Trinity Delta

Scancell

Outlook

Abundant development opportunities in immuno-oncology

17 November 2025

Scancell's investment case centres on the potential of the lead oncology programmes from its highly promising ImmunoBody and Moditope "off the shelf" platforms. Following the successful outcome from the SCOPE trial, iSCIB1+ has been selected as the optimal ImmunoBody candidate to progress into a potentially registrational trial. Forthcoming discussions with the FDA should define the registrational trial design. Other 2025 catalysts include further SCOPE data, plus early data from the RCC (renal cell carcinoma) cohort of the ModiFY study, which should provide useful insights into Modi-1's potential benefit when coupled with double checkpoint inhibitor (CPI) therapy. The formation of GlyMab Therapeutics will separate Scancell's immunotherapy and antibody platforms, reflecting their differing scientific needs and strategic direction. We expect the two businesses will attract different partners, investors and funding. Our rNPV valuation for Scancell is £382m, or 37p/share.

Year-end: April 30	2024	2025	2026E	2027E
Revenues (£m)	0.0	4.7	0.0	2.4
EBITDA (£m)	(17.3)	(14.1)	(18.4)	(8.0)
PBT (£m)	(9.1)	(15.3)	(20.7)	(9.7)
Net Income (£m)	(5.9)	(12.3)	(19.2)	(8.2)
EPS (p)	(0.68)	(1.26)	(1.85)	(0.79)
Cash (£m)	14.8	16.9	1.9	(3.5)

Source: Trinity Delta Note: Adjusted numbers exclude exceptionals

- **Next steps for iSCIB1+ on the horizon** Scancell is pioneering the next generation of immunotherapies, addressing hard to treat cancers. Its two distinct platforms, ImmunoBody and Moditope, are designed to overcome immune evasion, target tumour-specific vulnerabilities, and generate durable anti-tumour responses. ImmunoBody is the most advanced, with data from the Phase II SCOPE study of SCIB1/iSCIB1+ in combination with CPIs in advanced melanoma showing meaningfully improved outcomes across all key metrics. Upcoming FDA discussions will define the design and format of the potentially registrational study.
- **Modi-1 CPI combo data due in Q425** Results from the Phase I/II ModiFY trial RCC cohort, exploring Modi-1 with double CPIs, are expected in Q425. Similarly to SCIB1/iSCIB1+, the Modi-1/doublet CPI combination could be highly synergistic. Doublet CPI is SoC for advanced RCC and ModiFY data should establish whether the addition of Modi-1 could bring potential improvements in the first-line setting.
- **GlyMab Therapeutics provides strategic optionality** Establishment of GlyMab Therapeutics as a wholly owned Scancell subsidiary will separate the antibody and immunotherapy platforms into independent corporate entities. This reflects the reality that each will need distinct scientific expertise and strategic direction, and are likely to have different funding needs and attract different investors/partners.
- **Valuation of £382m ($477m), or 37p/share; cash through to Q326** iSCIB1+ is the most important contributor (worth >50%) and together with the platform, our model suggests ImmunoBody is worth £215m/$269m. This could increase to £346m/$432m when the iSCIB1+ registrational trial starts next year, with derisking events including further data, regulatory feedback, and securing trial funding.

Price	10.09p
Market Cap	£104.7m
Enterprise Value	£88.3m
Shares in issue	1,036.8m
12 month range	7.26-18.00p
Free float	56.5%
Primary exchange	AIM London
Other exchanges	N/A
Sector	Healthcare
Company Code	SCLP.L
Corporate client	Yes

Company description

Scancell is a clinical-stage immuno-oncology specialist. The key value drivers are iSCIB1+, the lead ImmunoBody programme, and Modi-1, the lead Moditope programme. The novel GlyMab glycan antibodies are earlier in development.

Analysts

Lala Gregorek
lgregorek@trinitydelta.org
+44 (0) 20 3637 5043

Philippa Gardner
pgardner@trinitydelta.org
+44 (0) 20 3637 5043

Investment case

A clinical stage immunotherapy specialist, with three distinct and broad ranging platforms

Scancell is a clinical-stage immuno-oncology specialist, founded in 1996 as a spin-out of research led by Professor Lindy Durrant at the University of Nottingham. It has two distinct immunotherapy platforms addressing oncology indications: **ImmunoBody** employs CD8 T-cell pathways, while **Moditope**'s activity is mediated via CD4 pathways. Both therapeutic platforms should have broad applicability in many forms of solid tumours. A third platform, **GlyMab**, generates novel high affinity anti-glycan antibodies, with two licensing deals with Genmab effectively validating the approach. Scancell initially listed on PLUS in 2008, moving to AIM in 2010. Sizeable investment by Redmile in 2020 transformed Scancell's ability to fund its activities. Current leading shareholders are Redmile (28.6%) and Vulpes (13.8%). Scancell is based in Oxford and Nottingham and has >50 employees.

Valuation

rNPV based valuation of £382m ($477m), equivalent to 37p per share (31p fully diluted)

We value Scancell using a sum-of-the parts, where the rNPVs of the three distinct technology platforms are summed together with cash. Within each, we have a standalone valuation for the clinical assets (ie iSCIB1+ and Modi-1) plus indicative placeholder platform valuations. Our Scancell valuation is £382m ($477m), or 37p per share (31p fully diluted), with iSCIB1+ the most important contributor, worth >50%. We value the ImmunoBody platform (which includes iSCIB1+) at £215m/$269m, with upside to our iSCIB1+ peak sales forecasts, in addition to upcoming de-risking events which could lift the ImmunoBody valuation alone to £346m/$432m; these include further iSCIB1+ survival data, FDA feedback on the registrational trial design, and clarity on the funding for this trial.

Financials

Cash runway is sufficient to Q326 beyond key value inflection points

Cash at end April 2025 was £16.9m (FY24: £14.8m), which (according to Scancell) provides a runway through to calendar Q326, beyond key near-term value inflections points for both iSCIB1+ and Modi-1. This cash runway could be extended by successful execution of any business development transaction(s), where management is proactively exploring various options across the pipeline. Current cash will not be sufficient to fund the planned iSCIB1+ registrational trial. Various funding options are available, however until there is clarity on this, our future forecasts (beyond ongoing spend on SCOPE and ModiFY) simply include an illustrative base level of R&D spend.

Sensitivities

Sizeable funds will be needed to progress iSCIB1+

Scancell faces the usual industry risks associated with drug development, including binary clinical trial results, navigating regulatory hurdles, ensuring sufficient financing is in place, progressing partnering discussions, and successful commercialisation. The main near-term specific sensitivity for Scancell, in our view, is on securing sufficient funds to progress iSCIB1+ into the planned registrational trial. Scancell does not currently have the resources to run this trial and there is no guarantee that funding from either investors or partners will become available.

Scancell: three distinct technology platforms

Scancell is a clinical-stage oncology specialist developing immunotherapies. Its differentiated portfolio is anchored around two novel platforms, ImmunoBody and Moditope, complemented by the GlyMab antibody platform that targets tumour-associated glycans. Collectively, these three platforms offer broad potential across a range of solid tumours. Investor focus is on the ImmunoBody platform pending FDA feedback on the design of the potentially registrational study for iSCIB1+. This follows promising SCOPE Phase II data, which are still maturing, with further updates expected over the coming 12 months. For Moditope, interim data from the renal cell carcinoma (RCC) cohort of the ModiFY study will provide important insights into the potential synergy of lead asset Modi-1 when combined with checkpoint inhibitor therapy. Meanwhile, GlyMab continues to produce compelling preclinical results, underscored by the recent establishment of GlyMab Therapeutics as a separate legal entity, which could offer strategic optionality in due course. Our rNPV valuation is £382m, or 37p/share, with further upside potential from the expected news flow.

Three platforms with broad applicability in oncology

Despite notable advances, many patients with advanced solid tumours still face poor outcomes. Tumour immune evasion, heterogeneity, and resistance to therapies leave substantial unmet clinical need. Scancell's three platforms each offer distinct, and diverse, approaches to create a differentiated immuno-oncology portfolio. ImmunoBody primes durable T-cell responses, optimised in iSCIB1+ for clinical and commercial scalability. Moditope targets stress-induced neoantigens across solid tumours and may similarly synergise with checkpoint inhibitors (CPIs). GlyMab, to be spun out as a separate entity, is generating encouraging preclinical data. In our view the combination of broad applicability, novel mechanisms, and emerging validation de-risks Scancell relative to single platform peers.

Lead immunotherapy programmes are iSCIB1+ and Modi-1

Scancell continues to advance a diverse pipeline of immuno-oncology assets. The lead ImmunoBody programme has successfully completed the SCOPE Phase II trial, with initial results demonstrating potent cytotoxicity and promising efficacy signals in metastatic melanoma. Maturing data expected through the next 12 months should reassure, as the optimised construct iSCIB1+, the main driver for our valuation, progresses into a potentially registrational trial. While iSCIB1+ is in the limelight, Modi-1 will soon also have data from the Phase I/II ModiFY study, which should determine whether use in combination with CPIs in RCC and head & neck cohorts shows similar incremental benefit for Moditope (as the SCOPE study has for ImmunoBody) and help determine potential future Modi-1 development plans. We expect these data to confirm that doublet CPI therapy is highly synergistic when coupled with Scancell's immuno-oncology approaches.

Further data are expected for both iSCIB1+ and Modi-1, with cash beyond upcoming catalysts

Further data from both the SCOPE and ModiFY studies, as well as regulatory clarity on the design and format of the potentially registrational study for iSCIB1+ following the imminent FDA meeting will be key near-term catalysts. Scancell's last reported cash at end April 2025 of £16.9m provides a runway through to calendar Q326, beyond these value inflection points; however, it is not sufficient to fund the planned iSCIB1+ registrational trial through to completion. Nevertheless, these data points should add momentum to Scancell's business development efforts as it seeks potential suitable out-licencing and partnering opportunities to optimally advance its assets.

Immunotherapy's future lies in combinations

Greater understanding of tumour mechanisms brings "vaccination" back into vogue

Scancell's technology platforms, ImmunoBody and Moditope, can be considered vaccines as they are administered in an analogous manner, but their activity is subtly different. Traditional vaccination is prophylactic: it harnesses the immune system to identify a threat, typically an infectious disease, and eliminates it. Such vaccination has been one of the most profound interventions in improving health outcomes and highlights how effective harnessing of the immune system can be. Vaccines to prevent certain cancers are now well established, but these tend to target viruses that initiate specific cancers, such as human papillomavirus (HPV) for cervical cancer and hepatitis B for some liver cancers.

Multiple tumour escape routes mean treatment combinations are more effective

While these represent great medical advances, there are many more challenges in creating an effective therapeutic oncology "vaccine". Historically it was thought that once the target cancer cell was identified accurately then the immune system could be "trained" to eliminate those cells. The many failures were attributed to a variety of factors but, as the understanding of how cancer immunoediting and the tumour microenvironment (TME) work has grown exponentially, we now appreciate that a successful tumour capitalises on multiple mechanisms to evade an immune response. This, in turn, means it is likely that a combination of treatments will be required to achieve successful, and durable, responses.

Exhibit 1: Cancer immunoediting underpins most solid tumours

The three key stages of cancer immunoediting are Elimination, Equilibrium, and Escape

- **Elimination:** also known as cancer immunosurveillance. The immune system, both innate and adaptive, works to detect and destroy transformed cells before they become clinically apparent. Markers, such as tumour-specific antigens (TSAs) and tumour-associated antigens (TAAs), on the surface of abnormal cells are detected. Then elements of the innate immune system (eg natural killer cells and macrophages) and adaptive immune system (eg cytotoxic T cells) are activated to kill the cancerous cells. If this phase is successful, the tumour is eradicated and never progresses to the later stages.
- **Equilibrium:** if the immune system does not completely eliminate all cancer cells during the elimination phase, the surviving variants may enter a prolonged period of dormancy. Immune control is where the adaptive immune system keeps the growth of the remaining tumour cells in check, preventing them from expanding rapidly. But, typically, a Darwinian-like selection process (tumour editing) sees the genetic instability of cancer cells and selective pressure from the immune system drive the emergence of new, less-immunogenic variants.
- **Escape:** immunologically "edited" tumour cells eventually breach the immune system's control and begin to grow progressively, becoming a clinically apparent and aggressive malignancy. The variants that survive have acquired characteristics that allow them to evade immune detection and destruction. Mechanisms of escape include reduced immunogenicity, creation of an immunosuppressive TME, and increased resistance to the immune system's attacks.

Source: Trinity Delta

Tumour progression can be viewed as a Darwinian process

Over the past decade, the role of the immune system in tumour initiation and progression has become better understood, as has the importance of the TME.

Cancer immunoediting is a dynamic process where the immune system interacts with developing tumours through the interaction between the immune system and developing tumour cells. It consists of three phases: **Elimination**, resulting in immune clearance; **Equilibrium,** immune control without complete eradication; and **Escape**, where the tumour develops resistance and grows (Exhibit 1). During this process, the immune system not only controls cancer but also "edits" the tumour, causing genetic changes that can then help it evade immune detection.

The TME can play a pivotal role in hindering anti-tumour activity

The TME is the complex cellular environment that surrounds a tumour, consisting of various immune cells, stromal cells, and extracellular matrix components. It can play a critical role in a tumour's progress, often becoming immunosuppressive during the Equilibrium and Escape phases and so hindering the body's anti-tumour immune responses and, also, impacting the efficacy of cancer therapeutics.

Clinical understanding means immuno-oncology has become first-line for many tumours

The appreciation of these multiple, and subtle, interactions has seen clinical research and therapy being switched from a tumour-centric to a TME-centric model. The principles underlying the cancer-immunity cycle were explored in previous notes, including how immunotherapy aims to identify and correct the imbalance so that the cycle becomes self-sustaining again and how the research effort has shifted to identifying and developing combination regimens that boost efficacy and limit treatment resistance, but do so with manageable side-effects.

Advent of checkpoint inhibitors brings combination therapies to the fore

Briefly, the TME can present significant impediments for immunotherapy efficacy. Certain tumours are immunologically "cold", or characterised by an absence of tumour-infiltrating lymphocytes (TILs). But even "hot" tumours, that are immunogenic and contain TILs, often see activity impeded by the presence of inhibitory checkpoint molecules such as PD-1 and CTLA-4, or immune suppressor cells like regulatory T-cells and myeloid-derived suppressor cells (MDSCs). The introduction of checkpoint inhibitors (CPIs) has markedly improved clinical outcomes for many cancer patients and has sparked considerable interest in novel immunotherapeutic combination strategies, including therapeutic cancer vaccines. The concurrent use of immunotherapies and CPIs is being investigated to enhance immunogenicity and augment patient response rates and survival outcomes.

Scancell's differentiated and distinct platforms progressing through clinical trials

Scancell's research targets adaptive immune mechanisms via two non-personalised ("off the shelf") platforms addressing distinct oncological pathways: ImmunoBody acts predominantly on CD8 T-cell mechanisms, whereas Moditope leverages CD4 pathways.

- **ImmunoBody** constructs have an elegant design that ensures efficient cross-presentation of specific epitopes (peptide sequences from proteins), and a consistently strong anti-tumour immune response. It is a flexible DNA immunotherapy that induces a high avidity cytotoxic CD8 T-cell response against epitopes with very restricted expression patterns. While promising activity was seen in an earlier monotherapy Phase I/II study in melanoma (offering future upside potential in earlier disease settings), the nearer-term opportunity lies in CPI combinations.
- **Moditope** is a different approach that stimulates a cytotoxic CD4 T-cell response. It effectively generates an immune response against cells undergoing autophagy (a vital process for most cancer cells) by targeting a modification on proteins. Exceptional preclinical results have been observed in preclinical studies, and the aim is to replicate these in the current clinical Phase II trial programme.

ImmunoBody's iSCIB1+ poised for pivotal trial

iSCIB1+ is the lead ImmunoBody programme

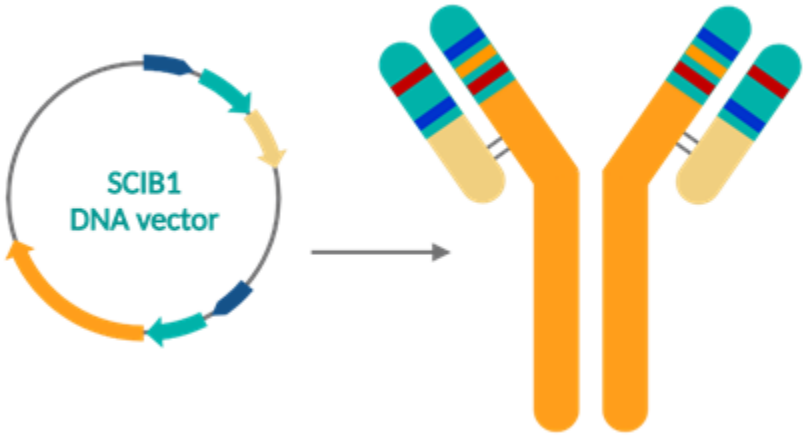
iSCIB1+ is the lead ImmunoBody programme, having been selected earlier this year (July 2025 Lighthouse) following encouraging results from the Phase II SCOPE study of SCIB1/iSCIB1+ in advanced melanoma. iSCIB1+ is the next-generation version of SCIB1 with the potential to address a larger patient population (c 80% of advanced melanoma patients vs c 30-40% with SCIB1). SCOPE's primary aim is to demonstrate that SCIB1/iSCIB1+ in combination with CPI doublet therapy (Yervoy [ipilimumab] plus Opdivo [nivolumab]) act synergistically and achieve improved clinical outcomes. The iSCIB1+ construct is designed to elicit broad, potent and specific immune responses, yet retaining a notably attractive side-effect profile.

ImmunoBody is a novel active immunotherapy

A versatile, flexible and robust immunotherapy platform

The ImmunoBody platform creates DNA plasmids that encode a human antibody framework, but where the native complementarity-determining regions (CDRs) are replaced with selected cytotoxic T lymphocyte (CTL) and CD4+ helper T-cell epitopes derived from tumour-associated antigens (TAAs) (Exhibit 2). Each IgG1 antibody scaffold can incorporate multiple, precisely defined T-cell epitopes to generate a chimeric antigen-antibody structure. These engineered constructs enable both direct and cross-presentation via antigen-presenting cells (APCs), leading to the priming and expansion of high-avidity, polyclonal T-cell responses, which in turn drives potent and broad-spectrum anti-tumour immunity.

Exhibit 2: The structure of ImmunoBody



Source: Scancell

Immunobody constructs carry multiple epitopes to elicit a broad immune response

ImmunoBody constructs are flexible, but with core features that include carefully selected synthetic epitopes that bind selectively to MHC (major histocompatibility complex) class I, to stimulate CD8+ CTLs, and MHC class II, to stimulate CD4+ helper T-cells. The highest avidity T-cell responses are generated if more than one pathway is used to present the same epitope. The targeting and activation of dendritic cells (DCs) is helped by selecting an Fc region of the protein form that targets activated DCs. DCs are considered the most efficient APCs being able to initiate, coordinate, and regulate adaptive immune responses. Each construct typically carries multiple epitopes to broaden the immune response and reduce the risk of immune escape.

Induction of both direct and cross-presentation by APCs

The expressed ImmunoBody protein is secreted and taken up by APCs, including dendritic cells, and induces dual modes of antigen presentation:

- **Direct presentation,** if APCs themselves take up the DNA and express the ImmunoBody they process and present epitopes via MHC class I, and
- **Cross-presentation**, if other cells express the ImmunoBody protein and secrete it, APCs can uptake the secreted protein and present the epitopes via both MHC class I and II pathways.

Epitope selection defines immune response durability, magnitude, and memory

This dual presentation is the key driver for priming naïve CD8+ T-cells (via MHC I) and activating CD4+ helper T-cells (via MHC II), which support CTL expansion and memory. This results in the generation of high-avidity, polyclonal CD8+ and CD4+ T-cell responses with strong cytotoxic potential. These T-cells in turn can recognise and kill tumour cells presenting the target antigen. The inclusion of helper epitopes enhances the durability, magnitude, and memory of the response. The outcome is stronger, longer-lasting immune responses with higher-quality T-cells, improved memory formation, and better tumour targeting compared to immunotherapies that only stimulate CD8+ T-cells.

SCOPE trial to assess SCIB1 and iSCIB1+ in melanoma

SCOPE study provides multiple data insights and guides registrational trial design

SCOPE is a Phase II, translational, open-label study assessing safety and efficacy of SCIB1/iSCIB1+ in advanced unresectable stage III/IV melanoma in combination with CPIs (either standard of care doublet therapy ipilimumab and nivolumab or pembrolizumab). The trial design consists of four cohorts (Exhibit 3) and enrolled over 130 patients across c 16 specialist oncology centres in the UK. The primary outcomes evaluated are safety and tolerability, with ORR (objective response rate) as the primary efficacy endpoint. Secondary endpoints include progression-free survival (PFS, likely to be the key endpoint in the registrational trial), duration of response (DoR), complete response rates (CR), disease control rate (DCR) ie stable disease or tumour regression, and overall survival (OS).

Exhibit 3: SCOPE study of SCIB1 and iSCIB1+ in advanced melanoma

Four cohorts to answer different questions on best combinations, ideal profile, and delivery route

- **Cohort 1** is evaluating 43 patients with SCIB1 plus SoC doublet therapy (nivolumab and ipilimumab) over 25 weeks, administered through a needle-free intra-muscular injection.
- **Cohort 2** is similarly investigating SCIB1, albeit combined with Keytruda (pembrolizumab). The low patient number enrolled (n=10) reflects how Keytruda has effectively been superseded as SoC in these indications.
- **Cohort 3** examines the next generation iSCIB1+ and SoC (nivolumab and ipilimumab), with 50 patients (all-comers) to reflect iSCIB1+'s broader activity profile.
- **Cohort 4** was added more recently, with a similar profile to Cohort 3 but using needle-free intra-dermal delivery, the rationale being that the dermal route could create stronger immune responses.

Source: Trinity Delta

SCIB1 is restricted to a specific HLA type, only found in 30-40% of melanoma patients

Aside from assessing the utility of SCIB1/iSCIB1+ in melanoma, the SCOPE study also aimed to determine the optimal ImmunoBody product (with iSCIB1+ selected). The differences between SCIB1 and iSCIB1+ may appear minor but

could be material clinically and commercially. SCIB1 incorporates specific epitopes from the proteins gp100 and TRP-2 which play key roles in the production of melanin in the skin and were identified from T-cells of patients who achieved spontaneous recovery from melanoma skin cancers. Although highly effective, SCIB1 is only suitable for the 30% to 40% of patients with the A2 HLA type.

iSCIB1+ was designed to work in a broader population than SCIB1

Following confirmation of SCIB1's efficacy, an additional three melanoma-specific epitopes from the same gp100 and TRP-2 proteins were incorporated into second-generation iSCIB1+ with the aim of targeting a broader range of HLA types and thus, potentially, a larger patient population. In addition, the AvidiMab platform was used to improve iSCIB1+ potency (providing better long-term protection and immunological memory), which also confers extended primary patent protection.

Consistent positive data across the SCOPE cohorts

Efficacy aim was to improve on the 48-50% ORR with SoC doublet therapy in advanced melanoma

The efficacy outcome goal of SCOPE was to materially improve on the ORR of 48-50% seen in patients receiving SoC doublet therapy alone in the real-world setting. The doublet therapy median PFS is 11.5 months (CheckMate-067 data) with 12-month PFS of 46%; in real-world data the median PFS was 7.9 months, with a CR rate of 16%, and DCR of 58%. For context, these responses set a high bar, as they were the highest observed in such advanced melanomas.

iSCIB1+ efficacy appears to be comparable to SCIB1, and improves on SoC

A summary of the SCOPE data across the cohorts is shown in Exhibit 4, highlighting the consistent benefits that have been observed with SCIB1/iSCIB1+. As the trial was open label, there are no directly comparable data, but historical data from other trials can be a useful benchmark, with the usual caveats around the limitations of cross trial comparisons. In this instance, the most relevant historical comparators are CheckMate-067 (which led to the approval and use of first-line nivolumab plus ipilimumab CPI doublet therapy, which is now SoC in the advanced melanoma setting) and real-world data on the use of ipi/nivo. We are not aware that data for doublet CPIs have been presented stratified by HLA-type, nor that there are any data to suggest certain HLA types may be more or less responsive to CPI doublet therapy.

Exhibit 4: Summary of SCOPE data shown against SoC

	Cohort 1: SCIB1	Cohort 2: SCIB1	Cohort 3: iSCIB1+	CheckMate-067	Real-world
n	41	10	31		
HLA types	A2 only	A2 only	Target HLAs		
CPI(s)	Ipi/nivo	Pembrolizumab	Ipi/nivo	Ipi/nivo	Ipi/nivo
PFS	56% at 23 months mPFS: not reached	57% at 12 months mPFS: 26.8 months	78% at 11 months mPFS: not reached	46% at 12 months; mPFS: 11.5 months	mPFS: 7.9 months
ORR	63%	70%	65%	50%	48%
DCR	83%	70%	81%		58%

Source: Scancell, Trinity Delta Note: CPI = checkpoint inhibitor; PFS = progression-free survival; ORR = objective response rate; DCR = disease control rate

Cohort 1 25-week data showed improvements with SCIB1 across all key measures

Looking at the elements in more detail, **Cohort 1** included 43 patients treated with intra-muscular SCIB1 plus doublet therapy, but restricted to the A2 HLA haplotype (and one of DR4, DR7, or DQ6). In the 41 evaluable patients (two were considered non-evaluable due to brain metastases and acral melanoma), PFS at 23 months was 56%, with 12/43 patients on trial for this entire period. The latest

DCR is 83% and ORR is 63%. As with prior interim data (November 2024 Lighthouse), meaningfully improved outcomes across all key metrics confirm the value of adding SCIB1 to CPI doublet therapy.

Cohort 2 impact is limited following changes in accepted standard of care

Cohort 2 follows Cohort 1's structure but evaluated SCIB1 in combination with pembrolizumab. However, pembrolizumab's importance has diminished as clinical practice shifted following compelling data from the landmark CheckMate-067 trial which highlighted the benefit of first-line nivolumab plus ipilimumab therapy. As a result, Cohort 2 recruitment was paused at 10 patients. PFS at 12 months was 57% for the SCIB1 combination vs 35% for pembrolizumab alone, with a DCR and ORR of 70% compared to an ORR of 41% for pembrolizumab alone. The merit, in our view, of this cohort are the additional insights and contribution to the safety and tolerability database. A major hope of adding SCIB1/iSCIB1+ to doublet therapy was not to simply improve response rates but, importantly, to potentially reduce the burden of the associated CPI toxicities.

Cohort 3 is critical as it examined iSCIB1+

Cohort 3 was a key element of the SCOPE study as it included the use of iSCIB1+ for the first time. This arm recruited 50 patients, who were treated with intra-muscular iSCIB1+ plus doublet CPI as SoC. To date 43 patients have reported data with a further seven patients awaiting their first verified scans. This cohort effectively included "all-comer" HLA types in order to test the theory that iSCIB1+ would be effective in a broader population.

Cohort 4 is evaluating intra-muscular vs intra-dermal delivery

Cohort 4 is noteworthy as it is evaluating 43 patients in the same format as Cohort 3 but using an intra-dermal route (PharmaJet's Tropis delivery system) and with an accelerated dosing regimen. This follows on from preclinical work suggesting that the activation and presentation to antigen presenting dendritic cells is superior intra-dermally and could also lead to an accelerated dosing schedule (three priming doses given in quick succession at weeks 0, 1 and 3, followed by a booster dose at week 7). The effect could be greater efficacy, but the accelerated dosing could also help address those patients who miss therapy due to CPI treatment toxicities (requiring the use of steroids). Patient recruitment has been bolstered by the partnership with the NHS Cancer Vaccine Launch Pad (CVLP) initiative. Initial Cohort 4 data remain on track for end-2025.

iSCIB1+ selected given the broader patient population

A target HLA population in which iSCIB1+ is effective has been defined

Cohort 3 is a critical element of SCOPE as it was the first test of the theory that iSCIB1+ would be effective in a broader population as it had recruited melanoma patients with "all-comer" HLA types. As outlined earlier in this report, the additional epitopes in iSCIB1+ were expected to broaden the addressable patient population and iSCIB1+ was predicted to work in A1, A2, A3, A31, A33, Bw4, B35, and B44 (with SCIB1 restricted to A2 only). All patients were HLA typed (through a readily available, simple, and rapid blood test) and responses were assessed and correlated. According to Scancell, iSCIB1+ was found to be effective in patients with A2, A3, A31, Bw4, B35 and B44, which has been thus defined as the "target" HLA population (with data from this cohort presented in Exhibit 4 stratified on this metric); iSCIB1+ did not stimulate a response in A1, and only a single patient with A33 was recruited so data were inconclusive.

iSCIB1+ data consistent with SCIB1…

Of the 43 patients that have reported data, 31 are in the target HLA population, 11 are in the non-target HLA population, and one was considered non-evaluable

due to brain metastases. In the target HLA group (n=31) the 11-month PFS was 78%, DCR was 81% and ORR was 65%. In the non-target HLA population (n=11), where iSCIB1+ was not found to be effective, the 11-month PFS was 50% and ORR was 27%, similar to doublet therapy alone. Interestingly, two patients in the target HLA group were rapid progressors, with tumours that advanced before their first week 13 scan. It is thought that an accelerated immunisation regime, allowing earlier doses of iSCIB1+ (as in Cohort 4) may help such patients. Data from the remaining seven patients are expected this year.

...but applicable to a much larger patient population

The target HLA population represents around 80% of all advanced melanoma patients, a much broader population, almost double that of SCIB1. Hence, iSCIB1+ was selected as the lead ImmunoBody candidate to take forwards into a future registrational trial.

Cohort 3 data stratified by T-cell response is enlightening

Positive T-cell responses led to an improved clinical outcome

In the SCOPE study, T-cell responses were evaluated to better understand clinical outcomes. Of the 50 patients enrolled in Cohort 3, T-cell data are available for 31 patients, with nine patients pending analysis. Of the remaining ten patients, five had no blood samples submitted due to being off study, and five were excluded following disease progression. Among the 31 evaluable patients, 19 demonstrated a positive T-cell response (Exhibit 5). The data show that a positive T-cell response is associated with improved clinical outcomes; specifically, patients with a T-cell response had an ORR of 79%, whilst with very few patients with progressive disease mounted a T-cell response

Exhibit 5: T-cell responses in Cohort 3

Clinical Response	N	Positive T-cell response	% positive
Complete/Partial Response	19	15	79%
Stable Disease	7	3	43%
Progressive Disease	5	1	20%
Total	**31**	**19**	

Source: Scancell, Trinity Delta

HLA type and CD8+ response linked to outcomes

Best responses were seen in patients with the target HLA type with a CD8+ response

Additional data show that all six epitopes included in iSCIB1+ induced antigen-specific T-cell responses. A CD8+ (killer T-cell) response was more strongly associated with clinical benefit, as measured by overall response rate (ORR) and defined as CR or PR. The best outcomes were observed in patients with the target HLA type who also mounted a CD8+ response:

- ORR if a positive T-cell response: 67% (12/18); and
- ORR if a CD8+ T-cell response: 83% (10/12).

A clean safety profile with no unexpected toxicities

SCIB1/iSCIB1+ are well tolerated without additional toxicities on top of SoC

Safety data from all SCOPE cohorts are shown in Exhibit 6. For both SCIB1 and iSCIB1+ there was a much lower incidence of Grade 3 and 4 treatment-related adverse events (AEs) compared to those related to the CPIs, and even fewer

serious adverse events (SAE), which are generally defined as being fatal, life-threatening, requiring hospitalisation or prolonged hospitalisation, or causing disability. Importantly, the addition of SCIB1/iSCIB1+ to CPIs does not appear to cause additional toxicities.

Exhibit 6: Safety data (TEAEs) from SCOPE across all Cohorts

	TOTAL EVENTS	EVENTS RELATED TO SCIB1 AND ISCIB1+	EVENTS RELATED TO CPI	EVENTS RELATED TO THE ADMINISTRATION PROCEDURE	NOT RELATED
All AEs (subjects)	1689	258	732	124	575
SAEs	123	11	92	0	20
AEs > G3	163	30	113	3	17
Grade Undefined	47	1	5	1	40

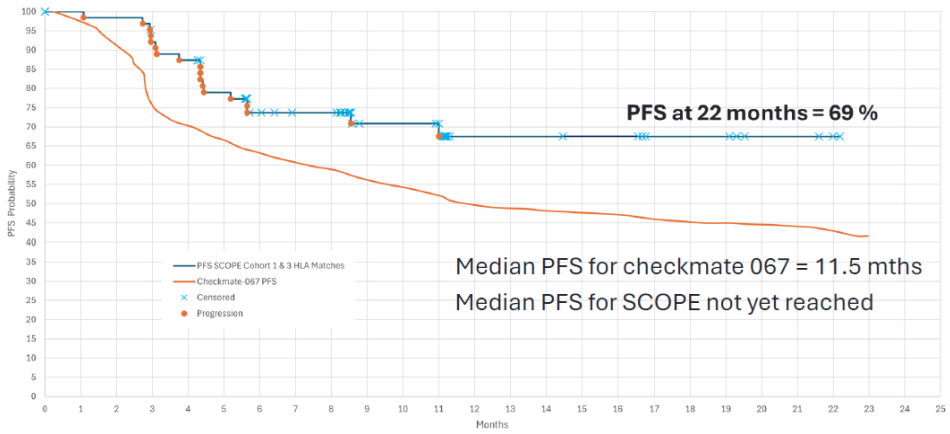
Source: Scancell Note: TEAE = Treatment emergent adverse event

Future plans accelerated based on impressive PFS

Impressive PFS improvement has accelerated development plans

Patient outcomes in SCOPE were primarily assessed by objective response rate (ORR). PFS (progression-free survival) and OS (overall survival) are also being measured but data are not yet mature. To date, PFS at 23-months in Cohort 1 is 56%, whereas in Cohort 3 the PFS is 78% at 11-months, with the combined cohorts shown in Exhibit 7 plotted against SoC data for doublet CPIs ipilimumab/nivolumab (from Checkmate-067). As they are not from the same study the data are not strictly comparable, however they do give an indication of the likely clinical benefit of combination therapy. With these caveats, there does appear to be a PFS improvement, and it is this outcome that has prompted the acceleration of future development activities for iSCIB1+.

Exhibit 7: Cohorts 1& 3 (target HLA) overlaid vs Checkmate-067 (illustrative)



Source: Scancell Note: Target HLA populations from Cohorts 1 and 3 combined (n=72) overlaid with doublet CPIs ipilimumab/nivolumab from Checkmate-067 trial as an illustration

Durable responses seen, hopefully maintained

Responses appear to be highly durable

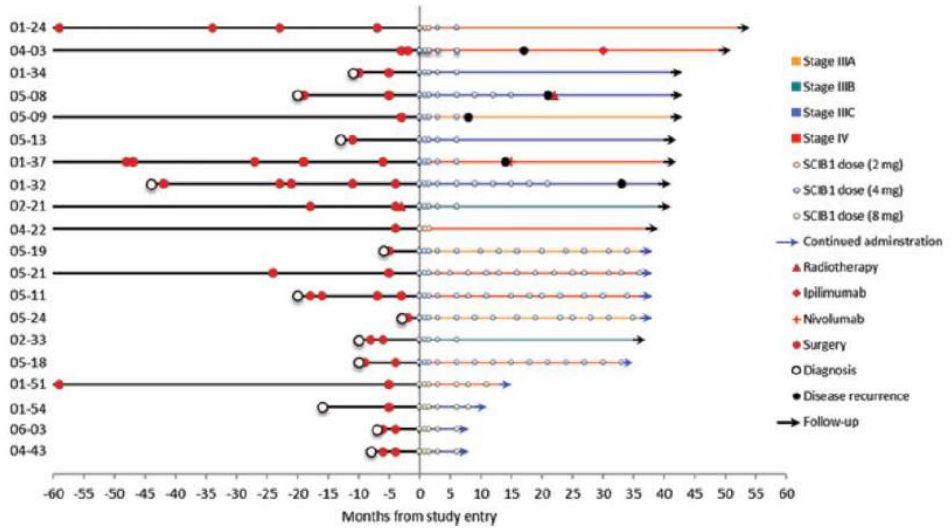
The data to date appear to suggest that responses achieved are highly durable and that once a patient has a response, this is maintained - longer follow up data will be important in this regard. This is perhaps due to the CD8+ killer T-cell responses observed in the majority of patients with a clinical response; these T-cells direct

tumour cytotoxicity and promote memory T-cell formation, which contribute to a prolonged clinical benefit. Durable responses have also been observed with SCIB1 as monotherapy (outlined below), where 14 patients (of 35 recruited) were still alive five years after the study had started.

SCIB1 has also shown durable activity as monotherapy

The SCIB1 monotherapy Phase I/II study included 35 patients with Stage III and IV metastatic melanoma (conducted before CPI therapy was approved). 15 patients had tumours present and 20 had fully resected disease, receiving doses ranging from 0.4mg to 8.0mg. The study showed a potent dose dependent immune response and an associated anti-tumour effect. All four patients who received the 8mg doses remained disease free. One of 15 patients with measurable disease showed an objective tumour response and 7/15 showed stable disease. Five of 20 fully resected patients experienced disease recurrence (Exhibit 8), but all remained alive at the cut-off date with a median observation time of 37 months.

Exhibit 8: Swimmer plot of 20 patients with fully-resected tumours



Source: Patel et al, Oncoimmunology 2018

Further SCOPE survival data are anticipated later this year

The expectation is that the effects of iSCIB1+ can be similarly maintained (ie are durable). This is a directly positive outcome for patients as it means they can live for longer without their disease worsening (a perhaps more meaningful real-world patient benefit than tumour shrinkage, ORR). Patients enrolled in SCOPE remain in the study for two years, so survival data (OS and PFS) will continue to be monitored. We expect an update on PFS, with longer follow-up, in Q425, as well as potential initial OS data.

Next steps for iSCIB1+ have been accelerated

Registrational study plans are being accelerated

Planning for a registrational trial was already underway in parallel to SCOPE, and the latest data, plus selection of iSCIB1+ have accelerated these efforts, with Scancell intending to discuss the trial design with the FDA imminently, ahead of Cohort 4 data. This is to ensure that the trial can start as soon as is practicable. Scancell also intends to discuss the clinical plans with multiple regulators (ie the MHRA in UK and EMA in Europe) and will seek to apply for any relevant accelerated pathways that could be applicable, to try and make iSCIB1+ available to patients as swiftly as possible.

Trial could potentially start in 2026 with a likely focus on PFS

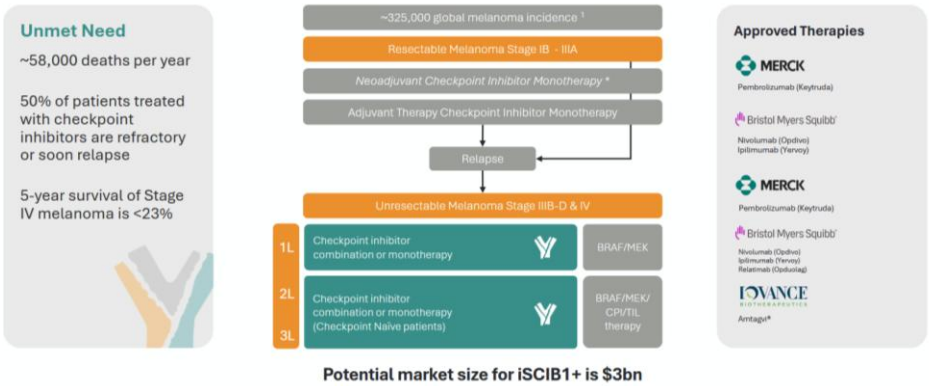
Irrespective of any accelerated approval pathways, a Phase III registrational trial will be needed to secure full approvals and our launch forecasts are based on completion of this. Scancell has previously outlined that the trial is likely to be a multinational, multi-centre, blinded study (with key centres in the US, UK, and Europe), perhaps in around 450-500 patients. A pre-specified interim data read-out would be typical in a registrational oncology trial, and if included this could potentially be used to seek accelerated approvals whilst the trial runs to completion. Given regulators tend to prefer survival endpoints as a more robust measure of patient benefit, we expect the primary efficacy endpoint of the trial will be PFS. We expect the trial to initially use the intra-muscular route of administration, with the potential for intra-dermal accelerated dosing incorporated should Cohort 4 data be supportive. A smooth regulatory clearance could see the trial start patient enrolment during 2026.

Looking beyond advanced melanoma

Potential for iSCIB1+ to also have a place in earlier-stage melanomas

While plans in the unresectable metastatic population are advanced, Scancell is at the very preliminary stages of exploring the potential to investigate iSCIB1+ in earlier stage neoadjuvant/adjuvant resectable melanoma. This is based on activity that has been observed in combination with various CPIs. In this less frail group, there is the possibility that even better outcomes could be achieved for patients. Exhibit 9 shows how iSCIB1+ could be positioned as a central element within various melanoma treatment pathways.

Exhibit 9: iSCIB1+ could become part of new standard of care in melanoma



Source: Scancell

Sizeable market for iSCIB1+ in late-stage melanoma; potential to expand into larger & earlier-stage markets

Based on the now clearly defined target patient population in advanced melanoma, management estimates that the addressable population is 38,000, with an addressable market size for iSCIB1+ in this indication of c $3bn. In the earlier-stage neoadjuvant/adjuvant setting the opportunity is larger, with a patient population of 129,000 and market potential of around $6-9bn. Other opportunities for iSCIB1+ could be as a preventative therapy in certain high-risk groups. However, at present, our peak sales assumption for iSCIB1+ focuses only on the advanced melanoma indication. There remains an unmet need in this group of patients; despite CPIs improving patient outcomes, only around 50% of patients maintain a long-term benefit, with the remainder either relapsing or becoming refractory to CPI treatment.

Moditope: an innovative approach to immunotherapy

Novel mechanism that is highly selective for tumour cells

Moditope is a novel approach that targets the modified self-antigens generated under conditions of cellular stress and exploits the normal immune responses responsible for the clearance of such compromised cells. In solid tumours, unregulated proliferation of malignant cells and the defining characteristics of the TME make such stresses a common feature; most cancer cells exist in hypoxic and nutrient-deprived conditions. To help survive in this hostile environment, they rely on autophagy to recycle proteins and eliminate damaged components that would otherwise be toxic.

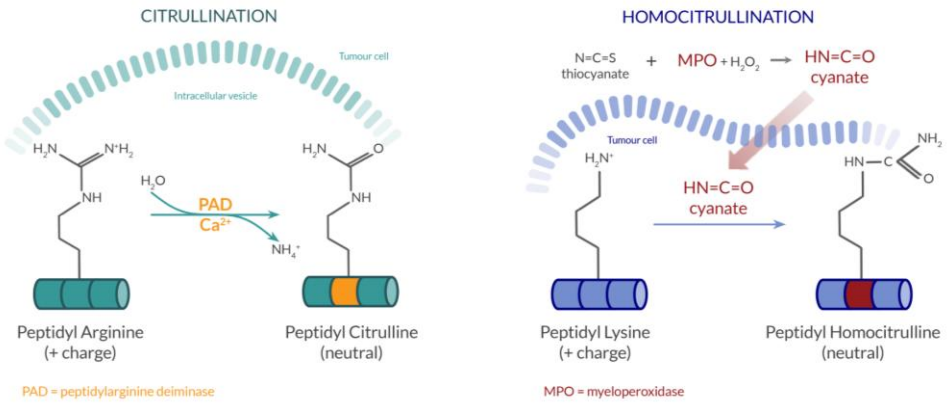
Targets modified peptides generated under conditions of cellular stress…

Autophagy is particularly pronounced in the hypoxic core of expanding tumours, prior to angiogenesis and the establishment of new vasculature. During this process, stress-induced post-translational modifications (siPTMs) and proteolytic cleavage generate modified peptides that accumulate at higher levels in tumours compared to normal tissues, where such stress responses are infrequent (as normal cells are rarely stressed in these ways).

…in particular citrullination and homocitrullination

Such PTMs are mediated by multiple enzymes, some of which are dysregulated specifically in tumour cells, making them potential tumour-selective targets. Notable examples of stress-induced PTMs include citrullination, the enzymatic conversion of arginine to citrulline, and homocitrullination (carbamylation), where lysine residues are converted to homocitrulline (Exhibit 10).

Exhibit 10: Schematic of the citrullination and homocitrullination pathways



Source: Scancell, Seminars in Immunology VA Brentville 2020

These modifications are enriched in tumour cells

Citrullination is catalysed by the PAD (peptidylarginine deiminase) family of calcium-dependent enzymes, which are broadly expressed across tissues. Within autophagosomes, PAD enzymes act on protein fragments, modifying arginine residues to citrulline. Homocitrullination (or carbamylation) is mediated by MPO (myeloid peroxidase), which converts lysine residues to homocitrulline. Both modifications are selectively enriched in tumour cells due to persistent environmental stress and elevated autophagic activity within the TME.

Dysregulation is poorly understood, but is known to impact cancer progression

Dysregulated citrullination pathways were first associated with autoimmune disorders, particularly rheumatoid arthritis. The breadth and depth of the biological functions mediated by citrullination is still poorly understood (especially whether its effects are context driven); however, it is known to affect pathways directly contributing to cancer progression. In particular, it has been shown to

influence the Wnt/β-catenin and androgen receptor signalling pathways. Beyond these effects, citrullination contributes to tumour progression, proliferation, and metastasis through several mechanisms, including promotion of EMT (epithelial-mesenchymal transition), modulation of apoptosis and cellular differentiation, facilitation of circulating tumour cell entrapment at secondary sites, and reactivation of dormant cancer cells.

Offers the potential for promising targets in cancer treatment

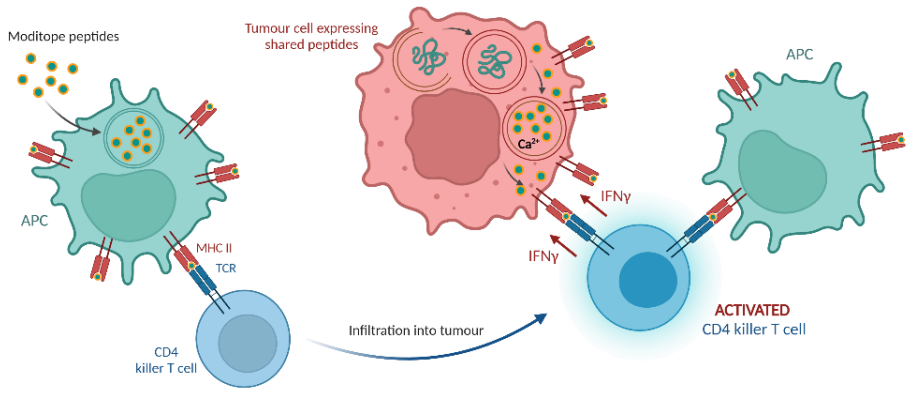
Citrullinated proteins are promising targets for tumour-directed immunotherapies. Candidate antigens being explored in a range of solid tumours include citrullinated forms of α-enolase (ENO1), vimentin (VIM), nucleophosmin (NPM1), matrix metalloproteinase-21 (MMP21), cytochrome P450 (CYP450), and glutamate receptor ionotropic (GRI). Under normal conditions, peptides presented on MHC class I molecules - and recognised by CD8+ cytotoxic T-cells - are generated through proteasomal degradation of intracellular proteins. In contrast, MHC class II bound peptides, which activate CD4+ helper T-cells, are typically derived from exogenous proteins internalised by antigen-presenting cells (APCs) and processed via lysosomal proteolysis. Importantly, neo-citrullinated peptides are presented through the MHC class II pathway, enabling direct recognition of stressed tumour cells by cytotoxic CD4+ T-cells.

Achieving the induction of high-avidity, cytotoxic T-cells

Off the shelf, with the potential for few side-effects…

The Moditope platform neatly harnesses the normal immune response that uses cytotoxic CD4 T-cells to eradicate stressed cells. Immunisation with citrullinated proteins has been shown to elicit durable CD4+ T-cell responses against tumour cells. Importantly, these T-cells recognise citrullinated epitopes that are absent from normal healthy tissues, and their activity is not thought to significantly affect cells implicated in autoimmune disease. Again, like ImmunoBody, the Moditope platform does not require personalisation and will be available “off the shelf”.

Exhibit 11: An illustration of the anti-tumour activity of Moditope



Source: Scancell, Seminars in Immunology VA Brentville 2020; Note: APC = antigen presenting cell; TCR = T-cell receptor; MHC = major histocompatibility complex; IFNγ = interferon gamma.

…and direct tumour killing activity

Exhibit 11 illustrates how the Moditope platform works, using Modi-1 as the example. Citrullinated (Modi-1) or homocitrullinated (Modi-2) peptides are directly conjugated to adjuvant to activate APCs. Following uptake, the modified peptides are processed and presented on MHC class II molecules. CD4+ T-cell receptors engage with these MHC-peptide complexes, leading to T-cell priming. The activated CD4+ T-cells then infiltrate the TME, where they recognise citrullinated

peptides presented by tumour-associated APCs. Upon activation, the CD4+ T-cells secrete IFN-γ, which drives upregulation of MHC class II expression on tumour cells. This amplifies antigen presentation, further activates CD4+ T-cells, and culminates in direct cytotoxic killing of tumour cells.

Potential for use as monotherapy or in combinations

Tumour cells typically create a protective immunosuppressive microenvironment in which MHC class II expression is downregulated, allowing them to evade immune surveillance. The secretion of IFN-γ by Moditope-activated CD4+ T-cells induces local inflammation and promotes MHC class II upregulation, effectively converting immunologically "cold" tumours into "hot" tumours that are more readily detected by the immune system. Through this mechanism Moditope generates cytotoxic CD4+ T-cells capable of overcoming tumour-induced immune suppression and directly eliminating malignant cells that would otherwise remain hidden. This suggests Moditope has potential both as a monotherapy and in combination with other immunotherapeutic agents, such as checkpoint inhibitors (CPIs), to target a broad spectrum of currently difficult-to-treat cancers.

Exhibit 12: A comparison of characteristics of Moditope and standard therapeutic vaccines

Reason for limited efficacy	Moditope	Standard therapeutic vaccines
Antigens targeted	Common proteins (eg cytoskeletal proteins) that have post-translational modifications	Tumour-associated antigens or neo-antigens
T-cell response	Cytotoxic CD4 T-cell and CD4 Th cell	Cytotoxic CD8 T-cell and CD4 Th cell
Synergistic with checkpoint inhibitors	Yes, although this may not be required	Yes
Delivery system	Peptide directly conjugated to adjuvant	DNA, RNA, unlinked peptides or virally encoded antigens

Source: Trinity Delta

Preclinical data show survival benefits in several aggressive cancers

Scancell has identified, and patented, a portfolio of siPTM modified epitopes. Preclinical studies show the Moditope platform can elicit robust immune responses against a wide range of solid tumours. *In vivo* experiments using a variety of citrullinated and homocitrullinated peptides have validated earlier findings from cancer cell line models and have shown significant survival benefits in several aggressive tumour models. Interestingly, tumour rechallenge assays show the induction of durable immune memory, highlighting the long-term protective potential of this approach. The strength of the anti-tumour response suggests that malignant cells possess limited mechanisms to resist cytotoxic CD4+ T-cell activity, in contrast to their well-documented strategies for evading CD8+ T-cell mediated killing.

Modi-1 is the lead Moditope programme

Modi-1 is the most advanced Moditope asset

Modi-1 is the lead Moditope programme and employs three citrullinated peptides, two derived from vimentin and one from α-enolase, with the combination selected specifically to minimise the possibility of tumour escape. These are conjugated to a synthetic toll-like receptor (TLR) 1/2 agonist (AMPLIVANT) which acts as a potent adjuvant and materially enhances activity (10-100 fold) via better dendritic cell antigen processing and presentation plus enhanced T-cell priming.

Targeting proteins have a role in various solid tumours

Vimentin is a cytoskeletal protein that is preferentially digested during autophagy. Vimentin plays a pivotal role in EMT and is associated with regulation of attachment, migration, and signalling in many solid tumours. Mesenchymal

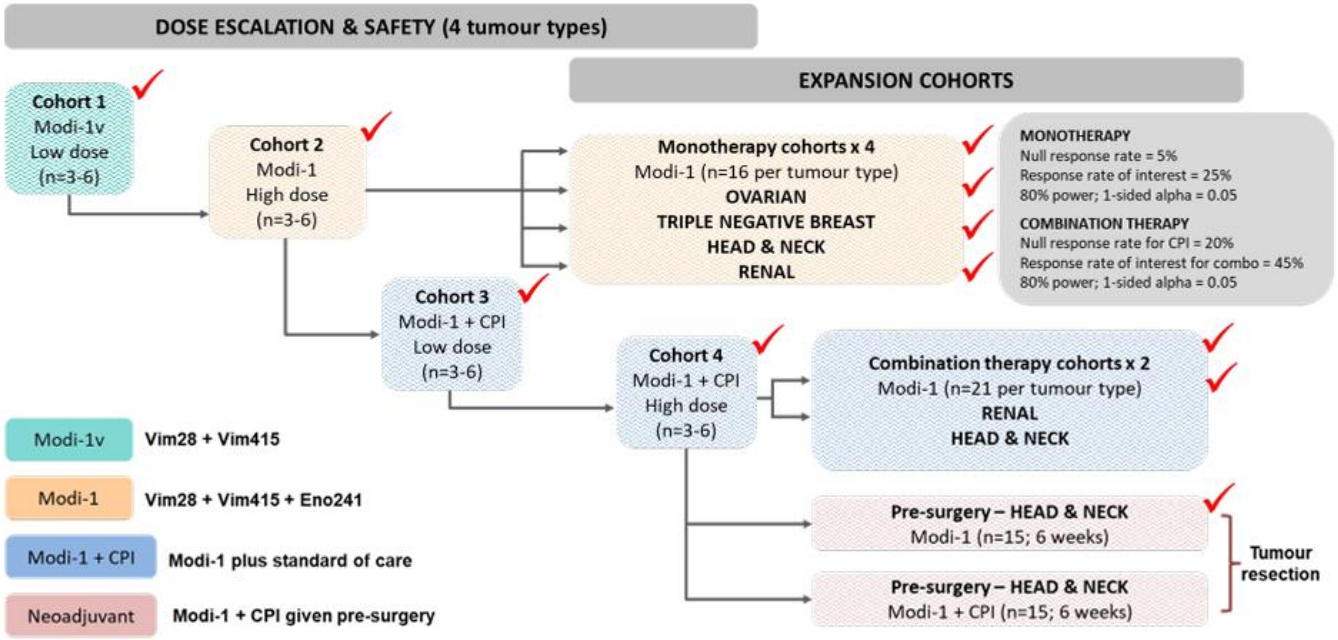
tumours such as endometrial, renal, sarcomas, lymphomas, and lung tumours express vimentin as their major cytoskeletal protein and, additionally, many epithelial tumours (eg breast, ovarian, renal, head & neck (H&N), gastrointestinal and prostate) switch from expression of cytokeratin to vimentin during metastasis. The second target is α-enolase, a metalloenzyme involved in glycolysis, that contributes to cancer cell proliferation, migration, invasion, and metastasis. Typically, cancer cells rely on aerobic glycolysis (the Warburg effect) for energy production, even when oxygen is not deficient. α-enolase is overexpressed in a range of cancer types, and it plays a key role in regulating tumour metabolism, proliferation, and survival in cancers such as ovarian, renal, H&N, lung, pancreatic and triple-negative breast cancer (TNBC), making it attractive as a target.

The ModiFY Phase I/II clinical trial is ongoing

Basket trial is examining Modi-1 across a range of solid tumours as monotherapy and in combination

The Phase I/II study (ModiFY) has been designed to generate meaningful clinical insights across multiple tumour settings, and is structured as two-stages (Exhibit 13). The first stage was an initial dose escalation and safety phase, followed by a series of indication-specific expansion cohorts designed to explore early efficacy signals in TNBC, ovarian cancer, H&N cancer, and renal cell carcinoma (RCC), both as monotherapy and in combination with CPIs, as well as in the neoadjuvant setting. The study plans to enrol over 120 patients across 14 UK sites. The 50 patients in the two cohorts of the first stage (Cohort 1 established the safety and tolerability of a low dose combination of the two vimentin peptides, with Cohort 2 employing a higher dose that also incorporated an additional enolase peptide) showed Modi-1 was well-tolerated at low and high doses as monotherapy in four tumour types and in combination with a CPI in two tumour types. No dose limiting toxicities were observed.

Exhibit 13: Modi-1 Phase I/II clinical trial design



Source: Scancell Note: CPI = checkpoint inhibitor

ModiFY trial addresses key safety elements

Importantly, the enrolled patients are expected to have progressed following first-line therapy, which introduces a clinically relevant consideration. Prior exposure to chemotherapy may induce stress responses in normal tissues, and could, in theory, influence Modi-1's tumour selectivity, potentially leading to on-target but off-tumour effects. Hence this trial will address a key safety element as efficacy data from ModiFY mature.

Early efficacy observations as monotherapy…

Encouraging early efficacy as monotherapy, with good T-cell responses, has been seen in various hard-to-treat cancers, including H&N, ovarian and TNBC. Despite failing prior treatments, 60% of patients receiving Modi-1 achieved stable disease for at least eight weeks, with some patients experiencing longer periods of SD. No safety concerns were reported, supporting its move into the combination settings.

…with durable responses even in hard-to-treat cancers

In the multiple specific expansion cohorts Modi-1 is administered alone or in combination with CPIs in patients with H&N cancer, TNBC or RCC, and as a monotherapy in patients with ovarian cancer, where there are no approved CPI therapies currently. In the ovarian cancer cohort, consisting of 16 patients, 44% of patients achieved stable disease for at least eight weeks, with some patients experiencing a longer duration of disease stability for four months or more. Although a small sample size and early data, the results are particularly encouraging as all patients had effectively exhausted existing treatment options, and their disease was actively progressing when they entered the study.

Promising early data in combination with a single CPI

Early data from the head and neck cohort, exploring Modi-1 in combination with a single CPI (pembrolizumab) in SCCHN (HPV negative head and neck squamous cell carcinoma), saw three of seven evaluable patients showing a PR at the 25-week scan. This is an encouraging ORR of 43% compared to typical ORRs of 19% for pembrolizumab and 13% for nivolumab as current SoC. The positive outcomes help underpin investigator interest in exploring Modi-1 in the neoadjuvant setting.

Data in RCC with double CPIs expected Q425

Recruitment into the RCC cohort, which combines Modi-1 with dual CPI therapy (ipilimumab + nivolumab), is continuing and is expected to render preliminary interim data in Q425. Similarly to SCIB1/iSCIB1+, the combination of Modi-1 with doublet CPIs could be highly synergistic and lead to improved patient outcomes. Doublet CPI is the standard of care for advanced RCC and used in the first-line setting. Hence, ModiFY data could help uncover the potential improvements a Modi-1/CPI combination could bring for first-line patients.

Preparatory work completed ready for next steps, pending data

Importantly, Scancell has now optimised the Modi-1 formulation for scalability and secured US patent protection for the Moditope platform, supporting its broader commercialisation potential.

GlyMab antibodies: novel and truly differentiated

A novel antibody approach

Monoclonal antibodies (mAbs) have quietly transformed clinical care, reshaping the way many chronic diseases are diagnosed and treated. From the earliest stages of a patient's journey, they enable accurate diagnosis and monitoring, while their unmatched specificity makes them ideal for precisely targeted therapies. Their influence extends far beyond the clinic: whether driving discovery in academic research or powering everyday consumer tests such as home pregnancy kits, their ubiquity underscores their immense value. Yet, despite this breadth, almost all approved mAbs remain confined to peptide and protein targets. A rare exception is dinutuximab (United Therapeutics' Unituxin), which binds to the glycan GD2 and is used to treat children with high-risk neuroblastoma.

Glycans are the target, a key element in many biological pathways

Yet carbohydrate binding antibodies, such as glycans, play key roles in biology. These endogenous antibodies recognise bacterial, fungal, and other microbial carbohydrates to prevent systemic infections and help maintain microbiome homeostasis. Their presence on proteins has profound influence on functions such as bioactivity, folding, trafficking, stability, half-life, signalling, and mediation of cell-cell interactions. Aberrant glycosylation is a hallmark of many cancers, shaping nearly every stage of tumour initiation, progression, and metastasis. Both glycoproteins and glycolipids can be altered in this way, giving rise to tumour-associated carbohydrate antigens (TACAs), which represent both a diagnostic signature and a therapeutic opportunity.

But creating effective antibodies has faced many challenges

Several features have made tumour-associated glycans difficult to harness as therapeutic targets. Glycans are structurally diverse and often heterogeneous, with subtle differences in linkage or branching that can confound antibody recognition. Unlike proteins, they are also less immunogenic, meaning that the immune system generates weak responses against them. Their inherent conformational flexibility adds a further layer of complexity, as glycans can adopt multiple orientations that obscure or diminish antibody binding. Additionally, it is more difficult to identify and create glycan antibodies that bind specifically to a glycan of interest, in contrast to an antibody that binds exquisitely to a protein epitope. Hence, despite the appeal of tumour-associated glycans, the challenges in producing high affinity antibodies have been significant.

GlyMab platform potentially addresses key issues

Scancell's in-house expertise has resulted in the unique GlyMab platform

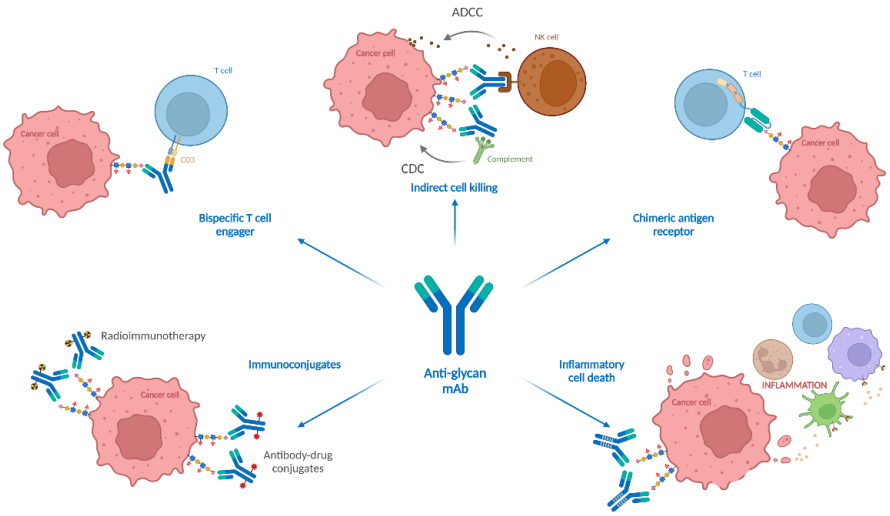
The GlyMab platform has potentially overcome these limitations and is very flexible, consistent, reproducible, and potent. The technology stems from Scancell's in-house expertise and can be employed to produce many differentiated mAbs that bind selectively to the target tumour-associated glycans. Preclinical studies have shown GlyMabs can achieve high affinity for glycans that are markedly overexpressed on cancer cells. Uniquely, these antibodies are capable of directly lysing tumour cells by disrupting the cell membrane and inducing oncotic necrosis, a form of immunogenic cell death (ICD) that bypasses the need for complement activation or immune effector cells. ICD is particularly significant in oncology, as it promotes the release of damage-associated molecular patterns (DAMPs) that engage receptors and ligands on dendritic cells, thereby initiating a cascade of immune activation. This not only facilitates immediate tumour clearance but also has the potential to generate durable, protective anti-tumour

immunity. In this way, anti-glycan mAbs may help remobilise the immune system's full repertoire, counteracting the immunosuppressive forces of the TME and restoring effective surveillance.

Each target can be developed into multiple modalities

The platform is highly flexible as these tumour-associated glycans can be expressed by a wide range of proteins and lipids. This means each anti-glycan antibody can be developed into multiple therapeutics products and formats such as antibody drug conjugates (ADC), bispecific antibodies, chimeric antigen receptor T-cells (CAR-T), as well as strategies that harness redirected T-cell killing both directly and indirectly (via ADCC antibody dependent cell cytotoxicity or CDC complement dependent cytotoxicity). Beyond these, anti-glycan antibodies may also be applied in radioimmunotherapy, broadening their reach across diverse therapeutic modalities (Exhibit 14). This adaptability enhances their clinical utility and positions them as a unifying approach for a wide range of cancers.

Exhibit 14: Illustrations detailing the various killing mechanisms of glycan antibodies



Source: Scancell, Vankemmelbeke M et al; OncoImmunology 5:1; January 2016

Early-stage pipeline with external validation

Industry interest is building, with antibody expert Genmab already a partner

Scancell's pipeline of differentiated antibodies is generating encouraging preclinical data. These GlyMabs are exquisitely tumour-specific and, in contrast to other approaches, have been shown in various models to have high affinity and good potency. The potential of this GlyMab platform has begun to attract industry validation. Antibody expert Genmab has licenced the development rights to two programmes; SC129 in 2022 (October 2022 Lighthouse) and SC2811 in 2024 (December 2024 Lighthouse):

- **SC129** targets sialyl-di-Lewis[a], showing strong selectivity for pancreatic tumours (74%), gastric cancers (50%), and colorectal cancers (36%). Lewis-based glycans are particularly attractive therapeutic targets due to their restricted expression in normal tissues and marked overexpression in epithelial-derived cancers. Preclinical testing has confirmed high binding affinity of SC129 to pancreatic and gastric tumour cells, with minimal binding to healthy tissue. Both direct and bystander killing effects have been demonstrated in internal and Genmab's preclinical studies reinforcing its potential as a first-in-class anti-glycan therapeutic.

- **SC2811** is a mAb that stimulates tumour infiltrating T-cells, having successfully shown target validation for glycolipid stage-specific embryonic antigen 4 (SSEA4) on human and mouse T stem memory cells. SC2811 could have clinical value in many solid tumours.

Combined milestones across the two deals of up to $1.25bn

Disclosed deal economics included substantial potential downstream milestones. The two deals follow a similar structure, and each one carries a potential value of up to $624m should all modalities be progressed, consisting of the $6m upfront payment and future milestone payments of up to $208m per product. Additionally, Scancell is eligible for low single-digit royalties on net sales of any commercialised products.

Three disclosed wholly-owned programmes

Scancell also has three disclosed, wholly-owned GlyMab programmes, together with other undisclosed earlier-stage preclinical programmes, which we expect will be progressed to preclinical validation points. The lead programme is SC134, which is gaining industry attention. While some may be partnered for further clinical development, Scancell is likely to take at least one into the clinic.

SC134 an attractive programme that will be further developed in-house before partnering

SC134 is gaining scientific momentum following a November 2024 paper highlighting strong preclinical data in models of SCLC (small cell lung cancer). SC134 is a bispecific T-cell engager that could offer "best in class" activity in SCLC. It targets Fucosyl GM1, a glycolipid overexpressed in most SCLC tumours but virtually absent from normal healthy tissues. Fucosyl GM1 is also the target for Bristol-Myers Squibb's BMS-986012 antibody and, following positive Phase II data (median OS of 15.6 months vs 11.4 months SoC), a 530 patient Phase III trial is actively recruiting patients. This progress is effective confirmation that fucosyl GM1 is an attractive target in SCLC and should help generate interest for SC134 among potential partners.

Earlier disclosed programmes are SC27 and SC79

Earlier stage programmes include: (1) **SC27** which targets Lewis^y for gastric cancers; preclinical studies have shown it to be more selective and potent than previous Lewis^y targeting approaches; and (2) **SC79** (target undisclosed) for various solid tumours.

GlyMab Therapeutics creates corporate clarity

GlyMab Therapeutics created to take GlyMabs forward, reflecting different needs to the immunotherapy platforms

Earlier in 2025, Scancell formed a new company, GlyMab Therapeutics, enabling the separation of the immunotherapy and antibody platforms into independent corporate entities to allow focus, resources, and investor attention to be appropriately directed to these distinct and unique platforms, potentially offering two independent value creation pathways. The timing reflects the progress of the lead wholly owned GlyMab asset, SC134, through preclinical development and the expectation of IND filing to start clinical studies in 2026.

Also helps attract different investors and makes partnering an easier process

Currently GlyMab Therapeutics is a wholly owned subsidiary of Scancell. The GlyMab platform, all relevant IP, and staff will be transitioned across to this entity over the next 12 months, or sooner if partnered. The near- to mid-term goal is to attract strategic and institutional investors to fund SC134 to clinical proof of concept data, to advance a second in-house GlyMab programme to IND filing, and to develop a further two to three preclinical assets (for either in-house development or partnering).

Sensitivities

Near-term sensitivity is on securing funds to further iSCIB1+ development

In common with most innovative healthcare companies, the main sensitivities for Scancell relate to clinical development and regulatory risks, successful commercialisation including executing partnership agreements, and the financial resources required to accomplish these. The main near-term specific sensitivity for Scancell, in our view, is on securing funds to progress iSCIB1+ into the planned registrational trial.

Funding will be needed for the registrational trial

The iSCIB1+ registrational trial is currently being planned, and although precise details on the size and scope are unknown, Scancell does not currently have the financial resources to run this trial. Whilst we believe data to date are compelling, and justify further development, the immuno-oncology field is crowded and hence it is always challenging for companies to stand out sufficiently to attract the appropriate level of interest to secure the required funding (be it directly from investors, and via an industry partner).

Misplaced perceptions about vaccines could be a headwind

There could be a perhaps misplaced perception by some observers that Scancell is a vaccine company. There has been negative rhetoric, mostly from the US, regarding vaccines. However, this largely relates to the more traditional prophylactic vaccinations that are designed to prevent diseases. Scancell's products are indeed injected, but their activity is subtly different, and they are designed to treat often incurable diseases. Nevertheless, this perception could limit the pool of potential investors/partners.

There have been prior failures in related fields that could cloud expectations

In terms of clinical trial risk, historic failures of previous therapeutic vaccines could cloud expectations of Scancell's programmes. Yet Moditope and ImmunoBody both have different mechanisms of action to any prior approaches and should be judged on their own merits. The design and execution of the clinical programmes is an important determinant of any study outcome, but this is particularly the case in immuno-oncology trials (especially when evaluating differing therapies in combination).

Competitors could overtake or steal the limelight

On the competitive front, ImmunoBody and Moditope would be complementary to many methods under investigation to enhance the activity of the immune system, with combination therapies increasingly accepted as standard of care for many solid tumours. However, not only is Scancell contending with novel alternative therapies, it is also competing directly against other therapeutic vaccine companies, including BioNTech and Moderna. Such players have greater resources, both physical and financial, and could leapfrog Scancell's progress. In addition, while Scancell's therapeutic platform technologies have demonstrable and attractive qualities, an unexpected breakthrough in an unrelated scientific area may side line one or more of its approaches.

GlyMab yet to enter the clinic thus development risk is high

The GlyMab antibody platform is at the earlier development stages and, understandably, carries greater uncertainties and risks. GlyMab has generated genuinely exciting preclinical data, which has attracted two licensing deals from Genmab, but the potential value of the platform will only be realistically demonstrated in appropriate clinical trials.

Valuation

rNPV based valuation of £382m ($477m), equivalent to 37p per share (31p fully diluted)

We value Scancell as a classic drug discovery and development play with three distinct platforms. We use a sum-of-the-parts model, which comprises risk-adjusted NPVs (net present value) which are summed together with cash. However, we have taken the opportunity to fully revisit our valuation, and within each platform we now have a separate standalone valuation for the clinical assets (ie iSCIB1+ and Modi-1), in addition to indicative placeholder valuations for the platforms. The clinical programmes (including those ready to enter the clinic) carry the greatest weight, with preclinical programmes discounted more aggressively to reflect the lower success probabilities. As always, we use conservative assumptions regarding market sizes and growth rates, net pricing, adoption curves, and peak market penetration. An overview of our valuation, together with key assumptions, is shown in Exhibit 15.

iSCIB1+ is the most important contributor to our overall Scancell valuation

Each rNPV includes an estimate of the potential development costs, and we also make the broad assumption at this stage that future commercialisation of each programme/platform will be via a partner, ie we assume future royalty streams. Whilst potential deal terms (on which we have limited visibility) will affect valuation, the variables that have the most impact on each rNPV are the peak sales, launch date, and probability of success; the latter are based on standard industry criteria for the respective stage of the clinical development process but are flexed to reflect the inherent clinical, regulatory, commercial, and execution risks. iSCIB1+ is the most important contributor to our overall Scancell valuation.

Exhibit 15: Scancell sum of the parts valuation

Programme	NPV (£m)	NPV ($m)	Probability	rNPV (£m)	rNPV ($m)	rNPV/ share (p)	Notes
iSCIB1+	652.1	815.2	30.0%	195.6	244.5	18.9	Launch year: 2030+; Peak sales: $1.5bn
Platform	389.1	486.4	5.0%	19.5	24.3	1.9	Launch year: 2031+; Peak sales: $1bn
ImmunoBody	**1,041.2**	**1,301.5**		**215.1**	**268.9**	**20.7**	
Modi-1	433.9	542.3	15.0%	65.1	81.3	6.3	Launch year: 2030+; Peak sales: $1bn
Platform	588.3	735.3	5.0%	29.4	36.8	2.8	Launch year: 2031+; Peak sales: $1.5bn
Moditope	**1,022.1**	**1,277.7**		**94.5**	**118.1**	**9.1**	
GlyMabs	**1,529.4**	**1,911.8**	**3.5%-5%**	**55.5**	**69.3**	**5.3**	Launch year: 2031+; Peak sales: $5bn NB: includes Genmab deals at 5%
Current cash	16.9	21.1		16.9	21.1	1.6	End FY25 (end April 2025)
Total	**3,609.7**	**4,512.1**		**381.9**	**477.4**	**36.8**	

Source: Trinity Delta Note: assumptions include a 12.5% discount factor, £/$ FX rate of 1.25; we assume all programmes will be partnered

ImmunoBody platform

ImmunoBody is worth £215m/$269m underpinned by iSCIB1+

In our new Scancell valuation, ImmunoBody in totality is worth £215m/$269m, with >90% underpinned by iSCIB1+, and the remainder a placeholder valuation for the platform, where we currently assign a token 5% probability of success. For iSCIB1+ our peak sales forecast is $1.5bn, which is based on advanced melanoma alone and does not include any contribution for neoadjuvant/adjuvant melanoma or any other indications. Scancell management estimates that the potential market size for iSCIB1+ in advanced melanoma is $3bn, hence there could be upside to our current forecasts, and if iSCIB1+ is developed in other sizeable indications.

ImmunoBody could be worth £346m/$432m by the time the iSCIB1+ trial commences

Given the compelling iSCIB1+ PFS data to date in the target population, we assign a 30% probability of success. Upcoming events that could help to derisk iSCIB1+ include: (1) further clinical data, particularly more mature survival data; (2) clarity from regulators regarding registrational trial design; and (3) visibility on funding of the trial. If Scancell can successfully navigate these events and the registrational trial is able to start next year and is fully funded, then a probability of success of around 50% for iSCIB1+ at that time would seem reasonable; all else being equal this would equate to an iSCIB1+ valuation of £326m/$408m, and a total ImmunoBody valuation of £346m/$432m.

Moditope platform

Moditope is worth £94m/$118m with upside as Modi-1 data become available

Our Moditope valuation is £94m/$118m, comprised of a standalone Modi-1 rNPV of £65m/$81m and an illustrative platform valuation of £29m/$37m. For Modi-1 our peak sales forecast is a placeholder $1bn, with this to be refined in the future as clinical data become available and development plans become clearer. We currently assign a 15% probability of success based on encouraging early efficacy as both monotherapy, and initial combination data in head and neck cancer earlier this year. Data in renal cell carcinoma later this year could help to de-risk Modi-1 and clarify future development plans. As with ImmunoBody, we assign a conservative 5% probability to the broader Moditope platform.

GlyMab platform

Genmab deals included within our GlyMab valuation

The GlyMab portfolio consists of wholly owned preclinical programmes with the most advanced SC134 for small cell lung cancer, and SC27 for a variety of solid tumours, in addition to several other earlier stage preclinical programmes. There are two additional programmes partnered with Genmab, with Scancell eligible for future potential development, regulatory and commercial milestones of up to $624m on each programme for development across all modalities (with $208m for each), plus single digit royalties on net sales. The Genmab deals provide external validation for the platform and hence for the wholly owned programmes we assign a success probability of 3.5% (and assume these will eventually be partnered), and a slightly higher 5% success probability to the Genmab partnered assets given deals are now in place, which reduces execution risk. For the Genmab programmes our valuation also reflects the disclosed deal terms.

The formation of GlyMab Therapeutics makes strategic sense

The formation of GlyMab Therapeutics confirms, in our view, the appropriateness of considering the platforms as individual entities. This not only achieves corporate clarity, as the platforms require differing scientific expertise and strategic direction, but their unique profiles are likely to attract different investors, reflecting their near- and medium-term funding needs, and commercial partners. We would consider this a natural evolution, which will allow focus, resources, and investor attention to be appropriately directed to these diverse and innovative platforms.

Financials

Potential for single digit milestone income from partner Genmab

Revenues for FY25 (12 months to 30 April 2025) were £4.7m (FY24: nil), which related entirely to the non-recurring $6m upfront from partner Genmab for the second GlyMab deal for SC2811 ($1m exclusive evaluation fee received in June 2024 and $5m in December 2024 on option exercise). SC129, the first GlyMab partnered with Genmab, is on track to enter the clinic in the near-term, which we believe could trigger milestone(s). As the timing is uncertain, we include these in FY27e, rather than in FY26e; hence we forecast £nil revenues in FY26e, and c £2.4m in FY27e (assuming milestones of $3m). Scancell is entitled to potential aggregate milestones of up to $624m from Genmab on each GlyMab deal, plus single digit royalties on net sales.

R&D spend is focused on clinical trials; G&A remains well controlled

R&D expenses in FY25 increased to £14.7m (FY24: £12.9m) which included continued spend on SCOPE and ModiFY, in addition to iSCIB1+ manufacturing scale up in readiness for future trials. G&A was well controlled, decreasing to £4.8m (FY24: £5.4m). Costs were offset to a degree by the Genmab milestone, leading to a narrower FY25 operating loss of £15.0m (FY24: £18.3m). The net financial loss in FY25 was £0.3m (FY24: income £9.2m), which included non-cash gains of £1.1m (FY24: 9.9m) relating to the convertible loan note. Together, this led to a wider net loss of £12.3m (FY24: £5.9m).

Our forecasts include a base R&D spend pending clarity on future plans and funding

Forecasting future R&D spend is challenging given there are a number of moving parts with both iSCIB1+ and Modi-1. On Modi-1, upcoming data will help to determine potential future development plans. Meanwhile, on iSCIB1+, Scancell is making plans for a registrational trial to start during 2026, and we estimate the trial could cost upwards of $100m given around 450 patients may be recruited with underlying doublet CPI costs of c $200k/patient, plus general running costs. Whilst Scancell does not currently have the cash resources to fund this trial to completion, various funding options could be available, including through business development activities (which could see the trial fully or partially funded by industry partners), capital markets, other financing routes, or a combination of these. Once there is clarity on funding of the iSCIB1+ registrational trial, and the next steps for Modi-1, then more realistic R&D forecasts will be possible. Until then, we assume a similar level of spend in FY26e vs FY25 as both SCOPE and ModiFY trials are still ongoing, whereas in FY27e, we simply forecast an illustrative base level of R&D spend. For G&A we forecast an incremental increase from £4.8m in FY25 to £4.9m in FY26e and £5.1m in FY27e.

Cash runway is sufficient to Q326, beyond key value inflection points

Cash at end April 2025 was £16.9m (FY24: £14.8m), which according to Scancell provides a runway through to calendar Q326; our model is consistent with this, forecasting a small amount of cash at end April 2026, and a cash shortfall during FY27e. This cash runway could be extended by successful execution of any business development transaction(s), where Scancell is proactively exploring various options across the pipeline. This is also beyond the key near-term value inflection points including further data on iSCIB1+, RCC data for Modi-1, and meeting with regulators regarding the iSCIB1+ registrational trial design, and provides Scancell with time to evaluate options for funding the iSCIB1+ trial.

Exhibit 16: Summary of financials

Year-end: April 30	£'000s	2023	2024	2025	2026E	2027E
INCOME STATEMENT						
Revenues		**5,271**	**0**	**4,711**	**0**	**2,400**
Cost of goods sold		(525)	0	(238)	0	(240)
Gross Profit		**4,746**	**0**	**4,473**	**0**	**2,160**
R&D expenses		(11,645)	(12,871)	(14,686)	(14,392)	(5,037)
General and administrative expenses		(5,021)	(5,396)	(4,788)	(4,884)	(5,128)
Other revenue/expenses		0	0	0	0	0
Operating Profit		**(11,920)**	**(18,267)**	**(15,001)**	**(19,276)**	**(8,005)**
EBITDA		**(11,018)**	**(17,301)**	**(14,122)**	**(18,416)**	**(7,992)**
Net Interest		(931)	(734)	(1,381)	(1,427)	(1,671)
Other financing costs/income		(1,453)	9,884	1,079	0	0
Profit Before Taxes		**(14,304)**	**(9,117)**	**(15,303)**	**(20,703)**	**(9,676)**
Adj. PBT		**(13,576)**	**(8,457)**	**(13,945)**	**(19,209)**	**(8,108)**
Current tax income		2,368	3,258	3,031	1,469	1,439
Net Income		**(11,936)**	**(5,859)**	**(12,272)**	**(19,234)**	**(8,237)**
EPS (p)		**(1.46)**	**(0.68)**	**(1.26)**	**(1.85)**	**(0.79)**
Adj. EPS (p)		**(1.37)**	**(0.60)**	**(1.12)**	**(1.71)**	**(0.64)**
Average no. of shares (m)		816.1	862.5	970.3	1,037.1	1,037.7
Gross margin		*90%*	*N/A*	*95%*	*N/A*	*90%*
BALANCE SHEET						
Current assets		**24,606**	**21,867**	**20,624**	**5,703**	**347**
Cash and cash equivalents		19,920	14,817	16,894	1,910	(3,516)
Accounts receivable		538	1,378	631	694	764
Inventories		0	0	0	0	0
Other current assets		4,148	5,672	3,099	3,099	3,099
Non-current assets		**2,249**	**1,709**	**2,466**	**1,619**	**1,619**
Property, plant & equipment		2,249	1,709	847	0	0
Intangible assets		0	0	1,619	1,619	1,619
Other non-current assets		0	0	0	0	0
Current liabilities		**(3,276)**	**(3,527)**	**(3,569)**	**(3,403)**	**(3,059)**
Short-term debt		0	0	0	0	0
Accounts payable		(2,970)	(3,099)	(3,178)	(3,280)	(3,059)
Other current liabilities		(306)	(428)	(391)	(123)	0
Non-current liabilities		**(33,227)**	**(23,554)**	**(23,356)**	**(22,233)**	**(22,233)**
Long-term debt		(32,481)	(23,088)	(23,233)	(22,233)	(22,233)
Other non-current liabilities		(746)	(466)	(123)	0	0
Equity		**(9,648)**	**(3,505)**	**(3,835)**	**(18,314)**	**(23,327)**
Share capital		61,514	72,856	83,440	83,440	83,440
Other		(71,162)	(76,361)	(87,275)	(101,754)	(106,767)
CASH FLOW STATEMENTS						
Operating cash flow		**(8,140)**	**(15,660)**	**(6,399)**	**(13,853)**	**(5,244)**
Profit before tax		(14,304)	(9,117)	(15,303)	(20,703)	(9,676)
Non-cash adjustments		4,014	(7,566)	2,568	3,781	3,253
Change in working capital		940	(711)	732	38	(289)
Interest paid		0	0	0	0	0
Taxes paid		1,210	1,734	5,604	3,031	1,469
Investing cash flow		**81**	**178**	**(1,203)**	**349**	**28**
CAPEX on tangible assets		(203)	(177)	(14)	(13)	(13)
Other investing cash flows		284	355	(1,189)	362	41
Financing cash flow		**(746)**	**10,390**	**9,690**	**(1,480)**	**(209)**
Proceeds from equity		166	11,342	10,584	0	0
Increase in loans		0	0	(450)	(1,000)	0
Other financing cash flow		(912)	(952)	(444)	(480)	(209)
Net increase in cash		**(8,805)**	**(5,103)**	**2,077**	**(14,984)**	**(5,425)**
Cash at start of year		28,725	19,920	14,817	16,894	1,910
Cash at end of year		**19,920**	**14,817**	**16,894**	**1,910**	**(3,516)**
Net cash at end of year		**(12,561)**	**(8,271)**	**(6,339)**	**(20,323)**	**(25,749)**

Source: Scancell, Trinity Delta Note: Adjusted numbers exclude share-based payments and exceptionals. FY27e R&D forecast is illustrative pending development plans.

Company information

Contact details

Scancell,
Unit 202 Bellhouse Building
Sanders Road,
Oxford Science Park,
Oxford,
OX4 4GD, UK

Tel: +44 (0) 1865 582 066

Web: www.scancell.co.uk

Top shareholdings

	% holding
Redmile Group	28.64
Vulpes Life Science Fund & Testudo Fund	13.83
Scancell directors and related holdings	0.26
Top institutional investors	**42.73**
Other shareholders	57.27
Total shareholders	**100.00**

Source: Scancell

Key personnel

Person	Position	Biography
Dr Jean-Michel Cosséry	Non-Executive Chair	Joined as Chair in February 2023. 25+ years of pharma and biotech experience, including commercial opertions, capital raising, IPOs, business development and M&A. Previously, VP North America Oncology at Eli Lilly, Chair of the Eli Lilly UK Board, and Chief Marketing Officer at GE Healthcare. Current NED at Malin Plc, Exact Therapeutics, Eracal Therapeutics, and Sophia Genetics; prior NED at Kymab, Immunocore.
Phil L'Huillier	CEO	Joined as CEO in October 2024. Prior leadership roles in pharma and biotech, driving growth and innovation, including as CEO of CatalYm GmbH (raising over $200m in financing and progessing its lead asset from Phase I to Phase IIb trials), Head of Merck Sharp & Dohme's European Innovation Hub & Business Development, and Executive Director of Cancer Research Technology Ltd. Previously at NED at Achilles Therapeutics, Artios Pharma, Blink Therapeutics, and PsiOxus Therapeutics. Holds a PhD in cellular and molecular biology from the University of Auckland and an MBA (University of Waikato).

Sath Nirmalananthan	CFO	Joined as CFO in August 2023. Over 15 years of finance experience across healthcare in FTSE and NASDAQ listed companies, investment banking, and audit. Previously CFO for Europe, Middle-East and Africa at Prenetics; Finance Director for eCommerce Health and for Group Reporting at Reckitt; plus senior financial planning/reporting roles at BTG. Former healthcare equity research analyst at Nomura and auditor at KPMG. Non-Executive member of the audit committee at The Institute of Cancer Research. Holds a BSc in Pharmacology from King's College London, and is an ACA (ICAEW) qualified Chartered Accountant.
Professor Lindy Durrant	Chief Scientific Officer	Founded Scancell in January 1996 as a spin-out from her work at the University of Nottingham (which she joined in December 1983). Roles at Scancell have included co-CEO, CSO, and CEO. Also Professor of Cancer Immunology at the Department of Clinical Oncology, University of Nottingham. Over 200 publications in peer-reviewed journals and over 143 patents filed. Holds BSc (Hons) Biochemistry and a PhD from Manchester University.
Dr Nermeen Varawalla	Chief Medical Officer	Joined as CMO in August 2024. Over 25 years of healthcare leadership experience in global biopharma, consultancy and clinical trial services. Prior roles include CMO at Relief Therapeutics and Atlantic Healthcare, SVP Head of Clinical Development at BTG. Currently Chair of CRISM Therapeutics and Atorvia Health Technologies. Holds an MD from the University of Mumbai, a DPhil in Clinical Medicine from the University of Oxford, where she was a Rhodes Research Fellow, and an MBA from INSEAD.

Philippa Gardner pgardner@trinitydelta.org
+44 (0) 20 3637 5042

Lala Gregorek lgregorek@trinitydelta.org
+44 (0) 20 3637 5043

Franc Gregori fgregori@trinitydelta.org
+44 (0) 20 3637 5041

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More information is available on our website: www.trinitydelta.org