain tumors such as GMB, tumors termed brain metastases can arise when tumors that arose elsewhere in the body spread to the brain. These secondary tumors are highly heterogeneous and are far more common than primary brain tumors. Paradoxically, these tumors are on the rise, and their occurrence is driven by powerful systemic anti-cancer therapies such as targeted monoclonal antibodies (mAbs) and ADCs. Briefly, these therapies help patients survive their systemic tumors and live longer – but these therapies are unable to enter the CNS (don't cross the BBB). In other words, mAbs like HERCEPTIN don't cause breast cancers to spread to the brain, but they do help patients live much longer and these tumors eventually make their way to the mAb-free CNS. As detailed below, the majority of secondary brain tumors arise from primary lung and breast cancers, so data for NEO212 in these tumor types could be exciting.

Most Common Primary Sources and Prognosis

- **Lung cancer** is a common site where tumors can spread to the brain and accounts for 40–50% of brain metastases. Both small cell lung cancers (SCLC) and non-small cell lung cancers (NSCLC) metastasize readily to the brain – often soon after initial diagnoses. Patients with EGFR mutations or ALK rearrangements can be treated with CNS-penetrant TKIs (osimertinib, alectinib) with an average survival of 2–3 years or more, while patients without targetable mutations (the vast majority) **have median survivals of** 6–12 months.
- **Breast cancer** accounts for about 15–25% of secondary brain cancers and is an extremely negative prognosis – although they often occur late in disease (breast cancer is generally detected much earlier than lung cancer due to regular monitoring). HER2-positive and triple-negative subtypes carry the highest risk of CNS spread. For Her2-positive cancers, there are small molecule treatments that can help, including and tucatinib, neratinib. If the BBB degrades, the ADC trastuzumab and deruxtecan can also reach brain metastases. Like lung cancers lacking clear driving mutations, **Triple-negative breast** cancer is difficult to treat with a median survival of 6–9 months after CNS detection.
- **Melanoma** is a rarer tumor type but has the highest propensity to metastasize to the brain. A staggering 75% of patients with advanced melanoma develop CNS involvement. Melanoma-derived CNS tumors are also prone to hemorrhage. Again, patients with relatively rare melanoma tumors that respond to small molecule BRAF/MEK inhibitors have better outcomes with median survivals of 12–18 months.

The BBB: an unusually tight endothelial junction

In most tissues, the endothelial cells that line the blood vessels are connected by a combination of adherens junctions and other loosely organized tight junctions. These junctions are intended to be leaky (termed pericellular gaps) and allow the sampling of small molecules, peptides and some proteins in the blood by local tissues and vice versa. Similarly, immune cells can use these small openings to squeeze out of blood vessels and

invade infected tissues. The situation in the CNS is quite different, and the endothelial junctions are designed to stop non-specific blood components from entering the CNS. A quick readout to give a sense of these differences is that the resistance of the junctions as measured by ion flow is about 1000 times higher than the resistance to ion flow for non-CNS junctions between endothelial cells.

BBB Tight Junctions — What Makes Them Different

- **Claudins** — claudin-5 is the dominant claudin at the BBB and considered a molecular signature. The molecule forms the primary seal of the paracellular cleft.
- **Occludin** — is also expressed at high levels at the BBB and plays a regulatory and structural role.

Suppression of Transcytosis

A critical and often underappreciated distinction is that the BBB doesn't just seal the paracellular space — it also **actively suppresses transcytosis**:

- Peripheral endothelial cells are rich in **caveolae** and pinocytic vesicles, allowing substantial vesicular transport
- BBB endothelial cells have **low caveolae density** and suppressed vesicular trafficking, partly regulated by pericyte signaling (Mfsd2a, a lipid transporter in BBB endothelial cells, is a key suppressor of caveolae formation)

NeOnc Key Patents

NeOnc has secured an exclusive worldwide license from the University of Southern California (USC) covering a broad patent portfolio supporting its lead candidates, NEO100 and NEO212, across oncological and neurological indications. In the U.S., the company holds rights to 28 issued patents and 14 pending applications, complemented by a significant international presence of 65 issued patents and 28 pending applications. All patents remain owned by USC and are exclusively licensed to NeOnc.

Patent Term Extension (PTE) eligibility may apply to select patents post-approval, representing a potential meaningful extension of commercial exclusivity. The breadth of international coverage alongside a healthy pipeline of pending applications suggests the company has prioritized protecting its IP across key global markets, while the dual-indication scope — oncology and neurology — provides flexibility for lifecycle management across the broader NeOnc product family.

Exhibit 29. NeOnc Patents

Product Candidate	Title [description]	US Application #	US Patent	Expiration Date	Foreign Counterparts
NEO100	Pharmaceutical Compositions Comprising Monoterpenes [Ultra Pure]	13/040,059 13/939,834 14/817,286 14/843,097 15/040,002 15/220,135 16/575,587 17/749,293	8,507,734[1,2] 9,133,085[1] 9,480,659[1] 9,498,448[1] 9,700,524[3] 10,457,618[1] 10,899,691[1] Pending[3]	8/29/2031 6/5/2031 3/3/2031 3/3/2031 3/3/2031 3/3/2031 3/3/2031 3/3/2031*	Canada, China, China, EU (France, Germany, United Kingdom, Ireland, and Italy)
NEO212	Pharmaceutical Compositions Comprising POH Derivatives [POH conjugated to temozolomide (TMZ)]	13/566,731 14/455,371 13/818,972 15/408,866	8,916,545[2] 9,580,372[3] 9,499,461[1,2,3] 10,092,562[3]	8/26/2031 8/26/2031 8/26/2031 8/26/2031	Brazil, China, EU (Germany, Spain, France, United Kingdom Switzerland, Ireland, Italy, Netherlands, Sweden), Japan, Hong Kong
NEO 214	Pharmaceutical Compositions Comprising POH Derivatives [POH conjugated to rolipram]	16/123,729 16/388,535 17/306,167	11,077,104[3] 11,013,804[2] pending[2,3]	8/26/2031 8/26/2031 8/26/2031*	Japan, EU (Spain, France, United Kingdom, Ireland, Italy, Netherlands, Sweden)
NEO 216	Pharmaceutical Compositions Comprising POH Derivatives and Methods of Use [POH conjugated to valproic acid]	16/606,520 18/150,933	11,559,508[2] pending[3]	11/24/2038 4/18/2038*	China, EU (pending in both)
NEO 218	A Perillyl Alcohol-3-Bromopyruvate Conjugate and Methods of Treating Cancer [POH conjugated to 3- Bromopyruvate]	16/465,081	10,858,305[1,2,3]	11/29/2037	China, EU, Japan (pending in all)
NEO 400	Pharmaceutical Compositions Comprising POH Derivatives [POH conjugated with linoleic acid (LA)]	17/251,452 17/313,258 17/573,693	pending[3] pending[3] pending[2]	8/26/2031* 8/26/2031* 8/26/2031*	China, EU (pending in both)
NEO 412	Pharmaceutical Compositions Comprising Perillyl Alcohol Derivatives [Triple conjugation of perillyl alcohol, linoleic acid, and temozolomide]	15/041,743 16/126,586	9,522,918[1,2] 10,696,680[3]	2/11/2036 2/11/2036	Australia, China, EU (Germany, France, United Kingdom, Ireland), Japan

1 Method of making pharmaceutical composition
2 Pharmaceutical composition
3 Method of using pharmaceutical composition

* 20 years from the earliest filing date, subject to patent term adjustment

Source: NTHI company reports

NeOnc Technologies Management Team

Amir F. Heshmatpour - CEO, Executive Chairman, and President

Amir F. Heshmatpour brings over 25 years of executive leadership to NeOnc Technologies, having founded AFH Holding & Advisory LLC in 2005 — a single-family office through which he has directed more than $6 billion in transactions across biotech and healthcare. Since acquiring NeOnc in 2023 and assuming the Presidency in 2025, he has focused on accelerating the company's clinical programs and expanding its global partnerships across the U.S., GCC/MENA, and India to advance treatment options for brain cancer patients.

David Choi - Chief Accounting Officer

David Choi joined NeOnc as Chief Accounting Officer in 2026, bringing over a decade of experience in accounting, financial reporting, and internal controls across public and private companies, including prior roles at Ernst & Young, Grant Thornton, and Blythe Global Advisors. A Certified Public Accountant with a Master of Professional Accountancy from UC Irvine, Mr. Choi is focused on building scalable financial infrastructure to support NeOnc's continued growth as a public clinical-stage biotechnology company.

Josh Neman, PhD - Chief Clinical Officer

Dr. Neman is an Associate Professor of Neurological Surgery, Neuroscience, and Physiology at the Keck School of Medicine of USC, where he also serves as Scientific Director of the USC Brain Tumor Center and Program Chair of the Cancer Biology and Genomics PhD Program. A UCLA-trained neurobiologist, he co-founded Synaptical Inc. in 2021 and CNSMENDER Consulting LLC in 2022, further extending his commitment to advancing cancer patient education and research.

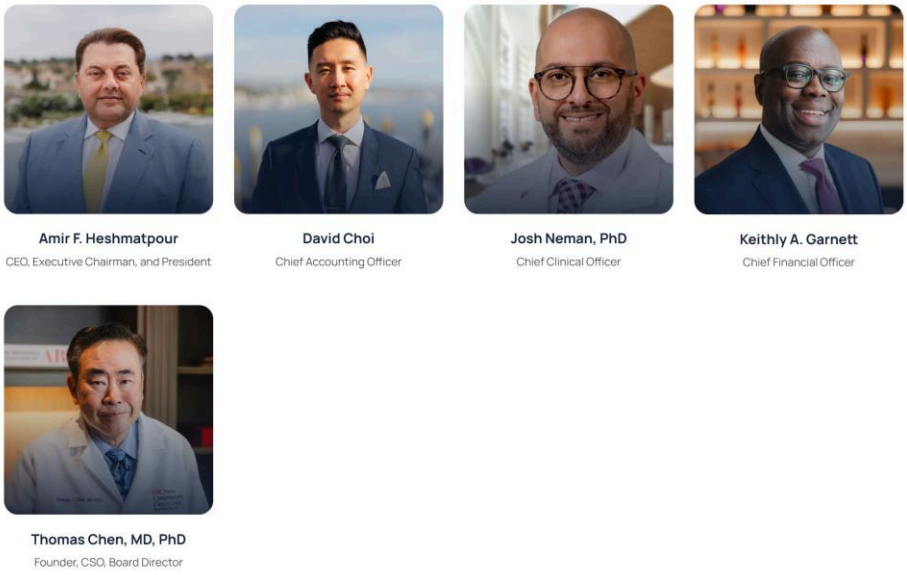
Keithly A. Garnett - Chief Financial Officer

Keithly Garnett has served as NeOnc's Chief Financial Officer and Director since 2023, leveraging over 17 years at Ernst & Young's Transaction Advisory Services practice where he advised major clients including Amgen, Edwards Life Sciences, and Medtronic on business valuation, financial strategy, and SEC reporting. He holds an MBA in Corporate Financial Management and a certificate from Columbia University's Executive Education CFO program, complemented by his concurrent role as CFO of AFH Holding & Advisory.

Thomas Chen, MD, PhD - Founder, CSO, Board Director

Dr. Thomas Chen is a board-certified neurosurgeon, tenured Professor of Neurosurgery and Pathology at USC, and Director of Surgical Neuro-Oncology, where he heads a research laboratory focused on glioma biology and holds a PhD in pathobiology with a thesis centered on immunotherapy for malignant brain tumors. A summa cum laude graduate of the University of Illinois, he is among a select few surgical neuro-oncologists in the country specializing in spine cancer surgery and serves on the editorial and review boards of leading journals including *The Spine Journal* and *Journal of Neurosurgery*.

Exhibit 30. Management team



Source: NTHI company reports

Exhibit 31. Income Statement

Thomas Shrader 212.527.3551
Jinnie Kim 212.882.2344

NeOnc (NTHI) Income Statement

Period Ending	Dec-24	Mar-25	Jun-25	Sep-25	Dec-25	Dec-25	Mar-26	Jun-26	Sep-26	Dec-26	Dec-26	Dec-27	Dec-28	Dec-29	Dec-30	Dec-31	Dec-32	Dec-33	Dec-34	Dec-35	Dec-36	Dec-37	Dec-38	Dec-39	Dec-40
$USD ('000s)	2024A	1Q25A	2Q25A	3Q25A	4Q25A	2025A	1Q26E	2Q26E	3Q26E	4Q26E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E	2034E	2035E	2036E	2037E	2038E	2039E	2040E
Product Revenue:																									
					-	-	-	-	-	-	-	-	-	-	26,863	91,163	168,969	287,049	458,535	513,890	542,992	573,744	606,239	640,576	676,859
															65,276	119,890	208,289	334,726	418,412	495,304	556,752	593,839	627,427	662,916	693,553
Revenue	83	40	-	-	-	40									-	80,553	147,788	256,784	412,620	515,635	610,386	686,067	737,678	787,204	840,057
Total Revenue	**83**	**40**	**-**	**-**	**-**	**40**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**92,139**	**291,606**	**525,046**	**878,558**	**1,289,567**	**1,524,829**	**1,710,130**	**1,853,650**	**1,971,343**	**2,090,695**	**2,210,469**
Operating Expenses:																									
COGS	-	-	-	-		-	0	0	0	0	0	0	0	0	13,821	43,741	78,757	131,784	193,435	228,724	256,520	278,047	295,702	313,604	331,570
Research and development	3,045	998	677	715	1,248	3,638	1,048	711	751	1,310	3,820	4,011	4,212	4,422	92,139	174,963	210,018	219,639	193,435	228,724	256,520	278,047	295,702	313,604	331,570
Legal and Professional	2,001	958	520	276	727	2,481	1,005	546	290	763	2,605	2,736	2,873	3,016											
General and administrative	1,680	849	984	903	2,081	4,818	892	1,033	948	2,185	5,059	5,312	5,577	5,856											
Share based compensation	0	23,074	3,526	5,040	3,915	35,555	0	0	0	0	0	0	0	0	184,278	437,409	472,542	658,918	386,870	304,966	256,520	278,047	295,702	313,604	331,570
Other	500	11,738	0	0	50	11,788																			
Total Operating Expenses	**7,226**	**37,617**	**5,708**	**6,935**	**8,021**	**58,280**	**2,946**	**2,291**	**1,989**	**4,259**	**11,484**	**12,059**	**12,662**	**13,295**	**290,238**	**656,113**	**761,317**	**1,010,342**	**773,740**	**762,415**	**769,559**	**834,142**	**887,105**	**940,813**	**994,711**
Operating Income (EBIT)	**(7,143)**	**(37,577)**	**(5,708)**	**(6,935)**	**(8,021)**	**(58,240)**	**(2,946)**	**(2,291)**	**(1,989)**	**(4,259)**	**(11,484)**	**(12,059)**	**(12,662)**	**(13,295)**	**(198,099)**	**(364,507)**	**(236,271)**	**(131,784)**	**515,827**	**762,415**	**940,572**	**1,019,507**	**1,084,239**	**1,149,882**	**1,215,758**
Interest Income	16	52	29	7	240	328	0	6	4	3	**13**	**16**	**18**	**18**	**245**	**(222)**	**(944)**	**(1,313)**	**(890)**	**880**	**3,250**	**6,021**	**9,010**	**12,194**	**15,574**
Interest Expense - related parties	(2,557)	(309)	(49)	(870)	(1,277)	(2,505)					**0**														
Other Income, net	(2,215)	(168)	48	(818)	(790)	(1,729)					**0**														
Income (Loss) before Taxes	**(11,898)**	**(38,002)**	**(5,680)**	**(8,616)**	**(9,848)**	**(62,146)**	**(2,945)**	**(2,285)**	**(1,985)**	**(4,256)**	**(11,471)**	**(12,043)**	**(12,644)**	**(13,276)**	**(197,854)**	**(364,729)**	**(237,215)**	**(133,097)**	**514,937**	**763,294**	**943,822**	**1,025,528**	**1,093,249**	**1,162,077**	**1,231,332**
Tax Provision		0	0	0	0																				
Net Income (Loss)	**(11,898)**	**(38,002)**	**(5,680)**	**(8,616)**	**(9,848)**	**(62,146)**	**(2,945)**	**(2,285)**	**(1,985)**	**(4,256)**	**(11,471)**	**(12,043)**	**(12,644)**	**(13,276)**	**(197,854)**	**(364,729)**	**(237,215)**	**(133,097)**	**514,937**	**763,294**	**943,822**	**1,025,528**	**1,093,249**	**1,162,077**	**1,231,332**
Basic EPS, GAAP	**(0.69)**	**(2.10)**	**(0.30)**	**(0.45)**	**(0.46)**	**(3.20)**	**(0.14)**	**(0.11)**	**(0.09)**	**(0.19)**	**(0.53)**	**(0.53)**	**(0.53)**	**(0.54)**	**(7.69)**	**(13.65)**	**(8.56)**	**(4.63)**	**17.33**	**24.85**	**29.75**	**31.34**	**32.42**	**33.47**	**34.47**
Diluted EPS, GAAP	**(0.69)**	**(2.10)**	**(0.30)**	**(0.45)**	**(0.46)**	**(3.20)**	**(0.14)**	**(0.11)**	**(0.09)**	**(0.19)**	**(0.53)**	**(0.53)**	**(0.53)**	**(0.54)**	**(7.69)**	**(13.65)**	**(8.56)**	**(4.63)**	**17.33**	**24.85**	**29.75**	**31.34**	**32.42**	**33.47**	**34.47**
Weighted average shares outstanding, basic	17,343	18,135	19,027	19,212	21,221	19,399	21,346	21,596	21,846	22,096	21,721	22,721	23,721	24,721	25,721	26,721	27,721	28,721	29,721	30,721	31,721	32,721	33,721	34,721	35,721
Weighted average shares outstanding, diluted	17,343	18,135	19,027	19,212	21,221	19,399	21,346	21,596	21,846	22,096	21,721	22,721	23,721	24,721	25,721	26,721	27,721	28,721	29,721	30,721	31,721	32,721	33,721	34,721	35,721

Source: BTIG Research estimates, NTHI company reports

Exhibit 32. Balance Sheet

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NeOnc (NTHI) Balance Sheet

Period Ending $USD ('000s)	Dec-24 2024A	Mar-25 1Q25A	Jun-25 2Q25A	Sep-25 3Q25A	Dec-25 4Q25A	Dec-25 2025A	Dec-26 2026E	Dec-27 2027E	Dec-28 2028E	Dec-29 2029E	Dec-30 2030E	Dec-31 2031E	Dec-32 2032E	Dec-33 2033E	Dec-34 2034E	Dec-35 2035E	Dec-36 2036E	Dec-37 2037E	Dec-38 2038E	Dec-39 2039E	Dec-40 2040E
	-																				
Cash and cash equivalents	65	5,439	125	1,513	59	59	489	1,369	1,693	1,434	23,432	(294,297)	(472,184)	(528,189)	52,197	876,654	1,881,933	2,973,224	4,136,647	5,373,183	6,683,284
Deferred offering costs - current	1,072	97	97	99	75	75	75	75	75	75	75	75	75	75	75	75	75	75	75	75	75
Debt issuance costs - current	672	672	672	672	672	672	672	672	672	672	672	672	672	672	672	672	672	672	672	672	672
Prepaid expenses and other current assets	410	1,152	760	677	583	583	852	894	939	986	4,354	9,842	11,420	15,155	11,606	11,436	11,543	12,512	13,307	14,112	14,921
Inventory	-					-															
Total Current Assets	**2,219**	**7,360**	**1,654**	**2,962**	**1,389**	**1,389**	**2,088**	**3,010**	**3,379**	**3,167**	**28,532**	**(283,708)**	**(460,017)**	**(512,287)**	**64,550**	**888,837**	**1,894,224**	**2,986,483**	**4,150,701**	**5,388,042**	**6,698,951**
Property and equipment, net	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Debt issuance costs – net of current portion	1,199	1,031	863	695	527	527	527	527	527	527	527	527	527	527	527	527	527	527	527	527	527
Deferred offering costs - net of current portion		51	27	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Right of use asset – operating lease	-	-	398	380	361	361	361	361	361	361	361	361	361	361	361	361	361	361	361	361	361
Intangible assets					500	500	500	500	500	500	500	500	500	500	500	500	500	500	500	500	500
Other non-current assets	-	0	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47	47
Total Assets	**3,417**	**8,442**	**2,988**	**4,083**	**2,824**	**2,824**	**3,523**	**4,445**	**4,814**	**4,602**	**29,967**	**(282,273)**	**(458,582)**	**(510,852)**	**65,985**	**890,272**	**1,895,658**	**2,987,918**	**4,152,135**	**5,389,477**	**6,700,386**
	-																				
Accounts payable and accounts payable - related parties	3,546	4,143	3,573	2,141	7,113	7,113	7,113	7,113	7,113	7,113	7,113	7,113	7,113	7,113	7,113	7,113	7,113	7,113	7,113	7,113	7,113
Accrued advisory fee – related party	-	8,829	5,883	3,676	1,757	1,757	1,757	1,757	1,757	1,757	1,757	1,757	1,757	1,757	1,757	1,757	1,757	1,757	1,757	1,757	1,757
Litigation settlement payable	4,641	4,649	4,698	4,746	4,892	4,892	4,892	4,892	4,892	4,892	4,892	4,892	4,892	4,892	4,892	4,892	4,892	4,892	4,892	4,892	4,892
Accrued compensation	735	445	255	255	255	255	255	255	255	255	255	255	255	255	255	255	255	255	255	255	255
Lease liability, current			69	70	71	71	71	71	71	71	71	71	71	71	71	71	71	71	71	71	71
Other current liability	-			4,701	5,952	5,952	5,952	5,952	5,952	5,952	5,952	5,952	5,952	5,952	5,952	5,952	5,952	5,952	5,952	5,952	5,952
Total Current Liabilities	**8,922**	**18,065**	**14,477**	**15,588**	**20,041**	**20,041**	**20,041**	**20,041**	**20,041**	**20,041**	**20,041**	**20,041**	**20,041**	**20,041**	**20,041**	**20,041**	**20,041**	**20,041**	**20,041**	**20,041**	**20,041**
	-																				
Lease liabilities, non-current	-	-	327	309	291	291	291	291	291	291	291	291	291	291	291	291	291	291	291	291	291
Other non-current liability	-	-			-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
	-																				
Total Liabilities	**8,922**	**18,065**	**14,804**	**15,897**	**20,331**	**20,331**	**20,331**	**20,331**	**20,331**	**20,331**	**20,331**	**20,331**	**20,331**	**20,331**	**20,331**	**20,331**	**20,331**	**20,331**	**20,331**	**20,331**	**20,331**
	-																				
Common Stock	2	1.903	1.903	1.948	2.198	2	2	2	2	3	3	3	3	3	3	3	3	3	3	4	4
Treasury stock					(2,706)	(2,706)	(2,706)	(2,706)	(2,706)	(2,706)	(2,706)	(2,706)	(2,706)	(2,706)	(2,706)	(2,706)	(2,706)	(2,706)	(2,706)	(2,706)	(2,706)
Additional paid in capital	45,102	78,985	76,797	85,414	97,951	97,951	108,870	121,834	134,847	147,911	371,130	423,619	484,524	565,351	627,250	688,243	749,808	816,539	887,507	962,772	1,042,349
Accumulated deficit	(50,608)	(88,610)	(88,615)	(97,231)	(112,755)	(112,755)	(122,974)	(135,017)	(147,660)	(160,937)	(358,790)	(723,519)	(960,734)	(1,093,831)	(578,893)	184,401	1,128,222	2,153,750	3,247,000	4,409,076	5,640,408
Total Stockholders' Equity (Deficit)	**(5,505)**	**(9,624)**	**(11,815)**	**(11,814)**	**(17,508)**	**(17,508)**	**(16,809)**	**(15,886)**	**(15,517)**	**(15,730)**	**9,636**	**(302,604)**	**(478,914)**	**(531,183)**	**45,653**	**869,941**	**1,875,327**	**2,967,586**	**4,131,804**	**5,369,146**	**6,680,055**
	-					-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total Liabilities and Stockholders' Equity	**3,417**	**8,442**	**2,988**	**4,083**	**2,824**	**2,824**	**3,523**	**4,445**	**4,814**	**4,602**	**29,967**	**(282,273)**	**(458,582)**	**(510,852)**	**65,985**	**890,272**	**1,895,658**	**2,987,918**	**4,152,135**	**5,389,477**	**6,700,386**

Source: BTIG Research estimates, NTHI company reports

Exhibit 33. Cash Flow statement

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NeOnc (NTHI) Cash Flow Statement

Period Ending $USD ('000s)	Dec-24 2024A	Mar-25 1Q25A	Jun-25 2Q25A	Sep-25 3Q25A	Dec-25 4Q25A	Dec-25 2025A	Dec-26 2026E	Dec-27 2027E	Dec-28 2028E	Dec-29 2029E	Dec-30 2030E	Dec-31 2031E	Dec-32 2032E	Dec-33 2033E	Dec-34 2034E	Dec-35 2035E	Dec-36 2036E	Dec-37 2037E	Dec-38 2038E	Dec-39 2039E	Dec-40 2040E
Net income (loss)	(11,898)	(38,002)	(5,680)	(8,616)	(9,848)	(62,146)	(11,471)	(12,043)	(12,644)	(13,276)	(197,854)	(364,729)	(237,215)	(133,097)	514,937	763,294	943,822	1,025,528	1,093,249	1,162,077	1,231,332
Adjustments to reconcile net loss to net cash used in operating activities:																					
Share based compensation expense - restricted stock	-	23,074	(2,150)	5,040	9,591	35,555	919	965	1,013	1,064	23,219	52,489	60,905	80,827	61,899	60,993	61,565	66,731	70,968	75,265	79,577
Depreciation and amortization	391	577	210	410	(51)	1,147	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Increase in bridge loan - expenses paid by bridge loan provider on behalf of the Company	476					-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Write off deferred issuance costs	704					-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Loss on extinguishment of bridge loan	2,070					-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Loss on change in fair value of derivative liability				380	309	690	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Other	2,557	300	-	821	1,131	2,252	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
						-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Changes in operating assets and liabilities:						-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Prepaid expenses and other current assets	(124)	(766)	416	83	71	(197)	(269)	(43)	(45)	(47)	(3,368)	(5,488)	(1,578)	(3,735)	3,549	170	(107)	(969)	(794)	(806)	(808)
Accounts receivable	-					-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Inventory	-				-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Other assets	-		(47)	-	-	(47)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Accrued compensation	56	(290)	(190)	-	-	(480)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Lease liability	(245)		20	(58)	25	(12)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Accrued advisory fee	-	8,829	(2,946)	(2,207)	(1,918)	1,757	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Accounts payable and accounts payable - related parties	1,799	628	(583)	(1,703)	2,775	1,117	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Other liabilities	-				1,251	1,251	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Net Cash Used in Operating Activities	**(4,214)**	**(5,650)**	**(10,949)**	**(5,849)**	**3,336**	**(19,113)**	**(10,821)**	**(11,120)**	**(11,675)**	**(12,260)**	**(178,002)**	**(317,728)**	**(177,887)**	**(56,005)**	**580,386**	**824,457**	**1,005,279**	**1,091,291**	**1,163,423**	**1,236,536**	**1,310,101**
Purchases of property and equipment	-	-				-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Purchases of intangible assets					(500)	(500)															
Net Cash Provided (Used in) by Investing Activities	**-**				**(500)**	**(500)**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**
Proceeds from the sale of common stock	4,616	11,324	-	-	0	11,324	10,000	12,000	12,000	12,000	200,000	-	-	-	-	-	-	-	-	-	-
Proceeds from the sale of common stock – private placement					1,000	1,000															
Proceeds from related party loan	892	300	-	-	-	300	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Repayment of related party loan	(791)	(600)	-	-	-	(600)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Deferred offering costs	(470)	-	-	-		-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Proceeds from sale of common stock pursuant to equity purchase agreement				3,197	1,637	4,833	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Proceeds from convertible notes payable	-			4,000	-	4,000	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
	-					-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Net Cash Provided by Financing Activities	**4,247**	**11,024**	**-**	**7,197**	**2,637**	**20,858**	**10,000**	**12,000**	**12,000**	**12,000**	**200,000**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**
Effect of exchange rate changes on cash, cash equivalents	-					-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Net Increase (Decrease) in Cash, Cash Equivalents	**33**	**5,374**	**(10,949)**	**1,347**	**5,473**	**1,245**	**(821)**	**880**	**325**	**(260)**	**21,998**	**(317,728)**	**(177,887)**	**(56,005)**	**580,386**	**824,457**	**1,005,279**	**1,091,291**	**1,163,423**	**1,236,536**	**1,310,101**
Cash Beginning of Period	**32**	**65**	**5,439**	**(5,510)**	**(4,163)**	**65**	**1,310**	**489**	**1,369**	**1,693**	**1,434**	**23,432**	**(294,297)**	**(472,184)**	**(528,189)**	**52,197**	**876,654**	**1,881,933**	**2,973,224**	**4,136,647**	**5,373,183**
Cash End of Period	**65**	**5,439**	**(5,510)**	**(4,163)**	**1,310**	**1,310**	**489**	**1,369**	**1,693**	**1,434**	**23,432**	**(294,297)**	**(472,184)**	**(528,189)**	**52,197**	**876,654**	**1,881,933**	**2,973,224**	**4,136,647**	**5,373,183**	**6,683,284**

Source: BTIG Research estimates, NTHI company reports

BTIG Covered Companies Mentioned in this Report

NeOnc Technologies (NTHI, Buy, $15 PT; Closing Price: $4.95)
ImmunityBio (IBRX, Buy, $13 PT; Closing Price: $8.19)
Verastem, Inc. (VSTM, Buy, $19 PT; Closing Price: $6.23)

Appendix: Analyst Certification and Other Important Disclosures

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I, Jinnie Kim, hereby certify that the views about the companies and securities discussed in this report are accurately expressed and that I have not received and will not receive direct or indirect compensation in exchange for expressing specific recommendations or views in this report.

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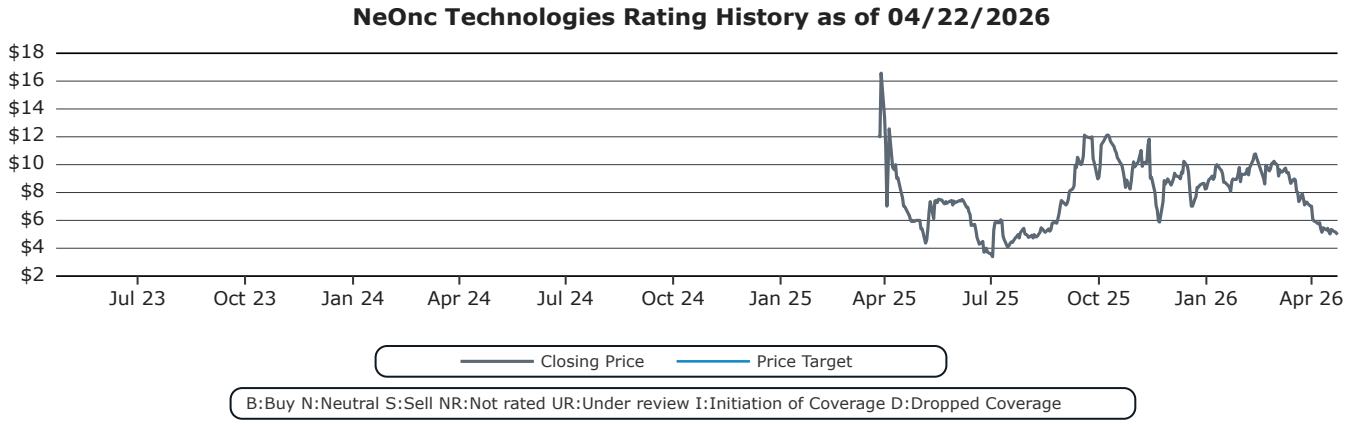
NeOnc Technologies (NTHI, Buy, $15 PT)

Valuation: We value NTHI using a DCF analysis (16% discount rate, 0.5% TG).
Risks: NeOnc is a clinical-stage biotech company. It faces all the standard upside and downside risks for that industry, including unexpected outcomes or safety signals from clinical readouts, regulatory uncertainty, and increasingly complex and price sensitive commercial markets for its product candidates.



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