INITIATING COVERAGE

Healthcare

Fair value: SEK7.0–12.0

Share price: SEK5.6

Elicera Therapeutics

Focused on the future of immuno-oncology

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We initiate coverage of Elicera Therapeutics, a Swedish clinical-stage immuno-oncology company developing next-generation cancer therapies. We see the company as an early-stage, platform-driven story where successful clinical validation of its iTANK technology could unlock meaningful long-term value. Our fair value range is SEK7–12 per share.

Platform-driven immuno-oncology strategy. Elicera's proprietary iTANK platform is designed to enhance CAR and TCR T-cell therapies by activating the broader immune system. The technology aims to address two major challenges in cell therapy: tumour heterogeneity (where tumours consist of different types of cancer cells) and the immunosuppressive tumour microenvironment (which can block or weaken the immune system's attack). By activating a broader anti-tumour immune response, iTANK may improve response durability and expand CAR and TCR T therapies into solid tumours. The biological rationale has been supported by preclinical data published in the peer-reviewed journal Nature Biomedical Engineering.

Lead asset approaching clinical validation. ELC-301 is an iTANK-armed CD20-directed CAR T therapy currently in Phase I/IIa development for relapsed/refractory diffuse large B-cell lymphoma (DLBCL), a setting with significant unmet medical need. Assuming successful development and launch around 2032e, we estimate peak sales potential of cUSD500m. Near-term catalysts include continued data from the CARMA study. Beyond ELC-301, the pipeline provides additional optionality through ELC-100, an oncolytic virus programme targeting neuroendocrine tumours that has reported data from a Phase I/IIa trial, and ELC-401, a preclinical CAR T programme targeting glioblastoma that is approaching the start of clinical trial.

Fair value range of SEK7–12 per share. Our fair value is based on a probability-adjusted SOTP valuation including ELC-301, ELC-100 and ELC-401, with ELC-301 representing the primary value driver (c40% of rNPV). We also assign a technology value to the iTANK platform in our SOTP valuation. In our view, further clinical de-risking of ELC-301 and validation of the iTANK platform would clearly support the equity story, although material risks related to clinical development and financing remain.

Changes in this report

	From	To	Chg
EPS adj. 2026e	n.a.	-0.52	n.a.
EPS adj. 2027e	n.a.	-0.84	n.a.
EPS adj. 2028e	n.a.	0.62	n.a.

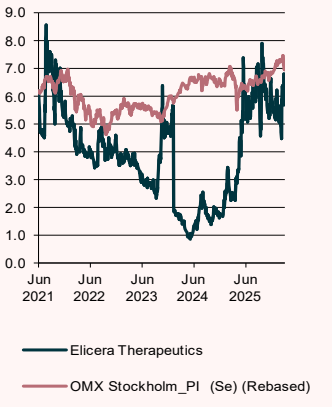
Upcoming events

Key facts

No. shares (m)	48.5
Market cap. (USDm)	30
Market cap. (SEKm)	274
Net IB Debt. (SEKm)	-52
Adjustments (SEKm)	0
EV (2026e) (SEKm)	222
Free float	76.8%
Avg. daily vol. ('000)	307
BBG	ELIC SS
Fiscal year end	December
Share price as of (CET)	12 Mar 2026 12:56

Key figures (SEK)	2025	2026e	2027e	2028e
Sales (m)	0	0	0	62
EBITDA (m)	-19	-25	-41	30
EBIT (m)	-19	-25	-41	30
EPS	-0.44	-0.52	-0.84	0.62
EPS adj.	-0.44	-0.52	-0.84	0.62
DPS	0.00	0.00	0.00	0.00
Sales growth Y/Y	n.a.	n.a.	n.a.	+chg
EPS adj. growth Y/Y	+chg	-chg	-chg	+chg
EBIT margin	n.m.	n.m.	n.m.	49.1%
P/E adj.	n.m.	n.m.	n.m.	9.0
EV/EBIT	neg.	neg.	neg.	8.0
EV/EBITA	neg.	neg.	neg.	8.0
EV/EBITDA	neg.	neg.	neg.	8.0
P/BV	12.3	5.8	42.6	10.0
Dividend yield	0.0%	0.0%	0.0%	0.0%
FCF yield	-7.1%	-9.2%	-14.9%	11.1%
Equity/Total Assets	76.2%	87.1%	48.0%	79.7%
ROCE	-85.6%	-72.4%	-152.0%	179.3%
ROE adj.	-85.6%	-72.4%	-152.0%	179.3%
Net IB debt/EBITDA	1.4	2.1	0.3	-1.1

Share price – 5-year



High/Low (12M) **SEK7.9/2.3**

Perf.	3M	6M	12M	YTD
Abs.	-4.08	-12.42	130.20	8.05
Rel.	-7.39	-20.27	123.44	6.68

Source: DNB Carnegie (estimates), FactSet, Infront & company data

This report has been commissioned and sponsored by Elicera Therapeutics. Commissioned research is considered to be marketing communication (i.e. not investment research under MiFID II). This material may be subject to restrictions on distribution in certain areas.

Equity story

Near term: within 12M

Elicera's near-term equity story is driven by clinical validation, with upcoming cohort readouts from the CARMA Phase I/IIa study the key catalyst. Data from additional cohorts is expected to further inform on safety, tolerability and early efficacy signals, which we believe is critical for de-risking the iTANK platform and supporting external interest. Beyond CARMA, and with increasing focus on ELC-401 in glioblastoma, the emphasis remains on strengthening the narrative around iTANK as a modular immune-enhancing platform applicable across multiple tumour types.

Long-term outlook: 5Y+

The success of CARMA is critical for Elicera, since it acts as a proof of concept in humans for the whole iTANK platform, which may in the end prove to be a springboard for development in solid tumours – a potentially significant opportunity in oncology, in our view. We believe Elicera's long-term equity story is anchored in the scalability of the iTANK platform across multiple cell-therapy modalities. Over a five-year horizon, we expect value creation to be driven by the continued clinical maturation of the CARMA programme into later-stage development, expansion into additional indications (including ELC-401 in glioblastoma) and potential partnering or out-licensing of the iTANK platform to third-party CAR T/TCR T developers.

Key risks:

- The most significant risk is related to clinical development – regulatory hurdles, potential delays and clinical trial failures
- As an early clinical stage biotech, Elicera relies on external financing, and we believe it will require additional financing to complete CARMA as well as support operations as a whole and continue developing the pipeline
- Partner dependence

Company description

Elicera Therapeutics is a Swedish clinical-stage biotechnology company developing next-generation immuno-oncology treatments, focusing on enhanced CAR T-cell therapies and oncolytic viruses. The company's proprietary and commercially available iTANK platform is an engineered 'arming' technology designed to boost the efficacy of CAR T therapies and oncolytic viruses, particularly in solid tumours. iTANK is already integrated into three of Elicera's lead candidates (ELC-301, ELC-401 and ELC-201) and is also available for out-licensing to other CAR T/TCR T developers.

Key industry drivers

- Increasing incidence and prevalence of cancer
- Ever growing demand for more effective treatments in difficult-to-treat population

Industry outlook

- The market for oncology drugs is expected to grow at double-digit rate over the coming five years (IQVIA)
- Larger pharmaceutical companies face patent cliffs, which we believe will continue to drive interest in in-licensing

Largest shareholders, capital

Avanza	7.9%
Di Yu	7.2%
Magnus Essand	7.1%

Cyclicality

Cyclicality: No

Not cyclical

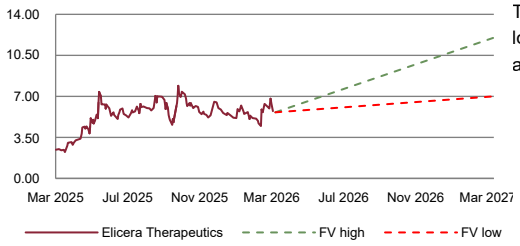
Key peers

BioInvent, Faron Pharmaceuticals

Valuation and methodology

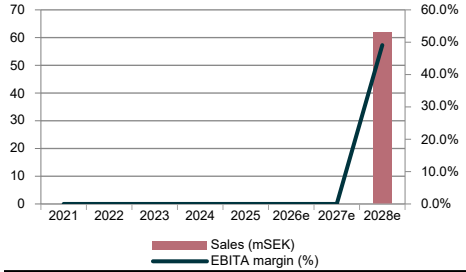
We value Elicera Therapeutics using a probability-adjusted cash flow model, evaluating each project individually and aggregating them in a SOTP model. Our valuation includes its lead assets ELC-301, ELC-401 and ELC-100, where we see a clear path forward, discounting future cash flows with a WACC of 14–20%. We also factor in the technology value of iTANK in our scenario analysis.

Fair value range 12M

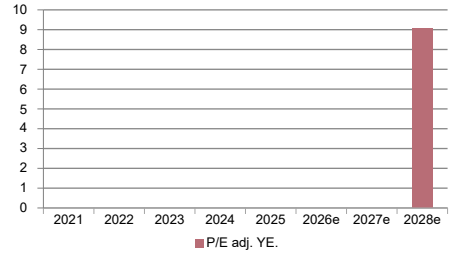


The upper end of our fair value range is derived from a SOTP analysis applying a WACC of 14%, while the lower end reflects a WACC of 20%. Our SOTP includes the company's key programmes ELC-301, ELC-401 and ELC-100, as well as the iTANK platform.

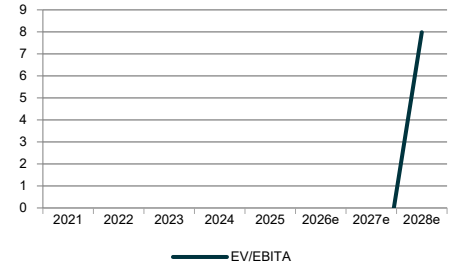
Key metrics



P/E 12-months forward



Long-term valuation trend



Source: DNB Carnegie (estimates) & company data

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Executive summary

We initiate coverage of Elicera Therapeutics with a fair value range of SEK7–12 per share. Elicera is a Swedish clinical-stage immuno-oncology company developing next-generation CAR T-cell therapies and oncolytic viruses, built around its proprietary iTANK immune-arming platform. The company currently has two clinical-stage programmes (ELC-301 and ELC-100) and two additional preclinical assets, offering exposure to a differentiated platform technology with potential applicability across multiple tumour types and cell therapy modalities. Overall, we believe Elicera offers high-risk, high-reward exposure to next-generation immuno-oncology through its modular platform strategy and focused pipeline. Successful demonstration of durable efficacy and manageable safety in the lead programme ELC-301 could represent the first human validation of the iTANK platform.

Differentiated immune-arming platform with broad applicability

In our view, Elicera's equity story centres on its proprietary iTANK platform, a modular technology designed to enhance the effectiveness of CAR and TCR T-cell therapies. The platform equips engineered T cells with an immune-activating component intended to strengthen the body's immune response against cancer within the tumour microenvironment. Cell therapies face two major biological challenges – first, cancer cells within the same tumour often differ, meaning therapies targeting a single marker may fail to eliminate all tumour cells. Second, the tumour environment can suppress immune activity, reducing the effectiveness of therapeutic T cells. iTANK is designed to address both challenges by strengthening immune activation and enabling a broader anti-tumour response.

Elicera's iTANK platform enhances engineered T cells with NAP to activate broader immune response. As a tumour-agnostic, add-on technology with early supporting data, we believe iTANK may offer scalability, partnering potential and strategic upside beyond a single clinical asset

The idea of iTANK is that it acts as an add-on layer that can be integrated into different cell therapy designs, potentially enhancing their ability to attack cancer cells. This modular approach makes the platform compatible with multiple tumour targets and therapy constructs. The biological rationale for the platform has been supported by preclinical data published in Nature Biomedical Engineering, and early clinical observations suggest the technology is active in patients. We believe this represents a key driver of the equity story, offering potential scalability, partnering opportunities and upside beyond any individual programme.

Clinical validation emerging from the lead programme

ELC-301: CD20-directed CAR T in r/r DLBCL. Early CARMA data shows promising activity and manageable safety at low doses. Targets CD20 to address relapse after CD19 therapies

We believe the value driver in the near term is the company's lead asset, ELC-301, an iTANK-armed CD20-directed CAR T therapy being developed in relapsed/refractory (meaning the cancer returns or does not respond to treatment, r/r) diffuse large B-cell lymphoma (DLBCL) in the third-line (3L) setting – an area of high unmet need despite recent therapeutic advances. DLBCL is the most common form of non-Hodgkin lymphomas – in the 7MM (the seven major markets: US, EU5 and Japan), there are approximately 65,000 new cases annually (source: LymphomaHub). Despite effective first- and second-line therapy, approximately 40% fail to respond, highlighting a significant unmet medical need in r/r DLBCL. Unlike approved CAR T products that target the 'usual' lymphoma antigen CD19, ELC-301 targets another antigen (CD20), offering a potential treatment alternative for patients who relapse after CD19-directed therapies due to antigen loss.

Early data from the ongoing Phase I/IIa CARMA study have shown encouraging signs of biological and clinical activity, including complete metabolic responses in heavily pre-treated patients at low dose levels, alongside a manageable safety profile. While patient numbers remain small and data immature, these early signals are important, in our view, as they provide first-in-human proof-of-concept for iTANK. The one-month follow-up data from all patients is planned for 2027, with full CARMA data readout expected in 2028–29, representing a key inflection point for both the asset and the broader iTANK platform.

Broader pipeline and platform optionality

Beyond ELC-301, Elicera's pipeline includes ELC-100, an oncolytic virus programme in neuroendocrine tumours (NET), and ELC-401, a preclinical CAR T programme targeting a type of brain cancer, glioblastoma – one of the most notorious and difficult-to-treat solid tumours in oncology. While ELC-401 remains an early-stage programme, preclinical development has now been completed and a preliminary Phase Ia/Ib design has been outlined, marking a tangible step toward clinical translation. However, the study remains subject to regulatory

feedback and manufacturing execution, and funding is not yet secured. We therefore view ELC-401 as a high-risk, high-reward opportunity that highlights iTANK's potential in solid tumours, particularly in challenging indications such as recurrent glioblastoma.

Business model rests on out-licensing – both assets and the platform

Elicera's broader pipeline includes ELC-100 for NETs and the early-stage, high-risk/high-reward ELC-401 CAR T programme for glioblastoma. Beyond internal development, the company aims to monetise its iTANK platform through non-exclusive out-licensing, offering potential upfronts, milestones and royalties.

Elicera's business model is not limited to internal development. iTANK is commercially available for out-licensing on a non-exclusive basis, enabling partners to integrate the technology into their own CAR T or TCR programmes. The company has already entered its first international research collaborations, and we see platform licensing as a potential source of future milestone and royalty revenues. While visibility into platform revenue potential remains low at this stage, we view iTANK as embedded strategic upside potential.

Attractive positioning for partnering in a deal-hungry market

With big pharma facing patent cliffs and seeking de-risked immuno-oncology assets, Elicera is positioned attractively for partnerships. Historical CAR T deals valued at USD1–6bn highlight strong demand for differentiated cell and gene therapy assets and platforms.

Large pharmaceutical companies continue to face patent cliffs and increasing R&D productivity pressure, reinforcing their reliance on external innovation. Despite cyclical volatility in biotech financing, partnering appetite has increasingly shifted toward de-risked, mid-stage assets, particularly within immuno-oncology. Assets that enhance existing modalities (rather than compete head-on with them) have historically been well received. Major players in the cell and gene therapy M&A field are among others Genetix Biotherapeutics, Roche, Astellas, Novartis and Takeda. Over the past 12 years, leading oncology-focused CAR T deals have reached total values of USD1bn–6bn, with higher values particularly for differentiated platforms partnered, which in our view further underscores the strong partnering appetite in the cell and gene therapy space. With ELC-301 approaching more mature clinical data and iTANK offering a differentiated, plug-in enhancement concept, we believe Elicera is well positioned for strategic partnering, both at the programme and platform level. Any such deal would not only provide non-dilutive capital but also serve as external validation of the technology.

Lean organisation backed by European CAR T pioneers

Elicera runs a lean organisation with strong scientific roots in early European CAR T development.

Elicera operates with a highly lean organisational structure, with only two full-time employees, while leveraging an extensive network of advisors. This capital-efficient setup keeps fixed costs low while maintaining access to deep expertise. We would like to note that the company's scientific foundation is particularly strong: in 2014, Prof Gunilla Enblad (advisor to Elicera) and her colleagues at Uppsala University were the first in Europe to treat cancer patients with CAR T therapy. Prof Magnus Essand from Uppsala University is Elicera's co-founder and Chief Scientific Officer. Founded with these deep roots in early European CAR T development, and with an experienced management team and board, we view Elicera as a small but scientifically well-anchored organisation with strong academic credibility.

High potential upside comes with high potential risk

Despite high expertise, the company faces significant risks: clinical execution challenges, reliance on future funding, limited institutional ownership and potential delays in development timelines.

Clinical and execution risk remains high, as oncology trials carry elevated attrition rates and setbacks in safety, efficacy, recruitment or operations could materially delay development. However, we believe main risk to the equity story is the financing risk. Elicera is pre-revenue and will require additional capital to advance its programmes, with non-dilutive grants insufficient to fund larger trials and not guaranteed. In the absence of a partnership, the company is likely reliant on equity markets, exposing shareholders to dilution risk. While we believe the current cash position supports operations into YE 2026, a capital raise appears probable without a strategic transaction.

Valuation

We reach a fair value range of SEK7–12 per share for Elicera. Our valuation is based on a risk-adjusted cash flow model (rNPV), where each project is assessed over its theoretical lifespan and then aggregated into a SOTP analysis. In our valuation, we include ELC-301, ELC-401, ELC-100, and the iTANK platform, with ELC-301 accounting for c40% of the rNPV. Our SOTP framework yields a valuation range of SEK7–12 per share, depending on discount rate assumptions. We have applied a WACC of 14–20% – we believe a high WACC is warranted given Elicera's early-stage clinical profile, lack of product revenues and dependence on external financing.

Expected news flow

As a company without marketed products and focused on developing innovative treatments, we believe Elicera's share price is highly sensitive to news flow. Over the short- and long-term, we expect a rich stream of updates, with clinical data and a potential licensing agreement being the key catalysts for the shares.

Potential news flow overview and our view on its impact on valuation

Timing	Impact	Event	Comment
2026e -	Major	Potential collaborations and licensing deals	iTANK
2026e -	Major	Potential collaborations and licensing deals	ELC-301
2026e -	Major	Potential collaborations and licensing deals	ELC-100
2026e -	Modest	Strategical update on way forward	ELC-100
2026e -	Modest	Strategical update on way forward	ELC-201
2027e -	Major	Potential trial start in GBM	ELC-401
2027e -	Major	Preliminary ORR data (1-month follow-up) – CARMA	ELC-301
2028–29e	Major	Full data readout for CARMA	ELC-301

Source: DNB Carnegie

Company introduction

Elicera Therapeutics is a Swedish clinical-stage biotechnology company developing next-generation immuno-oncology treatments, focusing on enhanced CAR T-cell therapies and oncolytic viruses. The company's proprietary and commercially available iTANK platform is an engineered 'arming' technology designed to boost the efficacy of CAR T therapies and oncolytic viruses. iTANK is already integrated into three of Elicera's lead candidates (ELC-301, ELC-401 and ELC-201) and is also available for out-licensing to other CAR T developers.

The company known today as Elicera Therapeutics originated in 2014, when it was founded as ViRex AB based on Prof Magnus Essand's research at Uppsala University. By 2016, the company had already filed a patent application for the iTANK platform and initiated its first Phase I/II clinical study using oncolytic viruses. Elicera Therapeutics was listed on Nasdaq First North in 2021, and in 2022 further strengthened its IP position for one of the lead candidates (ELC-100) through the acquisition of a patent from Immunicum.

The pipeline today consists of four programmes: two CAR T therapies (ELC-301 and ELC-401) and two oncolytic viruses (ELC-201 and ELC-100). ELC-100 is in completed Phase I/II trial with results recently published, while the Phase I/IIa CARMA study for ELC-301 was initiated in November 2024. Elicera is advancing ELC-401 towards a planned Phase Ib trial in H2 2027, pending regulatory alignment and manufacturing readiness, and ELC-201 remains in preclinical development. The programmes target indications with significant unmet need, including neuroendocrine tumours (NET), B-cell lymphoma (diffuse large B-cell lymphoma – DLBCL) and glioblastoma.

Elicera's business model is built on out-licensing both its iTANK platform and, over time, its individual drug candidates. iTANK is ready for commercialisation via non-exclusive licences, while the internal pipeline is expected to be partnered on a more exclusive basis. For smaller biotech firms, it is common to rely on partnerships for future income, and Elicera is no exception. The company expects its revenues to come from upfront payments, milestone payments and royalties.

Elicera Therapeutics' pipeline

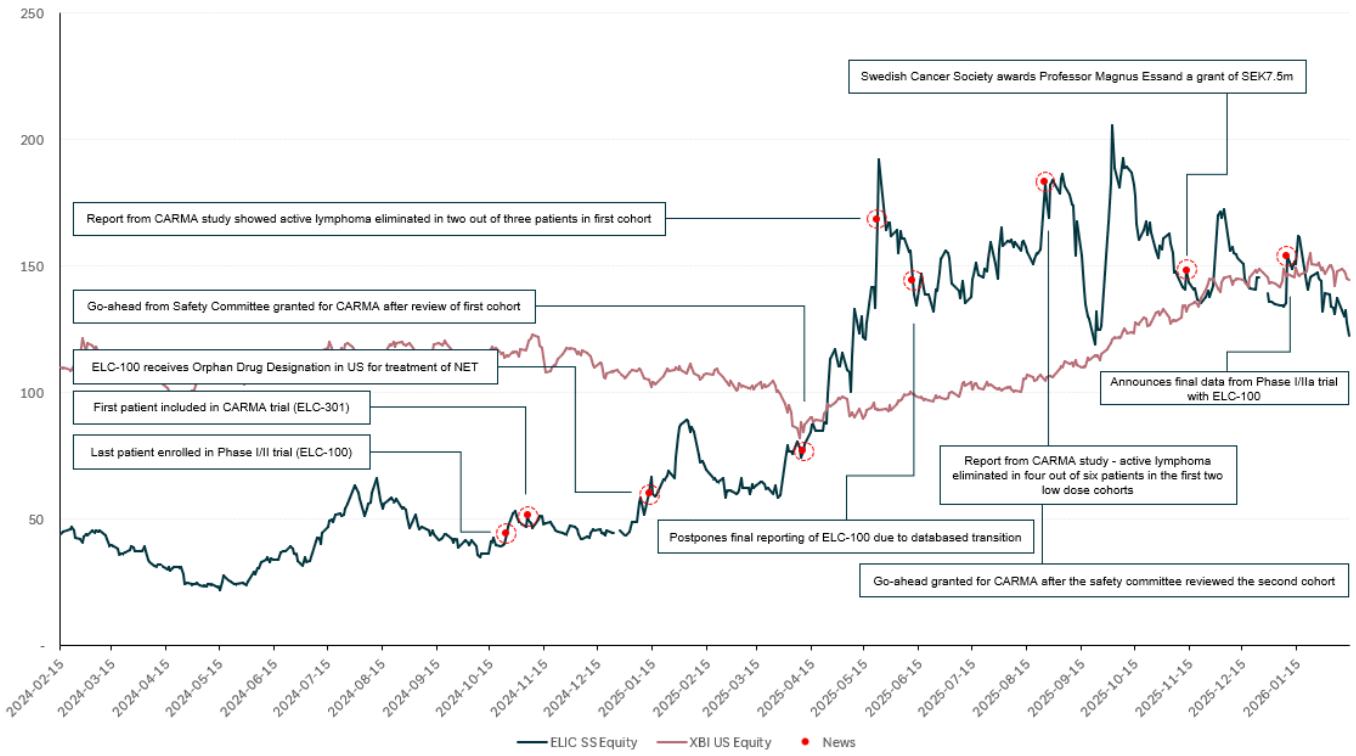
Project	Mechanism	Indication	Pre-clinical	Toxicology studies	Ph I/II	Ph II	Ph III	Market approval
ELC-100	**OV**	NET						2031e
ELC-301	**CAR T**	DLBCL						2032e
ELC-201	**OV**	-						-
ELC-401	**CAR T**	Glioblastoma						2035e

Completed
Ongoing

Source: Company, DNB Carnegie (time estimates)

News flow

News flow 2024–26



Source: Company, MFN, DNB Carnegie (chart visualisation)

In the past two years, we have seen a significant shift into clinical execution and progress across both lead programmes – ELC-100 and, more importantly, ELC-301. Below is a selection of key news topics.

2024–26

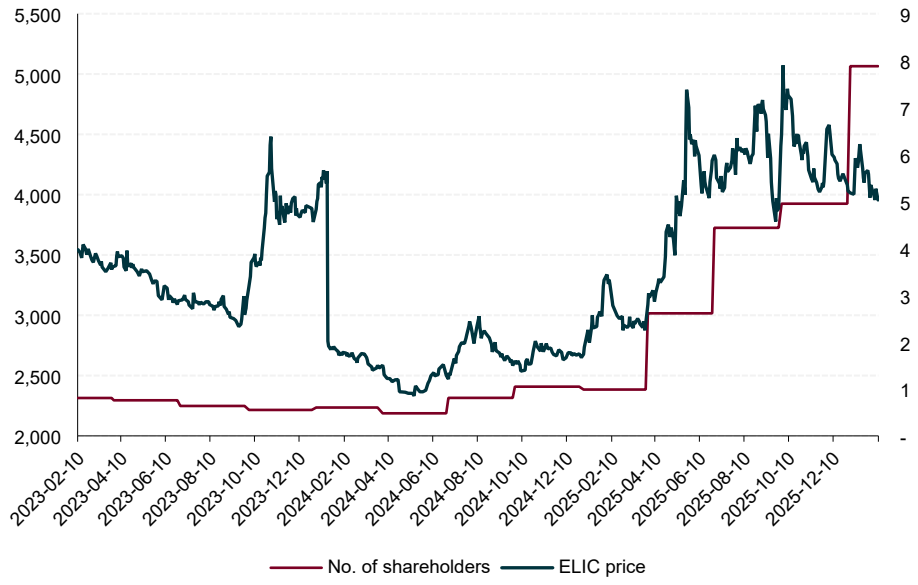
- Completed enrolment in the Phase I/II trial for ELC-100 (October 2024).
- Received approval to initiate the Phase I/II CARMA study with ELC-301; first patient dosed (November 2024).
- Obtained Orphan Drug Designation in the US for ELC-100 in NET (January 2025).
- Postponed final ELC-100 reporting due to a database transition (June 2025).
- CARMA (ELC-301) trial updates: safety committee green-light statements and early results showing elimination of active lymphoma in 4/6 patients in low-dose cohorts (August 2025), including heavily pre-treated patients and among these one individual who had previously failed standard CAR T therapy and achieved a complete metabolic response one month after treatment with ELC-301.
- Final data presented from the ELC-100 Phase I/IIa trial (January 2026), demonstrating partial responses in 2/8 efficacy-evaluable, heavily pre-treated and terminally ill patients.

Share price and ownership

Share price and ownership trends have shifted notably since 2022, a period during which the company moved from having a single clinical asset to progressing two lead candidates into clinical development. In our view, the most substantial shifts occurred in 2025.

In 2025, the share price climbed by around 200%, mainly on the basis of strong interest from retail investors. By the end of the year, the number of shareholders had more than doubled compared with January – from c2,200 to 5,000. Institutional ownership remains very low, however, making up only 0.02% of the total (Source: Holdings).

Share price and shareholder development – 2022–25



Source: Bloomberg, Holdings, DNB Carnegie (chart visualisation)

The iTANK platform

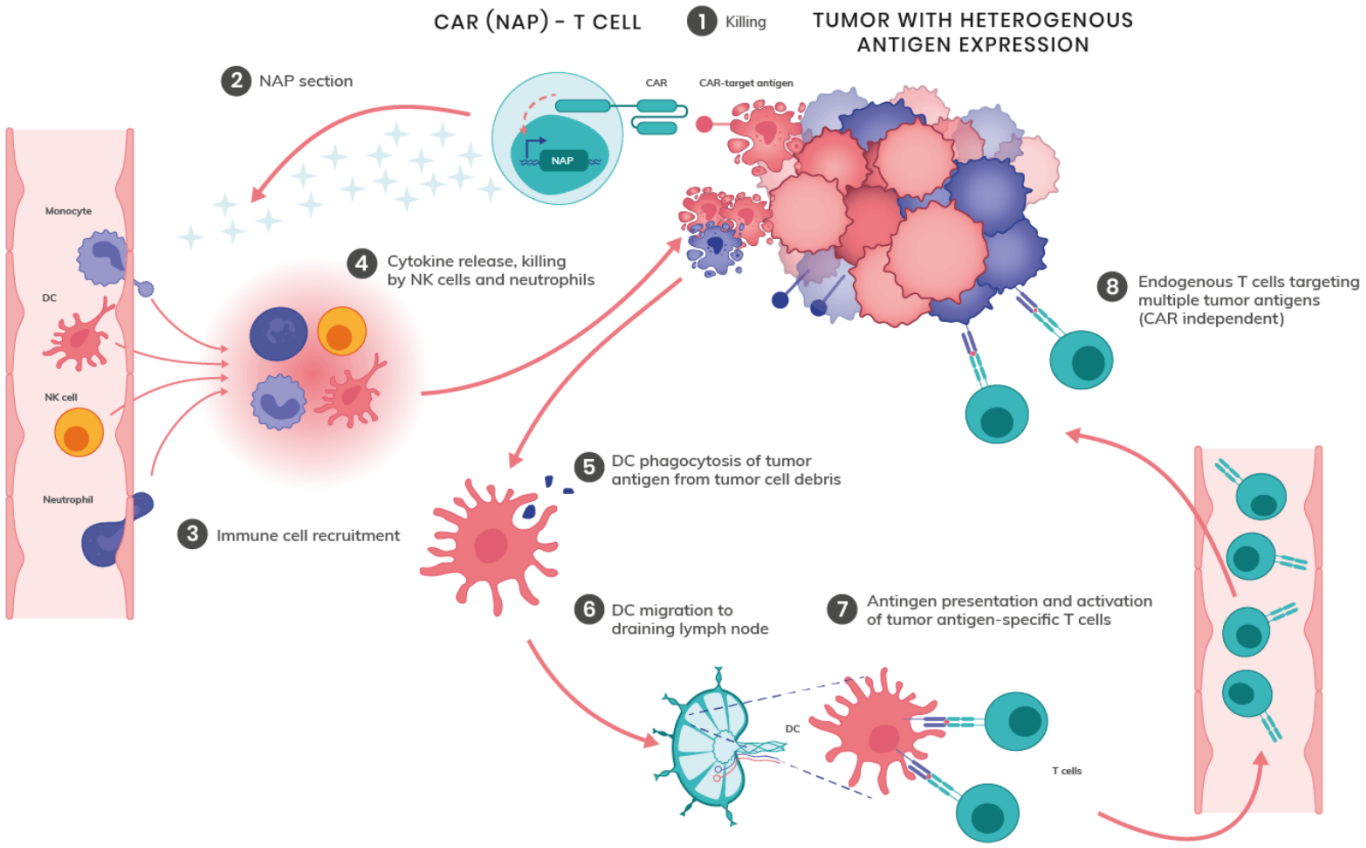
Elicera has developed a proprietary technology platform, iTANK (immunoTherapies Activated with NAP for efficient Killing), aimed at addressing two of the major challenges faced by therapies in the treatment of solid tumours – an immunosuppressive tumour microenvironment and heterogenous tumour target antigen expression. Put simply, the immunosuppressive tumour environment means that solid tumours create a 'hostile' neighbourhood for immune cells, releasing signals that weaken or shut down CAR T cells once they arrive, so the therapy cannot work as effectively or for as long as intended. When it comes to heterogeneous antigen expression, it essentially means that not all cancer cells 'look' the same. Some do not carry the specific marker (or target) that CAR T cells are designed to recognise, which means those cells can 'hide' from the treatment and continue to grow.

Mode of action

In general, CAR T cells are made by taking a patient's own T cells and genetically modifying them so they can better recognise cancer. The modification adds a new synthetic receptor to the T-cell surface, called a chimeric antigen receptor (CAR). The outer part of the CAR is designed to recognise and bind to a specific protein found on cancer cells, such as CD19 or CD20 on B-cell lymphomas. Once the CAR attaches to its target, the inner part of the receptor sends strong activation signals inside the T cell. This activates the T cell, makes it multiply, release immune signals and kill the cancer cell (source: Cancer Discovery). We will return to a more detailed discussion of how CAR T cells work later in the report.

Elicera's platform iTANK equips CAR or TCR T cells with a pluripotent pro-inflammatory neutrophil-activating protein (NAP) derived from Helicobacter pylori, which induces so-called 'bystander immunity' through epitope (a small part of an antigen) spreading. The system is independent of tumour type and target antigen. iTANK-enhanced CAR/TCR T cells induce a pro-inflammatory microenvironment that both strengthens CAR/TCR T-cell function and activates the patient's own cytotoxic T cells against a broad range of relevant tumour antigens (Source: company). The mechanism of action is described in a scientific article published in Nature Biomedical Engineering, see diagram and description below.

Mode of action – how CAR(NAP) T cells work



Source: Company, Nature Biomedical Engineering

- **Step 1–2:** The killing commences. Upon tumour engagement, CAR(NAP) T cell secretes the immune-activating protein NAP.
- **Step 3–4:** Immune cells are recruited and cytokines are released. Once engaged CAR(NAP) T cells engage with tumour cells, immune cells such as neutrophils and Natural Killer (NK) cells are recruited, which once activated attack tumour cells regardless of whether the tumour cells express the CAR target.
- **Step 5:** Clean-up from tumour cell debris. NAP also recruits monocytes and dendritic cells (DC) and activates them to release pro-inflammatory cytokines that counteract the immunosuppressive tumour microenvironment and further enhance effect. DCs phagocytose ("eat up") debris from the dying tumour cells and mature.
- **Step 6:** Once matured, DCs migrate to draining lymph nodes where tumour antigens are processed and presented to other T cells.
- **Step 7:** Activation of tumour antigen-specific T cells – in other words T cells with matching T cell receptors.
- **Step 8:** Further, these T cells circulate back to the tumour, where they "kill" tumour cells through so-called TCR-mediated recognition independent of the original CAR target.

In published preclinical studies, CAR T cells enhanced with iTANK performed better than standard CAR T cells, showing stronger tumour control and improved survival. The approach also worked well in tumours with mixed antigen expression and helped turn the tumour environment 'hotter' and more immune-active. This triggered broader immune responses,

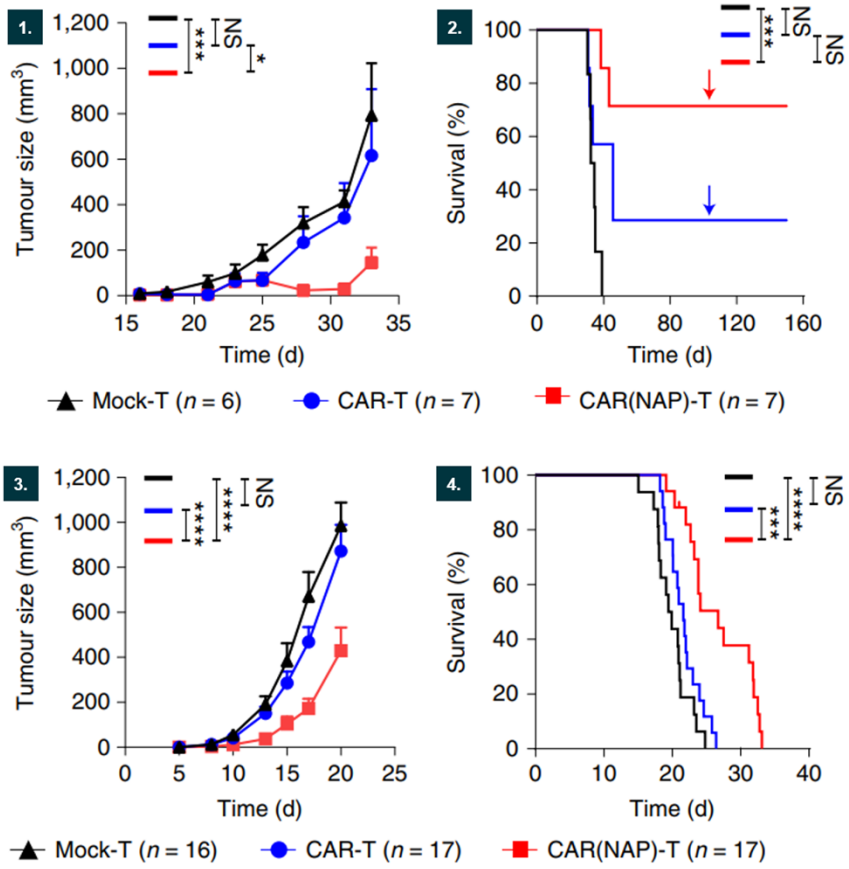
including epitope spreading and the recruitment of highly effective killer T cells. By creating a more inflammatory environment that reduces immune suppression, iTANK boosted CAR T activity across different tumour types and models, suggesting it can be combined with many different CAR designs (Source: company).

Preclinical data

Elicera's iTANK platform is supported by preclinical data. In a study published in Nature Biomedical Engineering in 2022, it was shown that CAR (NAP) T cells induced significantly stronger anti-tumour responses compared to conventional CAR T cells across multiple solid tumour models.

The addition of NAP reshaped the tumour microenvironment by promoting a pro-inflammatory, Th1-type immune response. This indeed led to increased recruitment and activation of innate immune cells such as dendritic cells, neutrophils and NK cells, as well as enhanced infiltration of cytotoxic CD8+ T cells (source: Nature Biomedical Engineering).

Preclinical data in tumour models



Source: Nature Biomedical Engineering, 2022 (https://doi.org/10.1038/s41551-022-00875-5)

Figures 1 and 2 show the treatment effect in a preclinical tumour model in mice (A20). Panel 1 presents the mean tumour size over time, measured until the first mouse in the group had to be euthanised due to tumour burden. This illustrates how different treatments influenced tumour growth dynamics. Panel 2 shows overall survival using a Kaplan–Meier curve. The arrows in this panel indicate that mice which had achieved complete tumour regression were later rechallenged with new A20 tumour cells to test whether immunological memory had developed. Figures 3 and 4 present corresponding data in a second tumour model, NXS2. Panel 3 shows the mean tumour size over time until the first mouse required euthanasia, reflecting tumour growth control. Panel 4 displays survival outcomes using a Kaplan–Meier curve. These panels demonstrate both tumour growth inhibition and survival benefit, as well as the ability of the therapy to induce durable immune protection in cured animals.

In an article published in Cancer Immunology and Immunotherapy, CD20-targeted CAR (NAP) T cells were evaluated on pre-clinical safety and efficacy. The study design combined both in vitro and in-vivo experiments, along with dedicated safety assessments. In vitro, the researchers tested rituximab-derived CD20 CAR T cells against multiple human CD20-positive lymphoma cell lines, as well as primary mantle cell lymphoma (MCL) cells obtained from a patient who had relapsed after rituximab treatment. In-vivo efficacy was assessed in murine

lymphoma models. This study demonstrated that CD20-targeted CAR T cells are functional and clinically relevant in B-cell lymphoma models, and that arming these cells with NAP enhances in vivo antitumour efficacy without introducing evident systemic toxicity in preclinical models. Taken together, the data support further clinical development of CD20-targeted, NAP-armed CAR T cells in relapsed or refractory B-cell lymphomas, particularly in settings where antigen escape may limit the durability of conventional CAR T therapies (source: Cancer Immunology, Immunotherapy, 2025).

Business model for iTANK

The platform is intended to be licensed out to international pharmaceutical companies developing CAR T-cell therapies, particularly within solid tumours. The business model is based on signing multiple licensing agreements for iTANK, enabling partners to advance future CAR T/TCR T programmes while generating upfront payments, milestone payments and royalties for Elicera. By building a growing portfolio of such agreements, the company aims to establish a recurring revenue stream while continuously developing and strengthening the iTANK platform and its market position.

In this area, Elicera has already entered into its first international collaboration agreements.

- In Germany, the company signed a material transfer agreement (MTA) with University Hospital Tübingen to include ELC-100 and ELC-201 (armed with iTANK) in testing aimed at developing a companion diagnostic for immuno-virotherapy in NETs, potentially helping identify patients most likely to respond to treatment.

- In China, Elicera partnered with the Fourth Affiliated Hospital of China Medical University to evaluate ELC-201 in preclinical proof-of-concept studies across multiple solid tumours using patient-derived organoids, generating data on efficacy and immune activation.

- In the US, Elicera entered an MTA with a leading cancer centre to assess the enhancing effect of iTANK when arming a novel TCR-T therapy targeting melanoma and lung cancer.

The company provides limited detail on the structure and scope of these agreements, and updates tend to be communicated on a sporadic basis. Nevertheless, we believe these collaborations remain strategically valuable as they generate external validation, broaden the potential applicability of the platform and may strengthen future partnering and licensing opportunities. These arrangements do not grant partners any intellectual property rights to the iTANK platform itself, while any patentable inventions arising from the collaborations are expected to be jointly owned by Elicera and the respective partner.

Technology platform valuation – not an easy task

Valuing a platform technology such as iTANK is inherently complex, in our view, as its potential extends beyond a single asset or indication. Unlike a defined CAR T programme with a specific target and clinical pathway, iTANK is positioned as a universal arming technology that can be applied across CAR and TCR-based therapies, independent of tumour type or antigen. This significantly broadens its theoretical addressable market – but also makes direct benchmarking difficult.

As described above, iTANK aims to overcome antigen heterogeneity and the immunosuppressive tumour microenvironment in solid tumours, with preclinical data supporting its potential as a broadly compatible, modular enhancement platform. However, translating this into valuation terms is challenging. Platform value depends on future validation across multiple programmes, potential out-licensing structures, breadth of IP protection, and the willingness of partners to integrate an external immune-arming component into their own CAR/TCR assets. We do not assign a standalone valuation range to iTANK at this stage. Instead, we view it as embedded strategic upside potential to the equity story.

Case study – Noile-Immune Biotech

To illustrate how a platform-based biotechnology business model may evolve, we highlight Noile-Immune Biotech as a relevant case example. Noile-Immune Biotech is a Japanese biotechnology company founded in 2015 and focused on developing next-generation cancer immunotherapies, particularly CAR T cell therapies.

The company's core technology is the PRIME CAR T platform, a next-generation CAR T approach designed to address key limitations of conventional CAR T therapy, particularly in solid tumours. According to the company website, the PRIME platform modifies CAR T cells so that, in addition to expressing a tumour-targeting CAR receptor, the engineered cells also secrete two immune-regulating molecules: interleukin-7 (IL-7) and the chemokine CCL19. These molecules play important roles in the immune system by supporting T-cell survival, expansion and immune cell trafficking (source: Noile-Immune Biotech)

Through this mechanism, PRIME CAR T cells aim to enhance CAR T cell proliferation and persistence, recruit additional immune cells such as endogenous T cells and dendritic cells to the tumour site, and improve immune cell infiltration into solid tumours. Conceptually, this approach is not fundamentally different from Elicera's iTANK platform, which also aims to enhance CAR T efficacy by stimulating a broader immune response within the tumour microenvironment. While the two technologies rely on different immune-modulating molecules, both platforms are designed to recruit and activate additional immune cells, and promote epitope spreading. In essence, the PRIME CAR T platform functions as a modular enhancement technology that can be incorporated into different CAR T or TCR therapies to improve their activity in solid tumours, illustrating a platform concept that we believe is comparable to Elicera's iTANK approach.

Noile has pursued a partner-driven development strategy, positioning its technology as a platform add-on that can enhance other companies engineered cell therapies. Through research collaborations and licensing agreements, the company has partnered with several leading cell-therapy developers and pharmaceutical companies, see a selection of partnerships below.

A selection of partnership agreements for Noile-Immune Biotech

Partner	Year	Deal type	Scope	Economics / Key terms
Takeda	2017	Research collaboration	Discovery and development of CAR T therapies using PRIME platform	Technology access payment, Takeda equity investment, option for licensing rights
Adaptimmune	2019	Co-development collaboration	Integration of PRIME platform into Adaptimmune's SPEAR TCR T therapies	Up to **USD312m** milestones across programs + mid-single-digit royalties
Autolus	2019	Exclusive license	Use of PRIME platform in selected CAR-T programs	Upfront payment, development milestones and royalties (undisclosed)
CHUGAI A member of the Roche group	2022	License agreement	Development of PRIME-based CAR T therapies for selected targets	Upfront payment, tech transfer fees, **cUSD132m in milestones**, royalties

Source: Noile-Immune Biotech press releases, Takeda, Chugai

Pipeline candidates

ELC-301

ELC-301 is being developed for patients suffering from a particularly severe form of DLBCL or who suffer relapse after several rounds of standard treatment (so-called relapsed/refractory disease, r/r).

In November 2024, Elicera started the CARMA study, a clinical Phase I/IIa trial with ELC-301 in patients with r/r DLBCL. CARMA is conducted in two parts: a dose-escalation phase (Phase I) and a dose-expansion phase (Phase IIa). The initial part was planned to include three cohorts (dosing groups) with three patients in the first and second dosing groups and six patients in the third dosing group. The objective of the first part of the trial is to identify the optimal dose for treatment with the CAR T-cell therapy ELC-301, which will then be tested in an additional six patients in the Phase IIa part of the study. CARMA has two locations – Karolinska University Hospital in Stockholm and Uppsala University Hospital – with Prof Gunilla Enblad as principal investigator.

CARMA is an open label study, in other words a type of study in which both the health providers and the patients are aware of the drug or treatment being given. This allows data to be reviewed and disclosed sequentially after each cohort without risking unblinding or introducing bias between treatment arms.

The CARMA study is partly financed with EUR2.5m in grants from the EIC Accelerator Fund. Our understanding is that the majority of the payout from the grant has been executed. The company is open with its strategy of looking for soft financing to continue execution on its projects. See more information on clinicaltrials.gov using the ID NCT06002659 to search the database.

CARMA trial design

Subjects	Patients with r/r DLBCL in 3L setting
Trial arm design	▪ Open study with 18 subjects (12 in Phase I – dose escalation and 6 in Phase IIa – maximum tolerable dose) ▪ 1 dose of ELC-301 (CD20 CAR(NAP) T-cell) ▪ Dose levels $2x10^5$, $6x10^5$, $2x10^6$
Primary endpoints	Incidence of dose limiting toxicity (DLT)
Secondary endpoints	▪ ORR, PFS, OS, DoR ▪ Additional efficacy endpoints including CAR T-cell persistence and immune status profiling

ORR (Overall Response Rate): *the proportion of patients whose tumours shrink or disappear (partial or complete response) following treatment.*

PFS (Progression-Free Survival): *the length of time during and after treatment that a patient lives without the cancer worsening.*

OS (Overall Survival): *the length of time from treatment start (or diagnosis) until death from any cause.*

DoR (Duration of Response): *how long a tumour response (partial or complete) lasts before disease progression.*

Source: Company, NCI National Cancer Institute

Clinical data – ELC-301

Elicera posted its latest update from CARMA in August 2025. Early efficacy signals were observed across the first two dose cohorts. In Cohort 1 (lowest dose; n=3), two patients achieved complete metabolic response (CMR) at one month, with one maintaining disease-free status at 12 months, while the third patient had stable disease at one month before progressing at three months. In Cohort 2 (n=3), two patients achieved CMR at one month, with one confirmed disease-free at three months, and the remaining patient achieved a partial response that was sustained at the three-month follow-up.

Updated data from CARMA was published on 6 March. The first two patients treated in cohort three (highest dose level) achieved complete metabolic response (CMR) at the one-month follow-up, with no dose-limiting toxicities observed. Across the eight patients treated to date, the disease control rate is 100%, the overall response rate is 7/8, and six patients achieved CMR at month one, of which four maintain CMR at their latest follow-up, with the longest responses lasting at least 12 months. Recruitment in cohort three (targeting six patients) is ongoing.

The company expects to present one-month follow-up data from all 18 patients in 2027, with the final report, including two-year follow-up data for all patients, planned for 2029.

Competitive positioning of ELC-301

Today's approved CAR T-cell therapies all target the tumour target CD19, which is a common B-cell protein that is overproduced on the surface of cancer cells in DLBCL. According to Elicera, in many of the individuals who suffer from relapse, this tumour target 'disappears' and further treatments with the same CAR T therapy therefore become ineffective. ELC-301 instead targets CD20, which is also overrepresented in B-cell lymphoma. By switching targets to CD20, ELC-301 enables the treatment of relapsed patients who need a new effective alternative.

Given the current treatment landscape of DLBCL (also see section Disease overview – DLBCL), ELC-301 is being developed as a third line (3L) or beyond therapy for DLBCL, i.e. intended for patients whose disease has relapsed or is refractory after at least two prior lines of standard treatment.

Later in the report, we highlight the highly competitive CAR T field, with many ongoing trials. However, if looking only at the DLBCL field, we do not see as many active CAR T trials. We have identified six CAR T programmes in the late-stage pipeline for treatment of r/r DLBCL, see summary below.

A selection of CAR T projects in the pipeline for B-cell malignancies

Asset	Company	Target(s)	Modality	Comment
ALLO-501	Allogene Therapeutics	CD19	Allogeneic CAR T	Off-the-shelf CAR T addressing manufacturing time and scalability limits of autologous CAR Ts
Zamtocabtagene autoleucel	Miltenyi Biomedicine	CD19, CD20	Autologous dual-target CAR T	Dual-antigen targeting designed to reduce relapse from antigen escape
CRG-022	CARGO Therapeutics	CD22	Autologous CAR T	Next-generation CAR T targeting CD22 in r/r B-cell malignancies
KITE-363; KITE-753	Gilead Sciences	CD19/20	Autologous CAR T	Multiple autologous CAR T candidates across r/r B-cell malignancies (Phase I–III)
Rapcabtagene autoleucel	Novartis	CD19	Autologous CAR T	CD19-directed CAR T in Phase I/II for r/r B-cell malignancies including DLBCL
JNJ-90014496	Jansen	CD19/20	Autologous CAR T	Dual-target CAR T for r/r B-cell NHL and high-risk DLBCL

Source: GlobalData, Bloomberg Intelligence, Gilead Sciences, Novartis

However, we believe the competitive landscape in DLBCL should also be looked at from the point of view of other modalities as well. It is worth noting that 3L treatment in DLBCL is evolving fast, and the rapid uptake of off-the-shelf bispecific antibodies, along with pricing pressure and differentiation requirements, could significantly change the landscape even once ELC-301 potentially gains approval. Rather than CAR Ts, we view ADCs and bispecific antibodies as the main competition to ELC-301. One direct competitor to ELC-301 in the 3L setting highlighted by Elicera is epcoritamab, a bispecific antibody that was developed by Genmab and AbbVie and was approved as a third-line therapy for DLBCL in 2023. In the Phase I/II EPCORE NHL-

1 study (NCT03625037), subcutaneous epcoritamab demonstrated strong antitumour activity in heavily pretreated patients with r/r large B-cell lymphoma, including those with prior CAR T exposure. Among 157 patients, the overall response rate was 63%, with a complete response rate of 39%. Responses were durable, with a median duration of response of 12 months, and the median duration among complete responders had not yet been reached at the time of analysis which was later published in Journal of Clinical Oncology (DOI: 10.1200/JCO.22.01725). Epcoritamab requires repeated doses, however, which is not the case for ELC-301.

Earlier in December 2025, ADC Therapeutics also published its data from LOTIS-7 phase Ib trial of its ADC drug Zynlonta in combination of with the bispecific antibody glofitamab. Results showed strong efficacy in r/r DLBCL. Among 49 efficacy-evaluable patients with at least six months of follow-up, the overall response rate was 89.8% and the complete response rate was 77.6% (source: ADC Therapeutics press release).

ELC-100

Elicera's oncolytic virus AdVince (ELC-100) is based on a genetically modified adenovirus, Ad5PTD, that has been optimised with regard to its ability to selectively infiltrate and replicate in neuroendocrine cancer cells. When new virus particles have formed in enough numbers, tumour cells burst through a so-called immunogenic oncolysis, releasing factors that can also activate the immune system. When the tumour cell dies, an immune response to the cancer is also triggered by the release of neoantigens which are picked up by the patient's antigen-presenting dendritic cells, which then train T-cells to attack cancer cells wherever they are in the body. In this way, AdVince is expected to achieve a local tumour-killing effect in the injected tumour while simultaneously triggering a long-term and systemic immune response through a T-cell attack on cancer cells in other parts of the body as well (Source: company).

In 2024, ELC-100 was granted Orphan Drug Designation (ODD) by the US FDA for the treatment of a subgroup of NETs – pancreatic neuroendocrine tumours (c10% of total NET population in the US (source: JAMA Oncology). ODD is awarded to therapies targeting rare diseases affecting fewer than 200,000 patients in the US and provides benefits such as tax credits for US clinical trials, fee waivers for marketing applications, and up to seven years of market exclusivity upon approval.

Elicera Therapeutics reported final data from its Phase I/IIa trial of the oncolytic virus ELC-100 in patients with advanced, end-stage neuroendocrine tumours (NET) who had exhausted all available treatment options. The study met its primary objective, demonstrating a favourable safety profile with no dose-limiting toxicities observed across 12 treated patients. Adverse events were consistent with the expected inflammatory response of an oncolytic virus, and the maximum tolerated dose was established at 1×10^{12} virus particles, the highest dose tested.

The trial also delivered early signals of clinical activity in a highly treatment-resistant population. Among the eight efficacy-evaluable patients, partial tumour responses were observed in two patients, and 75% remained progression-free 12 weeks after completion of the fourth treatment cycle. Patients were treated across four escalating dose cohorts and received up to eight administrations of ELC-100 via hepatic artery infusion or intratumoral injection.

These results provide initial proof of concept for both safety and anti-tumour activity of ELC-100 in advanced NET. Management has indicated that, based on these findings, the company will now evaluate strategic options for the continued development of the programme.

Competitive positioning of ELC-100

We consider the competitive and treatment landscape for ELC-100 to be quite different to that in DLBCL. Currently, our understanding is that NET research is focused on combination approaches and immune modulation. A wide array of clinical trials are exploring different combination therapies, ranging from radiopharmaceuticals and drug conjugates to viral therapies and T-cell engagers. While most programmes remain in early development, the common goal is to expand treatment options for patients with limited alternatives. We have identified several active trials in Phases I–III with oncolytic viruses in the field of NETs, e.g. one developed by Vyriad Inc (Voyager-V1) and one by Seneca Therapeutics (SVV-001). Another

active project with oncolytic viruses in NET is currently underway in University of Miami Cancer Centre.

Another company active in OV research is Finnish TILT Therapeutics. TILT Therapeutics is developing TILT-123, an oncolytic adenovirus engineered to express TNF-α and IL-2, designed to enhance immune activation within the tumour microenvironment. The strategy centres on combining the virus with established therapies to improve response rates in difficult-to-treat solid tumours. TILT is currently conducting an open-label, multicentre Phase I/Ib dose-escalation study in platinum-resistant or refractory ovarian cancer, evaluating TILT-123 in combination with pembrolizumab. The trial commenced in May 2022, with primary completion expected in March 2027 (Source: Clinicaltrials.gov, GlobalData).

Looking at ELC-100 from a potential positioning perspective, our understanding is that an OV in NET is likely best positioned in advanced or progressive disease after standard therapies have failed, where unmet need is highest. Elicera has stated that its aim with ELC-100 is to treat patients that have confirmed expression of somatostatin receptors and that have stopped responding to standard therapy.

ELC-401

Elicera also has another CAR T programme ELC-401, which is being developed for initial treatment of brain tumours, glioblastomas (GBM) in particular. ELC-401 targets the tumour antigen IL13Ra2, a receptor protein that is overrepresented in GBM.

In a preclinical study, the company has been able to show that IL13Ra2 is an effective tumour target for iTANK-enhanced CAR T-cells. The study, published in Nature Communications in 2023, analysed the antigen-binding domain (the chimeric antigen receptor, CAR) used in ELC-401 to target IL13Rα2, which Elicera intends to apply in the treatment of glioblastoma. Brain cancer is notoriously difficult to treat due to challenges such as a highly immunosuppressive tumour microenvironment and pronounced antigen heterogeneity. The article also highlights the critical importance of thorough screening and characterisation of CAR constructs to ensure the development of functional CAR T cells with sustained persistence and tumour-killing capacity over time, which is essential to reduce the risk of relapse following CAR T therapy (source: Nature).

Elicera has now completed preclinical development of ELC-401 and identified local administration as the preferred delivery route to overcome the blood-brain barrier and improve tumour targeting. It is now preparing a Phase I/Ib dose-escalation and expansion study in 12 patients across four arms and two dose levels, with repeated dosing and a design that includes treatment before surgery to assess immune cell infiltration and mode of action in resected tumour tissue. The proposed study design will be discussed with the Swedish MPA in mid-2026. Process development and manufacturing preparations are ongoing, and if regulatory alignment is achieved, our understanding is that the trial could start in H2 2027. The company is also seeking non-dilutive funding to support the programme.

Even if ELC-401 is still in its early stages of development and is yet to start a clinical trial, we believe the programme has the potential to be a meaningful game-changer, particularly for solid tumours and highly challenging indications such as glioblastoma, where effective treatment options remain very limited. It is intended that due to iTANK armament, ELC-401 is expected to be able to counteract the highly immunosuppressive microenvironment in GBMs and mobilise an immune response against other targets in this heterogeneous form of cancer (Source: company).

ELC-201

Elicera's next-generation oncolytic immunotherapy, ELC-201, is based on a genetically modified adenovirus vector, armed with the company's proprietary iTANK-technology and a trans gene for 4-1BBL. The treatment is expected to work synergistically with established immune checkpoint inhibitors and can theoretically be used for the treatment of all injectable solid tumours. We are awaiting details regarding strategic direction and indication selection for ELC-201.

Immuno-oncology

Immuno-oncology (IO) focuses on developing treatments that help the body's own immune system recognise and fight cancer. This is done by activating key immune cells (such as antigen-presenting cells, T-cells, and innate immune cells), reducing the immune-suppressive environment around tumours, and strengthening both the innate and adaptive immune responses. The innate immune system provides a rapid, non-specific first line of defence against invading pathogens and relies on immune cells such as natural killer cells and phagocytes. The adaptive immune system, in contrast, generates a targeted response by producing antibodies and activating specialised immune cells against specific antigens the body has encountered before. This adaptive response is highly specific and capable of evolving over time, allowing the immune system to respond to changing or mutating pathogens (Source: Oregon State University; Guyton and Hall, 2011). Unlike passive approaches that supply immune components directly and typically have short-lived effects, IO therapies aim to create a more durable anti-tumour immune response.

Immuno-oncology includes several therapeutic approaches, such as adoptive cell therapy, cancer vaccines, immunomodulators, oncolytic viruses and targeted antibodies (Source: Cancer Research Institute). Elicera Therapeutics is developing two types of immuno-oncology treatments: CAR T-cell therapies and oncolytic viruses. Both approaches attack and kill tumour cells but can also be genetically engineered to activate the patient's own T cells, enabling them to infiltrate and attack cancer cells.

Immuno-oncology classification

		elicera		elicera
Targeted antibodies	**Cancer vaccines**	**Cell therapies**	**Immunomodulators**	**Oncolytic viruses**
Targeted antibodies are a form of cancer immunotherapy that bind to specific antigens on cancer cells, disrupting tumour growth and helping the immune system recognise and eliminate malignant cells.	Cancer vaccines are a form of immunotherapy designed to train the immune system to recognise and attack cancer cells. Cancer vaccines rely on advanced approaches to identify tumour-specific antigens and stimulate a targeted immune response against cancer.	Cell-based immunotherapies use immune cells to fight cancer, either by expanding number of naturally occurring cells or by genetically engineering them to enhance anti-tumour activity.	Immunomodulators are therapies that influence the immune system by targeting pathways that control immune activation and suppression. By acting on the immune system's "brakes" and "accelerators," these agents enhance the body's ability to recognise and destroy cancer cells.	Use of viruses to selectively infect and kill cancer cells. Cancer cells often have weakened antiviral defences, these viruses can replicate inside tumours while sparing healthy tissue. As infected cancer cells burst, tumour antigens are released, helping activate the immune system to recognise and attack remaining cancer cells.
• Monoclonal antibodies • Antibody-drug(mAbs) conjugates (ADCs) • Bispecific antibodies	• Preventive cancer vaccines • Therapeutic cancer vaccines • Personalised neoantigen vaccines	• Tumour-infiltrating lymphocyte (TIL) therapy • Engineered T-cell receptor therapy • Chimeric antigen receptor (CAR) T-cell therapy • Natural Killer (NK) cell therapy	• Checkpoint inhibitors • Cytokines • Agonists • Adjuvants	

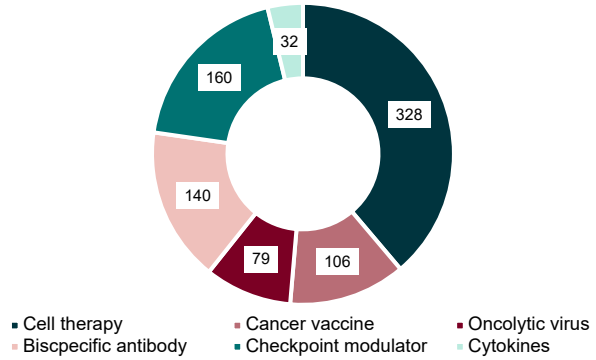
Source: Cancer Research Institute, DNB Carnegie (visualisation)

A crowded space: immuno-oncology pipeline

According to GlobalData's strategic update on immuno-oncology from 2025, there are currently 89 novel marketed immuno-oncology agents available in the eight major markets (8MM: US, France, Germany, Italy, Spain, UK, Japan and China) – and even more are on their way to a potential approval. As of May 2025, it was estimated by GlobalData that there were c1,500 industry-sponsored ongoing or planned clinical trials in the 8MM. Most of them are in early stages, with Phase I/II trials constituting the largest share (36%), followed closely by Phase II trials (35%).

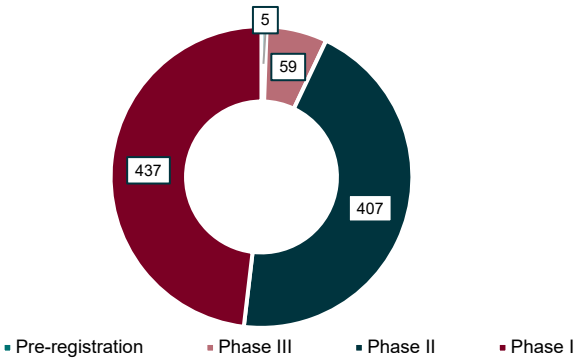
As for active trials, as of April 2025, there were c900 IO products in Phase I-III development in the 8MM, according to GlobalData. The leading modality is cell therapy, with c300 projects, followed by checkpoint modulators with c160 projects.

Pipeline by type (2025)



Source: GlobalData, 2025

Pipeline by phase (2025)



Source: GlobalData, 2025

In all phases of clinical development, the leading indication is unspecified solid tumours, with 301 IO agents in development, followed by lung cancer with 151 IO agents and gastric cancer with 93 IO agents. At the bottom of the activity list for immunotherapies in the pipeline are relapsed multiple myeloma (c54 agents), ovarian cancer (c59), and DLBCL with c60 agents in development.

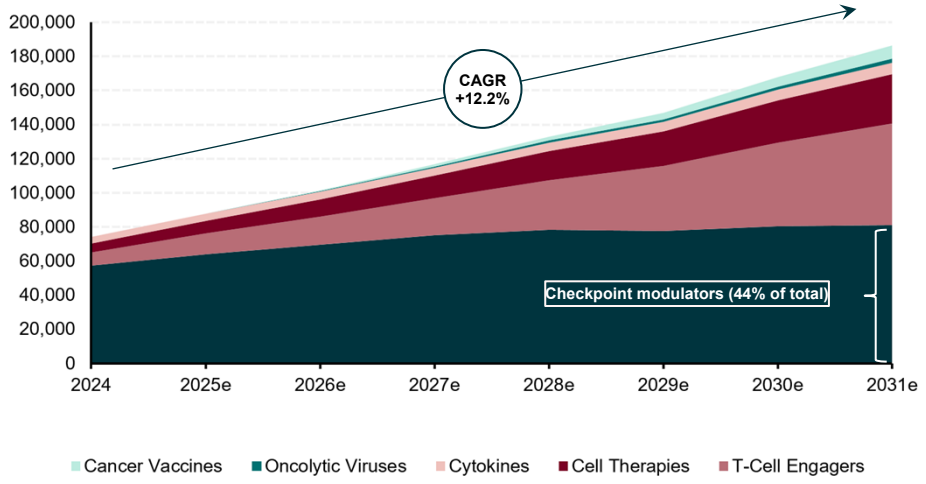
The market for immunotherapies

Market forecasts point to a strong and sustained expansion of the IO space. According to a Coherent Market Insights (CMI) report published on BioSpace, the global IO drugs market is expected to more than triple, from cUSD32bn in 2025 to cUSD107bn by 2032, implying a CAGR of c19%. CMI estimates that checkpoint modulators will continue to occupy the largest treatment segment, with revenues projected to reach around USD10bn by 2025.

Complementing this view, GlobalData estimates that sales across the major IO therapy classes may reach an even higher number – USD186bn by 2031, with checkpoint modulators also accounting for the largest treatment segment with around 44% of total sales. While checkpoint inhibitors continue to dominate in absolute terms, the widening gap between total IO market growth and checkpoint-only revenues highlights the increasing contribution from next-generation modalities such as cell therapies, T-cell engagers, oncolytic viruses and cancer vaccines.

Overall, these forecasts highlight the scale of the IO space opportunity, with new approaches supporting longer-term growth. At the same time, the size of the market and breadth of the pipeline reflects intense competition. Based on historical likelihood-of-approval rates (LoA), only a limited number of programmes are likely to translate into meaningful commercial success.

Immuno-oncology market value (USDm)



Source: GlobalData

Challenges in immuno-oncology

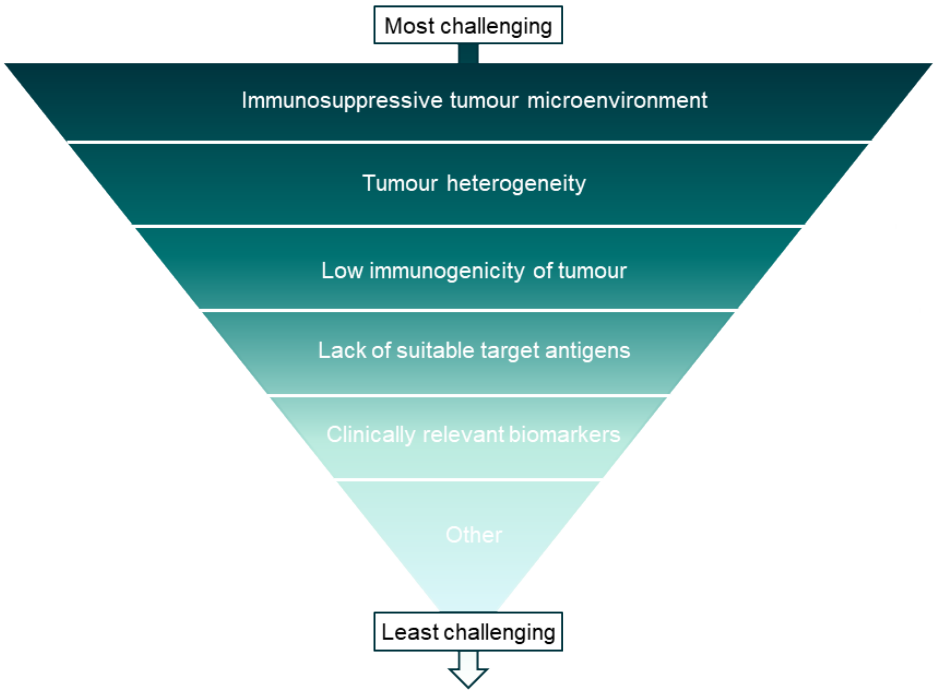
The field of immuno-oncology faces several challenges, such as treatment resistance, insufficient response rates, and side effects, among others. Far from all patients benefit meaningfully from immunotherapies, as tumour cells can evade or counteract the immune responses induced by these treatments.

Solid tumours represent a particularly significant challenge within immuno-oncology. They are biologically complex and highly heterogeneous, often characterised by diverse antigen expression and mutational profiles. This heterogeneity makes it difficult to develop a single therapy capable of targeting all tumour cells. In addition, the tumour microenvironment (TME) in solid tumours is frequently immunosuppressive (so-called ‘cold’), limiting immune cell infiltration and effective immune activation and therefore making them particularly difficult to treat with immunotherapy as a monotherapy (source: Cell Communication and Signaling, 2025).

Tumours may also develop multiple immune-evasion mechanisms, including downregulation of antigen expression, upregulation of immunosuppressive molecules, and activation of inhibitory signalling pathways. These factors contribute to resistance to immunotherapy and represent a key obstacle in the treatment of solid tumours. In many cases, solid tumours also lack truly tumour-specific antigens, further complicating the development of effective immunotherapies.

As previously noted, the iTANK platform aims to address two of the major challenges for IO therapy: immunosuppressive tumour microenvironment and tumour heterogeneity. Other key challenges in immune-oncology treatment include the lack of suitable target antigens and the absence of clinically relevant biomarkers, among others.

Challenging aspects of optimising IO therapy

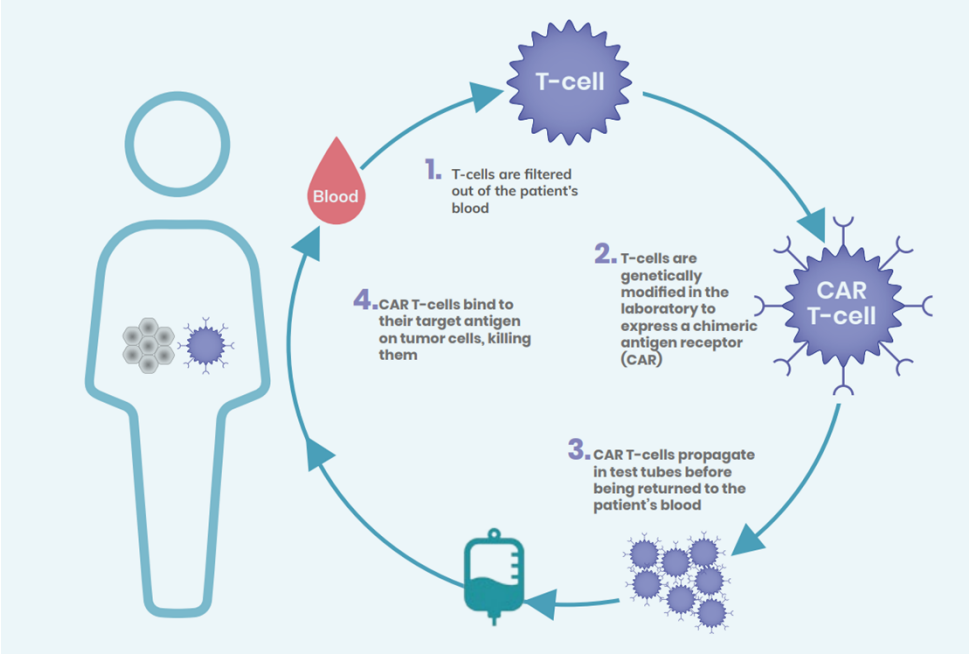


Source: GlobalData High-Prescriber Survey (March 2025)

CAR T therapies

Within the broader immuno-oncology landscape, CAR T-cell therapy is one of the leading and clinically validated modalities. CAR T-cell therapy is a form of cancer immunotherapy in which a patient's own T cells are collected, genetically engineered to express chimeric antigen receptors (CARs), expanded ex vivo, and reinfused to target and kill cancer cells. CARs enable T cells to recognise specific antigens on tumour cells, triggering potent and sustained immune-mediated cell killing. CAR T-cell therapies have shown particular success in blood cancers such as leukaemia, lymphoma and multiple myeloma, where they have delivered durable clinical responses (sources: Nature, signal transduction & targeted therapy, 2023; DNB Carnegie research).

CAR T-cell therapy treatment – an overview



Source: Company

All approved CAR T-cell therapies in oncology are autologous, meaning T cells are collected from the patient, genetically modified to express a CAR targeting a specific tumour antigen, and then reinfused into the same patient. Autologous CAR T-cell therapies are time-consuming to manufacture and present significant logistical challenges, contributing to very high production costs. Allogeneic CAR T-cell therapies, which use T cells from healthy donors, offer faster and potentially simpler manufacturing; however, they may carry a higher risk of triggering unwanted immune reactions in the treated patient (source: ASTCT – American Society for Transplantation and Cellular Therapy). Elicera's ELC-301 is an autologous therapy.

Overview of advantages and disadvantages of autologous versus allogeneic cell therapies

Therapy type	Advantages	Disadvantages
Autologous cell therapy	**Immune compatibility:** Minimal risk of immune rejection, as cells are derived from the patient's own body.	**Time-consuming and costly:** Patient-specific manufacturing leads to long production times and delayed treatment. **Heterogeneity:** Variability in starting material can result in inconsistent manufacturing efficiency and product quality.
Allogeneic cell therapy	**Scalability:** Cells from a single donor can be expanded and used for multiple patients, improving cost efficiency and reducing variability. **Readiness:** "Off-the-shelf" products enable rapid treatment without manufacturing delays.	**Immune compatibility risks:** Risk of graft-versus-host disease (GvHD) and other immune complications, often requiring immunosuppression. **Matching requirements:** Donor–recipient matching may be necessary to reduce rejection risk.

Source: Ladd, B. 2025

Approved CAR T-cell therapies, in order of approval by the FDA

Product	Treatment modality	Main approved indications
Kymriah	CD 19-directed (autologous)	• Relapsed/refractory (R/R) B-cell acute lymphoblastic leukaemia (ALL) in patients ≤25 years • R/R large B-cell lymphoma (LBCL) after ≥2 prior lines • R/R follicular lymphoma (FL) after ≥2 prior lines
Yescarta	CD 19-directed (autologous)	• R/R large B-cell lymphoma (including DLBCL, PMBCL, high-grade B-cell lymphoma) • LBCL refractory to first-line or relapsing within 12 months • R/R follicular lymphoma after ≥2 prior lines
Tecartus	CD 19-directed (autologous)	• R/R mantle cell lymphoma (MCL) • R/R B-cell precursor acute lymphoblastic leukaemia (ALL) in adults
Breyanzi	CD 19-directed (autologous)	• R/R large B-cell lymphoma after ≥2 prior lines or ineligible for HSCT • R/R chronic lymphocytic leukaemia (CLL)/small lymphocytic lymphoma (SLL) after ≥2 prior lines • R/R follicular lymphoma after ≥2 prior lines • R/R mantle cell lymphoma after ≥2 prior lines
Abecma	BCMA-directed (autologous)	• R/R multiple myeloma after ≥2 prior lines
Carvykti	BCMA-directed (autologous)	• R/R multiple myeloma after ≥1 prior line, refractory to lenalidomide
Aucatzyl	CD 19-directed (autologous)	• R/R B-cell precursor ALL in adults

Source: FDA, Alliance for Cancer Gene Therapy, DNB Carnegie (research)

The first CAR T-cell therapy, tisagenlecleucel (Kymriah), was approved by the FDA in August 2017, for the treatment of paediatric and young adult patients with acute lymphoblastic leukaemia (ALL). Since then, progress in the field has been rapid.

Development in CAR T therapies is commonly divided into three to four generations, reflecting increasing levels of engineering sophistication. First-generation CAR T cells, developed in the 1990s and early 2000s, demonstrated limited clinical efficacy due to poor expansion and persistence after infusion (source: Cancer Discovery, 2013; Company).

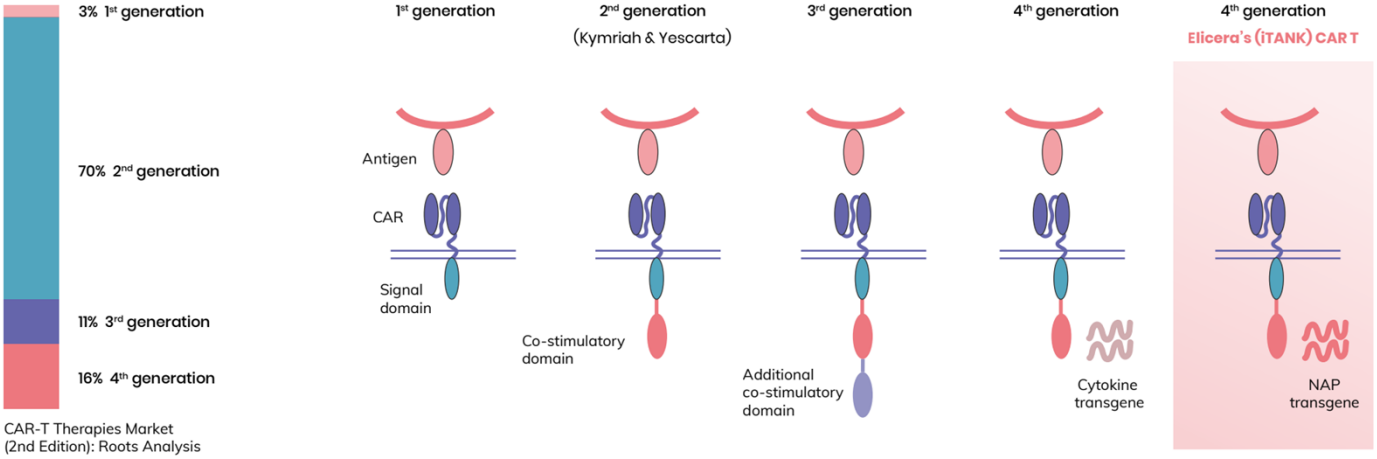
Second-generation CAR T cells, introduced in the mid-2000s, incorporated a single co-stimulatory domain, which markedly improved T-cell activation, survival, and antitumour activity. All currently approved CAR T-cell therapies, as well as around 70% of programmes currently in clinical development, belong to this second generation, including those approved for B-cell lymphomas.

Third-generation CAR T cells, developed in the early 2010s, include two co-stimulatory domains simultaneously and have shown further improvements in function, persistence, and immune activation compared with second-generation constructs. However, their incremental clinical benefit has yet to be fully established.

Research is now focused on fourth-generation CAR T cells, which build on second-generation designs but add transgenes encoding immunostimulatory molecules or other enhancements, such as cytokine production or multi-antigen recognition. These modifications aim to activate

the innate immune system, recruit endogenous CD8+ T cells, and broaden CAR T efficacy across a wider range of tumour types (Source: Biomarker Research).

The four generations of CAR Ts



Proliferation	Insufficient	Improved	Improved +	Improved +	Improved +
T cell survival/ongoing response	Brief	Extended	Extended	Extended +	Extended +
Immune activation	Insufficient	Improved	Improved	Improved +	Improved +++

Source: Company

A selection of long-term outcomes following CAR T therapy in DLBCL and other indications

CAR T product, indication and patient population in trials	ORR (%)	CRR (%)	PFS / EFS (months)	Duration of response (DOR) (months)
Kymriah; DLBCL (24), FL (14)	66	55	5-year PFS: DLBCL 31%	60% maintained DOR at 5 years
Yescarta; DLBCL (77), PMBCL (8), tFL (16)	74	54	Median EFS: 5.7; 24-month EFS: 38%	NR
Kymriah; DLBCL, HGBCL, tFL (115)	53	39	Median PFS: 2.9; Median EFS: 2.8	Median DOR: NE
Breyanzi; LBCL (270)	73	53	Median PFS: 6.8	Median DOR: 26 (CR responders)
Yescarta; DLBCL (126), HGBCL (31)	83	65	Median PFS: 15	Median DOR: 27

ORR (Overall Response Rate) - % of patients who achieve a meaningful tumour reduction, including complete and partial responses.

CRR (Complete Response Rate) - % of patients with complete disappearance of all detectable cancer.

PFS (Progression-Free Survival) - Time from treatment start until disease progression or death from any cause.

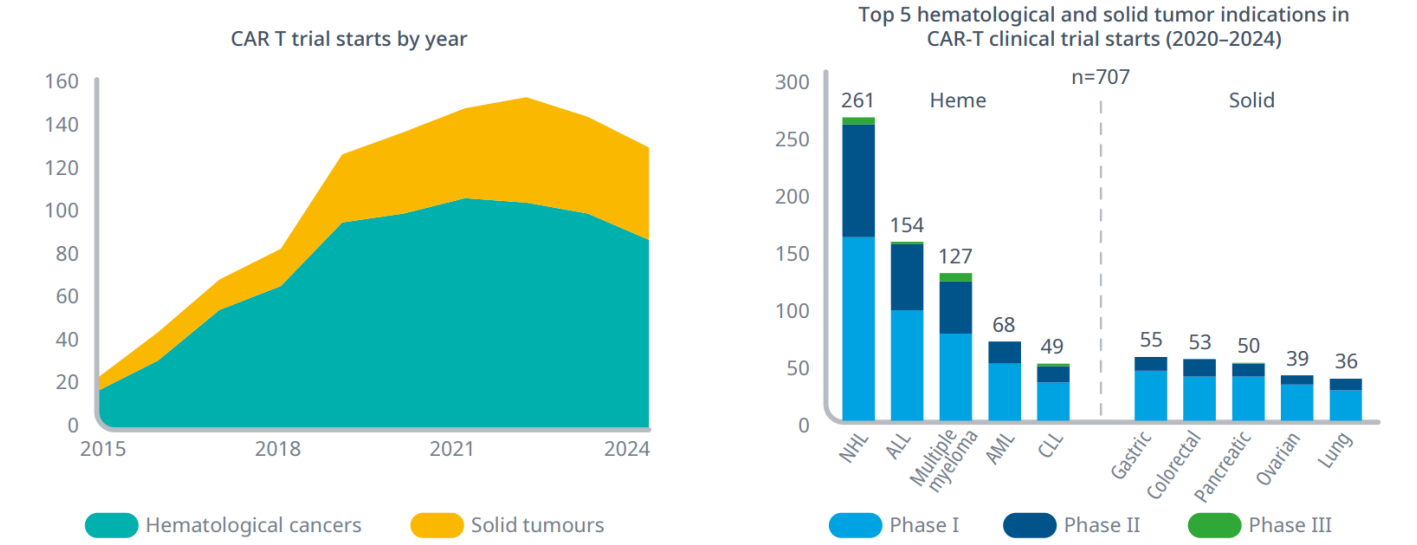
EFS (Event-Free Survival) - Time from treatment start until a predefined event occurs, such as progression, relapse, treatment failure, or death.

Source: LymphomaHub, FDA, DNB Carnegie

Pipeline for CAR Ts

CAR T clinical development has expanded rapidly over the past decade, with a sharp rise in trial initiations, particularly after the first approvals of Kymriah and Yescarta, and initially dominated by haematological indications, but increasingly complemented by growing activity in solid tumours also. However, trial starts have declined by around 15% from the 2022 peak and remain largely concentrated in haematological cancers (Source: IQVIA). The largest indication in CAR T development is non-Hodgkin lymphoma (NHL) (of the 707 CAR T trials started in the last five years, 261 look at patients with NHL), followed by ALL and multiple myeloma according to IQVIA – all haematological malignancies, which we believe makes this a very crowded space. Most CAR T clinical programmes remain in Phase I or II, with few Phase III trials. At YE 2025 there were c300 active clinical trials (+5% Y/Y from c280 active trials at YE 2024) registered in GlobalData's Clinical Trials database.

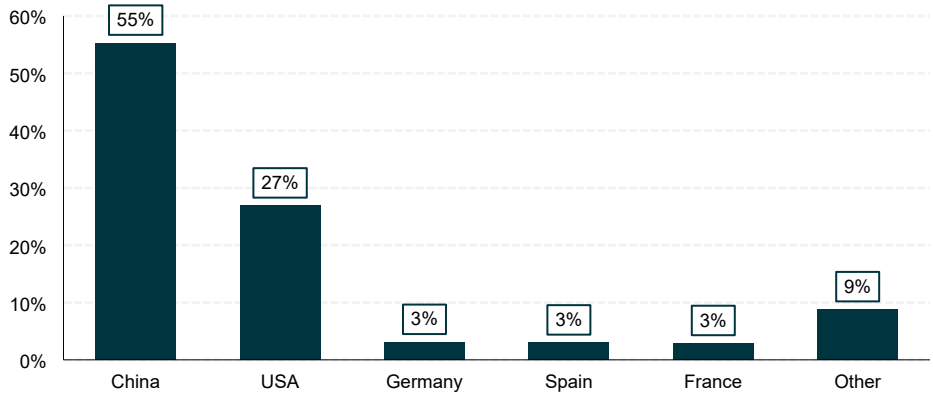
CAR T-cell therapy clinical trial starts and ongoing trials by top indications



Source: IQVIA (2025)

Elicera is the only company developing CAR T-cell therapies in the Nordics, and according to GlobalData Europe more broadly is lagging in this area. China currently dominates CAR T development, accounting for c55% of all trial starts, followed by the US with c27%. The high number of CAR T trials in China likely reflects strong government support for bringing innovative therapies to market, with development activity now outpacing that in the West. Chinese companies are investigating roughly x1.5 more in CAR T therapies than their US counterparts (source: GlobalData, IQVIA), driven by initiatives such as Healthy China 2030.

Percentage of clinical trials for CAR T therapies by country



Source: GlobalData

Challenges and limitations of CAR T therapies

CAR T-cell therapies have delivered transformative efficacy in certain haematological malignancies, as shown above, but they are also associated with meaningful clinical, operational and regulatory challenges. As with most oncology treatments, side effects remain a key limitation. CAR T treatment can trigger immune overactivation, commonly presenting as cytokine release syndrome (CRS) with flu-like symptoms such as fever, fatigue and muscle pain. In severe cases, CRS may impair organ function and require intensive care. Neurological toxicities can also occur, sometimes leading to transient or persistent cognitive impairment.

Additional adverse events include pancytopenia, neutropenia, and other haematological toxicities, all of which require careful monitoring and specialised clinical management (source: Cell Reports Medicine, 2025; Mayo Clinic; JAMA Oncology).

From a development and commercial perspective, the field is becoming competitive, as demonstrated in the number of active CAR T trials ongoing. Established CAR T products dominate current approved indications, raising the bar for new entrants to demonstrate clear clinical differentiation. At the same time, solid tumours remain a major challenge due to immunosuppressive tumour microenvironments and the absence of well-defined tumour-specific antigens. Regulatory expectations have also tightened, with agencies increasingly favouring large, randomised trials and overall survival endpoints, which can extend development timelines and increase costs. Finally, while allogeneic CAR T approaches may offer logistical advantages, they may also carry higher risks of immune complications and potentially lower efficacy compared with autologous products.

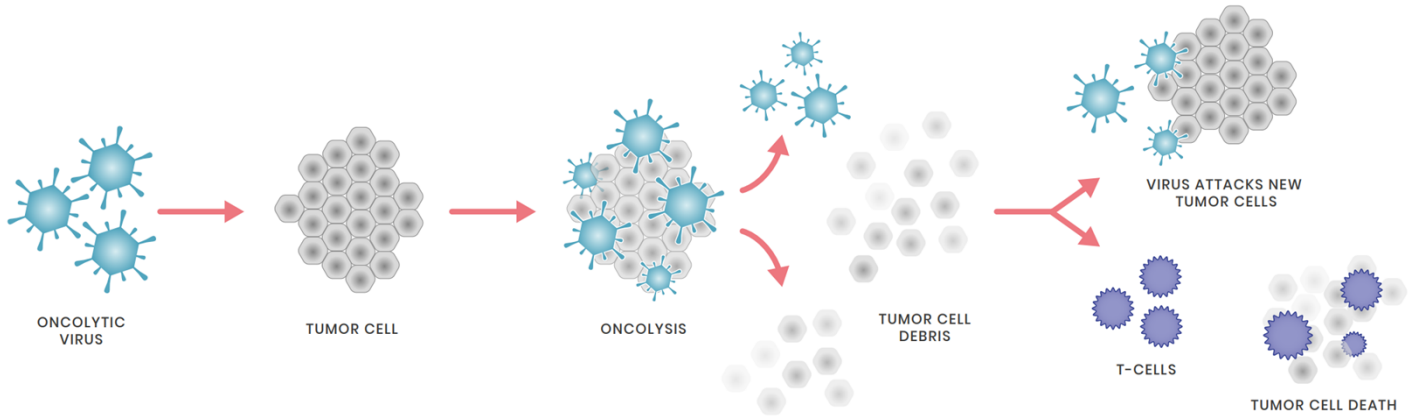
Key challenges in CAR T therapy

- Significant and sometimes severe safety risks (CRS, neurotoxicity, haematological toxicities).
- Complex patient selection
- Limited access due to treatment at specialised centres and high production costs.
- An intensely-competitive field and increasing regulatory requirements.
- Persistent difficulty in addressing solid tumours.
- Trade-offs between scalability and efficacy in allogeneic CAR T approaches.

Oncolytic viruses

Oncolytic viruses (OV) are a form of immuno-oncology therapy based on genetically engineered viruses designed to selectively infect and destroy tumour cells while largely sparing healthy tissue. Although the concept of oncolytic viruses was identified several decades ago, it was not until the 2010s that advances in genetic engineering and immunology enabled these therapies to transition from preclinical research into clinical development.

Mechanism of action of oncolytic virus therapy



Source: Company

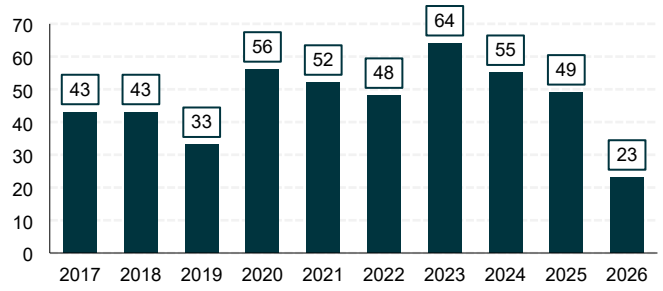
Several virus backbones have been explored in this field, most commonly adenoviruses, herpes simplex viruses, measles morbilliviruses and human enteroviruses. Depending on their route of administration, oncolytic viruses are broadly categorised as intratumoral therapies, administered directly into tumours, or systemic therapies, designed to reach disseminated disease following intravenous delivery.

A major milestone for the field was the approval of talimogene laherparepvec (T-VEC) by the EMA and FDA in 2015 for the treatment of unresectable metastatic melanoma, validating the clinical potential of live-virus cancer therapies. Earlier approvals in China, including Oncorine and Gendicine, further demonstrated proof of concept, and more recent late-stage programmes (such as Genelux's Olvi-Vec, Replimune's RP1–RP3, CG Oncology's cretostimogene grenadenorepvec, and Oncolytics Biotech's pelareorep) are primarily focused on solid tumours.

Pipeline for oncolytic viruses

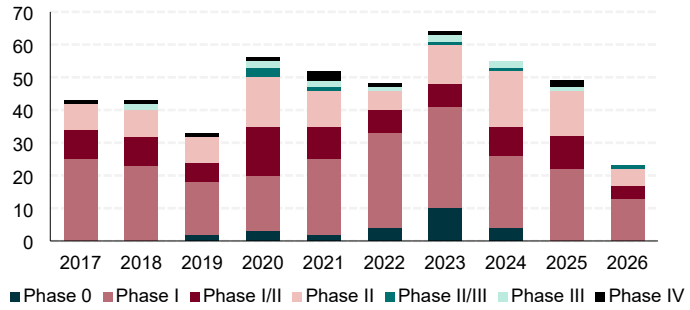
The clinical pipeline scene for oncolytic viruses is less lively compared with CAR T-cell therapies. As of YE 2025, there were 28 active oncolytic virus clinical trials, representing a c10% Y/Y decline according to GlobalData Clinical Trials. Trial initiation activity has also been relatively modest, with oncolytic virus programmes averaging around 50 new trial starts per year during 2017–25, significantly lower than the level observed for CAR T therapies. This reflects a less mature and less crowded development landscape but also highlights the more cautious pace of clinical progress within the oncolytic virus field.

Clinical trial starts by year (OV)



Source: GlobalData

Clinical trial starts by year and phase (OV)



Source: GlobalData

Challenges with oncolytic viruses

Our understanding is that the muted level of activity in oncolytic viruses (as compared to CAR T therapies) reflects a combination of scientific, clinical, and industrial hurdles rather than a lack of conceptual appeal. While oncolytic viruses may offer clear theoretical advantages, for instance the 'off-the-shelf' availability and much lower production costs, their practical execution has proven challenging (Source: key opinion leader (KOL) interviews, GlobalData). Manufacturing at scale remains complex, as small variations in viral replication or purity can materially affect efficacy, and regulators remain highly cautious around genetic stability and the risk of unintended mutations.

Clinically, KOLs note that efficacy signals have so far been modest and inconsistent, particularly beyond locally injected lesions. Although local tumour regression has been observed in multiple indications, these responses have not translated reliably into durable systemic benefit. A central limitation is systemic delivery: viruses are often rapidly neutralised by the immune system, struggle to penetrate poorly vascularised or fibrotic tumours, and face difficulty reaching metastatic sites after local administration. However, rather than signalling failure, KOLs state that the current clinical plateau highlights the need for re-engineered approaches, including rational combination strategies and improved delivery platforms. Advances in patient selection, trial design, and synthetic biology may enable these therapies to overcome earlier limitations and unlock broader clinical potential (Source: Molecular Science, Achim et al.).

Competition from other immunotherapies is also a structural challenge for OV development. Checkpoint inhibitors, bispecific antibodies and cell therapies have already demonstrated robust response rates, durable remissions and, in some indications, overall survival benefits. These therapies are also supported by large datasets, established regulatory pathways and growing clinical familiarity. Against this backdrop, OV therapies must not only show activity, but clearly differentiate on clinically meaningful endpoints such as durability, depth of response, safety, or ease of use. As a result, our understanding is that OVs are often positioned as adjunct or combination agents rather than standalone therapies, raising the bar for further development and potential launches.

Disease overview

Elicera's pipeline spans both haematological and solid tumour indications, including diffuse large B-cell lymphoma (DLBCL), neuroendocrine tumours (NET) and glioblastoma. In this report, we place primary emphasis on DLBCL, which we view as the near-term value driver. We also review the NET opportunity, where Elicera has advanced most and recently announced final data from the Phase I/IIa trial of ELC-100. We also touch upon glioblastoma, which represents a high-risk and very early-stage project (ELC-401), but a potentially high-reward solid tumour opportunity given the substantial unmet medical need.

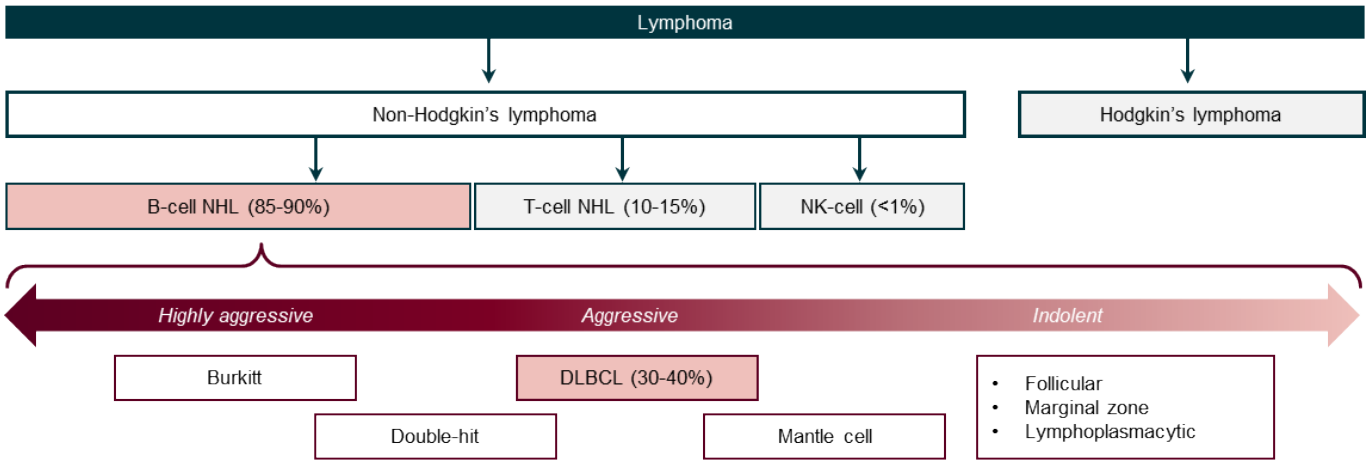
DLBCL

Epidemiology and disease background

Lymphoma is a type of blood cancer that originates in the lymphatic system, a network of tissues, vessels and organs involved in immune defence. The disease arises from malignant transformation of lymphocytes, a class of white blood cells. Lymphomas are broadly classified into Hodgkin lymphoma and non-Hodgkin lymphoma, with more than 70 recognised subtypes. NHL is the most common type of lymphoma, representing around 85–90% of diagnosed lymphomas, and is the 10th most common cancer globally, with more than 500,000 cases diagnosed each year (WHO, 2024).

Clinically, these subtypes of lymphoma can be classified as indolent (slow-growing) and aggressive (fast-growing) forms, with markedly different treatment strategies and prognoses. Diffuse large B-cell lymphoma (DLBCL) is the most common aggressive subtype of non-Hodgkin lymphoma and represents a key focus in lymphoma research due to its high relapse rate and significant unmet medical need in relapsed/refractory disease.

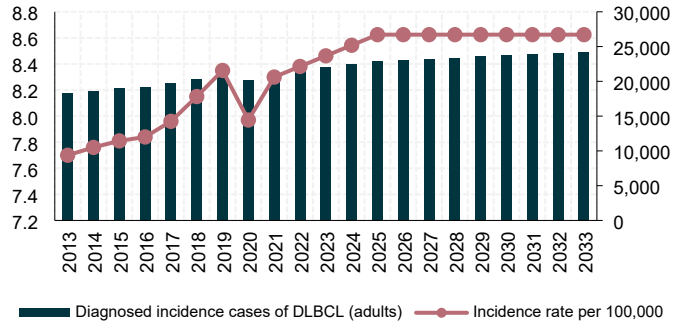
Lymphoma classification



Source: Lymphoma and leukaemia society data, Brigham Health, National Cancer Institute, DNB Carnegie (visualisation)

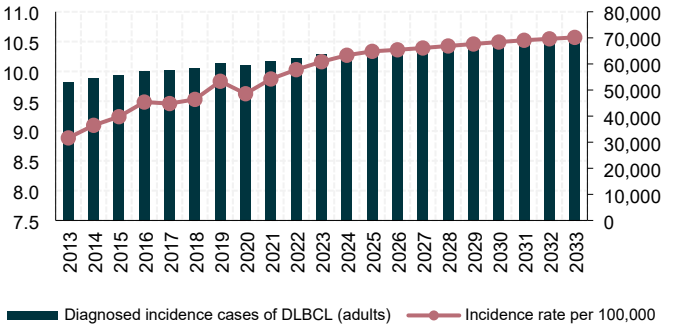
According to different sources, DLBCL accounts for c30-40% of all NHL cases (sources: Lymphoma Research Foundation, GlobalData, Cancer Research Institute). While the incidence (number of new cases) increases with age (half of the patients are over 60 years old), DLBCL can also occur in younger adults and, rarely, in children. SEER data indicates that DLBCL incidence has been declining over time, and the overall pattern mirrors that of non-Hodgkin lymphoma, with incidence rising through the first half of the 20th century before largely plateauing in its decline rate. Unfortunately, many countries lack robust, country-specific incidence data for DLBCL. As a result, data aggregators (such as GlobalData) often estimate DLBCL incidence using proxy assumptions, typically modelling it as a percentage of the total NHL population based on better-characterised markets. According to these estimations, yearly, approximately 65,000 adults are diagnosed with DLBCL in the 7MM (Source: GlobalData), of which c23,000 are diagnosed in the US. See historical data and forecasts we have used in our models below.

Incident diagnosed cases (n) and incidence rate (per 100,000) – US



Source: SEER data (historical data) & GlobalData forecasts

Incident diagnosed cases (n) and incidence rate (per 100,000) – 7MM



Source: GlobalData (historical data and forecasts)

Symptoms and staging

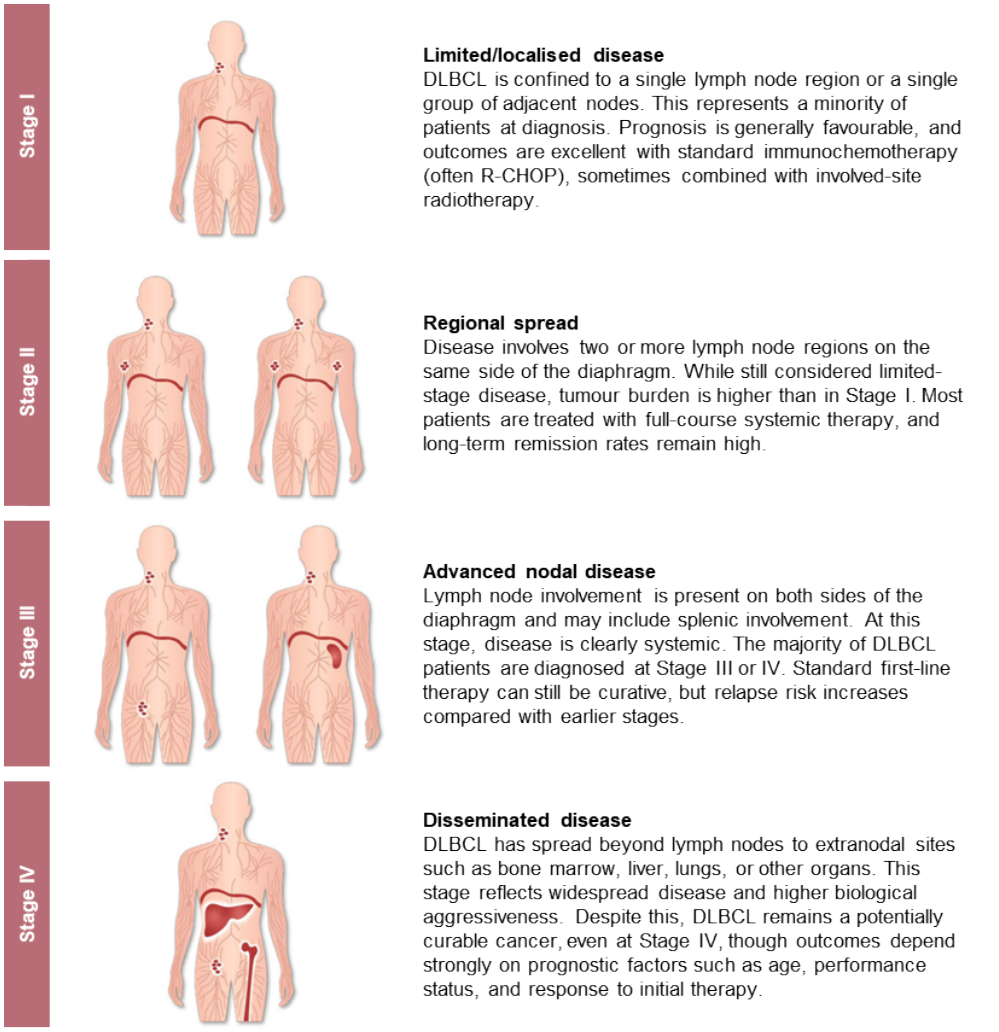
The first sign of DLBCL is usually a painless, rapid swelling in the neck, underarms or groin caused by the enlarged lymph nodes. For some patients, however, this swelling may be painful. Symptoms of an aggressive B-cell lymphomas, such as DLBCL, typically develop over a relatively short period, ranging from a few weeks to a few months. The most common presenting symptom of DLBCL is also enlarged lymph nodes (>1.5 cm) but other symptoms include fatigue, general malaise, unintentional and unexplained weight loss, night sweats, and fever (the so-called 'B-symptoms') (source: Cleveland Clinic).

DLBCL is usually diagnosed through a biopsy of a suspected affected lymph node, blood tests assessing levels of different blood cell types, and radiological investigations, most commonly positron emission tomography (PET) combined with computed tomography (CT).

Once the diagnosis of DLBCL is confirmed, the next step is to understand the location of the disease in the body, and stage correctly. Staging may also include a bone marrow biopsy and a lumbar puncture. See diagram below for NHL staging according to the Lugano system. This system categorises NHL from stage I–III (limited disease) to III–IV (advanced disease), based on the cancer spreading in lymph nodes, bone marrow and other organs.

- **Stage I:** Cancer is limited to a single lymph node region or a single, localized group of nearby lymph nodes.
- **Stage II:** Two or more lymph node regions are involved, but all are on the same side of the diaphragm (either above or below).
- **Stage III:** Lymph node involvement occurs on both sides of the diaphragm, and the spleen may also be affected.
- **Stage IV:** Widespread disease with involvement beyond lymph nodes, including organs such as bone marrow, liver, lungs, or bones.

Lymphoma staging



Source: Lymphoma research foundation, DNB Carnegie (visualisation)

Current treatment landscape

NHL in general has seen the most innovation in haematological cancers, with 19 new drugs launched since 2015, including four CAR T-cell therapies, three bispecific antibodies and two antibody drug conjugates predominantly for the treatment of relapsed or refractory large B-cell lymphoma (Source: IQVIA). Thus, the treatment landscape has evolved significantly over the past decade, particularly in the second-line (2L) and third-line (3L) settings.

Approximately two-thirds (c66%) of patients with DLBCL are cured with standard first-line therapy. The most widely used regimen is R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, and prednisone). If the lymphoma responds well and the patient achieves remission (meaning no detectable disease remains) treatment is discontinued (source: Lymphoma research foundation; Treatment guidelines).

Approximately 10–15% of patients have primary refractory disease, where DLBCL does not respond to standard therapy, and an additional 20–30% experience relapse, most commonly within the first two years following initial treatment. Refractory and relapsed DLBCL is more challenging to treat, although several effective therapeutic options remain available and can still induce remission (source: Lymphoma research foundation).

First-line therapy is, as mentioned, dominated by chemoimmunotherapy, most commonly R-CHOP. Dose-adjusted regimens (e.g. R-EPOCH) or reduced-intensity R-mini-CHOP may be considered for selected or frail patients, while radiotherapy is applied in specific clinical settings.

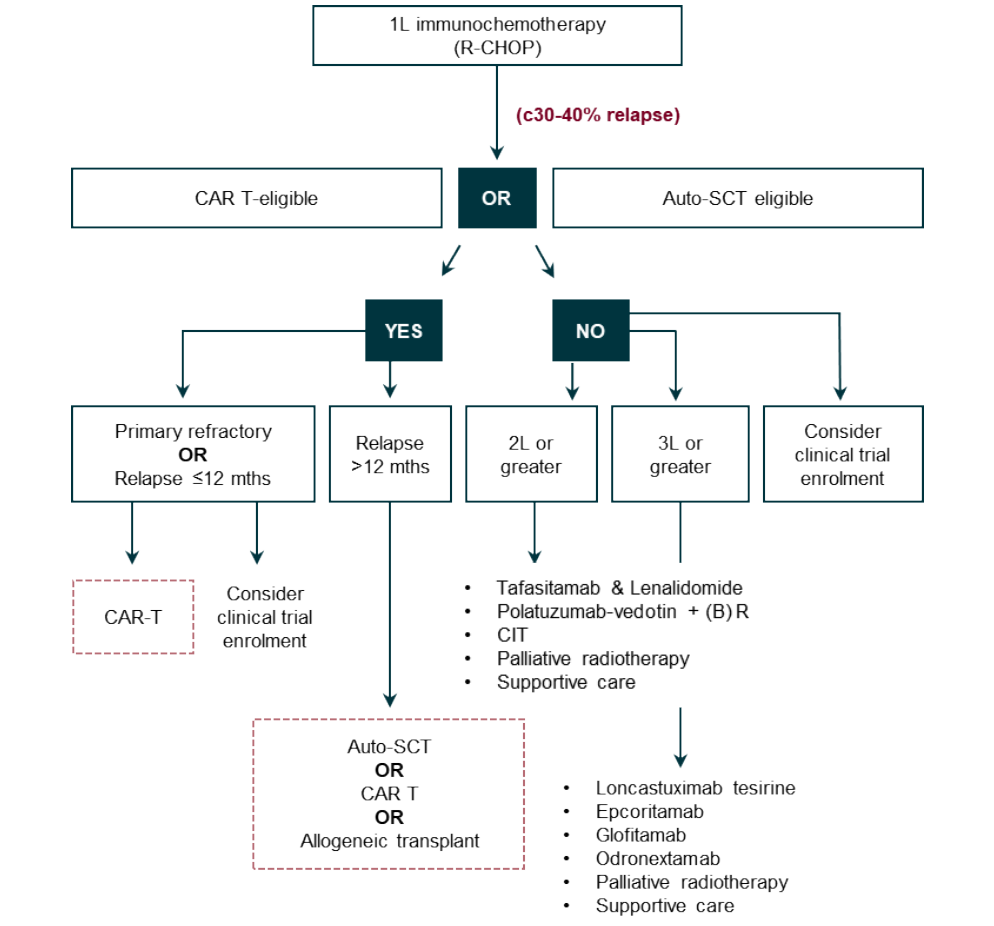
In primary refractory disease or relapse within 12 months, CAR T-cell therapy is on its way to becoming the standard of care in the second-line setting, with Yescarta, Kymriah, and Breyanzi replacing historical salvage chemotherapy and autologous stem cell transplant (ASCT) in eligible patients. Transplant eligible patients move on to an autologous stem cell transplantation (auto-SCT). If the patient is not eligible for either CAR T or ASCT, additional treatment options include among others bispecific antibodies, monoclonal antibodies, antibody-drug conjugastes (ADCs) (source: Lymphoma Research Foundation; Treatment guidelines; Journal of Hematology & Oncology).

But even if a patient is CAR T eligible, in real world practice not all patients are able to receive it. CAR T-cell therapy treatment is typically restricted to highly specialised centres with the infrastructure and expertise required to manage complex manufacturing logistics and potentially severe toxicities, which limits access. In addition, the high cost of CAR T therapies remains a significant barrier for healthcare systems and payors which constrains uptake further.

This variability in access is reflected in real-world treatment rates. According to IQVIA, the proportion of referred DLBCL patients who ultimately receive CAR T therapy ranges widely – from around 39% in Canada to as high as 88% in Italy. The reasons for non-treatment differ across countries, but disease progression while awaiting treatment is consistently cited as the most common limiting factor.

For patients who relapse after 12 months and are not eligible for either CAR T or ASCT, the traditional approach remains salvage chemotherapy and other treatment options listed above. Five-year relative survival is approximately 79% in Stage I and around 55% in Stage IV (Source: Cancer Research UK).

Treatment algorithm for r/r DLBCL



Source: Journal of Hematology & Oncology (2025)

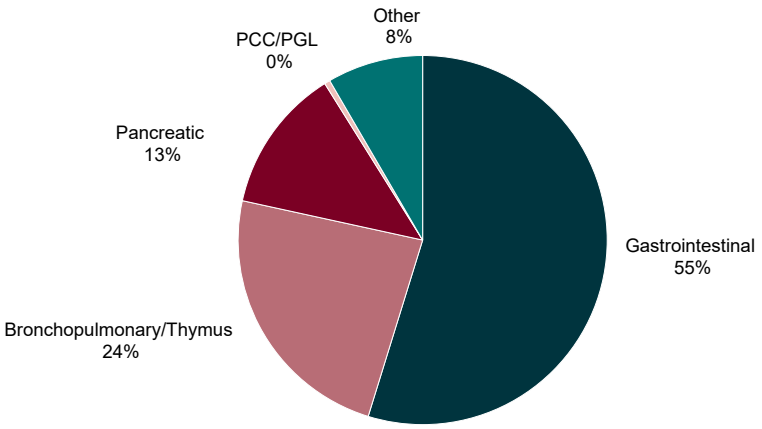
NET

Epidemiology and disease background

Neuroendocrine tumours (NETs) are a relatively rare and heterogeneous group of cancers that arise from neuroendocrine cells (specialised cells that share features of both nerve cells and hormone-producing endocrine cells). These cells are distributed throughout the body but are most commonly found in the gastrointestinal tract, pancreas, and lungs. While the majority of NETs are malignant, they vary widely in their biological behaviour, ranging from slow-growing, indolent tumours to aggressive, fast-progressing cancers (Source: Cleveland Clinic).

NETs are typically classified along two main dimensions: hormonal activity and site of origin. Functional NETs secrete excess hormones and can cause characteristic hormone-related syndromes, whereas non-functional NETs do not produce hormones and often present later due to tumour growth or mass effects on surrounding tissues. Tumours are also grouped by their primary site, which has important implications for prognosis, symptom profile, and treatment strategy. The most common subtypes are gastrointestinal NETs (GI-NETs c55%) (historically also referred to as 'carcinoid' tumours), which most frequently arise in the intestines, rectum or appendix. Lung NETs represent the second most common group (c24%), developing in the lungs or bronchial airways, while pancreatic NETs (P-NETs) originate in the pancreas and account for roughly 10% of all NETs (Source: JAMA Oncology). Less common neuroendocrine malignancies include Merkel cell carcinoma of the skin and neuroblastoma, which primarily affects children and arises from immature nerve cells.

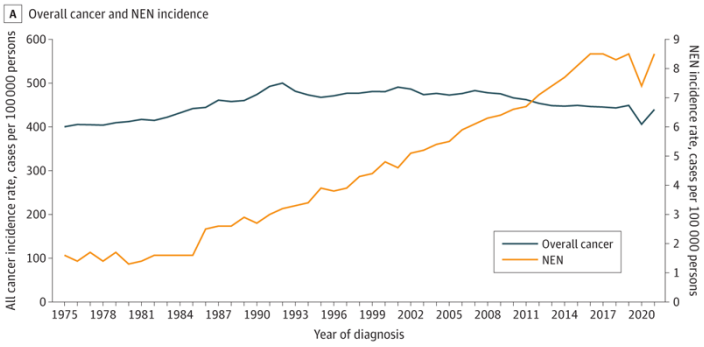
NETs by site of origin



Source: GlobalData

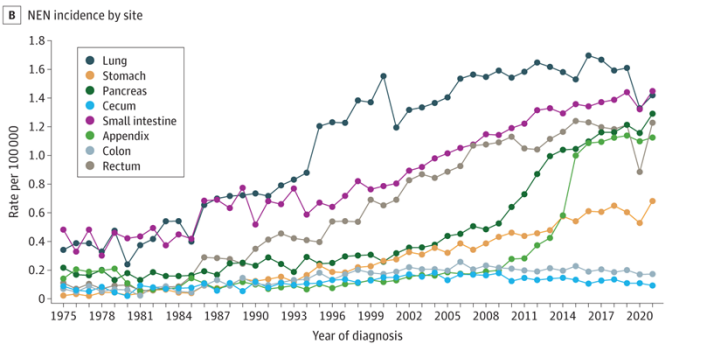
An article analysing SEER data for NETs recently published in the Journal of the American Medical Association (JAMA; Dasari et al., 2025) highlights a structurally expanding NET population in the US, driven primarily by earlier detection. Based on an analysis of 145,447 NET cases the age-adjusted incidence rate increased more than fivefold, from 1.64 per 100,000 in 1975 to 8.52 per 100,000 in 2021, with a temporary dip in 2020 likely related to Covid-19-associated diagnostic disruption. The rise was observed across all tumour sites, stages and grades, but was particularly pronounced in localised disease, well-differentiated tumours, and appendiceal and rectal primaries. The authors concluded that this increase rather suggests improved awareness and diagnostic intensity rather than a shift toward more aggressive biology of the tumour. Since 2000, the highest incidence rates have been observed in GI NETs and lung NETs.

Overall cancer and NET* incidence – US data



NEN or NET is used as synonyms in different articles and databases
Source: JAMA Network, Dasari et al. (2025)

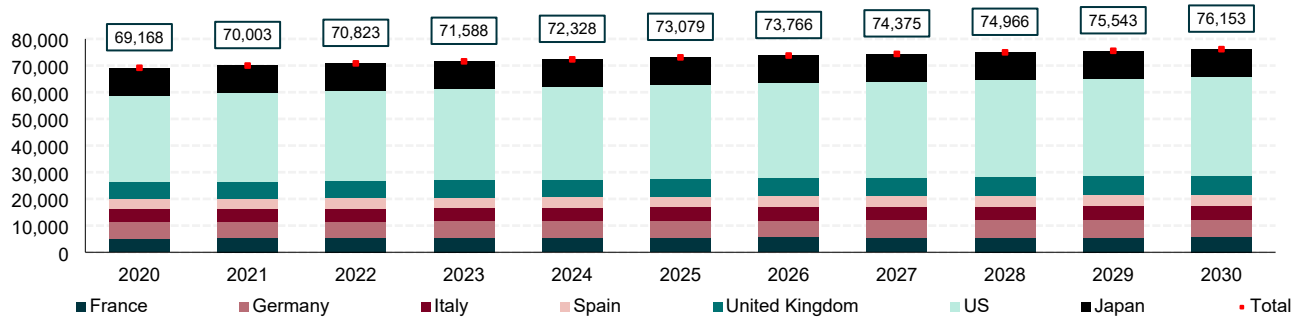
NET incidence rate by origin site – US data



Source: JAMA Network, Dasari et al. (2025)

While NET epidemiology is well documented in the US through large registries such as SEER, comparable, high-quality incidence and prevalence data across the rest of the 7MM are limited, with estimates often relying on fragmented national sources or extrapolations. In our analysis we have used data from GlobalData. GlobalData estimates that in 2025 there were c35,000 incident cases of NET in the US, c27,000 in EU4 and UK, and c10,500 in Japan.

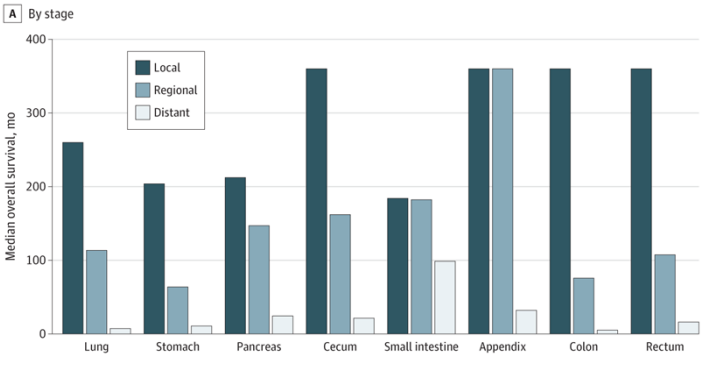
NET – diagnosed incident cases (US, EU4 + UK and Japan)



Source: SEER database, GlobalData, DNB Carnegie (chart visualisation)

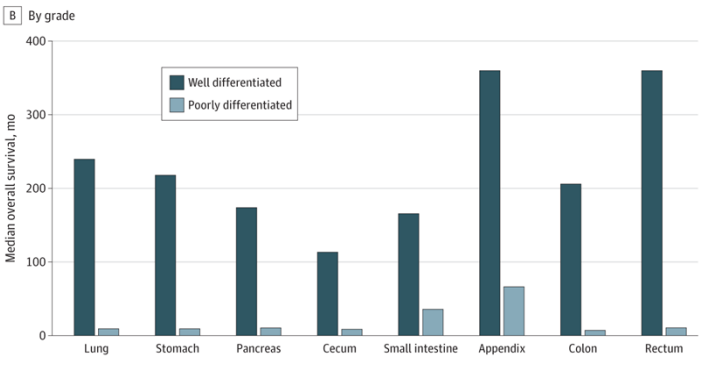
On a positive note, overall survival (OS) has improved meaningfully over time, with median OS of 11.8 years across all NETs and 6.7 years in distant-stage, well-differentiated disease, although outcomes remain highly dependent on age, stage, grade, and primary tumour site.

Overall survival by stage – US data



Source: JAMA Network, Dasari et al. (2025)

Overall survival by NET grade – US data



Source: JAMA Network, Dasari et al. (2025)

Symptoms and staging

Clinical presentation is highly variable – many NETs develop slowly over several years and may be asymptomatic in early stages, contributing to delayed diagnosis. Symptoms, when they do appear, will depend on tumour location and hormone secretion, and may include non-specific complaints such as fatigue, weight loss and reduced appetite.

- Gastrointestinal NETs can cause abdominal pain, diarrhoea, bowel obstruction or bleeding, flushing, and wheezing.
- Pancreatic NETs may lead to blood glucose disturbances, ulcers, gastrointestinal symptoms, and unexplained weight loss.
- Lung NETs often present with respiratory symptoms such as recurrent infections, coughing up blood, shortness of breath, or chest pain.

The underlying causes of most NETs remain poorly understood, although genetic predisposition and certain pre-existing conditions may increase risk. Diagnosis can be challenging and is sometimes incidental, as symptoms are frequently non-specific or mistaken for more common conditions. Work-up typically involves a combination of blood and urine tests (including neuroendocrine markers such as chromogranin A), imaging modalities such as CT, MRI and PET scans, endoscopic procedures where appropriate, and definitive confirmation through biopsy. The diversity in biology, presentation and clinical course underscores the need for site-specific and personalised management approaches in neuroendocrine cancers.

Our analysis focuses on gastroenteropancreatic NETs, as they represent the most common sub-group and account for the majority of clinical development, epidemiological data and approved therapies.

WHO 2019 classification of gastroenteropancreatic NETs

Morphology	Grade	Ki-67 index (%)
Well-differentiated NETs	G1	<3
Well-differentiated NETs	G2	3-20
Well-differentiated NETs	G3	>20
Poorly differentiated NECs • Small-cell • Large-cell	G3	>20
MiNEN Tumour-like lesions		

Source: National Cancer Institute, ESMO, WHO
Note: MiNEN – mixed neuroendocrine/nonendocrine neoplasm; NEC – neuroendocrine carcinoma; Ki-67 index – A way to measure how fast cancer cells in a tumour are dividing. Ki-67 is a protein found only in cells that are dividing. A high Ki-67 proliferation index means many cells are dividing quickly and that the cancer is likely to grow and spread. A Ki-67 proliferation index over 30% is typically considered high.

Well-differentiated NETs are graded G1 to G3 based on the Ki-67 proliferation index, a marker of how rapidly tumour cells are dividing. G1 tumours are slow-growing (Ki-67 <3%), G2 show intermediate proliferation (Ki-67 3–20%), while G3 NETs display higher proliferative activity (>20%) but retain well-differentiated morphology. Poorly differentiated NETs are inherently high-grade (G3), characterised by aggressive growth and thus high Ki-67 levels.

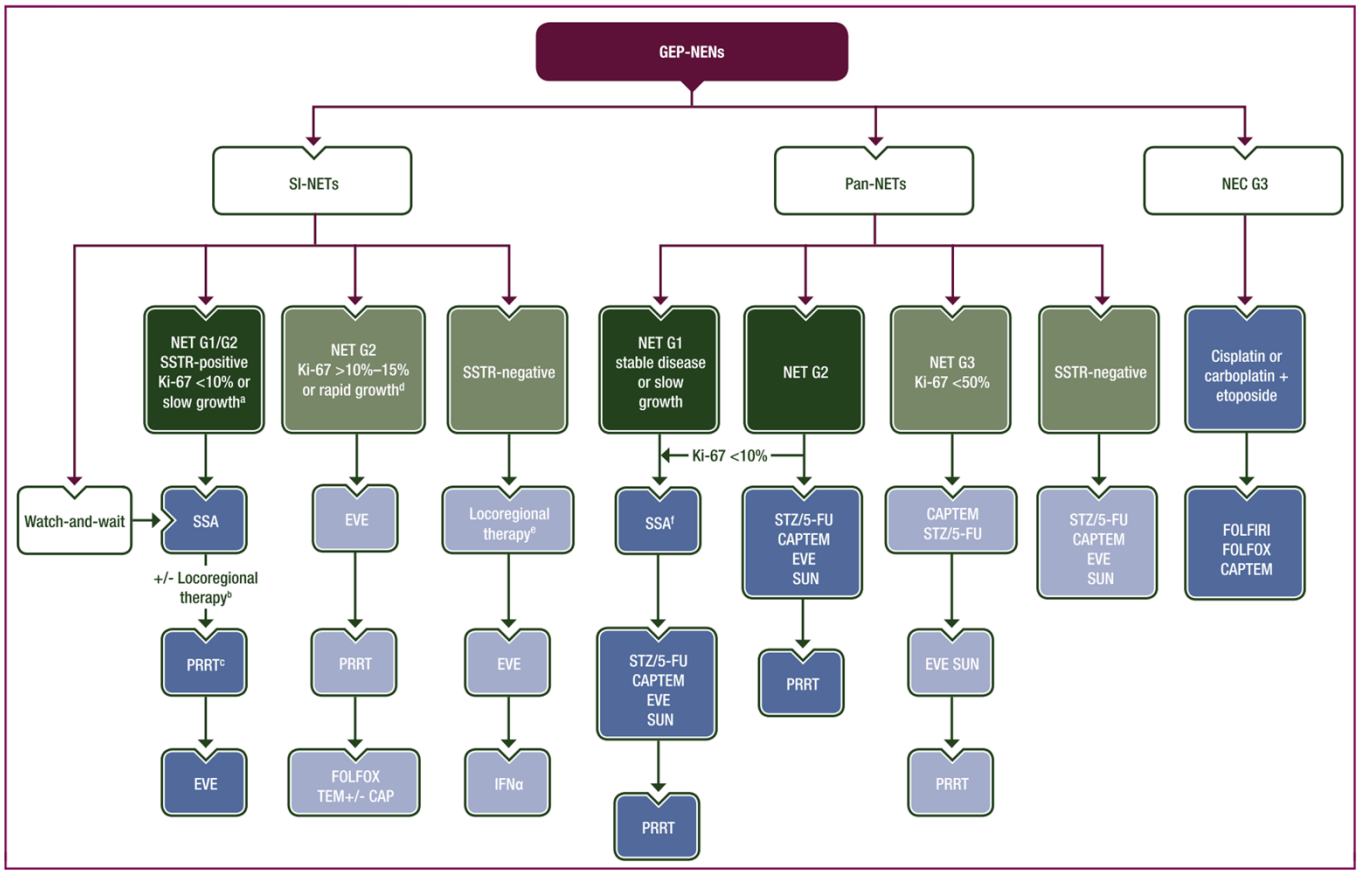
Current treatment landscape

Treatment of neuroendocrine tumours depends on the tumour type, location, whether it produces excess hormones, how aggressive it is, and whether it has spread to other parts of the body (Source: Oncology Times). In general, surgery is the main treatment modality (used in c71% of patients), followed by somatostatin analogues, chemotherapy, and radioactive isotopes and other targeted therapies (Source: European Neuroendocrine Therapy). The goal of treatment with somatostatin analogues is to suppress hormone production and slow tumour growth; many patients can live for long periods with this form of therapy.

Over the past decade, relatively few new treatments for progressive metastatic NETs have received FDA approval. For example, sunitinib (a tyrosine kinase inhibitor) and everolimus (an

mTOR inhibitor) were approved by the FDA for pancreatic NETs in 2011. This was followed by the approval of lanreotide, a somatostatin analogue (in 2014) for a specific class of NETs – gastroenteropancreatic NETs (GEP-NETs). Most recently, in 2018, the FDA approved Lutathera, a radioactive isotope therapy, for the treatment of progressive, somatostatin receptor–positive GEP-NETs (Source: Oncology Times).

Therapy overview in GEP-NETs



Source: Pavel et al. (2020)

Treatment of GEP-NETs is highly individualised and driven by tumour biology rather than a fixed treatment sequence. Key factors include tumour grade and Ki-67 index, growth rate, somatostatin receptor (SSTR) expression, symptom burden and disease distribution (liver involvement in particular).

In low-grade, well-differentiated NETs (G1 and selected G2), a watch-and-wait approach is appropriate for asymptomatic patients with stable disease. When treatment is needed, somatostatin analogues (SSAs) are the backbone in SSTR-positive disease. Locoregional treatment (for instance surgery, embolisation therapies or radionuclide therapies) may be used early in patients with liver-only or liver-dominant disease if extrahepatic disease is stable.

For NET G2, treatment choice depends largely on tumour growth rate. Slower-growing disease may be managed with SSAs or targeted therapy (e.g. everolimus), while progressive disease may require chemotherapy or PRRT in SSTR-positive patients. PRRT may be considered comparable to everolimus in SI-NET G2 and as an option after SSA or chemotherapy in SSTR-positive Pan-NET G1/G2.

Pan-NET G3 with moderate Ki-67 behaves closer to G2 disease and is treated accordingly, with streptozotocin- or temozolomide-based chemotherapy selected based on tumour growth. In contrast, poorly differentiated NEC G3 is an aggressive entity treated primarily with platinum-based chemotherapy.

Overall, therapy escalation is guided by documented disease progression, with an expanding role for PRRT and targeted agents enabling more tailored treatment strategies. To summarise,

- Management is biology-driven (grade, Ki-67, growth rate, SSTR status)
- Watch-and-wait is appropriate in selected low-grade, asymptomatic NETs
- SSAs are first-line in SSTR-positive disease
- Locoregional therapy is important in liver-dominant disease
- PRRT is a key option in SSTR-positive progressive NETs

Future treatment landscape

Several challenges remain to R&D of new drugs for NETs. Although significant resources have been and are being invested in oncology R&D, the majority of funding has historically been directed toward the most common cancers, such as breast, colorectal and lung. NETs, being classified as a rare cancer type, have received less attention and consequently less funding.

In a recent review in the Journal of Clinical Medicine, the pipeline for GEP-NETs was called “A new era of landmark trials”. New treatment modalities (radioligand therapies, tyrosine kinase inhibitors, immune checkpoint inhibitors and antibody-drug conjugates (ADCs)) are expanding options beyond conventional therapy. However, the authors note several structural challenges. While many novel agents demonstrate encouraging response rates and PFS, toxicity and quality-of-life impacts are non-trivial, particularly in indolent disease, where long treatment duration is common. High treatment costs, access constraints, and limited data on long-term disease control and resistance mechanisms have further limited broad adoption.

ADCs stand out as a particularly promising emerging class in advanced, treatment-refractory NETs, aiming to improve efficacy while limiting systemic toxicity by delivering potent cytotoxics directly to tumour cells (Source: Journal of Clinical Medicine). Early clinical data show durable responses, even in heavily pre-treated patients, positioning ADCs as a potential next growth driver in the space.

We found clinical trial data for c145 active clinical trials in NET indications on GlobalData – however this is data from 2020. At that time, data was heavily weighted toward early-stage development, with most assets in preclinical, Phase I or Phase II, and only a limited number of late-stage programmes nearing potential registration.

The pipeline is also largely driven by small-molecule therapies targeting established oncology pathways (e.g. VEGF, mTOR/PI3K, DNA damage), reflecting mostly incremental innovation. In contrast, SSTR-targeted therapies (including peptides and radioligand treatments) represent the most clinically advanced and validated segment, with several programmes in Phase II–III and forming the backbone of current NET treatment. Other modalities (such as monoclonal antibodies, checkpoint inhibitors, ADCs, cell and gene therapies, vaccines, and oncolytic viruses) represent a much smaller share of the pipeline and are mostly in early development. These approaches remain higher-risk, with limited clinical validation in NETs to date.

Glioblastoma

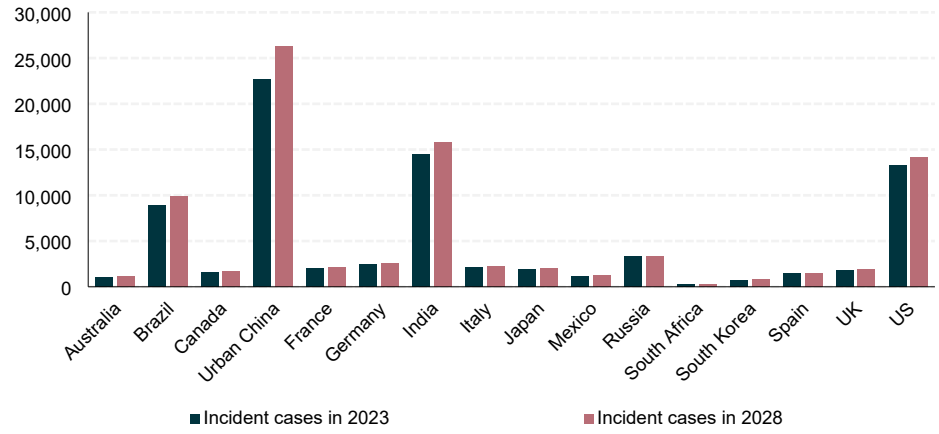
Epidemiology and disease background

Glioblastoma (GBM), also referred to as glioblastoma multiforme, is a WHO Grade IV astrocytoma and is the most common/aggressive primary malignant brain tumour in adults, accounting for approximately 48% of all malignant brain tumours. It originates from astrocytes (supportive glial cells within the central nervous system) and is characterised by rapid proliferation, diffuse infiltration into surrounding brain tissue, necrosis, and abnormal angiogenesis (Source: American Brain Tumour Association). Established risk factors include prior exposure to ionising radiation and rare inherited cancer syndromes (e.g. Li-Fraumeni, neurofibromatosis), which together account for <5% of cases. The vast majority of GBMs arise sporadically and there is no convincing evidence linking GBM to lifestyle factors, environmental toxins or mobile phone use (Source: Mayo Clinic).

GBM is highly invasive at the microscopic level, meaning tumour cells extend beyond the visible tumour mass, making complete surgical resection almost impossible.

GlobalData epidemiologists estimated 79,389 incident cases of GBM in 16 major markets in 2023, which they expect to grow to approximately 87,000 cases by 2028 (in 16MM – see graph below). Incidence increases with age, with most diagnoses occurring between 45 and 70 years.

Incident cases of GBM in 16MM in 2023 and forecast for 2028



Source: GlobalData

Despite aggressive treatment, prognosis remains poor. Median overall survival is approximately 12–15 months, with 2-year survival around 7% and 5-year survival in low single digits. Some sources note that survival declines markedly with age, and fewer than one in 10 patients survive beyond five years following diagnosis (Sources: American Brain Tumour Association, Mayo Clinic).

Symptoms and staging

Clinical presentation varies depending on exact tumour location, but typically reflects raised intracranial pressure and disruption of normal brain function. Common symptoms include persistent or worsening headaches, seizures, cognitive decline, personality or behavioural changes, focal neurological deficits (e.g. weakness or sensory loss), and speech or visual disturbances.

Diagnosis is primarily based on MRIs, which provide high accuracy of tumour detection, followed by histopathological confirmation via biopsy or surgical specimen. Molecular profiling is increasingly standard and includes different biomarkers.

The conventional TNM system (which classifies solid cancers based on tumour size and local extent (T), lymph node involvement (N), and distant metastases (M)) is not applicable to GBM, as the disease rarely spreads beyond the central nervous system and does not follow typical metastatic patterns. Instead, GBM is classified using the WHO grading system, which reflects tumour biology rather than anatomical spread. Brain tumours are graded from I to IV based on how abnormal the cells appear under the microscope and how aggressively they are expected to grow (Source: Glioblastoma Foundation). All glioblastomas are designated WHO Grade IV, the highest grade, reflecting their highly aggressive behaviour, rapid proliferation and poor patient prognosis.

Current treatment landscape

GBM treatment is individualised and will depend on tumour size, location, patient fitness and goals of care. When possible, management starts with maximal safe surgical resection, but complete removal is rarely achievable due to diffuse infiltration. Standard therapy consists of postoperative radiotherapy combined with chemotherapy temozolomide (TMZ).

In older or frail patients, hypofractionated radiotherapy regimens are commonly used, with TMZ added in selected candidates. Tumour Treating Fields (TTFields/Optune) are an approved adjunct in the adjuvant setting and have shown a statistically significant OS benefit, but real-world uptake remains limited by device burden, cost and adherence requirements. In the event of recurrence, options are limited and largely non-curative, but can typically include repeat surgery and/or re-irradiation in selected patients, and systemic approaches such as nitrosoureas (e.g. lomustine/carmustine). Bevacizumab is mainly used as a steroid-sparing, symptom-relief strategy rather than for durable survival benefit, while regorafenib is an NCCN-listed option but is not uniformly adopted. Overall, the paradigm remains anchored in surgery + radiochemotherapy, with incremental refinements (radiotherapy tailoring, TTFields, select chemo combinations; Source: Mayo Clinic, Journal of Clinical Oncology).

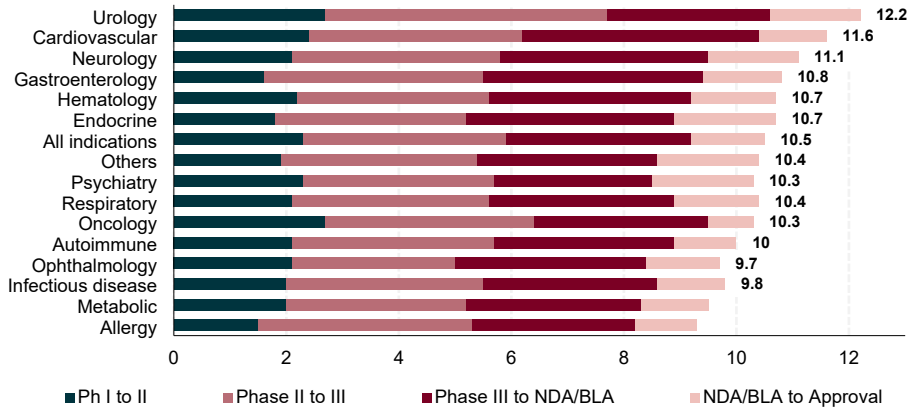
When it comes to GBM and immune-oncology, despite significant interest, immune checkpoint inhibitors have not improved survival in large randomised GBM trials (nivolumab programmes in newly diagnosed and recurrent settings were negative overall), underscoring the immunosuppressive microenvironment and biology-driven resistance. That said, a review published in Journal of Clinical Oncology highlights pockets of signal: neoadjuvant PD-1 blockade has shown immune activation and a survival signal in small studies, and vaccines remain active – most notably dendritic-cell approaches (including DCVax-L reporting longer OS in some cohorts) and surviving-targeting strategies (SurVaxM moving into randomised testing). Cell therapies (CAR T/CAR-NK) and oncolytic viruses have shown feasibility and occasional durable responses in early-phase work, but remain investigational, with mixed results to date.

Development pathway

Timelines

In general, for all indications, data from more than 6,000 successful phase transitions between 2011 and 2020 show that moving from Phase I to regulatory approval takes, on average, around 10.5 years. Oncology and haematology estimated transition times are 10.3 and 10.7 years, respectively (Source: Thomas et al., 2021). However, some sources indicate the typical development time for an oncology drug may be around 6.7 years (Source: Demicri et al., 2023). These timelines are highly indicative – some oncology drugs benefit from expedited approval pathways, such as Breakthrough Therapy and Accelerated Approval designations, which can shorten the development time.

Phase transition durations from Phase I – based on disease area (years)



Source: Thomas et al. (2021), DNB Carnegie (chart structuring)

That said, we stress that oncology trials are notoriously lengthy and often delayed, with long follow-up times, costly and prone to delays in general. A key factor of this inefficiency is patient recruitment: numerous studies show that more than 80–85% of trials are delayed, often due to difficulty identifying eligible patients, low enrolment rates, and poor retention (Source: Current Oncology, 2024). In cancer wards in general, fewer than 5% of adults participate in trials, and many referrals never progress to actual enrolment.

Operational bottlenecks exacerbate these issues. Trial start-up (site selection, contracting, and regulatory and ethics approvals) is one of the slowest phases, taking nearly eight months on average (Source: Current Oncology, 2024), potentially compressing downstream activities, further hindering recruitment and increasing the likelihood trials miss enrolment targets. There are also structural barriers, where many trials fail to enrol representative patients due to under-referral, limited health literacy, travel burdens or restrictive eligibility criteria. Older adults, ethnic minorities, and patients with comorbidities remain consistently underrepresented. Investigators themselves may face time constraints or limited awareness of available studies. Broader assessments, including those by the European Clinical Research Infrastructure Network (ECRIN), highlight recurring issues such as funding gaps, complex regulation, insufficient infrastructure and inadequate understanding of clinical research – all of which contribute to delays and, in some cases, even premature trial closure.

Regulatory levers for faster development

In immuno-oncology (IO) and advanced therapies, regulatory frameworks in both the US and EU offer multiple pathways designed to shorten development timelines and accelerate access to promising treatments. Unlike traditional drug development, IO and cell therapy programmes can sometimes move straight from early studies (Phase I/II) to large confirmatory trials (Phase III), significantly shortening the time to market. In the US, the FDA helps speed up promising treatments through programmes such as Accelerated Approval, Fast Track, and Breakthrough

Therapy designation. These pathways allow for closer dialogue with the FDA, the possibility to submit data on a rolling basis, and faster review timelines. Breakthrough Therapy status requires early clinical evidence showing that the treatment may offer a clear benefit compared with existing options. The FDA's Real-Time Oncology Review (RTOR) adds another layer of efficiency by enabling earlier submission of critical data, allowing reviewers to begin assessment ahead of the full application *(*Source*:* GlobalData, FDA, EMA*)*.

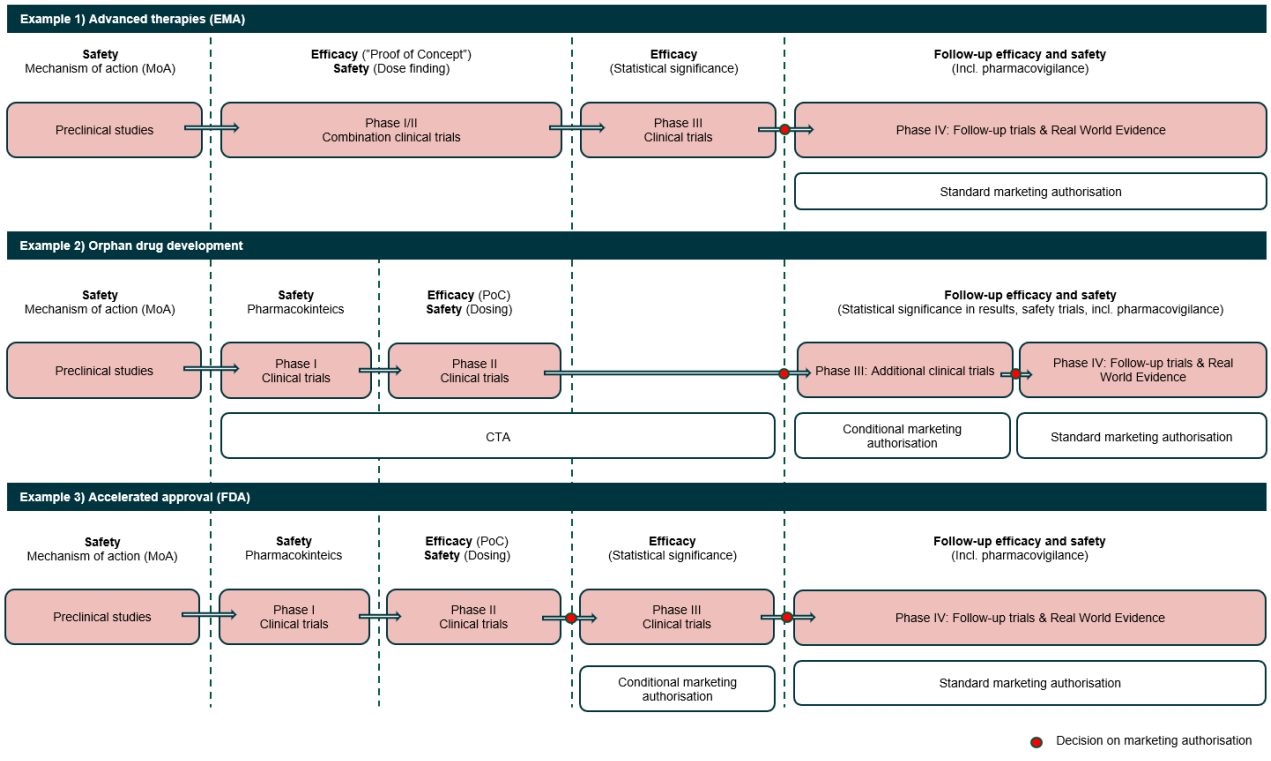
The FDA and EMA both provide orphan drug designation (ODD) with incentives such as extended market exclusivity and reduced regulatory fees for therapies addressing rare diseases. In the EU, the PRIME (priority medicine pathway) scheme supports early and enhanced regulatory guidance to expedite development, while cell-based therapies are evaluated under the ATMP framework, which offers specialised regulatory routes. Recent joint EMA and European Organisation of Research & Treatment of Cancer (EORTC) efforts include incorporation of patient-reported outcomes into oncology assessments that may further broaden the evidence base considered for approval.

Orphan Drug Designation

ODD provides several incentives for pharmaceutical companies developing treatments for rare diseases, including tax credits for clinical trial expenses, reduced patient requirements in clinical studies, exemption from certain FDA fees, and extended market exclusivity on approval. These incentives are designed to encourage investment in areas with limited commercial appeal due to small patient populations. ODD is granted for drugs targeting diseases affecting fewer than 200,000 patients in the US or fewer than 5 per 100,000 people in the EU (Source: FDA, EMA).

We find these pathways particularly relevant for Elicera. All three candidates (ELC-301, ELC-401 and ELC-100) target indications and patient groups with a significant unmet need. ELC-100 has already secured ODD in the US for the treatment of pancreatic neuroendocrine tumours, strengthening its regulatory position and supporting a potentially accelerated route to market. Additional expedited designations could materially shorten development timelines, reduce regulatory risk and enhance the company's attractiveness for partnering.

Illustrative development timelines



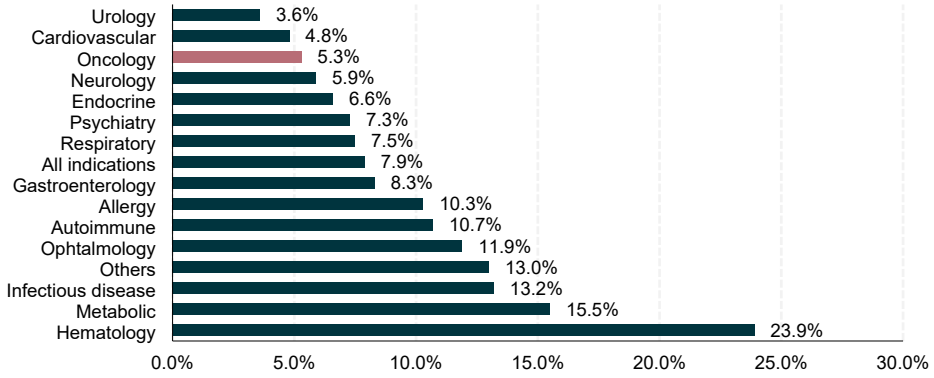
Source: DNB Carnegie (visualisation and graph structuring), Detela & Lodge 2019 (original graphics), GlobalData

LoA (likelihood of approval)

When we assess LoA (likelihood of approval (the estimated probability that a drug candidate will ultimately receive regulatory approval) across Elicera's pipeline, we consider each asset in the context of its modality and stage of development. To guide these assessments, we reference historical benchmarks that illustrate typical success rates across different programme types and development strategies. These benchmarks provide important context and form the basis for the risk-adjustments we apply to our sales forecasts at each stage.

A report analysing 12,728 clinical and regulatory phase transitions from 9,704 development programmes over 2011–20 showed overall approval likelihood from Phase I has historically been low at 7.9%, with Phase II posing the highest barrier, with fewer than a third of candidates advancing. Success rates vary widely across indications: haematology shows the strongest LoA from Phase I at 23.9%. Within oncology, IO stands out with a higher LoA of 12.4%, versus 5.3% for oncology programmes overall (Source: Thomas et al., 2021).

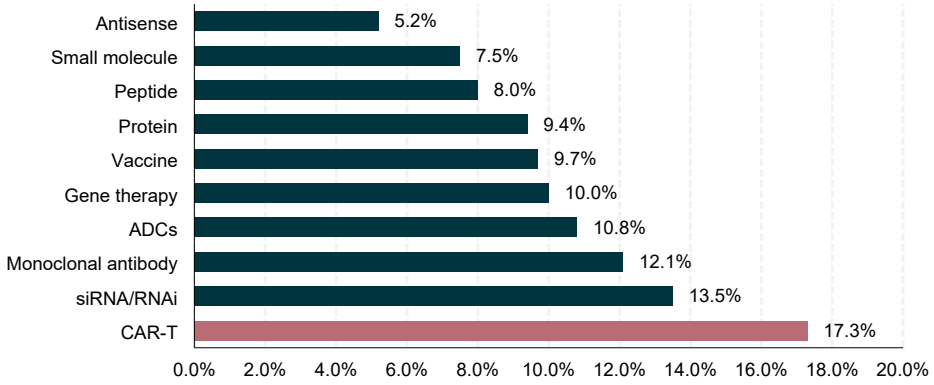
LoA from Phase I – by disease area (2011–20)



Source: Thomas et al., 2021

We need to view LoA for ELC-301 against the backdrop of low historical success rates for oncology programmes; however, CAR T development has seen the most success among selected novel modalities. At 17.3% (n=67), the Phase I LoA for CAR T-therapies is more than double the 7.9% average across all diseases, and more than triple the 5.3% Phase I LoA for all of oncology (Source: Thomas et al., 2021).

LoA from Phase I – novel modalities



Source: Thomas et al., 2021

Other more up-to-date sources, such as an extensive collection of data for phase transition success rates from GlobalData (over the past 18 years), indicate that LoA from Phase I to Approval is 10% for oncology (n=13,000), 7% for DLBCL (n=c700) and 15% for neuroendocrine cancers (n=300). For brain cancers, LoA is estimated at only c3% from Phase I, significantly below the broader oncology average, which reflects historically high failure rates in brain tumour drug development (Source: GlobalData). Only four drugs have been approved by the FDA for the treatment of GBM over the past five decades (Source: Advanced drug delivery reviews, 2026).

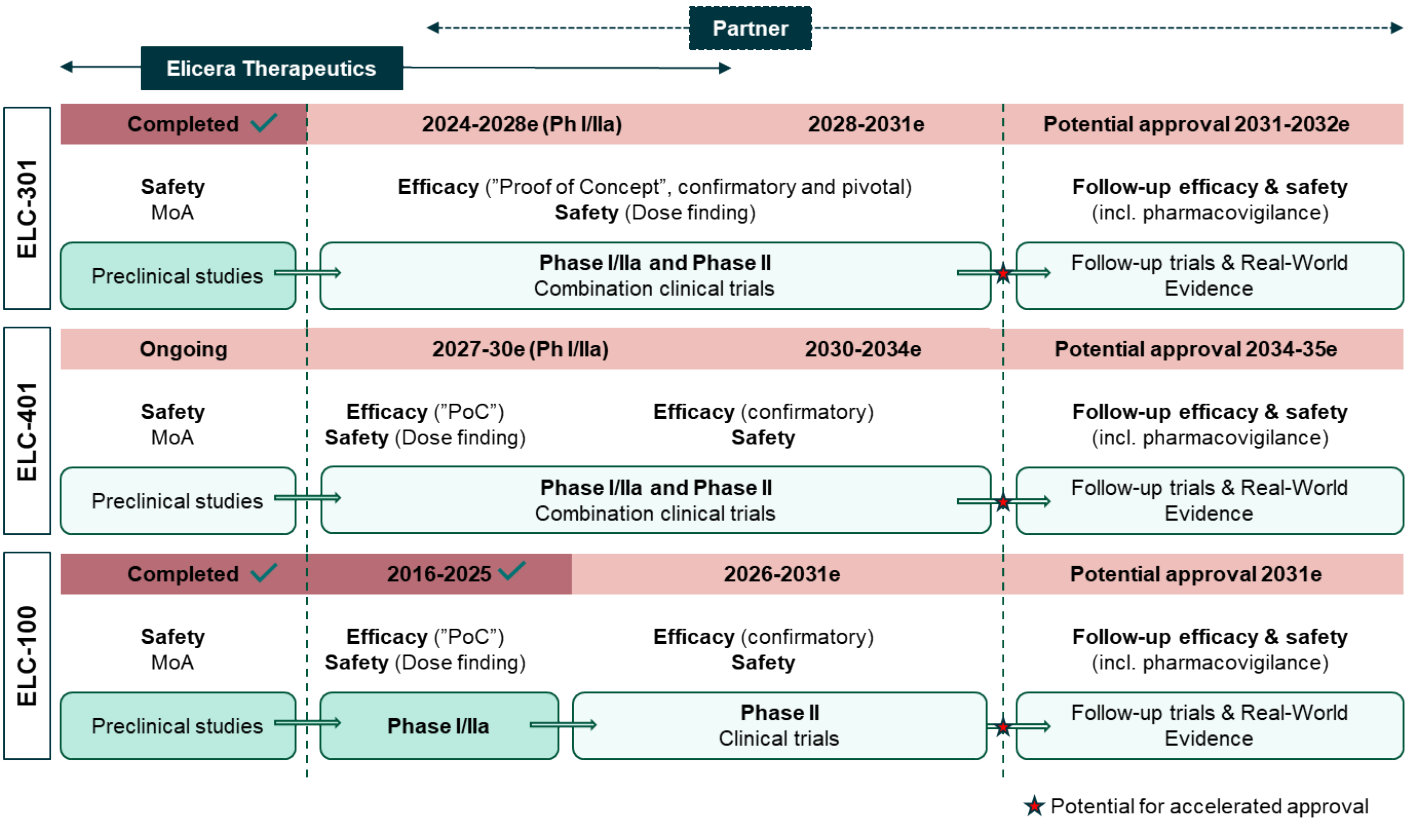
In our risk adjustment of Elicera Therapeutics, we incorporate LoA rates based on the CAR T specific data, CNS oncology data (brain cancer) and data on NETs – 17% for ELC-301, 3% for ELC-401 and 15% for ELC-100.

Phase transition success rate (%)

	PTSR				
	PI-PII	PII-PIII	PIII-Pre-registration	Pre-registration-Approval	PI-LoA (cumulative)
Oncology (for reference)	67%	38%	44%	84%	**9%**
DLBCL (for reference)	69%	27%	48%	75%	**7%**
Brain cancer	54%	18%	33%	99%	**3%**
Neuroendocrine cancer	74%	29%	87%	82%	**15%**
CAR T					**17%**

Source: GlobalData, Thomas et al. (2021)

Indicative timeline for ELC-301 and ELC-100 development



Source: DNB Carnegie (estimates and visualisation)

Higher bar ahead for next-generation CAR Ts?

One potential risk for the future of development of CAR Ts could be the FDA signals on tougher regulatory framework. The FDA has been signalling a tougher regulatory stance for new CAR T therapies, with full randomised controlled trials (RCTs) increasingly becoming the expected standard for approval. While earlier CAR Ts were often approved based on single-arm studies, the growing number of marketed products has prompted the agency to require controlled trials with robust comparators and survival or time-to-event endpoints to clearly demonstrate an incremental benefit.

According to an article in JAMA, trial design must carefully account for existing standards of care (including approved CAR Ts) as well as ethical considerations and confounding factors such as disease natural history and treatment expectations. Single-arm studies may still be acceptable in rare cancers or heavily pretreated populations, and response rates can support accelerated approval, but we believe full approval will increasingly hinge on stronger endpoints such as overall survival generated in RCTs.

This may potentially be a negative development for the future of ELC-301 and ELC-401 that may increase development costs further, and extend timelines and competitive hurdles. However, to our understanding, these FDA signals are not immediately relevant for Elicera. ELC-301 is aimed at patients with r/r DLBCL while ELC-401 targets glioblastoma – both settings characterised by high unmet medical needs and limited effective treatment alternatives. In such heavily pre-treated or aggressive indications, single-arm studies and response-based endpoints have historically remained acceptable and, in some cases, “good enough” for accelerated pathways.

Market dynamics and deal activity

As already mentioned, the market for advanced IO therapies is, unsurprisingly, defined by significant development costs, long lead times and ever-increasing demands on manufacturing and regulatory execution. In this landscape, we believe striking a partnership deal is not optional, but central. A strategic partner would provide capital, industrial capabilities and commercial reach, materially reduce execution risk and could accelerate the path to market. Below, we outline current market trends starting from a broad perspective on overall activity in pharma and biotech, then the cell and gene therapy space, and lastly, we highlight relevant benchmark deals in CAR T and oncolytic viruses to frame the partnership environment and what it could mean for Elicera Therapeutics.

Overall activity – M&A and licensing deals

We start by looking at the pharma-biotech landscape as a whole and the broader oncology space. Robust balance sheets, interest rate-cut expectations and looming patent cliffs are setting the stage for continued elevated deal activity in 2026, Bloomberg Intelligence (BI) wrote in its annual summary of pharma-biotech activities in 2025. BI expects pharma-biotech M&A to remain strong in 2026, supported by potential interest rate cuts and the roughly USD200bn in cash and balance-sheet flexibility held by large pharmaceutical companies.

Big pharma cash chests

Company	Currency	Period	Cash (USDm)	Net debt* (m)	Leverage ratio (x)
AstraZeneca	USD	Q3 2025	8,182	24,450	1.0
Bayer	EUR	Q3 2025	8,498	32,708	2.7
GSK	GBP	Q3 2025	4,445	14,444	1.2
Novartis	USD	Q3 2025	9,753	20,365	0.8
Novo Nordisk	DKK	Q3 2025	5,122	68,631	0.8
Roche	CHF	H2 2025	15,100	21,005	0.8
Sanofi	EUR	Q3 2025	10,450	11,245	0.7
AbbVie	USD	Q3 2025	5,671	63,072	2.0
Bristol-Myers	USD	Q3 2025	16,908	32,070	1.5
Eli Lilly	USD	Q3 2025	9,914	32,593	1.6
J&J	USD	Q3 2025	18,562	27,233	0.8
Merck & Co	USD	Q3 2025	18,214	23,160	0.8
Pfizer	USD	Q3 2025	14,984	46,728	1.5
Amgen	USD	Q3 2025	9,445	45,142	2.1
Biogen	USD	Q3 2025	3,960	2,325	0.7
Gilead	USD	Q3 2025	9,354	15,587	1.0
Regeneron	USD	Q3 2025	18,729	-17,464	n.a.
Vertex	USD	Q3 2025	12,010	-12,010	n.a.

Source: Bloomberg Intelligence
Note: *Net debt in local currency

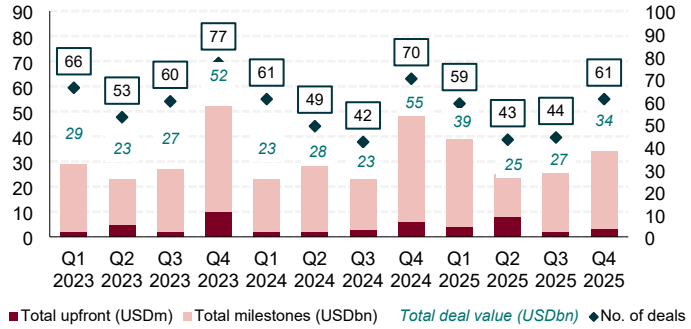
M&A activity in 2025 was close to historical peak levels. As of 1 December, 28 acquisitions valued at USD1bn or more had been announced or completed, compared with 25 during all of 2024, with a total deal value of USD104bn versus USD63bn. However, while deal count was close to the 2015 peak (29 transactions), the aggregate value was roughly 60% below the 2019 high of USD260bn (Source: Bloomberg Intelligence).

More granular data from CapIQ and BiomedTracker suggests M&A activity picked up fairly late into the year, and strengthened sharply in Q4, reaching a post-pandemic high, led by Phase III and commercial-stage transactions. Buyers showed a clear preference for more de-risked assets, reflecting focus on near-term revenue generation. This willingness to pay for reduced risk was also evident in pricing, with the median deal size for commercial-stage targets up from USD750m in 2024 to USD935m in 2025 (Source: DNB Carnegie//Back Bay).

Neurology accounted for a large share of the deal value, the bulk in just two transactions (J&J's acquisition of Intra-Cellular (USD14.6bn) and Novartis' acquisition of Avidity (USD12bn)).

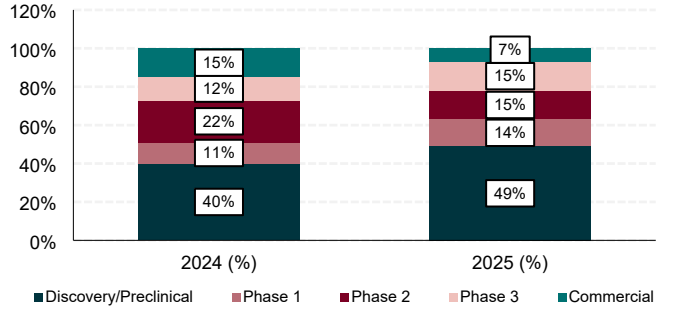
Aside from M&A, licensing is still the preferred deal type, since drug in-licensing remains a less-risky and favoured way to boost pipelines, BI writes. Though activity eased relative to the pandemic-fuelled highs of 2020–21, the deal volume in 2025 was still robust, with oncology programmes remaining a highly sought-after field (about 25–30% of all deals (Source: BI)). Overall licensing activity in 2025 fell slightly in volume terms compared with 2024 (207 versus 222 deals); however, the total deal value was broadly stable, supported by a handful of large partnerships and milestone-heavy structures. In contrast to the late-stage bias seen in M&A, licensing was skewed earlier in development, as companies moved to secure differentiated mechanisms of action before further clinical catalysts could drive valuations higher. Discovery and pre-clinical programmes accounted for 49% of deal volumes (up from 40% in 2024), while commercial-stage deals declined to 7% (from 15%), reflecting a strategic preference for earlier entry points combined with milestone-based risk sharing.

Licensing deals by quarter (USDm and number of deals)



Source: BiomedTracker, DNB Carnegie//Back Bay Life Science Advisors

Licensing deals based on development stage – 2024 vs. 2025



Source: BiomedTracker, DNB Carnegie//Back Bay Life Science Advisors

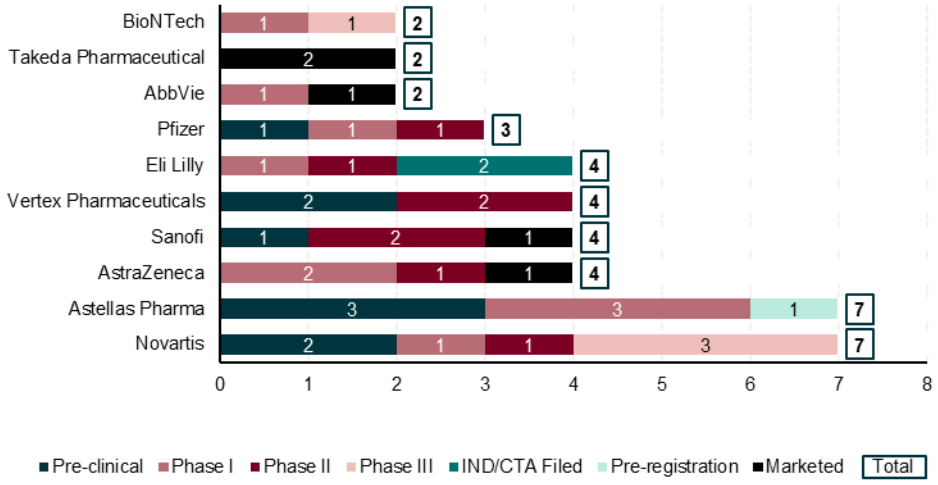
BiomedTracker data also shows that oncology dominated the licensing landscape in 2025, with 53 deals representing cUSD57bn in total value. Activity was particularly concentrated around PD-1 x VEGF bispecifics following strong data from Summit's ivonescimab, triggering a wave of competitive deal-making. China-origin innovation also remained a defining theme, highlighting the growing recognition of China-based R&D as a high-quality and cost-efficient source of assets for Western companies (Source: DNB Carnegie//Back Bay Healthcare Update, February 2025).

Cell and gene therapy landscape

Unlike the overall trend described above, where licensing has proved a popular route for big pharma to get access to new technologies, the picture in cell and gene therapies (CGT) is a little different. Over the past 10 years there has been a strategic shift away from licensing deals toward M&As in the CGT space. Only four CGT licensing deals were signed in H1 2025. Historically, companies such as J&J, AstraZeneca, Novartis, BMS and Roche all pursued strategic CGT acquisitions to strengthen pipeline differentiation.

The absolute number of M&A deals with CGTs peaked in 2023, with 25 completed that year, while that year, the number of licensing deals dipped below M&A for the first time since 2015 (Source: GlobalData).

M&A top acquirers in cell and gene therapy – by phase (number of deals, 2015–25)



Source: GlobalData

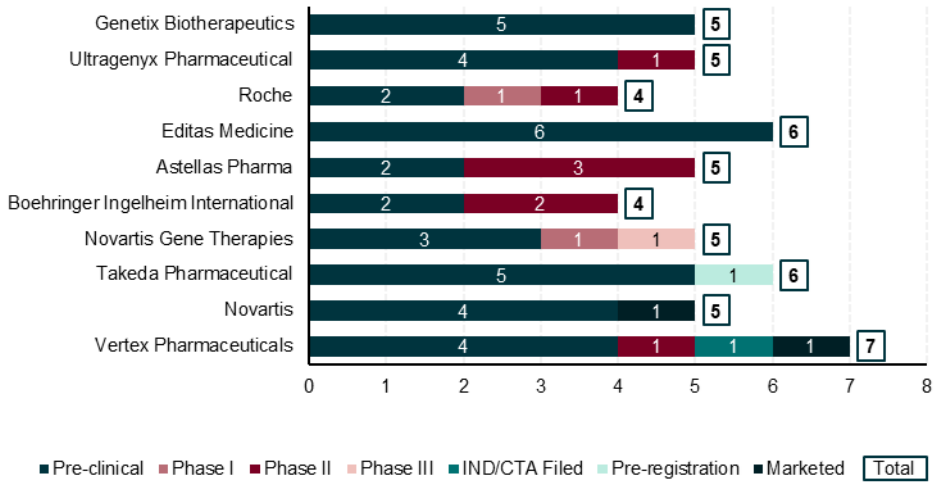
Novartis and Astellas lead by total number of acquisitions. Acquisition data from GlobalData spans multiple phases but is skewed towards later-stage assets (Phase II, Phase III and beyond), indicating a preference for more de-risked programmes. Companies such as AstraZeneca, Sanofi, Vertex and Eli Lilly have also been active, often targeting mid- to late-stage assets where clinical validation had been established.

On the other hand, the licensing landscape differs somewhat, even including top licensors. In licensing, activity is more heavily weighted towards earlier-stage programmes, particularly pre-clinical and IND/CTA-filed assets. Companies like Vertex, Editas, Takeda and Ultragenyx stand out for the volume of licensing deals, often securing pipeline optionality earlier in development. Compared with M&A, licensing appears more focused on accessing innovation earlier in the value chain, while acquisitions are used to secure more advanced, de-risked assets.

Looking at data for deal values, and not just volumes over 2015–25, Roche stands out with roughly USD14bn in licensing commitments, driven by major platform-focused transactions such as Poseida (USD6bn), Shape (USD3bn) and Sarepta (cUSD3bn). Vertex has similarly pursued a platform-consolidation strategy, entering seven high-value licensing and co-development deals (cUSD5bn in total) to strengthen gene-editing and mRNA delivery among others (cUSD5bn in total). Bristol Myers Squibb has expanded its oncology and in vivo editing pipeline through alliances in the CGT space for a total deal value of USD4bn. Allogene has concentrated on securing intellectual property around allogeneic CAR T and donor-cell platforms to improve scalability and reduce manufacturing costs.

Zooming in from the large landscape of CGT deals, let us now look more closely at relevant deals in the CAR T and oncolytic virus spaces, which we later use as a benchmark in our forecasts for Elicera.

Top licensors in cell and gene therapy – by phase (number of deals, 2015–25)



Source: GlobalData

Some of the deals in the CAR T and oncolytic virus space

CAR T deals

With help from GlobalData registry over 2015–25, we identified 58 Phase I-II CAR T licensing deals of which we highlight 10 below where detailed, publicly available terms were disclosed. Over the past 12 years, leading oncology-focused CAR T transactions have reached total deal values of USD1bn–6bn, particularly for differentiated platform technologies.

Upfront payments varied significantly depending on development stage and asset maturity. Earlier-stage or option-based deals (e.g. Keyi-ERIGEN; Precision-TG) typically carry relatively modest upfront payments in the low double-digit millions of USD; in contrast, clinical-stage assets with stronger validation (e.g. Legend-Novartis at USD100m upfront and CBMG-Janssen at USD245m upfront) have made materially higher initial commitments (Source: GlobalData).

Based on these recent CAR T licensing benchmarks, we believe a reasonable deal range for Elicera's early-stage CAR T programme ELC-301 would reflect its current development stage and single-asset positioning. Comparable early clinical or IND-stage oncology CAR T transactions typically feature upfront payments in the USD10m–30m range and total deal values of approximately USD200m–400m, with the majority of value back-end loaded through development and commercial milestones.

Against this backdrop, an upfront payment of around USD15m–30m and a total potential deal value of cUSD300m appears reasonable to us for ELC-301 and ELC-401 in a base-case scenario. Should complete Phase I/IIa data demonstrate clear differentiation in efficacy, durability and/or safety, we see scope for improved deal terms, potentially moving upfront payments higher and increasing the total deal value accordingly. In this context, our current assumption of USD20m upfront and USD300m total deal value sits at the conservative end of the observed benchmark range and appears defensible, while leaving room for upside upon strong clinical validation.

Some of the licensing deals in CAR T space

Year	Originator	Sponsor	Upfront (USDm)	Total deal value (USDm)
2022	Poseida Therapeutics	Roche	110	6,000
2019	Cellectis	Allogene Therapeutics	Undisclosed	2,800
2025	Keyi (Zhejiang) Pharmaceutical	ERIGEN LLC	15	1320 (+ up to 800 in sales royalties)
2014 (amended 2024)	Cellectis	Servier (sublic. Allogene)	66	Up to 1,106
2024	Umoja Biopharma	AbbVie	Undisclosed	Up to 1,440 (+ sales milestones & royalties)
2024	Precision BioSciences	TG Therapeutics	7.5 (part of 17.5 near-term)	Up to c300
2016	Transposagen Biopharmaceuticals	Janssen Biotech (J&J)	Undisclosed	Up to 292 per product
2017 (revised 2022)	Kite Pharma (Gilead)	Daiichi Sankyo	50	Up to 200
2023	Cellular Biomedicine Group (CBMG)	Janssen Biotech (J&J)	245	Undisclosed
2023	Legend Biotech	Novartis	100	Up to 1,010 (+ royalties)
Average*			**85**	**1,060**

Source: DNB Carnegie (estimates) & company
Note: Excluding Roche-Poseida deal from the total deal value average

Oncolytic viruses deals

Publicly available deal data for early-stage oncolytic virus programmes in oncology is more limited than CAR T and broader cell therapy transactions. Nevertheless, we believe the following provide relevant reference points:

2010 – Jennerex/Transgene (Phase I/II, regional Europe): Strategic collaboration for Pexa-Vec with up to USD116m in milestones plus equity participation. Transgene secured European rights and committed to mid-/late-stage development.

2017 – Oncolytics Biotech/Adlai Nortye (ANBC): Phase III oncolytic virus (REOLYSIN) licensed for Asia-Pacific. The deal included USD5.3m upfront (USD21.2m near-term total) and a USD86.6m total deal value including milestones plus double-digit royalties. Regional commercialisation structure, with ANBC leading development and launch in licensed territories.

2020 – ImmVira/Shanghai Pharmaceuticals: Phase I intratumoral oncolytic HSV-1 (MVR-T3011) licensed for Greater China. Total deal value of USD165.5m (CNY1.15bn), milestone-heavy with royalties up to 12%. Shanghai Pharma responsible for regional development and commercialisation; ImmVira retains ex-China rights.

2020 – KaliVir/Astellas (Preclinical, global): Worldwide licence and research collaboration for intravenously delivered vaccinia virus (VET2-L2) plus a follow-on product. Up to USD56m upfront/research support and up to USD634m total potential across both products, plus royalties.

2021 – Genelux/Newsoara (Phase III, regional): Exclusive Greater China licence for Olvi-Vec and a broader vaccinia platform. USD11m upfront/near-term payments and up to USD160.5m per product in milestones, plus tiered royalties. Regional commercialisation structure, with the partner funding development locally despite late-stage asset status.

Based on the transactions listed above and GlobalData deal data, we believe ELC-100, having completed Phase I/IIa, would most closely benchmark against early Phase II regional licensing deals rather than large multi-asset platform collaborations. Historically, such transactions have been milestone-heavy, with modest upfront payments and total deal values typically in the USD100m–300m range, depending on scope and differentiation.

For ELC-100, in this context we assume an upfront payment of USD10m–15m and a total potential deal value of USD150m–200m as a reasonable base-case range.

Financial forecasts

We include the two lead candidates, ELC-301 and ELC-100 in our forecasts (ELC-401 and ELC-201 are excluded given their early development stage). We view partnering as the most likely route to market for both programmes. Despite increasing competition in the immuno-oncology field, we believe ELC-301 has a clear opportunity in a defined niche – heavily pre-treated DLBCL patients in the third-line setting – and we forecast peak sales of approximately USD500m across the 7MM markets by 2037. For ELC-401, we assume peak sales of above USD1bn in the 7MM by 2041, reflecting the high unmet need in recurrent glioblastoma and the potential applicability of the therapy in the second-line setting and beyond. However, given that the programme remains in an early stage of development, we apply a substantial risk adjustment in our model, which significantly reduces the contribution to the overall rNPV. For ELC-100, which targets an even more niche population, we estimate peak sales of around USD200m by 2037. Given the capital-intensive path through clinical trials, our estimates assume that a strategic partner advances both assets through late-stage development and commercialisation. Below, we outline our assumptions on pricing, sales and costs.

Geographical scope

When estimating sales for Elicera, we include the US and EU4 countries (Germany, Spain, France and Italy) + the UK in our models for ELC-301 and ELC-100. Additionally, we include Japan in our forecasts for ELC-301 sales, since we have available epidemiological data for the region for DLBCL. The US and Europe have many similarities in their regulatory framework, and we believe that clinical data generated in each region can be leveraged for market submissions in both markets. We also include Japan in our forecasts, as the Japanese regulatory framework is also broadly aligned with US and EU standards, often accepting foreign clinical data with limited bridging requirements. This enables potential regulatory submissions based on largely the same clinical package, making Japan an attractive and capital-efficient third core market opportunity in our model.

ELC-301 for DLBCL

Based on the company's current timelines, with the full CARMA trial readout planned for 2028, we estimate that Elicera could initiate a confirmatory Phase II trial in 2028e. Assuming a positive outcome, a potential accelerated approval pathway could enable market entry in 2031–32e. This timeline is highly dependent on the strength of both the Phase I/IIa data and subsequent trial results. The company does not provide its own projections regarding time to market.

In modelling the potential addressable market, we use the incidence of DLBCL in the 7MM (US, EU4, UK and Japan) as the key metric. According to GlobalData, DLBCL incidence in 2024 amounted to approximately 65,000 patients across the 7MM, alongside a substantially larger prevalent diagnosed population of around 190,000 patients. Of total incident cases, we assume that 30–40% progress to second-line (2L) treatment, and that 45–55% of these patients fail therapy and require alternative options, thereby moving into the third line (3L) setting. This results in an estimated target population of approximately 8,900 patients in 2026e.

DLBCL incidence (men and women, >18 y.o.)

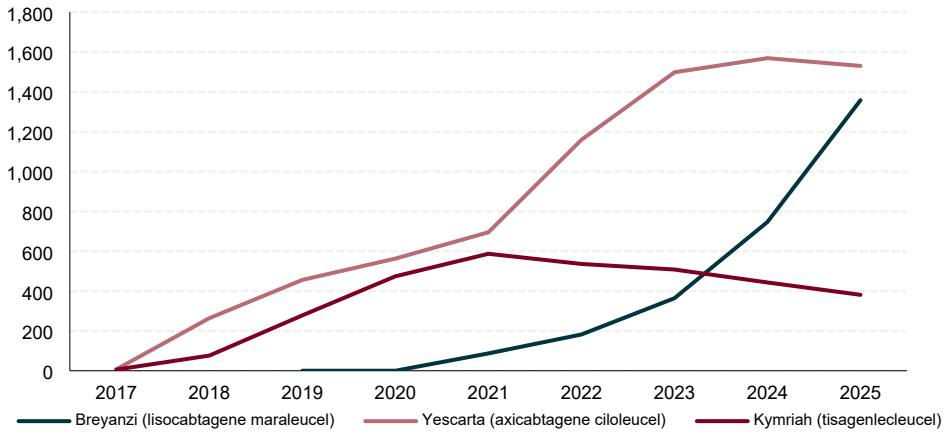
	2024	2025	2026	2027	2028	2029	2030	2031	2032	2033
USA	22,487	22,885	23,068	23,245	23,418	23,584	23,745	23,902	24,054	24,203
France	5,455	5,476	5,499	5,523	5,546	5,570	5,594	5,617	5,640	5,661
Germany	8,721	8,706	8,689	8,672	8,657	8,641	8,625	8,609	8,592	8,577
Italy	4,092	4,136	4,184	4,233	4,283	4,337	4,390	4,444	4,495	4,548
Spain	1,923	1,956	1,988	2,021	2,059	2,095	2,131	2,169	2,206	2,246
UK	7,199	7,300	7,406	7,513	7,616	7,718	7,819	7,920	8,015	8,109
Japan	14,739	14,825	14,868	14,941	15,039	15,130	15,215	15,275	15,304	15,318
Total incident cases (no. of patients)	**64,616**	**65,284**	**65,702**	**66,148**	**66,618**	**67,075**	**67,519**	**67,936**	**68,306**	**68,662**

Source: GlobalData

Further, we assume launch in 2031–32e, a market penetration of 15% and a gradual ramp-up of sales up until peak of cUSD500m in 2037e. For pricing, we assume pricing in line with average marketed list prices of Yescarta, Kymriah and Breyanzi in Europe, the US and Japan – USD325,000 for Europe, USD466,000 for the US and USD176,000 for Japan, respectively (Source: GlobalData). In our view, the fourth-generation design and iTANK arming could justify parity pricing if CARMA yields positive data. However, we do not model any premium at this stage, as pricing will ultimately depend on demonstrated clinical benefit and competitive dynamics at the time of launch.

We assume that ELC-301 reaches peak sales five to six years post-launch (i.e. 2037, assuming launch in 2031–32), which is broadly in line with historical CAR T ramp profiles, see graph below. It is worth noting that Kymriah, Yescarta, and Breyanzi are all approved for multiple B-cell malignancy indications (including various types of large B-cell lymphoma and follicular lymphoma, and Kymriah also approved for acute lymphoblastic leukemia and Breyanzi for mantle cell lymphoma) and the below graph represents sales for all indications. Yescarta launched in 2017 and reached cUSD1.6bn in annual sales by 2024, implying c7 years to peak. Kymriah ramped to peak sales in 4 years to cUSD600m by 2021, before declining due to competitive pressure. Breyanzi, which launched later, shows a steep ramp from 2021 onward.

Sales build for Breyanzi, Yescarta and Kymriah (USDm)



Source: GlobalData, Gilead, BMS and Novartis

The above ramp-up illustration broadly reflects overall commercial trajectories of oncology drugs once launched. A report by GlobalData, focused on drugs approved in a single oncology indication, had shown that oncology drugs typically reach peak sales within five to seven years: the median time to peak was five years in the US, six years in the EU and seven years in the rest of the world, with a global median of approximately 5.5 years. Faster uptake in the US is largely driven by earlier and more streamlined reimbursement following FDA approval, whereas longer timelines in Europe and other regions reflect more protracted pricing and reimbursement processes.

Market penetration for ELC-301 is one of the most challenging factors to estimate. When assuming a 15% market penetration, we keep in mind that ELC-301 is meant to be positioned as a potentially transformative therapy in a setting characterised by high unmet medical need and poor prognosis – 3L and beyond.

Unlike broader oncology settings where patients are frequently managed in community hospitals (c80% of oncology patients in the US are diagnosed and treated in community centres), r/r DLBCL patients who would typically require a CAR T treatment are treated at highly specialised centres. Treatment decisions are concentrated among a limited number of highly specialised physicians, which may facilitate relatively rapid adoption if clinical data are compelling. Centralisation in this case may therefore support, rather than limit, uptake.

With a 15% market penetration assumption we do not imply market dominance or replacement of standard of care in 3L. We rather assume that ELC-301 secures a meaningful position within a defined treatment line. We believe it reflects infrastructure realities and competitive risk, while remaining consistent with concentrated academic adoption patterns in high unmet-need oncology settings.

Sales build for ELC-301

		2032	2033	2034	2035	2036	2037	2038	2039	2040
Addressable population for ELC-301										
USA	3,227	3,247	3,267	3,299	3,330	3,362	3,394	3,427	3,459	3,493
EU4 + UK	3,882	3,908	3,934	3,962	3,991	4,020	4,049	4,079	4,109	4,140
Japan	2,062	2,066	2,068	2,083	2,098	2,113	2,129	2,144	2,160	2,175
	9,171	**9,221**	**9,269**	**9,344**	**9,419**	**9,495**	**9,572**	**9,650**	**9,728**	**9,808**
USA										
Addressable population (n)	3,227	3,247	3,267	3,299	3,330	3,362	3,394	3,427	3,459	3,493
		START					**PEAK**			
Launch curve										
Market penetration		0%	2%	5%	8%	12%	15%	12%	11%	9%
ASP (USD)		466,000	470,660	475,367	480,120	484,921	489,771	494,668	499,615	504,611
Price increase		1%	1%	1%	1%	1%	1%	1%	1%	1%
No. of patients treated (n)		5	49	148	275	403	509	411	363	314
Sales USA (USDm)		**2**	**23**	**71**	**132**	**196**	**249**	**203**	**181**	**159**
EU4 + UK										
Addressable population (n)	3,882	3,908	3,934	3,962	3,991	4,020	4,049	4,079	4,109	4,140
		START					**PEAK**			
Launch curve										
Market penetration		0%	2%	5%	8%	12%	15%	12%	11%	9%
ASP (USD)		325,000	328,250	331,533	334,848	338,196	341,578	344,994	348,444	351,928
Price increase		1%	1%	1%	1%	1%	1%	1%	1%	1%
No. of patients treated (n)		6	59	178	329	482	607	489	431	373
Sales EU4 + UK (USDm)		**2**	**19**	**59**	**110**	**163**	**207**	**169**	**150**	**131**
Japan										
Addressable population (n)	2,062	2,066	2,068	2,083	2,098	2,113	2,129	2,144	2,160	2,175
		START					**PEAK**			
Launch curve										
Market penetration		0%	2%	5%	8%	12%	15%	12%	11%	9%
ASP (USD)		176,000	177,760	179,538	181,333	183,146	184,978	186,828	188,696	190,583
Price increase		1%	1%	1%	1%	1%	1%	1%	1%	1%
No. of patients treated (n)		3	31	94	173	254	319	257	227	196
Sales Japan (USDm)		**1**	**6**	**17**	**31**	**46**	**59**	**48**	**43**	**37**
TOTAL SALES (USDm)		**5**	**48**	**147**	**274**	**405**	**516**	**420**	**375**	**327**

Source: DNB Carnegie (estimates)

Below, we have conducted a sensitivity analysis to visualise the impact of changes on peak sales in two key parameters: market penetration and list price in the US.

Sensitivity analysis on effect on peak sales (USDm) – ASP (USD) versus market penetration (%)

		ASP (USD)			
		200,000	300,000	466,000	500,000
Market penetration (%)	5%	125	142	171	178
	10%	249	285	342	356
	15%	374	427	**516**	534
	20%	498	569	684	712
	25%	623	712	854	890

Source: DNB Carnegie (estimates)

ELC-401 in Glioblastoma

Based on the company's latest published plans for ELC-401, we understand that preclinical development has now been completed. The next step is to advance the programme into clinical development, with a planned Phase Ia/Ib dose-escalation and expansion trial in patients who have previously undergone standard treatment (2L and beyond). Elicera is currently pursuing several opportunities to secure non-dilutive funding, such as grants, and responses to these applications are expected throughout 2026. In our estimates, we assume that the clinical trial could begin in 2027e, with a potential market entry in 2035e.

In modelling the potential addressable market, we look at GBM incidence across the EU4, the UK, the US, and Japan (7MM) as the key starting point. According to GlobalData, GBM incidence was c22,980 patients across the 7MM in 2024. To estimate the addressable population for ELC-401, we adjust for IL13RA2 expression, as IL13RA2 is a GBM-restricted receptor expressed in c50–75% of GBM patients, according to published sources (Oncotarget; Materials Today: Bio). In the planned Phase Ia/Ib trial, patients will be eligible for treatment if their tumours are IL13RA2-positive and they have recurrent disease scheduled for a second surgery.

Recurrence rates in GBM are very high. The journal Cancers notes that “recurrence is the usual way of progression,” while other sources suggest that c70% of newly diagnosed patients experience recurrence within one year of diagnosis (GlobalData). In our model, we therefore estimate the addressable population by applying IL13RA2 expression rates and assuming use of ELC-401 in the second-line setting and beyond. This results in an estimated target population of c12,700 patients by 2035e.

GBM incidence (men and women, >18 y.o.)

	2024	2025	2026	2027	2028	2029	2030	2031	2032	2033
USA	12,145	12,318	12,483	12,638	12,785	12,931	13,082	13,200	13,319	13,438
EU4 + UK	9,061	9,143	9,224	9,307	9,392	9,473	9,555	9,638	9,721	9,805
Japan	1,774	1,778	1,778	1,782	1,787	1,789	1,792	1,787	1,781	1,776
Total incident cases (no. of patients)	**22,980**	**23,239**	**23,485**	**23,727**	**23,964**	**24,193**	**24,429**	**24,624**	**24,821**	**25,020**

Source: GlobalData

Further, we assume a potential launch in 2035e, with market penetration reaching 15–25% over time and a gradual ramp-up of sales to peak revenues of cUSD1.1bn by 2041e. For pricing, we assume a level comparable to ELC-301, reflecting the advanced therapy modality and the high unmet need in this patient population. We assume treatment setting for ELC-401 in second line and beyond (2L+), targeting patients with recurrent disease after standard-of-care treatment.

Sales build for ELC-401

	2035	2036	2037	2038	2039	2040	2041
EU4 + UK							
Adressable population (n)	4,988	5,031	5,075	5,119	5,163	5,208	5,253
	START						**PEAK**
Launch curve	0.01	0.10	0.25	0.45	0.75	0.85	1.00
Market penetration	0%	2%	4%	7%	11%	13%	15%
ASP (USD)	325,000	325,000	325,000	325,000	325,000	325,000	325,000
No. of patients treated (n)	7	75	190	346	581	664	788
Sales EU4 + UK (USDm)	**2**	**25**	**62**	**112**	**189**	**216**	**256**
USA							
Adressable population (n)	6,841	6,902	6,964	7,027	7,090	7,154	7,219
	START						**PEAK**
Launch curve	0.01	0.10	0.25	0.45	0.75	0.85	1.00
Market penetration	0%	3%	6%	11%	19%	21%	25%
ASP (USD)	466,000	466,000	466,000	466,000	466,000	466,000	466,000
No. of patients treated (n)	17	173	435	791	1,329	1,520	1,805
Sales USD (USDm)	**8**	**80**	**203**	**368**	**620**	**708**	**841**
Japan							
Adressable population (n)	883	880	877	875	872	869	867
	START						**PEAK**
Launch curve	0.01	0.10	0.25	0.45	0.75	0.85	1.00
Market penetration	0%	2%	4%	7%	11%	13%	15%
ASP (USD)	176,000	176,000	176,000	176,000	176,000	176,000	176,000
No. of patients treated (n)	1	13	33	59	98	111	130
Sales USD (USDm)	**0**	**2**	**6**	**10**	**17**	**20**	**23**
TOTAL SALES (USDm)	**11**	**107**	**270**	**491**	**826**	**944**	**1,120**

Source: DNB Carnegie (estimates)

Potential impact of US pricing reforms

Given the high pricing expectations typically associated with advanced oncology therapies (including our assumed ASP for ELC-301) and the resulting sensitivity of long-term valuation to pricing assumptions, we consider it relevant to discuss how evolving drug-pricing reforms could affect Elicera's future commercial potential.

In recent years, US drug pricing has been subject to numerous policy changes, political initiatives and headline-driven debates, ranging from the implementation of the Inflation Reduction Act's Medicare negotiation framework to renewed pricing proposals under the Trump administration. One thing is clear: uncertainty around US drug-pricing reform remains elevated.

In 2025, US drug-pricing policy entered a more aggressive and fragmented phase, driven by parallel executive action, agency rulemaking and targeted congressional intervention (Source: Sidley, Stanford Department of Medicine). The Trump administration has issued a series of executive orders (EOs) aimed at lowering US drug prices, with a strong focus on reintroducing Most-Favoured-Nation (MFN) pricing and expanding federal leverage across Medicare, Medicaid and private markets (Source: GovInfo, Deloitte).

Executive Order 14297 (The MFN pricing EO) directs federal agencies to explore value-based pricing models within Medicare through pilot programmes, potentially broadening the government's ability to negotiate drug prices. While less prescriptive than the IRA's formal price negotiation process for selected high-cost drugs, the order signals an ambition to expand federal leverage in pricing discussions (Source: GovInfo). Our understanding is that such initiatives can be implemented within Medicare through existing statutory authority, for instance through demonstration programmes run by the Center for Medicare & Medicaid Innovation (CMMI), without requiring new congressional legislation. However, extending mandatory pricing mechanisms to the broader commercial market would require congressional approval, which remains politically challenging.

The Trump-aligned America First Policy entails that US drug prices should converge downward toward international levels, suggesting that pharmaceutical companies could offset lower US prices by increasing prices abroad. In practice, we believe this appears challenging. Markets such as the EU, Japan, and Canada operate under entrenched cost-containment frameworks and centralised price negotiations, limiting their ability to absorb materially higher prices (Source: DNB Carnegie Research).

On the other hand, elements of the proposed policy platform targeting pharmacy benefit managers (PBMs) could be more constructive for manufacturers. Efforts to increase transparency and reduce intermediary margins could strengthen net realised pricing for innovative therapies, particularly in high-cost oncology segments. For companies such as Elicera, this could partially offset pressure from federal price negotiation mechanisms.

We believe the implications for Elicera depend heavily on the future positioning. Advanced cell therapies such as ELC-301 (CAR T) or oncolytic virus programmes such as ELC-100 would probably be priced at the higher end of the oncology spectrum if approved, making them potential candidates for future Medicare negotiation frameworks. However, cell and gene therapies often benefit from strong clinical differentiation (high ORR, durable responses), which can support value-based pricing arguments. Just like in Elicera's case, many advanced therapies initially target smaller, high unmet-need populations, potentially delaying exposure to broader price control mechanisms. So we do not believe this to be a primary risk factor in the Elicera case; on the contrary, in our view, long-term valuation remains more sensitive to clinical execution, differentiation, and partnering strategy than to near-term US pricing reform risk. We therefore view pricing-policy uncertainty as a relevant but currently a secondary variable in the overall investment story.

ELC-100 for NETs

We currently await further communication from the company regarding the path forward for ELC-100. Assuming a partnering scenario, we estimate that a partner could initiate a confirmatory Phase II trial in 2026–27e, with a potential market entry in 2031e.

In modelling the potential addressable market for ELC-100, we use the incidence of NETs in the US, EU4 and UK as the key metric. According to GlobalData, NET incidence in 2024 was approximately 60,000 patients in these markets. Of these, we assume that 30% are diagnosed with metastatic disease at presentation, resulting in an estimated addressable population of around 19,000 patients in 2026e. As previously discussed, data on NET are limited, and we therefore exclude Japan from these estimates due to the absence of sufficiently granular information and datasets.

NET incidence (men and women, >18 y.o.)

	2024	2025	2026	2027	2028	2029	2030	2031	2032	2033
USA	34,592	35,081	35,533	35,945	36,336	36,735	37,153	37,598	38,048	38,504
EU4 + UK	27,288	27,542	27,784	28,005	28,233	28,436	28,646	28,879	29,114	29,350
Total incident cases	**61,880**	**62,623**	**63,317**	**63,950**	**64,569**	**65,171**	**65,799**	**66,477**	**67,162**	**67,854**

Source: GlobalData

Further, we assume a market penetration of 15% and gradual ramp up in sales until peak in 2037e. For pricing, we assume an ASP of USD50,000 in Europe and USD65,000 in US.

Sales build for ELC-100

	2031	2032	2033	2034	2035	2036	2037	2038	2039	2040
EU4 + UK										
Diagnosed incident cases of NET	28,879	29,114	29,350	29,589	29,829	30,072	30,316	30,562	30,811	31,061
Patients with metastatic cancer	8,664	8,734	8,805	8,877	8,949	9,021	9,095	9,169	9,243	9,318
Addressable population (n)	8,664	8,734	8,805	8,877	8,949	9,021	9,095	9,169	9,243	9,318
	START						PEAK			
Launch curve	0.01	0.10	0.25	0.45	0.75	0.85	1.00	0.75	0.60	0.40
Market penetration	0%	2%	4%	7%	11%	13%	15%	11%	9%	6%
ASP (USD)	50,000	50,750	51,511	52,284	53,068	53,864	54,672	55,492	56,325	57,169
Price increase	1.5%	1.5%	1.5%	1.5%	1.5%	1.5%	1.5%	1.5%	1.5%	1.5%
No. of patients treated (n)	13	131	330	599	1,007	1,150	1,364	1,031	832	559
Sales EU4 + UK (USDm)	**1**	**7**	**17**	**31**	**53**	**62**	**75**	**57**	**47**	**32**
USA										
Diagnosed incident cases of NET	37,598	38,048	38,504	38,965	39,432	39,904	40,382	40,865	41,355	41,850
Patients with metastatic cancer	11,279	11,414	11,551	11,689	11,829	11,971	12,114	12,260	12,406	12,555
Addressable population (n)	11,279	11,414	11,551	11,689	11,829	11,971	12,114	12,260	12,406	12,555
	START						PEAK			
Launch curve	0.01	0.10	0.25	0.45	0.75	0.85	1.00	0.75	0.60	0.40
Market penetration	0%	2%	4%	7%	11%	13%	15%	11%	9%	6%
ASP (USD)	65,000	65,975	66,965	67,969	68,989	70,023	71,074	72,140	73,222	74,320
Price increase	1.5%	1.5%	1.5%	1.5%	1.5%	1.5%	1.5%	1.5%	1.5%	1.5%
No. of patients treated (n)	17	171	433	789	1,331	1,526	1,817	1,379	1,117	753
Sales USA (USDm)	**1**	**11**	**29**	**54**	**92**	**107**	**129**	**99**	**82**	**56**
TOTAL SALES (USDm)	**2**	**18**	**46**	**85**	**145**	**169**	**204**	**157**	**129**	**88**

Source: DNB Carnegie (estimates)

Income statement

In the near term, as Elicera continues to advance ELC-301 and potentially initiates a clinical trial for ELC-401 in 2027e, we expect trial-related costs to increase. We estimate that the planned ELC-401 trial could entail additional costs of cSEK40m–50m.

While a licensing agreement could theoretically occur at any stage of development, including at the preclinical stage, we believe the probability of securing such an agreement increases meaningfully once clinical efficacy data has been demonstrated for ELC-301 and potentially also for ELC-401. Our sales estimates assume a partnership agreement for ELC-301 in 2028, including an upfront payment of USD20m. All revenue and cost estimates related to the clinical programs are risk-adjusted.

Elicera has no interest-bearing long-term debt on its balance sheet, and therefore interest expenses are negligible. Elicera has no interest-bearing long-term debt on its balance sheet, and consequently interest payments are negligible.

P&L overview – on a quarterly and yearly basis

	2025Q1	2025Q2	2025Q3	2025Q4	2025	2026e	2027e	2028e
Net sales	0	0	0	0	0	0	0	62
Other op. Income	0	3	8	0	11	4	0	0
Personnel costs	-2	-2	-2	-2	-7	-8	-8	-8
Other external expenses (including R&D)	-7	-4	-6	-6	-23	-21	-33	-23
Total opex	-8	-6	-8	-8	-30	-29	-41	-31
EBIT	**-8**	**-3**	**0**	**-8**	**-19**	**-25**	**-41**	**30**
EBIT margin (%)	-	-	-	-	-	-	-	49%
Net profit	**-8**	**-3**	**0**	**-8**	**-18**	**-25**	**-41**	**30**
Sales growth Y/Y (%)	N.m.	N.m.	N.m.	N.m.	N.m.	N.m.	N.m.	N.m.
Opex growth Y/Y (%)	-2%	-18%	165%	38%	23%	-1%	39%	-23%

Source: DNB Carnegie (estimates) & company (historical data)

Cash flow and balance sheet

Since Elicera is in a pre-commercial stage, it does not bind much working capital. The business is run in a typical for biotech asset-like manner.

Financing

As Elicera is currently pre-revenue, operations are funded through external sources, including non-dilutive grants and capital market financing. At year-end 2025, the company reported cash of cSEK25m.

Based on our estimates, the current cash position may fund operations through 2026. However, to fully execute the development plan and advance the pipeline beyond year-end 2026, we believe additional capital will be required.

Strategically, we would prefer to see funding secured through a partnership – potentially related to the iTANK platform or any of the three assets with a defined indication (ELC-301, ELC-100 and ELC-401). That said, in the case of ELC-301, we believe additional clinical data from the ongoing CARMA study may be required to optimise partnering terms. As such, our base case assumes that capital will be needed before a partnership agreement for ELC-301 can be secured.

In parallel, the company is actively pursuing non-dilutive financing in the form of soft funding and grant programmes, including applications intended to support the clinical development of ELC-401. Successful grant funding could help partially offset the cost of initiating and conducting the planned clinical trial, thereby reducing the near-term financing need.

A partnership related to iTANK or ELC-100 or ELC-401 would meaningfully improve the funding outlook and could reduce or eliminate the need for equity financing. Absent such an agreement, we see a high likelihood of a capital raise. In our forecasts, we model an additional funding requirement of approximately SEK50m in the near-term.

Valuation

We value Elicera Therapeutics with a fair value range of SEK7–12 per share. Our valuation is based on a risk-adjusted cash flow model (rNPV), where each project is assessed over its patent lifespan and then aggregated in a sum-of-the-parts (SOTP) analysis. We have applied a WACC range of 14–20%, with the lower end of our valuation range derived using a WACC of 20%, and the upper end using a WACC of 14%, reflecting varying levels of market risk appetite.

SOTP

In our SOTP analysis of Elicera, we include the company's three programmes ELC-301, ELC-401 and ELC-100, which we believe have a clear path forward either through continued development by the company (which would likely require additional financing) or through potential partnerships. We also include a theoretical value for the iTANK platform in our SOTP. Our forecasts are risk-adjusted based on our assessment of development risk, taking into account development stage, indication and mechanism of action. Balancing these assumptions, we derive a fair value range of SEK7–12 per share.

WACC assumptions

We expect that Elicera may require additional external financing before a potential partnership could be secured. While the timing, size, structure and market conditions of such a transaction are difficult to predict, the company's current cash runway does not fully cover the clinical milestones required to meaningfully de-risk its assets.

Given this uncertainty, and in line with our house valuation framework, we apply a higher WACC to reflect elevated financing risk. If sentiment is weak, dilution could be higher and terms less favourable and if raised after strong clinical data, conditions could improve. As this timing is difficult to predict, we believe it is prudent to reflect this uncertainty through a higher discount rate. Thus, we assume a WACC range of 14–20%.

ELC-301 assumptions

As outlined previously in this report, we believe ELC-301 has the potential to receive approval in DLBCL in 2031–32e at the earliest. We forecast peak sales of cUSD500m in this indication, which we believe may be reached by 2037. We also pencil in a partnership deal, which we believe may materialise once the Phase I/IIa complete data set is finished in 2028e, including an upfront payment of USD20m and a total deal value of USD300m and additional royalty income of 15% on net sales. We assume the partner will take over further development of ELC-301 from then onwards. Our estimates are risk-adjusted based on a LoA of 17%.

ELC-401 assumptions

As for the GBM indication, we believe ELC-401 has the potential to enter clinical development in 2027e, with a possible market approval in recurrent GBM in 2035e at the earliest. Given the high unmet medical need in this indication and the limited effective treatment options available at recurrence, we forecast peak sales of above USD1bn in the 7MM, which we estimate may be reached c2040–41e.

Similar to ELC-301, we believe the programme could attract partnering interest once clinical proof-of-concept has been demonstrated, although the timing remains uncertain at this early stage of development. We do not rule out that a partnership could materialise following early clinical efficacy signals from the planned Phase Ia/Ib study, which could support further clinical development and commercialisation.

Our estimates are risk-adjusted based on a LoA of 3%, reflecting the early stage of the programme and the historically low likelihood of approval in brain cancer indications. As a result, despite the attractive commercial potential, the current contribution of ELC-401 to our overall rNPV remains relatively limited at this stage.

ELC-100 assumptions

As for ELC-100, we believe the project has the potential to receive approval in NET in 2030–31e at the earliest. We forecast peak sales of cUSD200m in this indication, which we believe may be reached also by 2037. With similar logic, we pencil in a partnership deal. However, as the company has not yet communicated how the project will continue from here on forward, we choose to assume a partnership deal is relevant only once more data for ELC-100 is generated, potentially in an upcoming trial where more efficacy data will be shown. We pencil in a deal with a total value of USD150m, including an upfront payment of USD10m and royalties of 14%. Our estimates are risk-adjusted base on a LoA of 15%.

iTANK assumptions

We believe the iTANK platform possesses attributes that could make it attractive for external partnering and, to capture this potential value, we model a future licensing deal with a total headline value of USD200m. To reflect partnering risk, we apply a 50% probability of success and discount future milestone payments. The assumed deal value is based on benchmark transactions involving Noile-Immune presented earlier in this report.

SOTP valuation – lower and higher end (SEKm)

Project	Launch	Probability	Peak sales (USDm)	Valuation approach	rNPV (SEKm)	rNPV/share (SEK)
ELC-301	2032e	17%	516	DCF, WACC 20%	142	3
ELC-100	2031e	15%	170	DCF, WACC 20%	70	1
ELC-401	2035e	3%	1,120	DCF, WACC 20%	43	1
iTANK		50%			116	2
Unallocated costs					-46	-1
Enterprise value (EV), SEKm					**324**	**7**
Net cash Q4 2025					27	1
Total rNPV					**351**	**7**

Project	Launch	Probability	Peak sales (USDm)	Valuation approach	rNPV (SEKm)	rNPV/share (SEK)
ELC-301	2032e	17%	516	DCF, WACC 14%	245	5
ELC-100	2031e	15%	170	DCF, WACC 14%	109	2
ELC-401	2035e	3%	1,120	DCF, WACC 14%	63	1
iTANK		50%			199	4
Unallocated costs					-58	-1
Enterprise value (EV), SEKm					**558**	**12**
Net cash Q4 2025					27	1
Total rNPV					**585**	**12**

Source: DNB Carnegie (estimates) & company

Below we present a sensitivity analysis based on iTANK theoretical total deal value (USDm) versus WACC (%).

Sensitivity analysis – total deal value iTANK (USDm) versus WACC (%)

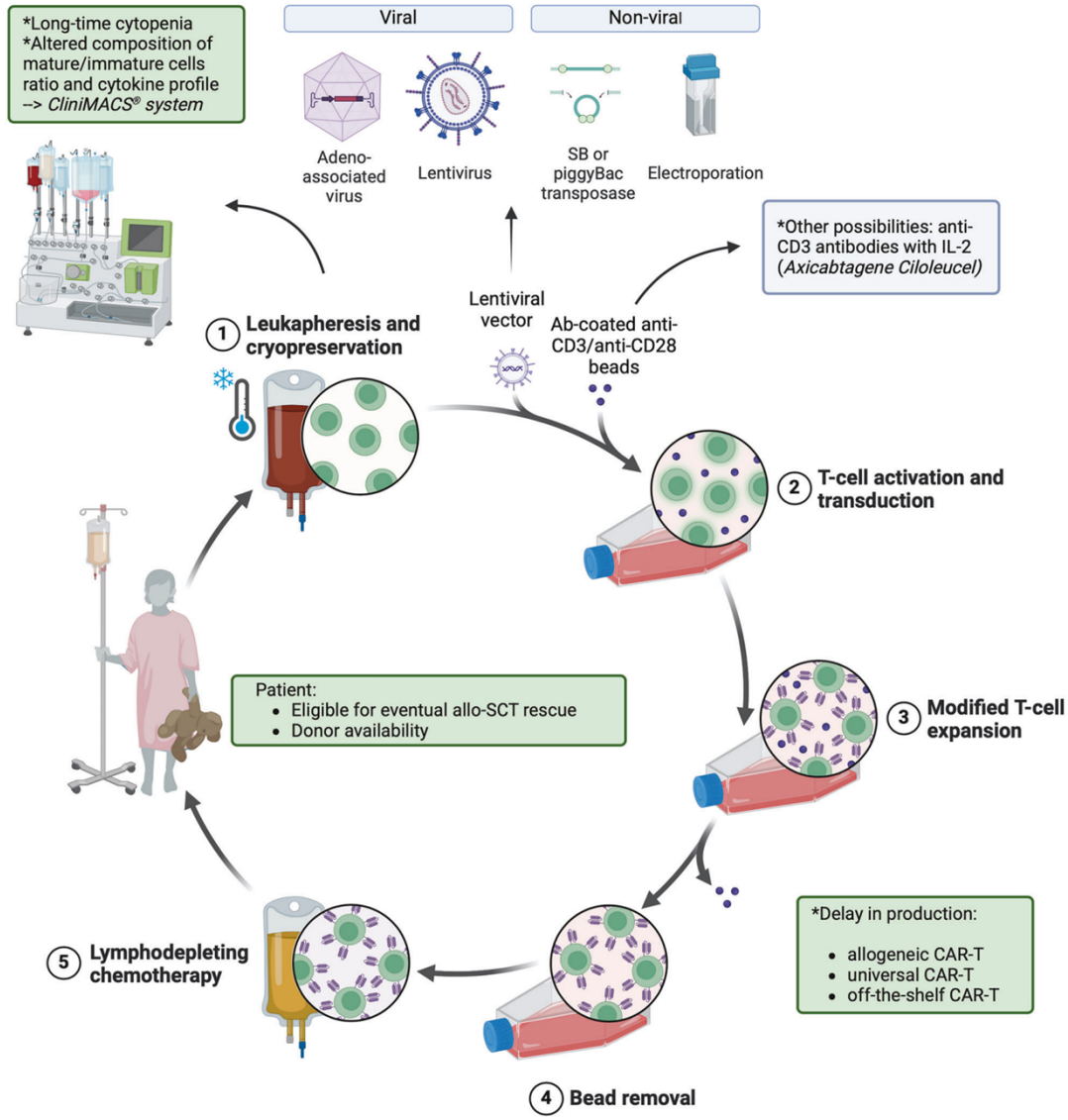
	Theoretical licensing deal value - iTANK (USDm)				
WACC (%)	0	100	200	300	400
12%	9	12	14	17	19
14%	8	10	**12**	14	16
16%	7	8	10	12	14
18%	6	7	9	10	11
20%	5	6	**7**	8	10
22%	4	5	6	7	8

Source: DNB Carnegie (estimates)

Chemistry manufacturing and control (CMC)

The CAR T-cell manufacturing process is largely standardised and typically consists of four main steps: collection and selection of T cells, activation and initial expansion, genetic modification to introduce the CAR construct, and final expansion followed by cryopreservation.

Automated CAR T cell manufacturing process



Source: Zugasti et al., 2025

In the first step, T cells are collected from the patient's blood through apheresis (a blood collection procedure). However, in patients with advanced or relapsed disease, low T-cell counts or prolonged cytopenias (abnormally low levels of blood cells) can complicate cell collection and reduce manufacturing success. The quality and composition of the starting material can also influence the final CAR T-cell product. To address this, some manufacturing processes use T-cell selection systems, such as magnetic bead-based technologies, to better control T-cell purity.

T-cell activation is typically achieved using antibody-coated beads (commonly anti-CD3/anti-CD28), after which the CAR gene is introduced into the cells. Currently, all approved CAR T therapies use viral vectors (lentiviral or retroviral) to insert the CAR gene, although alternative non-viral gene delivery methods are under investigation (Source: Nature: Signal transduction and targeted therapy).

One important step in manufacturing – viral vectors

On viral vectors – one of the most important and a critical safety parameter in CAR T manufacturing is vector copy number (VCN). Since viral vectors permanently integrate into the T-cell genome, too many inserted copies may increase certain risks. Such risks are for instance uncontrolled integration, potentially causing insertional mutagenesis by disrupting nearby host gene transcription. Even though secondary malignancies were reported for patients who received gene-engineered cell therapies, the risk of secondary malignancy is, however, regarded as low and not significantly different from patients who received standard-of-care (Source: ESMO, 2025). Because of these risks, regulators require robust and precise VCN monitoring before a product can be released to the markets.

Against this backdrop, it is notable that one of Elicera's founders has co-authored a recent peer-reviewed publication describing a validated ddPCR-based assay for precise quantification of vector copy number (VCN) in CAR T and TCR T products. The study not only demonstrates high accuracy and reproducibility but also introduces an adjusted VCN calculation to better reflect the true gene copy number in transduced cells. See the full article here.

Manufacturing time – a possible paradigm shift coming

Manufacturing time remains a critical limitation, particularly in aggressive cancers, as delays can negatively affect patient outcomes. This has driven interest in allogeneic, or "off-the-shelf," CAR T-cell therapies, which use donor-derived cells and can be made available immediately. Several such approaches are already in early clinical development.

Since the first FDA approval of a CAR T therapy for acute lymphoblastic leukemia (ALL) in 2017, cell therapy has become a leading modality in hematologic oncology. As mentioned above, seven commercial autologous CAR T products are now available and have demonstrated transformative efficacy in B-cell lymphomas and multiple myeloma. However, despite strong clinical outcomes, the field faces serious operational and access challenges, notably manufacturing complexity, high costs, safety risks, and limited patient accessibility.

A key structural bottleneck is the so-called "vein-to-vein" (V2V) time – the period between leukapheresis (a procedure where a patient's white blood cells are collected from the blood to be used for cell therapy manufacturing) and reinfusion of the final drug product. Today, V2V time typically ranges from 3–5 weeks, with a median of approximately 31 days. This delay is clinically important, especially for heavily pretreated patients with aggressive disease, as the cancer may progress so quickly that some patients are no longer eligible for treatment before they receive the infusion – exactly the patient indication that Elicera's ELC-301 is aiming to treat. It is estimated that c30% of patients prescribed CAR T never actually go through with leukapheresis, and c20% of those who do undergo leukapheresis never receive the final therapy, largely due to disease progression or clinical deterioration during the waiting period (Source: Cell and Gene Therapy Insights).

Vein-to-vein time for commercial CAR T-cell therapies

Product name	Commercial name	Indication	Vein-to-vein time
Tisagenlecleucel	Kymriah®	FL, DLBCL, ALL	3–4 weeks
Axicabtagene ciloleucel	Yescarta®	FL, DLBCL	3.5 weeks
Brexucabtagene autocel	Tecartus®	MCL, ALL	2–3 weeks
Lisocabtagene maraleucel	Breyanzi®	FL, LBCL, MCL, CLL, SLL	3–4 weeks
Obecabtagene autoleucel	Aucatzyl®	ALL	3 weeks
Idecabtagene vicleucel	Abecma®	MM	4 weeks
Ciltacabtagene autoleucel	Carvykti®	MM	4–5 weeks

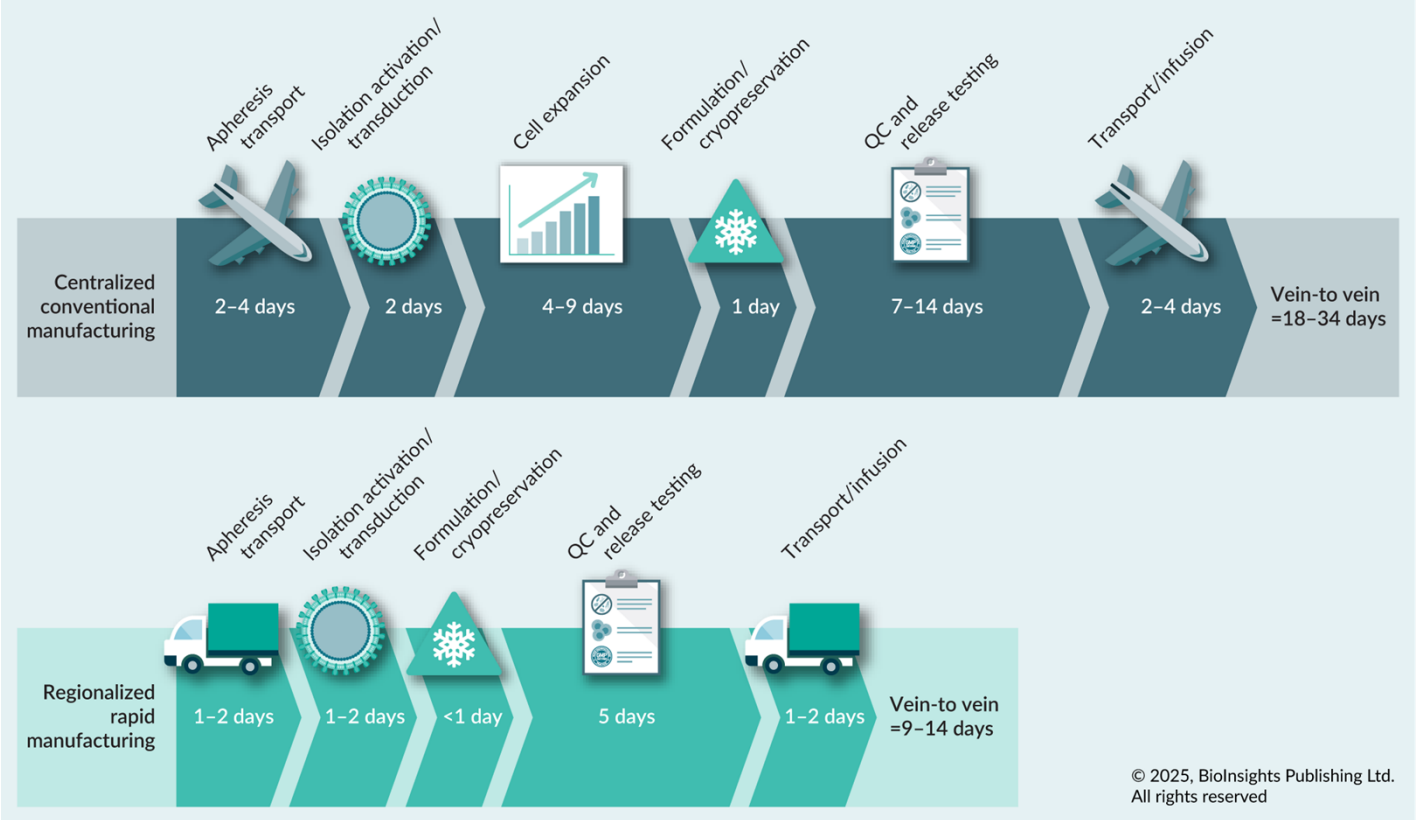
Source: Cell & Gene Therapy Insights (2025)

These challenges are receiving increasing attention across the industry. One way to address this is development of rapid manufacturing platforms capable of producing CAR T cells within 24–72 hours. This would represent a paradigm shift. Instead of relying on lengthy ex vivo expansion, newer manufacturing approaches shorten production time by collecting and preparing the cells before a prolonged growth phase, with the expectation that most of the cell multiplication happens inside the patient after infusion.

Early clinical data from platforms such as Novartis' T-Charge and Bristol Myers Squibb's NEX-T suggest that rapidly manufactured CAR T cells may retain a less differentiated, more "naïve" state – meaning the cells are biologically younger and less specialised. This more youthful cell profile is associated with a greater ability to multiply, survive longer in the body, and maintain anti-tumour activity over time (Source: Cell & Gene therapy Insights, 2025).

Another strategic consideration is manufacturing geography. Most commercial CAR T products rely on centralised manufacturing facilities to maintain quality control and scale efficiency. However, centralised models extend V2V time even more due to shipping and cryopreservation. A decentralised or near-patient manufacturing model could reduce transport delays and enable fresh drug product administration, potentially shortening V2V time by at least 7 days.

Overview of process development stages in conventional (top) and rapid (bottom) manufacturing settings



Source: DNB Carnegie (estimates) & company

Elicera's positioning

For Elicera, as an autologous CAR T developer, manufacturing speed and reliability are not peripheral operational topics. These topics are central to the equity story, we believe. In autologous cell therapy, the ability to move a patient from leukapheresis to infusion efficiently and predictably may be just as critical as the underlying biology. The structural bottlenecks described in CAR T manufacturing directly influence clinical execution, competitive positioning, cost structure, and ultimately valuation.

Here, Elicera is supported by its academic partners. Vecura, the GMP unit at the Karolinska Center for Cell Therapy and Elicera's academic partner, has more than 30 years of experience in advanced therapy medicinal product (ATMP) manufacturing. Having produced over 50 different gene and cell therapy products, Vecura is regarded as one of Europe's leading academic production facilities, providing significant manufacturing expertise and regulatory know-how to the collaboration (Source: Karolinska Institute).

Already in January 2021, the company and academic partners Karolinska Institute, Vecura and Uppsala University were awarded SEK5m from Vinnova to develop an automated, GMP-

compliant CAR T manufacturing process in preparation for the Phase I/II study of ELC-301. The stated goal was to simplify production, reduce failure rates and potentially decentralise manufacturing – thereby improving accessibility.

This work has since translated into tangible operational progress. Elicera has achieved a competitive V2V time of 17 days for ELC-301 in the CARMA study. In January 2024, the collaboration was further supported by SEK850k in funding to develop a similar automated production process for ELC-401, including tech transfer to Vecura's GMP facility. The focus is on reducing contamination risk, shortening lead times and improving product quality.

Risks and considerations

Here we outline the key risks relevant to Elicera Therapeutics and, more broadly, to companies operating in the biotechnology and IO sectors. The list is not exhaustive but highlights the areas we consider most relevant to Elicera Therapeutics. Main risks to our estimates are associated with the outcomes of clinical trials, competition and financing needs.

Clinical development risk

Elicera's valuation is highly dependent on the progression of its two lead programmes, ELC-100 and ELC-301, both of which remain early in clinical development. The main risk is that clinical outcomes may fail to demonstrate the efficacy or safety required to justify continued investment, we believe. Oncology trials are particularly exposed to delays, recruitment challenges, and high attrition rates. Any negative or inconclusive data from the ELC-100 or ELC-301 studies would materially weaken the company's prospects.

Financing risk

Elicera does not currently generate product revenue and is dependent on external financing to execute its clinical strategy. While the company has historically secured significant non-dilutive support from the Swedish Cancer Society, Vinnova, EU Horizon funding and other grants, these sources are episodic and insufficient to support late-stage trials. Should larger pivotal studies be required, particularly for ELC-301, Elicera would need substantial additional capital or a partnering agreement. Unfavourable market conditions, delays, or an inability to attract a strategic partner would heighten financing risk.

Partner dependence

Elicera's business model is centred on partnering: out-licensing the iTANK platform broadly to CAR T developers, and securing programme-level partnerships for its drug candidates. While the platform's mechanism is differentiated, the timing and likelihood of partnership deals are inherently uncertain. Partner priorities, competitive pressures, and shifting strategic focus within large pharmaceutical companies may influence deal appetite. A slower-than-expected out-licensing execution could affect both financial runway and long-term value creation.

Regulatory risk

Cell therapies and oncolytic viruses face rapidly evolving regulatory requirements in both the US and EU. While expedited pathways may shorten timelines, the FDA may be simultaneously raising the evidentiary bar for new CAR T therapies. In a recent JAMA article, senior CBER officials stated that future CAR T products will generally need randomised controlled trials demonstrating superiority over existing therapies to obtain traditional approval. Single-arm studies (previously sufficient for all approved CAR Ts) will likely only support accelerated approval in settings with no alternatives. These higher standards may require larger or longer studies, increasing costs and delaying approvals. This is in addition to existing expectations around long-term follow-up, safety monitoring, and manufacturing controls.

Competing in a crowded space

The field of immuno-oncology in general, and CAR T in particular, is highly competitive, with many academic and industry groups pursuing novel combinations and next-generation approaches. Several competing CAR T platforms are attempting to overcome solid-tumour barriers, and numerous oncolytic virus constructs are under investigation globally. If competitors demonstrate superior safety, efficacy or convenience, or secure faster regulatory trajectories, Elicera's commercial opportunity could narrow. Furthermore, pricing pressure in oncology could limit long-term revenue potential.

Intellectual property and technology platform risk

Elicera's commercial potential depends on strong IP protection for the iTANK platform and its product candidates. While the company holds multiple patents, IP disputes or challenges are common in the biotech industry. A weakened IP position could negatively impact the attractiveness of partnering opportunities, particularly for platform out-licensing. Any delays in patent issuance, regional limitations, or competitive filings could restrict freedom-to-operate.

Manufacturing and supply-chain risk

Elicera depends on external partners for vector production, cell therapy manufacturing and other critical components. Manufacturing of CAR T cells and oncolytic viruses is complex, capacity-constrained, and tightly regulated. Any supply-chain disruptions or production failures could delay clinical timelines, increase costs, or create compliance risks.

Legal, regulatory compliance and broader market risks

As with all pharmaceutical companies, Elicera is exposed to regulatory compliance risk, including potential changes in healthcare legislation, reimbursement frameworks, or political scrutiny of drug pricing. Failure to adhere to GDPR, GMP, GCP or other regulatory standards could result in sanctions, delayed trials, or reputational damage.

Extended timelines and operational delays

The ELC-100 (AdVince) Phase I/II study has progressed more slowly than typical oncology trials, reflecting the broader structural challenges of conducting complex early-phase studies in rare tumours. In general, delays in patient recruitment, academic-site dependencies, and reliance on stepwise public funding may delay the timelines. Additional operational setbacks – such as the database transition we have seen affecting ELC-100 result publication, and site activation processes – could extend the potential delays further. We view the risk of extended timelines due to operational delays as high.

Appendix 1: SWOT

Strengths: Elicera's main strength is its differentiated technology platform, iTANK. The platform is designed to enhance immune cell therapies, particularly in solid tumours, where traditional CAR T therapies have struggled. In simple terms, iTANK helps engineered immune cells recruit and activate additional immune cells once they reach the tumour. One of the biggest challenges in solid tumours is that cancer cells are biologically diverse and often suppress the immune system. A technology that can attract "backup" immune cells and create a broader immune response could meaningfully improve the durability and depth of treatment effects.

Importantly, early clinical signals suggest that the platform is biologically active in patients. In the lymphoma programme (ELC-301), some patients have shown no detectable active disease after treatment, even at relatively low doses, and with an acceptable safety profile so far. While based on small numbers, early human data is a crucial step in demonstrating that the platform works beyond preclinical models.

Elicera is also not dependent on a single project. With more than one clinical programme, the company reduces the risk of complete failure if one asset does not succeed. In addition, iTANK is positioned as a technology that can potentially be added to other companies' cell therapies. If validated, this creates the possibility of a licensing and partnering model, which may be more scalable and less capital-intensive than developing all products internally.

Weaknesses: The most significant weakness is the very early stage of development. Most of Elicera's value rests on early clinical or preclinical data involving small patient populations and no control groups. Early-stage results in oncology often appear promising but do not always hold up in larger, more rigorous trials. The probability of clinical failure remains high.

Elicera is also a small biotechnology company with limited financial and operational resources compared with large pharmaceutical competitors. This makes it more dependent on external funding, partnerships, and capital markets access.

As a development-stage company without commercial revenue, Elicera will likely require additional funding before reaching potential commercialisation. This creates financing risk and the possibility of shareholder dilution.

Furthermore, its cell therapies are autologous, meaning they are manufactured individually from each patient's own cells. This process is expensive, complex, and difficult to scale. Finally, while iTANK is presented as broadly applicable, its value has not yet been independently validated in large partner-led programmes, leaving some uncertainty around external adoption.

Opportunities: Elicera operates in areas of significant unmet medical need. Solid tumours and aggressive cancers such as glioblastoma have limited effective treatment options and poor survival outcomes. Even modest improvements in durability or survival can carry substantial medical and commercial value.

There is also growing industry interest in next-generation cell therapies. Many companies are actively seeking ways to improve existing CAR T and TCR therapies, particularly in solid tumours. A technology that enhances existing therapies rather than replacing them may be easier for partners to adopt.

If iTANK continues to generate promising clinical signals, Elicera could pursue multiple non-exclusive licensing agreements. This could provide non-dilutive revenue streams without requiring the company to fund and run multiple large-scale clinical programmes independently.

In addition, several of Elicera's target indications are rare or hard-to-treat cancers that may qualify for orphan drug incentives. Regulatory incentives could potentially reduce development timelines, lower costs, and improve commercial exclusivity.

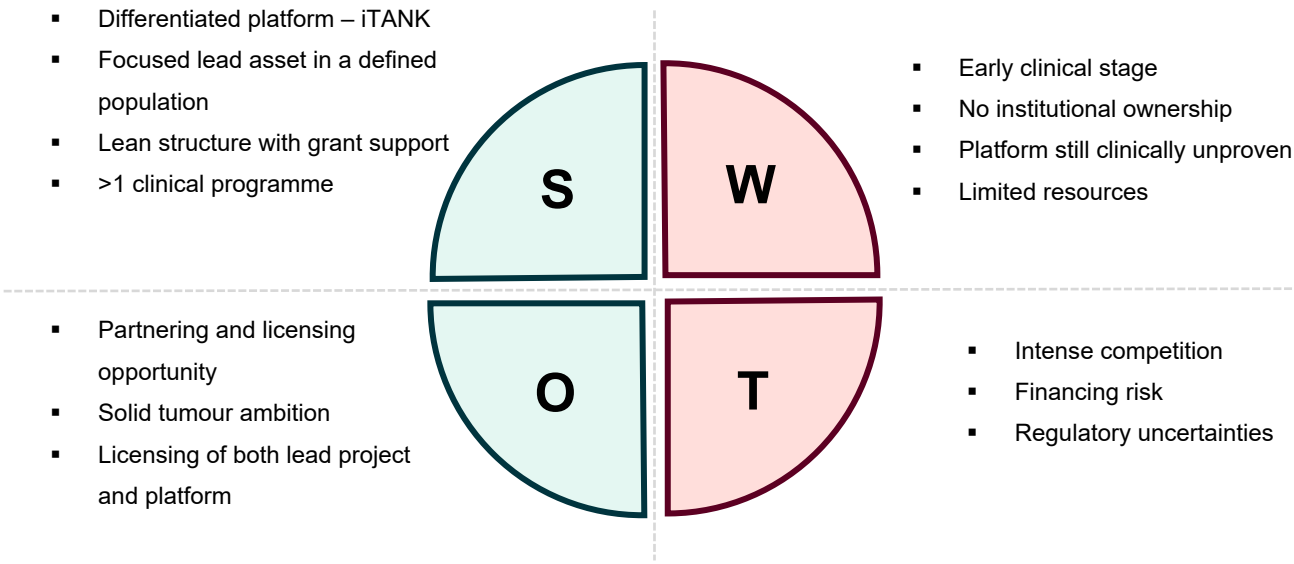
Threats: Cell and gene therapies historically have high failure rates, particularly in later-stage trials. Promising early data does not guarantee clinical success, and setbacks in efficacy, safety, or manufacturing can significantly delay or halt development.

Competition in the immuno-oncology space is intense. Many companies are developing alternative strategies to enhance or replace existing cell therapies, including bispecific antibodies and other next-generation immune approaches. If competitors demonstrate similar or superior outcomes more quickly, Elicera's bargaining power and relevance could weaken.

Regulatory and safety risks are also significant. Immune cell therapies can cause serious side effects, including cytokine release syndrome and neurotoxicity. Regulators remain cautious, especially in high-risk indications such as brain cancer.

Finally, financing remains an ongoing structural threat. Development of advanced therapies is capital-intensive, and if partnerships or capital markets access weaken, the company may face dilution or strategic constraints.

SWOT



Source: DNB Carnegie

Appendix 2: Sustainability

Elicera Therapeutics does not explicitly present a structured sustainability strategy in its reporting. However, several elements of its operations naturally align with responsible and ethical business practices. As a clinical-stage biotech company, Elicera's primary contribution to sustainability lies in its mission to develop next-generation immuno-oncology therapies for patients with high unmet medical need – an area closely connected to the UN Sustainable Development Goal 3 (Good health and well-being).

Governance practices follow Swedish corporate standards, and the company emphasises responsible behaviour in its interactions with employees, suppliers and research partners. As Elicera progresses toward later-stage development, we expect more detailed ESG reporting may increase, particularly regarding environmental impact, supply-chain responsibility and long-term patient access considerations.

Sustainability-related risks

Elicera shares the common sustainability risks of early-stage biotech firms. Social risks relate to future pricing and patient access, while environmental risks involve the handling of biological materials and laboratory waste within partner facilities. No sustainability-related incidents have been reported to date.

The UN Sustainable Development Goals



Source: United Nations

Appendix 3: Management and Board of directors

Management

Jamal El-Mosleh – CEO and Co-Founder

Mr. El-Mosleh joined Elicera in 2020 and has since served as the company's CEO. Mr. El-Mosleh is recognised as a co-founder through a patent-acquisition from Immunicum. Prior to leading Elicera, Mr. El-Mosleh was the CEO of First North-listed biotech company Annexin Pharmaceuticals between 2017–19. He also served as the CEO and co-founder of the immuno-oncology company Immunicum AB (Mendus) listed on Nasdaq Small-Cap. At Immunicum, he was responsible for the company's listing on Nasdaq First North and the initiation of a broad international clinical programme.

Mr. El-Mosleh holds an MSc degree in Industrial Engineering and Management from Chalmers University of Technology and an MSc degree in Innovation and Entrepreneurship from Chalmers School of Entrepreneurship.

Holdings in the company: 2,712,200 shares (incl. related parties)

Ingvar Karlsson – CFO

Mr. Karlsson has served as the CFO of Elicera since 2021. Alongside his work at Elicera, Mr. Karlsson works part-time as the CFO at Amferia AB and is a board member of Oxcia AB (publ.). Prior to joining Elicera, Mr. Karlsson has vast experience within financial management including as CFO of Lekolar Group, CFO of Doro AB, and Group Controller at Gambro Group.

Mr. Karlsson holds a Master's degree in Economics from Lund University.

Holdings in the company: 130,800 (incl. related parties)

Magnus Essand – Chief Scientific Officer and Co-Founder

Dr. Essand co-founded Elicera in 2014 and has since served as the company's Chief Scientific Officer. Alongside his work at Elicera, Dr. Essand is a professor of gene therapy at Uppsala University, a position he has held since 2009. Dr. Essand is an accomplished researcher who has authored/co-authored peer-reviewed scientific articles and received several large research grants. Prior to co-founding Elicera and becoming a professor, Dr. Essand's experience includes being an Associate Professor at Uppsala University (2000–09) and a Postdoctoral Fellow at the American National Cancer Institute (1996–00).

Dr. Essand earned his PhD in Physical Biology at Uppsala University.

Holdings in the company: 3,425,589 (incl. related parties)

Di Yu – Head of Translational Research and Co-Founder

Dr. Yu co-founded Elicera in 2014 as member of Dr. Magnus Essand's research team at Uppsala University. He worked as researcher at Elicera prior to becoming the Head of Translational Research in 2020. Alongside his work at Elicera, Dr. Yu is a Post-Doctoral Fellow and researcher in immuno-oncology at Uppsala University. Dr. Yu has co-invented Elicera's patents and is an awarded researcher.

Dr. Yu earned his PhD in Virology and Immunology at Uppsala University. He also holds an MSc degree in Applied Biotechnology from Uppsala University and a BSc in Biology from Shaanxi Normal University.

Holdings in the company: 3,473,705 (incl. related parties)

Anna Koptina Gültekin – Head of Regulatory Affairs

Dr. Koptina joined Elicera in 2023 and has since served as the company's Head of Regulatory Affairs. Dr. Koptina has extensive experience within regulatory affairs and is currently and independent consultant via her company Exellerium AB. Alongside her work at Elicera she is the Head of Regulatory Affairs at Mendus AB (formerly Immunicum). Dr. Koptina's previous experience includes work as a Regulatory and Medical Affairs Consultant at MedicoRent AB and as a Global Regulatory Affairs Manager at Galderma.

Dr. Koptina earned her PhD at the All-Russian Russian Research and Technological Institute of Biological Industry. She also holds an MSc in Biotechnology from Kazan State Technological University.

Holdings in the company: None

Board of directors

The board of directors of Elicera Therapeutics consists of the following five members:

Agneta Edberg – Chairman of the Board

Agneta Edberg has served as the Chairman of the Board of Elicera Therapeutics since 2020. Ms. Edberg has over 25 years of experience within the life sciences industry. Her previous positions include Managing Director and VP (Nordic Countries) at Mylan AB, CEO of LFF Service AB, Svenska Läkemedelsförsäkringen AB and NM Pharma AB. She is currently a member of several boards of directors in the healthcare industry.

Ms. Edberg holds a degree from the University of Sundsvall.

Holdings in the company: 202,623 (incl. related parties)

Christina Herder – Board Member

Dr. Herder has served as a member of the board of Elicera Therapeutics since 2020. Dr. Herder has over 30 years of experience of pharmaceutical and business development within the life science industry. Her previous experience includes acting CEO of Idogen AB, Executive Vice President of Medivir, CEO of Modus Therapeutics, and Director of Corporate Development at SOBI. She currently works as a board professional.

Dr. Herder earned her PhD in Physical Chemistry at KTH Royal Institute of Technology. She also holds an Executive MBA from Stockholm University.

Holdings in the company: 112,300 (incl. related parties)

Margareth Jorvid – Board Member

Mrs. Jorvid has served as a member of the board of directors of Elicera Therapeutics since 2020. Mrs. Jorvid has extensive experience of working with Regulatory Affairs within life science has held positions at the Medical Products Agency (MPA), Roussel Nordiska, Hoechst Marion Roussel, and Neopharma. Since 2006, she works as a consultant in regulatory affairs and quality assurance in the pharmaceutical industry.

Mrs. Jorvid holds an MSc in Pharmacy from Uppsala University, an MBA from the Stockholm School of Economics, and an MSc in Medical Technology Regulatory Affairs from Cranfield University.

Holdings in the company: 124,400 (incl. related parties)

Magnus Essand – Board Member

Dr. Essand has served as a member of the board of directors of Elicera Therapeutics since 2014. Alongside his work at Elicera, Dr. Essand is professor of gene therapy at Uppsala University, a position he has held since 2009. Dr. Essand is an accomplished researcher who has authored/co-authored peer-reviewed scientific articles and received several large research grants. Prior to co-founding Elicera and becoming a professor, Dr. Essand's experience includes being an Associate Professor at Uppsala University (2000–09) and a Postdoctoral Fellow at the American National Cancer Institute (1996–00).

Dr. Essand earned his PhD in Physical Biology at Uppsala University.

Holdings in the company: 3,425,589 (incl. related parties)

Sharon Longhurst

Dr. Longhurst has served as a member of the board of directors of Elicera Therapeutics since 2024. Dr. Longhurst has more than 20 years of experience in CMC development of biological

medicinal products, specialising in Advanced Therapy Medicinal Products (ATMP). Her previous experience includes being a VP of Development at Gadeta BV, Head of CMC at Immunicum AB (now Mendus), and Akari Therapeutics. Dr. Longhurst is currently a Director at Advanced Biologics Consulting Ltd.

Dr. Longhurst earned her PhD in Virology at the University of Warwick and holds a BSc in Biochemistry from the University of Surrey.

Holdings in the company: None.

Ownership

Ownership overview

Company shareholder	No. of shares	Capital (%)	Votes (%)
Avanza	3,818,129	8%	8%
*Di Yu	3,473,705	7%	7%
*Magnus Essand	3,425,589	7%	7%
*Jamal El-Mosleh	2,712,200	6%	6%
Sc Holding AB	1,379,380	3%	3%
Tuvedalen Ltd	1,278,075	3%	3%
IBKR Financials, CH	1,110,476	2%	2%
Stefan Henriksson	1,084,512	2%	2%
Göran Persson	1,067,911	2%	2%
Ing-Britt Turban	1,024,170	2%	2%

Source: Company website (https://www.elicera.com/investors-2/the-share)
Note: *denotes company insiders

Appendix 4: IP portfolio

Below is an overview of Elicera Therapeutics' patent portfolio for the key drug candidates. Patent terms are typically 20 years from the filing date, but in some cases may be extended through mechanisms such as Patent Term Adjustment (PTA) in the US and Supplementary Protection Certificate (SPC) in the EU, which compensates for delays in the patent office's examination process. The scope of protection varies by patent family and includes composition-of-matter claims, as well as use in specific combinations. Pending applications are included in the table below as well, where grants have not yet been issued.

IP portfolio

Drug Candidate	Title	Year of application	Patent approved	Expiration date
ELC-100/AdVince	Hexon TAT-PTD Modified Adenovirus and uses thereof	2013	USA	2033
ELC-201	Adenovirus for treatment of cancer	2021	–	2041
ELC-301 and ELC-001 (the iTANK-platform)	T-Cell Immunotherapy	2016	*–Turkey, United Kingdom, Switzerland, Europe (except Cyprus, Greece, Ireland, Croatia, Romania, Slovakia, Czech Republic and Hungary) and China.*	2036
ELC-401	CAR T IL-13Ra2	2020	–	2040

Source: Company

Financial statements

Profit & loss (SEKm)	2019	2020	2021	2022	2023	2024	2025	2026e	2027e	2028e
Sales	0	0	0	0	0	0	0	0	0	62
COGS	0	0	0	0	0	0	0	0	0	0
Gross profit	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**62**
Other income & costs	0	-3	-13	-19	-17	-17	-19	-25	-41	-31
Share in ass. operations and JV	0	0	0	0	0	0	0	0	0	0
EBITDA	**0**	**-3**	**-13**	**-19**	**-17**	**-17**	**-19**	**-25**	**-41**	**30**
Depreciation PPE	0	0	0	0	0	0	0	0	0	0
Depreciation lease assets	0	0	0	0	0	0	0	0	0	0
Amortisation development costs	0	0	0	0	0	0	0	0	0	0
Amortisation other intangibles	0	0	0	0	0	0	0	0	0	0
Impairments / writedowns	0	0	0	0	0	0	0	0	0	0
EBITA	**0**	**-3**	**-13**	**-19**	**-17**	**-17**	**-19**	**-25**	**-41**	**30**
Amortization acquisition related	0	0	0	0	0	0	0	0	0	0
Impairment acquisition related	0	0	0	0	0	0	0	0	0	0
EBIT	**0**	**-3**	**-13**	**-19**	**-17**	**-17**	**-19**	**-25**	**-41**	**30**
Share in ass. operations and JV	0	0	0	0	0	0	0	0	0	0
Net financial items	0	0	0	0	1	1	0	0	0	0
of which interest income/expenses	0	0	0	0	1	1	0	0	0	0
of which interest on lease liabilities	0	0	0	0	0	0	0	0	0	0
of which other items	0	0	0	0	0	0	0	0	0	0
Pre-tax profit	**0**	**-3**	**-13**	**-19**	**-16**	**-16**	**-18**	**-25**	**-41**	**30**
Taxes	0	0	0	0	0	0	0	0	0	0
Post-tax minorities interest	0	0	0	0	0	0	0	0	0	0
Discontinued operations	0	0	0	0	0	0	0	0	0	0
Net profit	**0**	**-3**	**-13**	**-19**	**-16**	**-16**	**-18**	**-25**	**-41**	**30**
Adjusted EBITDA	0	-3	-13	-19	-17	-17	-19	-25	-41	30
Adjusted EBITA	0	-3	-13	-19	-17	-17	-19	-25	-41	30
Adjusted EBIT	0	-3	-13	-19	-17	-17	-19	-25	-41	30
Adjusted net profit	0	-3	-13	-19	-16	-16	-18	-25	-41	30
Sales growth Y/Y	na	na	na	na	na	na	na	na	na	+chg
EBITDA growth Y/Y	na	-chg	-chg	-chg	+chg	+chg	-chg	-chg	-chg	+chg
EBITA growth Y/Y	na	-chg	-chg	-chg	+chg	+chg	-chg	-chg	-chg	+chg
EBIT growth Y/Y	na	-chg	-chg	-chg	+chg	+chg	-chg	-chg	-chg	+chg
EBITDA margin	nm	nm	nm	nm	nm	nm	nm	nm	nm	49.1%
EBITA margin	nm	nm	nm	nm	nm	nm	nm	nm	nm	49.1%
EBIT margin	nm	nm	nm	nm	nm	nm	nm	nm	nm	49.1%
Tax rate	na	na	na	na	na	na	na	na	na	na

Cash flow (SEKm)	2019	2020	2021	2022	2023	2024	2025	2026e	2027e	2028e
EBITDA	0	-3	-13	-19	-17	-17	-19	-25	-41	30
Paid taxes	0	0	0	0	0	0	0	0	0	0
Change in NWC	0	2	-1	11	1	-8	-1	0	0	0
Interests paid	0	0	0	0	0	0	0	0	0	0
Actual lease payments	0	0	0	0	0	0	0	0	0	0
Non cash adjustments	0	0	0	0	0	0	0	0	0	0
Discontinued operations	0	0	0	0	0	0	0	0	0	0
Total operating activities	**0**	**-1**	**-14**	**-9**	**-15**	**-23**	**-19**	**-25**	**-41**	**30**
Capex tangible assets	0	0	0	0	0	0	0	0	0	0
Capitalised development costs	0	0	0	0	0	0	0	0	0	0
Capex - other intangible assets	0	0	0	0	0	0	0	0	0	0
Acquisitions/divestments	0	0	0	0	0	0	0	0	0	0
Other non-cash adjustments	0	0	0	0	0	0	0	0	0	0
Total investing activities	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
Dividend paid and received	0	0	0	0	0	0	0	0	0	0
Share issues & buybacks	0	12	55	0	0	20	20	50	0	0
Change in bank debt	0	0	0	0	0	0	0	0	0	0
Other cash flow items	0	0	0	0	0	0	0	0	0	0
Total financing activities	**0**	**12**	**55**	**0**	**0**	**20**	**20**	**50**	**0**	**0**
Operating cash flow	0	-1	-14	-9	-15	-23	-19	-25	-41	30
Free cash flow	0	-1	-14	-9	-15	-23	-19	-25	-41	30
Net cash flow	0	12	41	-9	-14	-3	1	25	-41	30
Change in net IB debt	0	11	41	-9	-15	-3	1	25	-41	30
Capex / Sales	nm	nm	nm	nm	nm	nm	nm	nm	nm	0.0%
NWC / Sales	nm	nm	nm	nm	nm	nm	nm	nm	nm	-7.5%

Source: DNB Carnegie (estimates) & company data

Financial statements, cont.

Balance sheet (SEKm)	2019	2020	2021	2022	2023	2024	2025	2026e	2027e	2028e
Acquired intangible assets	0	0	0	0	0	0	0	0	0	0
Other fixed intangible assets	0	0	0	0	0	0	0	0	0	0
Capitalised development	0	0	0	0	0	0	0	0	0	0
Tangible assets	0	0	0	0	0	0	0	0	0	0
Lease assets	0	0	0	0	0	0	0	0	0	0
Other IB assets (1)	0	0	0	0	0	0	0	0	0	0
Other non-IB assets	0	0	0	0	0	0	0	0	0	0
Fixed assets	**0**	**1**	**1**	**1**	**0**	**0**	**0**	**0**	**0**	**0**
Inventories (2)	0	0	0	0	0	0	0	0	0	0
Receivables (2)	0	0	0	0	0	1	0	0	0	0
Prepaid exp. & other NWC items (2)	0	0	2	2	0	0	2	2	2	2
IB current assets (1)	0	0	0	0	0	0	0	0	0	0
Other current assets	0	0	0	0	0	0	0	0	0	0
Cash & cash equivalents (1)	0	12	52	44	29	26	27	52	11	32
Current assets	**0**	**12**	**54**	**46**	**30**	**28**	**29**	**54**	**13**	**34**
Total assets	**0**	**13**	**55**	**46**	**30**	**28**	**29**	**54**	**13**	**34**
Shareholders' equity	0	10	52	33	16	21	22	47	6	27
Minorities	0	0	0	0	0	0	0	0	0	0
Other equity	0	0	0	0	0	0	0	0	0	0
Total equity	**0**	**10**	**52**	**33**	**16**	**21**	**22**	**47**	**6**	**27**
Deferred tax	0	0	0	0	0	0	0	0	0	0
LT IB debt (1)	0	0	0	0	0	0	0	0	0	0
Other IB provisions (1)	0	0	0	0	0	0	0	0	0	0
Lease libilities	0	0	0	0	0	0	0	0	0	0
Other non-IB liabilities	0	0	0	0	0	0	0	0	0	0
LT liabilities	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**	**0**
ST IB debt (1)	0	0	0	0	0	0	0	0	0	0
Payables (2)	0	2	2	1	1	2	2	2	2	2
Accrued exp. & other NWC items (2)	0	0	0	13	13	5	5	5	5	5
Other ST non-IB liabilities	0	0	0	0	0	0	0	0	0	0
Liabilities - assets held for sale	0	0	0	0	0	0	0	0	0	0
Current liabilities	**0**	**2**	**3**	**14**	**14**	**7**	**7**	**7**	**7**	**7**
Total equity and liabilities	**0**	**13**	**55**	**46**	**30**	**28**	**29**	**54**	**13**	**34**
Net IB debt (=1)	0	-12	-52	-44	-29	-26	-27	-52	-11	-32
Net working capital (NWC) (=2)	0	-2	-1	-12	-13	-6	-5	-5	-5	-5
Capital employed (CE)	0	10	52	33	16	21	22	47	6	27
Capital invested (CI)	0	-2	-1	-12	-13	-6	-5	-5	-5	-5
Equity / Total assets	nm	81%	95%	71%	54%	75%	76%	87%	48%	80%
Net IB debt / EBITDA	nm	4.1	4.0	2.3	1.7	1.6	1.4	2.1	0.3	-1.1

Per share data (SEK)	2019	2020	2021	2022	2023	2024	2025	2026e	2027e	2028e
Adj. no. of shares in issue YE (m)	0.00	0.00	19.78	19.78	19.78	35.09	48.54	48.54	48.54	48.54
Diluted no. of Shares YE (m)	0.00	0.00	19.78	19.78	19.78	35.09	48.54	48.54	48.54	48.54
EPS	na	na	-1.33	-0.98	-0.83	-0.59	-0.44	-0.52	-0.84	0.62
EPS adj.	na	na	-1.33	-0.98	-0.83	-0.59	-0.44	-0.52	-0.84	0.62
CEPS	na	na	-1.32	-0.98	-0.83	-0.59	-0.44	-0.52	-0.84	0.62
DPS	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
BVPS	na	na	2.64	1.66	0.83	0.59	0.46	0.97	0.13	0.56

Performance measures	2019	2020	2021	2022	2023	2024	2025	2026e	2027e	2028e
ROE	nm	-55.4%	-42.0%	-45.7%	-66.6%	-86.6%	-85.6%	-72.4%	-152.0%	179.3%
Adj. ROCE pre-tax	na	na	-42.0%	-45.4%	-66.3%	-86.3%	-85.6%	-72.4%	-152.0%	179.3%
Adj. ROIC after-tax	na	na	1071.1%	318.9%	139.6%	181.3%	368.0%	547.1%	886.5%	-658.9%

Valuation	2019	2020	2021	2022	2023	2024	2025	2026e	2027e	2028e
FCF yield	0.0%	-0.4%	-5.2%	-3.1%	-5.4%	-8.6%	-7.1%	-9.2%	-14.9%	11.1%
Dividend yield YE	na	na	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Dividend payout ratio	na	na	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
Dividend + buy backs yield YE	na	na	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%
EV/Sales YE	na	na	nm	nm	nm	nm	nm	nm	nm	3.92
EV/EBITDA YE	na	na	neg.	neg.	neg.	neg.	neg.	neg.	neg.	8.0
EV/EBITA YE	na	na	neg.	neg.	neg.	neg.	neg.	neg.	neg.	8.0
EV/EBITA adj. YE	na	na	neg.	neg.	neg.	neg.	neg.	neg.	neg.	8.0
EV/EBIT YE	na	na	neg.	neg.	neg.	neg.	neg.	neg.	neg.	8.0
P/E YE	na	na	nm	nm	nm	nm	nm	nm	nm	9.0
P/E adj. YE	na	na	nm	nm	nm	nm	nm	nm	nm	9.0
P/BV YE	na	na	1.90	2.12	5.50	2.82	11.35	5.80	42.59	10.00
Share price YE (SEK)			5.02	3.52	4.56	1.67	5.22	5.64		

Source: DNB Carnegie (estimates) & company data

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