UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 6-K

Report of Foreign Private Issuer
Pursuant to Rule 13a-16 or 15d-16
of the Securities Exchange Act of 1934

For the month of June 2026

Commission File Number: 001-37643

PURPLE BIOTECH LTD.
(Translation of registrant's name into English)

4 Oppenheimer Street, Science Park, Rehovot 7670104, Israel
(Address of principal executive offices)

Indicate by check mark whether the registrant files or will file annual reports under cover Form 20-F or Form 40-F.

Form 20-F ☒ Form 40-F ☐

Purple Biotech

On June 23, 2026, Purple Biotech Ltd. (the "**Company**" or the "**Registrant**") issued an updated Company presentation, "**Purple Biotech Corporate Presentation June 2026**", which is attached hereto as Exhibit 99.1.

Exhibit	
99.1	Purple Biotech Corporate Presentation June 2026

SIGNATURES

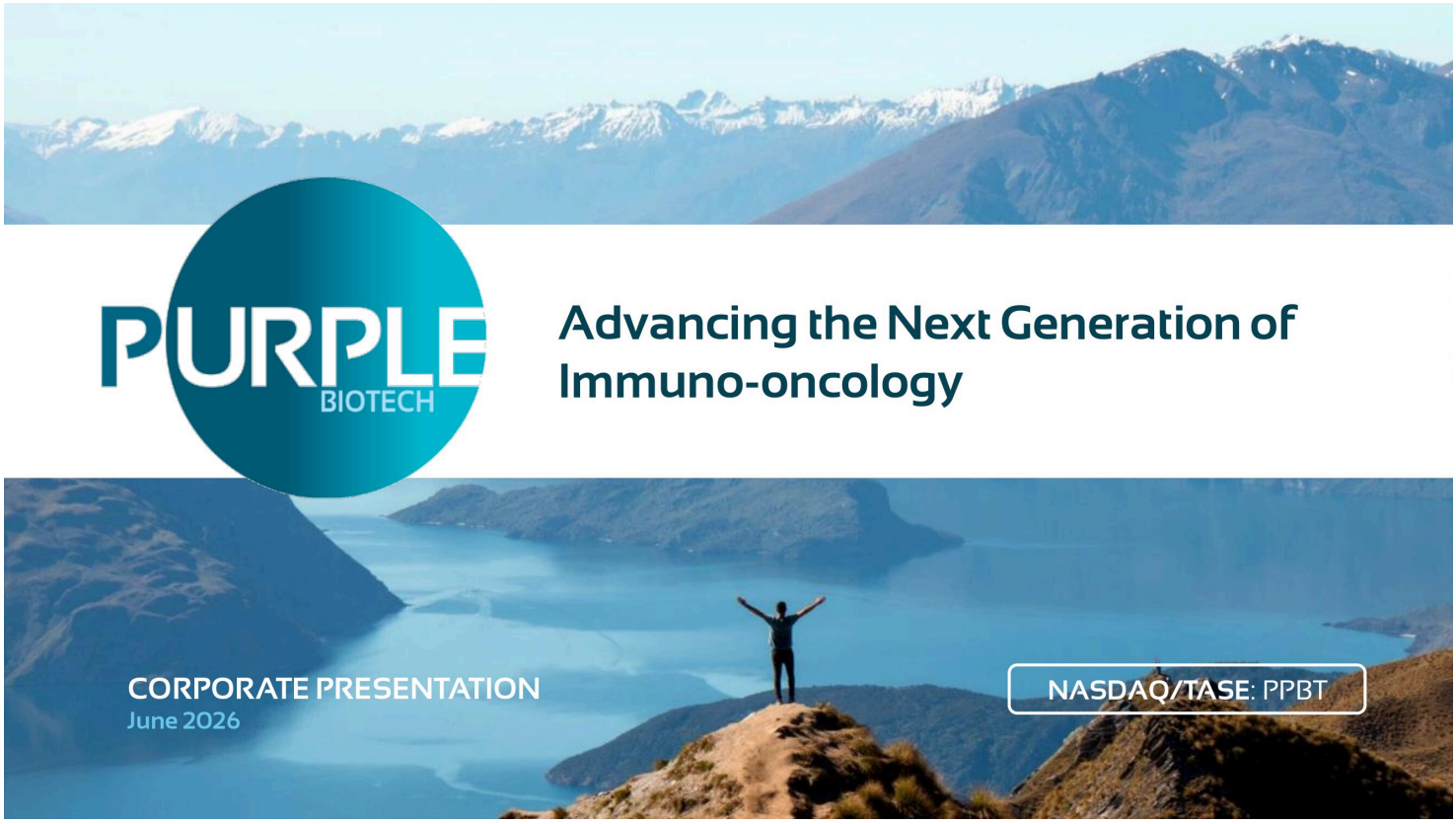
Pursuant to the requirements of the Securities Exchange Act of 1934, the Registrant has duly caused this report to be signed on its behalf by the undersigned, thereunto duly authorized.

June 23, 2026

PURPLE BIOTECH LTD.

By: */s/ Gil Efron*
Gil Efron
Chief Executive Officer

Exhibit 99.1


PURPLE
BIOTECH
Advancing the Next Generation of Immuno-oncology
CORPORATE PRESENTATION
June 2026
NASDAQ/TASE: PPBT

Forward-looking Statements and Safe Harbor

Certain statements in this presentation that are forward-looking and not statements of historical fact are forward-looking statements within the meaning of the safe harbor provisions of the Private Securities Litigation Reform Act of 1995. Such forward-looking statements include, but are not limited to, statements that are not statements of historical fact, and may be identified by words such as "believe", "expect", "intend", "plan", "may", "should", "could", "might", "seek", "target", "will", "project", "forecast", "continue" or "anticipate" or their negatives or variations of these words or other comparable words or by the fact that these statements do not relate strictly to historical matters. You should not place undue reliance on these forward-looking statements, which are not guarantees of future performance. Forward-looking statements reflect our current views, expectations, beliefs or intentions with respect to future events, and are subject to a number of assumptions, involve known and unknown risks, many of which are beyond our control, as well as uncertainties and other factors that may cause our actual results, performance or achievements to be significantly different from any future results, performance or achievements expressed or implied by the forward-looking statements. Important factors that could cause or contribute to such differences include, among others, risks relating to: the plans, strategies and objectives of management for future operations; product development for NT219, CM24 and IM1240; the process by which such early stage therapeutic candidates could potentially lead to an approved drug product is long and subject to highly significant risks, particularly with respect to a joint development collaboration; the fact that drug development and commercialization involves a lengthy and expensive process with uncertain outcomes; our ability to successfully develop and commercialize our pharmaceutical products; the expense, length, progress and results of any clinical trials; the impact of any changes in regulation and legislation that could affect the pharmaceutical industry; the difficulty in receiving the regulatory approvals necessary in order to commercialize our products; the difficulty of predicting actions of the U.S. Food and Drug Administration or any other applicable regulator of pharmaceutical products; the regulatory environment and changes in the health policies and regimes in the countries in which we operate; the uncertainty surrounding the actual market reception to our pharmaceutical products once cleared for marketing in a particular market; the introduction of competing products; patents obtained by competitors; dependence on the effectiveness of our patents and other protections for innovative products; our ability to obtain, maintain and defend issued patents; the commencement of any patent interference or infringement action against our patents, and our ability to prevail, obtain a favorable decision or recover damages in any such action; and the exposure to litigation, including patent litigation, and/or regulatory actions; the impact of the economic, public health, political and security situation in Israel, the U.S. and other countries in which we may operate or obtain approvals for our products or our business, and other factors that are discussed in our Annual Report on Form 20-F for the year ended December 31, 2025 and in our other filings with the U.S. Securities and Exchange Commission ("SEC"), including our cautionary discussion of risks and uncertainties under "Risk Factors" in our Registration Statements and Annual Reports. These are factors that we believe could cause our actual results to differ materially from expected results. Other factors besides those we have listed could also adversely affect us. Any forward-looking statement in this press release speaks only as of the date which it is made. We disclaim any intention or obligation to publicly update or revise any forward-looking statement or other information contained herein, whether as a result of new information, future events or otherwise, except as required by applicable law. You are advised, however, to consult any additional disclosures we make in our reports to the SEC, which are available on the SEC's website, https://www.sec.gov. The trademarks, tradenames, service marks and logos included herein are the property of the owners thereof and are used for reference purposes only. Such use should not be construed as an endorsement of the products or services of the Company

Snapshot: Company Highlights

1. **CAPTN-3: differentiated tri-specific platform activating both innate and adaptive immune systems**
   The only platform engaging both T cells AND NK cells while incorporating a masking technology

2. **Lead program expected to enter the clinic in 2027**
   IM1240 (cappedCD3x**5T4**xNKG2A), a first-in-class CAPTN-3 tri-specific antibody
   Second program, IM1305 (cappedCD3x**TROP2**xNKG2A)

3. **Safety and efficacy demonstrated in IND enabling Studies**
   Strong anti-tumor activity and 300× improved safety profile versus the non-masked comparator

4. **Focused on key inflection point and significant market potential to create value**
   Comparable masked TCE programs secured >$1B deals at preclinical stage.
   Key catalyst - results from P1 study starting from 1H 2028

5. **Solid tumors remain largely untreated by T cell engagers**
   A large unmet need with no approved tri-specific option

FINANCIALS

Cash
$6.4M
As of March 31, 2026

Runway
INTO H1 2027

Exchange
NASDAQ + TASE: PPBT

The Problem: T Cell Therapy Challenges in Solid Tumors

Multi-specific T cell engagers show promise in blood cancers but face major hurdles in solid tumors

Imprecise Targeting

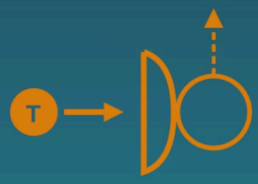
Many T cell engagers are 'always on'—activating systemically instead of selectively at tumor sites

51-90%

CRS rates with current TCEs*

Severe Toxicity

Cytokine release syndrome (CRS) limits dosing and tolerability



Limited Potency

Tumors escape single-mechanism therapies through adaptive resistance

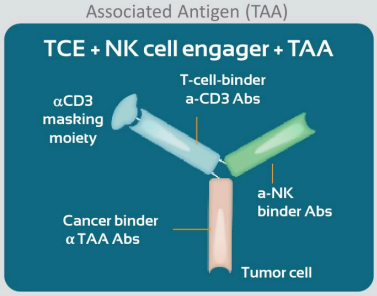
*Synnott et al., Cancer, 2025 https://acsjournals.onlinelibrary.wiley.com/doi/full/10.1002/cncr.70069

CAPTN-3: A Tri-specific Platform Designed for Safer, Broader Tumor Killing

Solid tumors meet tri-specific antibodies:
CAPTN-3 activates immune cells selectively at tumors combining safety with potent anti-tumor activity

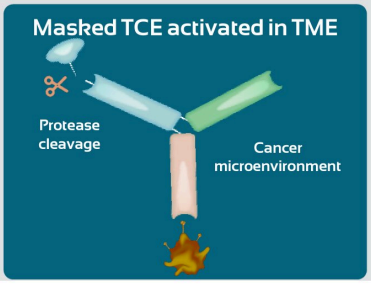
STEP 1: SAFE-INACTIVE IN BLOOD

TCE is "capped" —not triggering immune activation-while targeting the Tumor Associated Antigen (TAA)



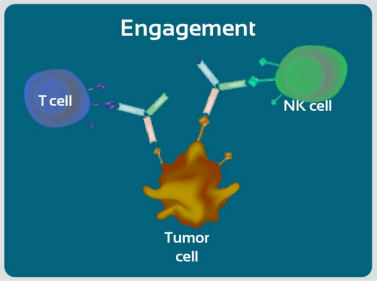
STEP 2: ACTIVATED AT TUMOR

Tumor enzymes remove cap, activating drug precisely where needed



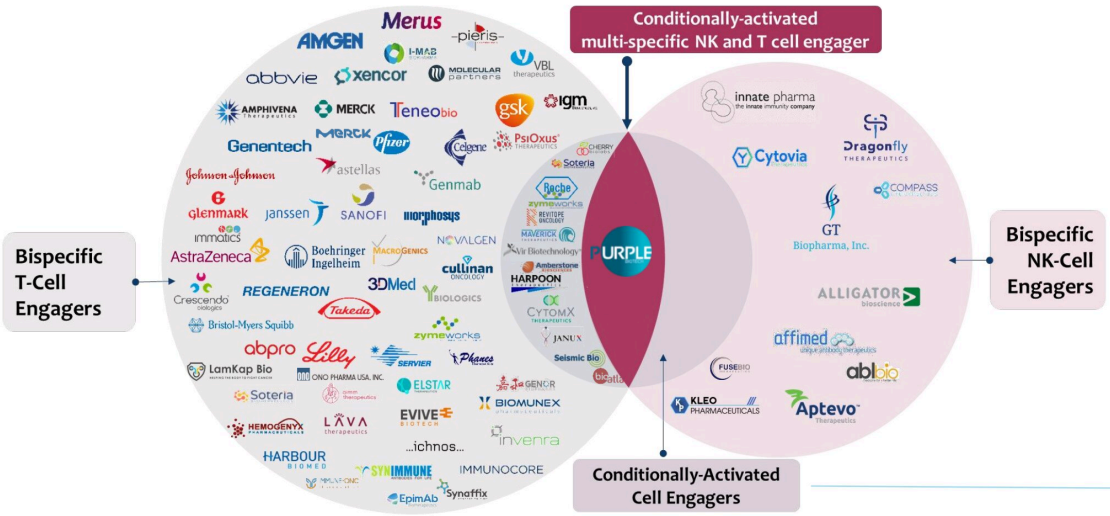
STEP 3: DUAL IMMUNE ATTACK

Engages both T cells AND NK cells for roader killing while binding to the TAA



IM1240, our lead program, demonstrates improved safety at doses 300x higher than the non-capped variant with potent anti-tumor activity across preclinical studies

CAPTN-3: Built to Stand Out
Enhanced Efficacy with Improved Safety



astellas
Vir Biotechnology
- PSMA **masked** TCE
- Phase 1
- **$1.7b** deal size

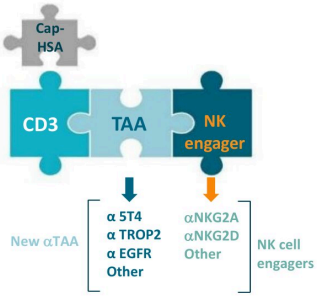
Bristol Myers Squibb
Janux
- nondisclosed **masked** TCE
- Preclinical
- **$850M** deal size

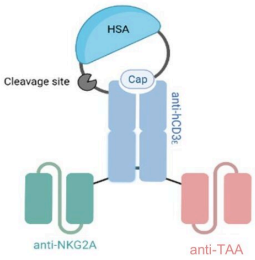
abbvie
xilio THERAPEUTICS
- **Masked** TCE
- Preclinical
- **$2.1b** deal size

PURPLE

*Based on publicly available information

Plug & Play Tri-specific Scaffold: Differentiated by Capped TCE with an NK Cell Activating Arm





Modular TCE architecture

- Integrates multiple TAAs or NK-activating arms
- Robust pipeline of novel candidates
- Multiple development and partnership opportunities

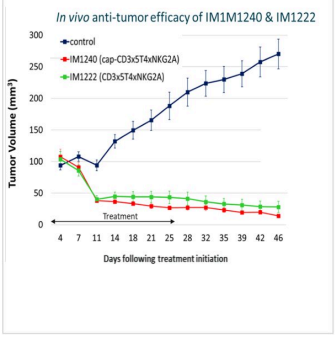
Differentiated TCE Platform

- **Dual immune activation** (T cells and NK cells) drives synergistic efficacy
- **Activates highly cytotoxic T-cell subsets** ($CD8^+$, γδ2), further enhanced by checkpoint modulation (NKG2A/HLA-E axis)
- **Conditionally activated design** delivers superior safety with enhanced antitumor activity
- **Enhanced pharmacokinetics and stability** via proprietary capping design and **human serum albumin (HSA)**
- Tumor-selective activation via **multi-protease**–mediated cap cleavage; **non-capped TAA design** enables efficient targeting across multiple tumor types and optimal immune engagement

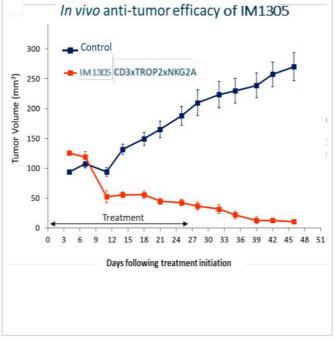
Plug & Play Platform Capabilities *In-Vivo* Validation

Pipeline and Platform Candidates Demonstrations

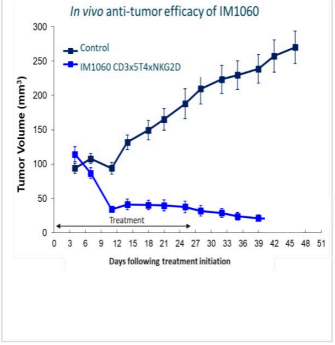
CD3x**5T4**xNKG2A



CD3x**TROP2**xNKG2A

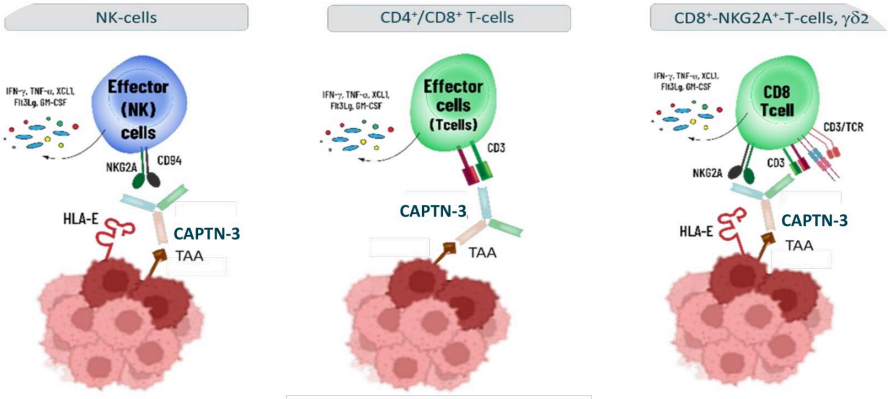


CD3x5T4x**NKG2D**



Sustained tumor regression across multiple tri-specifics targeting TAAs and NK receptors

Activating Both Innate and Adaptive Immune Responses



Activates NK cells by blocking the inhibitory NKG2A–HLA-E interaction

Stimulates both helper (CD4+) and killer (CD8+) effector T cells via CD3 targeting

Dual mechanism: turns on killer T cells and blocks an immune "off switch" to unleash a stronger attack on tumors

Multi MOA: Activation of T cells through CD3 engagement, NKG2A inhibition and NK cell activation. Dual role for NKG2A inhibition in unleashing both T-cell and NK-cell activity

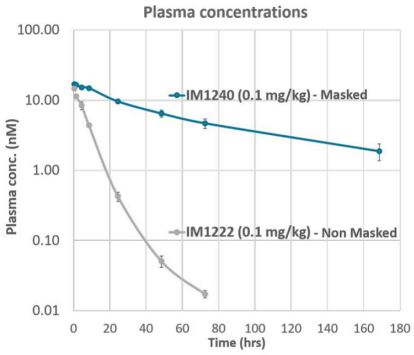
A CAPTN-3 Pipeline Dedicated to Advancing Oncology Therapies

PROGRAM	INDICATION	PRECLINCAL	PHASE 1	PHASE 2	COLLABORATOR	KEY 2026-27 MILESTONES
IM1240 Capped-CD3x**5T4**xNKG2A	Solid Tumors				Mount Sinai	TOX, IND, Ph 1 Initiation
IM1305 Capped-CD3x**TROP2**xNKG2A	Solid Tumors				Mount Sinai	

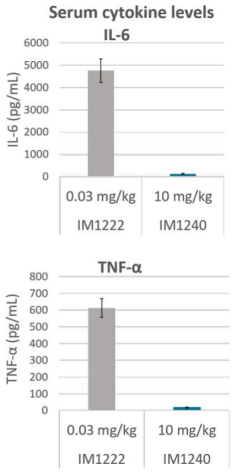
Major Inflection Point Expected After Proof-of-Concept Study within Two Years : Comparables are +$1B

IM1240 Non-Human Primate Data:

Masking extends half-life, increases exposure and reduces cytokine release at ~300-fold higher dose vs non capped



Pk parameter		IM1240 / IM1222 0.1 mg/mL
$T_{1/2}$	hrs	X 8
$AUC_{0-\infty}$	h·µg/mL	X 16
Clearance	mL/h/kg	X 0.07



Non-GLP dose range finding (DRF) study in NHP

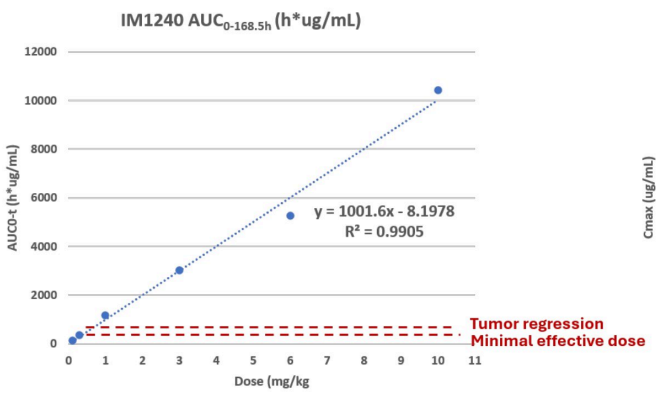
- **Safety**: Improved safety at doses up to 300-fold higher than a non-capped comparator (IM1222)
- **Pharmacokinetics**: Higher exposure and extended half life via masking technology with ~14-fold slower clearance than a non-capped comparator.
- **Dose flexibility**: Higher dosing without toxicity, potentially improving efficacy

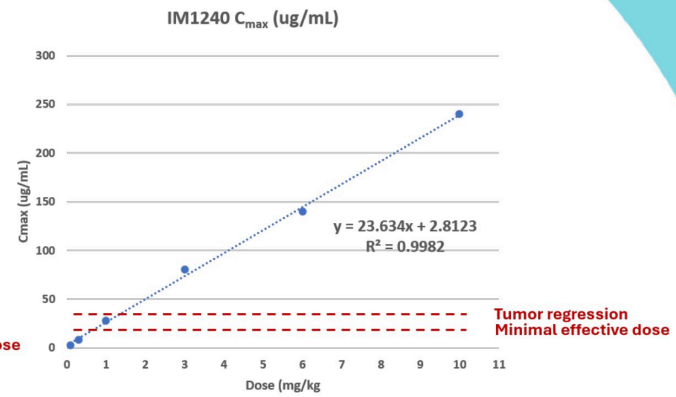
CAPTN-3 doesn't just improve safety — it unlocks doses unmasked TCEs cannot reach

GLP - Good laboratory Practice; DRF – Dose range finding; NHP – Non-human primates. Cytokine Release Syndrome - key safety concern with TCEs manifested by high IL-6 and TNF-α driving systemic inflammation

IM1240 Non-Human Primate Toxicology Data:

Dose-proportional pharmacokinetics and a broad therapeutic window





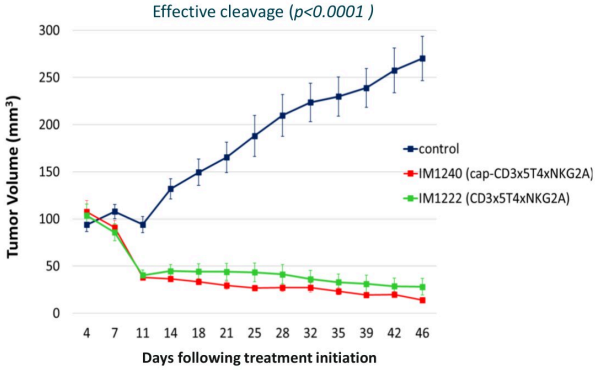
- IM1240 demonstrated linear, dose-proportional pharmacokinetics in NHPs, with systemic exposure and Cmax scaling predictably with dose
- Exposure levels associated with tumor regression and minimal effective doses in mouse model remained well below tolerated levels in NHPs, indicating a broad therapeutic window

IM1240: capped-CD3x5T4xNKG2A

In vivo efficacy and synergy of the CD3 and NKG2A arms

In vivo anti-tumor efficacy of IM1240 & IM1222*

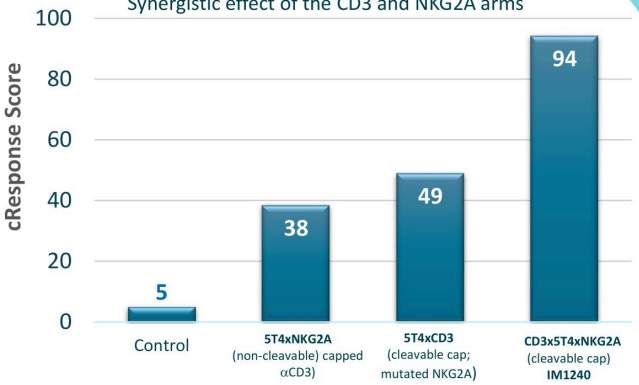
Effective cleavage (*p<0.0001)*



- IM1240 achieved tumor regression with no recurrence
- Comparable activity between capped IM1240 and non-capped IM1222 confirms efficient cap-cleavage *in vivo*

Patient-derived explants (PDE) of 5T4⁺NSCLC **

Synergistic effect of the CD3 and NKG2A arms



- Disabling either arm reduced tumor killing (cResponse 38–49); the full tri-specific IM1240 nearly doubled the response (cResponse 94)

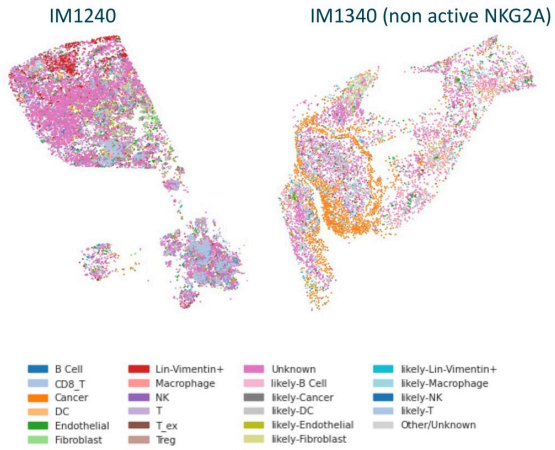
*TNBC MDA-MB-231 cells were s.c. inoculated 1:1 with PBMCs in IL-15 KI mice on day 0. Treatments: IV QOD 1mg/kg non-capped (IM1222) or equimolar dose 1.7 mg/kg capped (IM1240) till day 24, 8 mice per group, Follow up 20 days following treatment completion

**5T4⁺NCSLCand aPD1-resistant Patient-Derived Explant (PDE)* at 10nM concentration

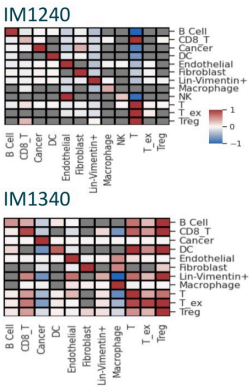
****Ex vivo patient-derived tumor explants (PDE) involve the culture of resected tumor fragments that retain the TME native architecture and immune cell array, tumor heterogeneity and the proliferative capacity (Golan 2023, Powley 2020). Fresh NSCLC patient-derived biopsy was cultured as 250um slices, treated for 96hr, fixed, and H&E slides were blindly scored for response based on cell viability and damage to the cancerous tissue according to pathological criteria. A scale of 0–100 was created with a score of 0 representing completely viable cancer tissue and a score of 100 representing no viable cancer cells. The analysis included functional (cell death) score of response (cResponse) using proprietary artificial intelligence (AI) algorithm (Curesponse).*

IM1240 Induces Mature TLS Formation and Drives Immune Activation NKG2A blockade is required for the effect

Spatial plot



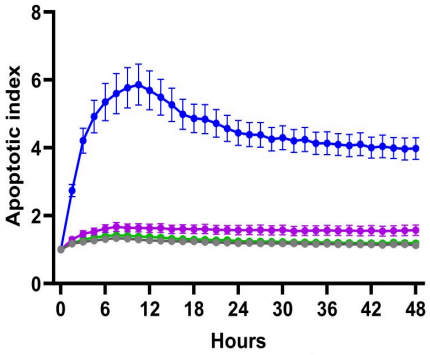
Interaction Analysis



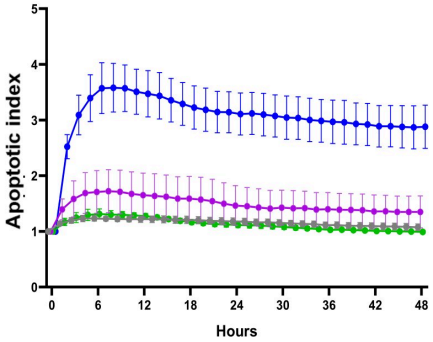
- **Tissue reorganization-** IM1240 treatment, compared with NKG2A loss-of-function variant IM1340, induced pronounced immune cell reorganization within the tissue.
- **TLS induction –** IM1240 induced mature tertiary lymphoid structures (TLS), which are associated with strong anti-tumor immunity and improved clinical outcomes
- **Immune-cell shift -** IM1240 increased CD8 T cells and NK cells while reducing Tregs and tumor cells, shifting the tumor microenvironment toward immune activation

IM1240 Induces Apoptosis in Patient-Derived Biopsies from PD-1 Resistant Tumors Both the CD3 and NKG2A arms are required for full activity

Neo.Adj. chemo+αPD1 resistant metastatic lymph node HNSCC (n=6)



Enfortumab vedotin/pembro Resistant Bladder Cancer (n=1)



IM1240 capped CD3X5T4XNKG2A 100nM
IM1340 NKG2A mutated 100nM
IM1242 non-cleavable cap 100nM
Medium

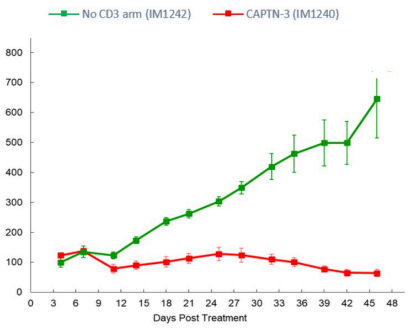
- **Activity in resistant tumors:** IM1240 induced apoptosis in patient-derived samples from PD1-resistlant metastatic HNSCC (n=6) and bladder cancer (n=1)
- **Both arms required:** Loss of function in either the CD3 or NKG2A diminished activity, confirming the trispecific design drives the effect

Neoadjuvant-treated biopsies/resections were dissociated and analyzed by Incucyte in 9 replicates. Y-axis shows arbitrary units as fold change.

Both CD3 and NKG2A Contribute to Anti-tumor Activity *In Vivo* (EACR 2025)

CD3 arm — primary driver

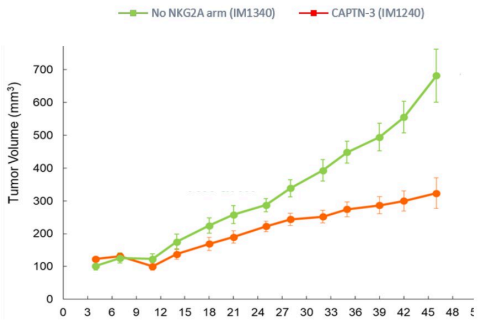
IM1240 vs. IM1242 (no CD3) · 1.5 mg/kg



p < 0.0001

NKG2A arm — significant benefit

IM1240 vs. IM1340 (no NKG2A) · 0.75 mg/kg

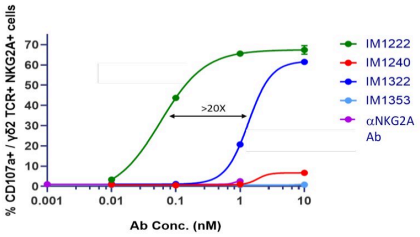


p = 0.002

CD3 engagement drives tumor killing | NKG2A blockade adds meaningful benefit, consistent with its co-expression in CAPTN-3 target tumors

Significant activation of NKG2A+γδ2 T cells and enhanced anti-cancer activity by CAPTN-3 NKG2A Blockade Amplifies activity

A. NKG2A+γδ2 T cell activation & contribution of NKG2A arm



IM1222 - CD3x5T4xNKG2A
EC_{50} (IM1222) ~ 60pM

IM1322 - CD3x5T4xNKG2Amutated
EC_{50} (IM1322) ~ 1350pM

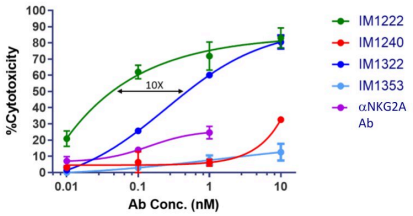
IM1353 CD3mutatedx5T4xNKG2A

IM1240 CappedCD3x5T4xNKG2A

Monalizumab – anti NKG2A

No activity

B. γδ2 T cell-mediated anti-cancer activity & contribution of NKG2A arm



IM1222 - CD3x5T4xNKG2A
EC_{50} (IM1222) ~ 70pM

IM1322 - CD3x5T4xNKG2Amutated
EC_{50} (IM1322) ~ 700pM

IM1353 CD3mutatedx5T4xNKG2A

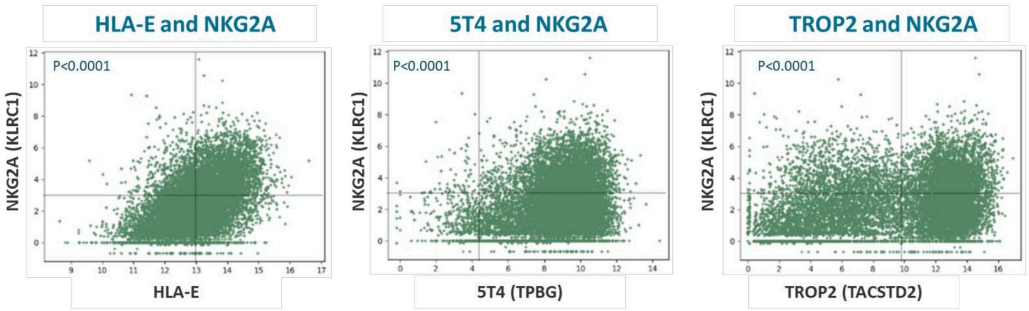
IM1240 CappedCD3x5T4xNKG2A

Monalizumab – anti NKG2A

- CD3 arm engagement is essential as **capped CD3 (IM1240) and CD3-mutated (IM1353) variants show no cytotoxic activity** in vitro
- The bispecific CD3x5T4 (NGK2A-mutated, IM1322) required 10-20x higher concentration to achieve comparable killing, demonstrating that **NKG2A arm blockade amplifies potency**
- Together, these data support a model in which **CD3 initiates the response and NGK2A substantially enhances the potency**

NKG2A is Relevant in the Targeted Patient Population

Transcriptomic analysis of ~11,000 primary human tumors (TCGA) (ESMO-IO 2025)



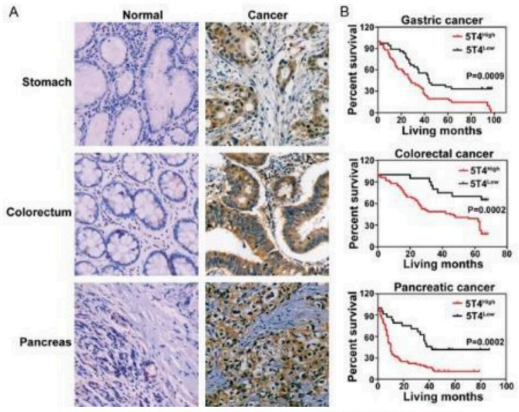
- The Axis is Active - NKG2A and its inhibitory partner HLA-E are co-expressed across human tumors
- NKG2A expression strongly correlated to 5T4– NKG2A arm will be active in CAPTN-3 target population
- NKG2A expression strongly correlated to TROP2– reinforcing CAPTN-3 relevance across indications

NKG2A correlation with IM1240 target tumors is confirming its relevance in the intended patient population

Work performed by Dr. Amir Horowitz, Associate Professor of Immunology and Immunotherapy, and Oncological Sciences, and a member of the Marc and Jennifer Lipschultz Precision Immunology Institute and The Tisch Cancer Institute, Icahn School of Medicine at Mount Sinai

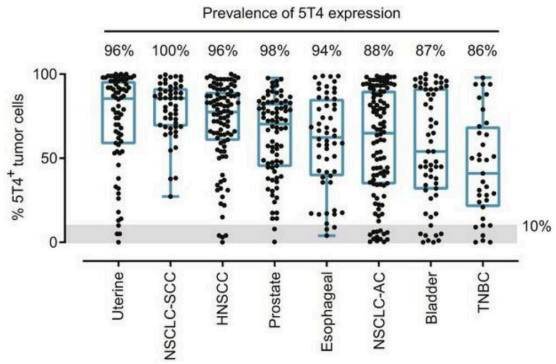
5T4: Sought-after Oncology Target Expressed on Solid Tumors

5T4 is highly expressed on common cancer types and relates to a poor prognosis



Am J Cancer Res 2018;8(4):610-623 www.ajcr.us /ISSN:2156-6976/ajcr0074519

5T4 is a Tumor Associated Antigen prevalent in several large indications



Kemper et al. LSA 2022;5:e202201481

Strong biological rationale

- Transmembrane glycoprotein, functions during **embryonic development** and **limited expression in normal** adult tissues[1]
- Associated with **cancer stem cells** → linked to relapse and resistance[2]

1.https://doi.org/10.1016/j.semcancer.2014.07.004 2.https://doi.org/10.1158/1078-0432.CCR-16-1834

5T4: A Novel TAA with Renewed Therapeutic Potential

Broad industry interest in targeting 5T4

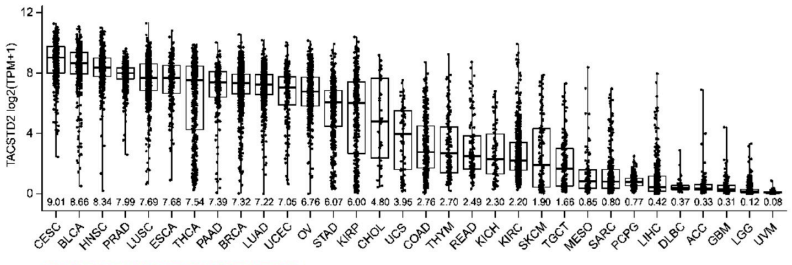
ADC			Bispecific Ab	TCE
TUB-030 Tubulis/ Gilead Phase 1/2	JK-06 Salubrious Early efficacy signals (ASCO 2026)	XB010 Exelixis phase 1	ALG.APV-527 Alligator phase 1	CBA-1535 Chiome phase 1

IM1240 first in class tri-specific T and NK cell engager targeting 5T4 – advantages:

- *Masked TCE* -> Tumor-selective activation, improved PK profile, reduces on-target/off-tumor toxicity and systemic T cell activation
- *NKG2A* -> overcomes immune suppression, addressed tumor variability (NK, T cells) -> increased rate of response
- Expanded therapeutic window

TROP2 is Broadly Expressed Across Solid Tumors

Supports its appeal as an oncology target

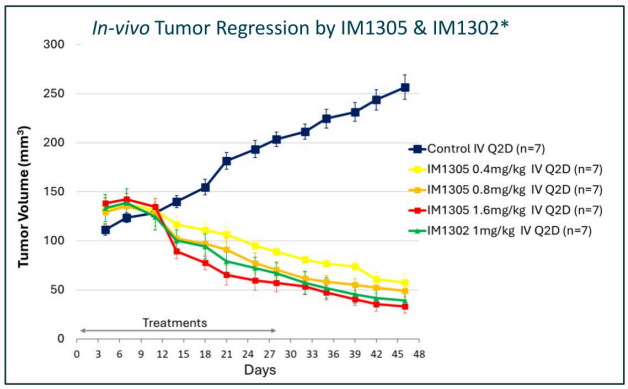


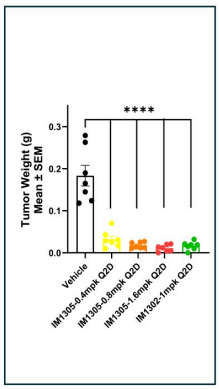
Kuo, Peiwen, et al. PloS one 20.4 (2025): e0321555

- Broadly expressed across multiple solid tumors
- Clinically validated ADC target

CESC, cervical squamous cell carcinoma and endocervical adenocarcinoma; BLCA, bladder urothelial carcinoma; PRAD, prostate adenocarcinoma; LUAD and LUSC, NSCLC; ESCA, esophageal carcinoma; THCA, thyroid carcinoma; PAAD, pancreatic adenocarcinoma; UCEC, uterine corpus endometrial carcinoma

IM1305: Dose-Dependent, Sustainable Efficacy
Capped CD3xTROP2xNKG2A *In vivo* Proof of Concept

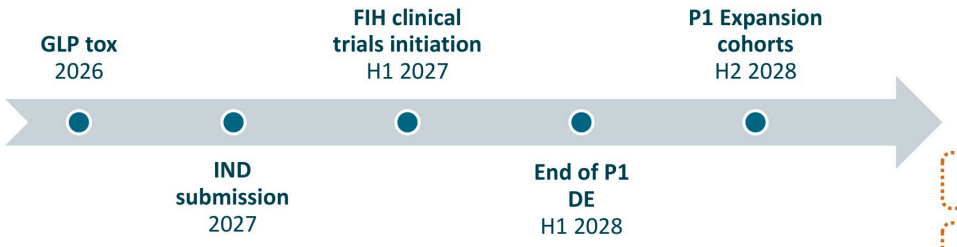




- Sustained tumor regression by IM1305. No tumor recurrence observed.
- Similar results were obtained with equimolar doses of the non-capped variant, IM1302, suggesting efficient cleavage of the cap in the TME.

*IM1302 is uncapped-CD3xTROP2xNKG2A. TNBC MDA-MB-231 cells were s.c. inoculated 1:1 with PBMCs on day 0 in IL15-KI BNDG mice (8 mice per group). 20 days following treatment completion tumor was removed and weighed (day 48). Statistics performed by 2-side Anova test.

Next Steps:
IM1240 and Other CAPTN-3 Antibodies and Beyond

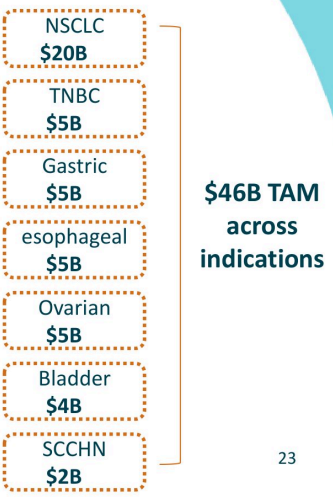


Planned IM1240 Phase 1/2 Study

- Basket trial P1 Dose escalation (**n~36)** in expected 5T4 expressing solid tumors (TNBC, esophageal, SCCHN, NSCLC, ovarian, gastric, bladder)
- Primary Objectives: Safety, tolerability and MTD
- 6 expansion cohorts (**n~127**) in selected indications to assess efficacy
- Pre-IND meeting completed with clear development roadmap

Preclinical Development Advancing with IM1305: capped-CD3xTROP2xNKG2A

One IM1240 molecule. Two immune arms. Seven indications. $46B opportunity.



T cell Engagers are Advancing: Solid Tumors Remain the Largest Unmet Need Purple Biotech: CAPTN-3 entering the clinic in 2027

$1B+

Comparable masked TCE deals closed at preclinical stage

300x

Higher dosing with minimal cytokine release

2027-2028

First-in-human trial initiation
Study results

Only tri-specific T and NK cell engager with conditional activation — built for solid tumors

FDA pre-IND completed— clear, de-risked path to the clinic

Modular platform with two candidates across complementary tumor targets

Snapshot: Company Highlights

1. **CAPTN-3: differentiated tri-specific platform activating both innate and adaptive immune systems**
   The only platform engaging both T cells AND NK cells while incorporating a masking technology

2. **Lead program expected to enter the clinic in 2027**
   IM1240 (cappedCD3x**5T4**xNKG2A), a first-in-class CAPTN-3 tri-specific antibody
   Second program, IM1305 (cappedCD3x**TROP2**xNKG2A)

3. **Safety and efficacy demonstrated in IND enabling Studies**
   Strong anti-tumor activity and 300× improved safety profile versus the non-masked comparator

4. **Focused on key inflection point and significant market potential to create value**
   Comparable masked TCE programs secured >$1B deals at preclinical stage.
   Key catalyst - results from P1 study starting from 1H 2028

5. **Solid tumors remain largely untreated by T cell engagers**
   A large unmet need with no approved tri-specific option

FINANCIALS

Cash
$6.4M
As of March 31, 2026

Runway
INTO H1 2027

Exchange
NASDAQ + TASE: PPBT

Leadership Team

Eric K. Rowinsky, MD
Chairman of the Board
Former CMO at ImClone, Stemline, Board member at Biogen Inc.

Gil Efron
Chief Executive Officer
Former Deputy CEO & CFO at Kamada (NASDAQ:KMDA)

Shai Lankry
Chief Financial Officer
Former CFO at Gamida Cell Ltd. (NASDAQ:GMDA)

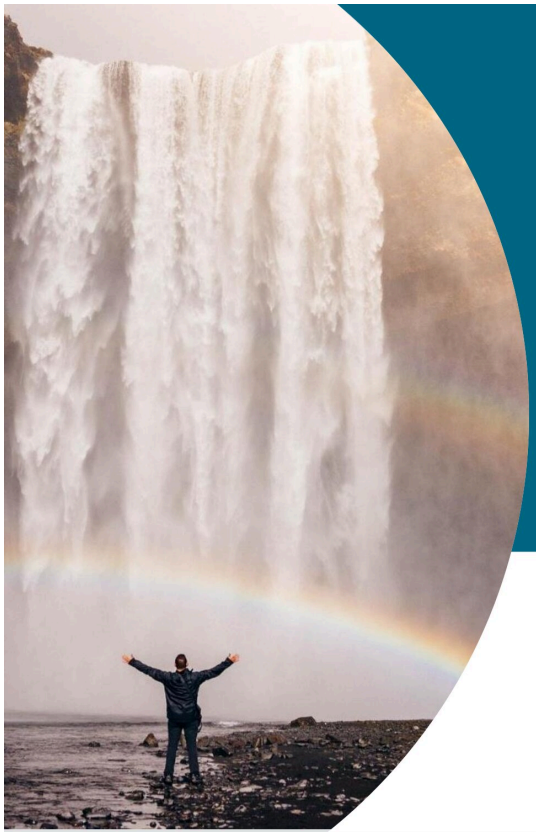
Michael Schickler, PhD
Head of Clinical and Regulatory Affairs
Formerly at Hoffmann-La Roche, CEO at CureTech

Hadas Reuveni, PhD
VP Research & Development
Formerly at Keryx (NASDAQ:KERX)

PURPLE BIOTECH

THANK YOU

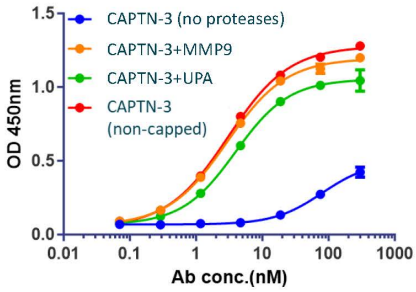
Contact Us:
ir@purple-biotech.com



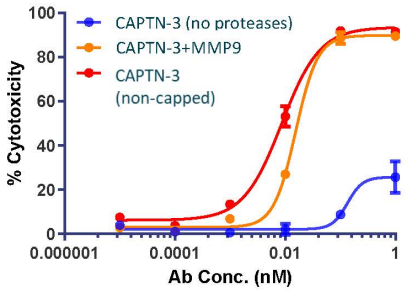
Appendix: CAPTN-3

CD3 Cap inhibits T cell binding and cleavage restores activity:
CAP advantages: improved safety, PK, and therapeutic window

ELISA CD3 binding assay
Binding of CAPTN-3 to CD3 is dependent on protease cleavage



PBMCs mediated cytotoxicity assay
Cytotoxic effect is restored following protease cleavage

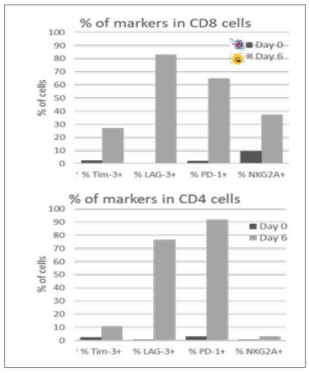


- Binding to CD3 and cytotoxic effect is retained following protease cleavage of the CD3 cap
- CAPTN-3 uses multiple TME-specific proteases – increasing likelihood for cleavage by broad-tumor types

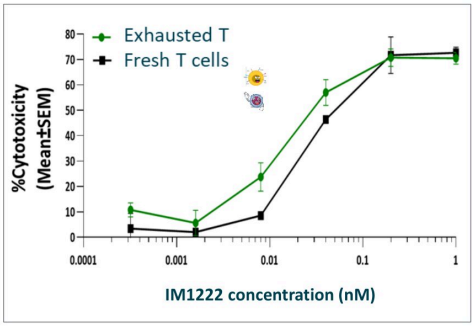
CAPTN-3 Induces Reinvigoration of Exhausted T cells

Added value of the NKG2A arm

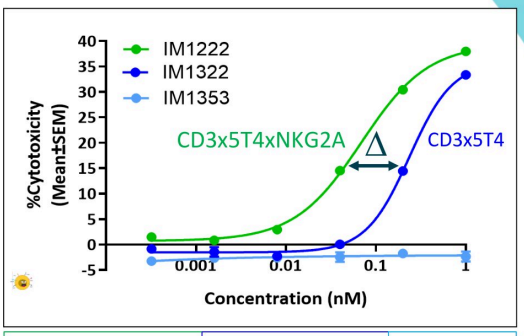
Production of exhausted T cells by repeated stimulation



IM1222 induced reinvigoration of exhausted T cells; similar effect to fresh T cells



Added value of the NKG2A arm

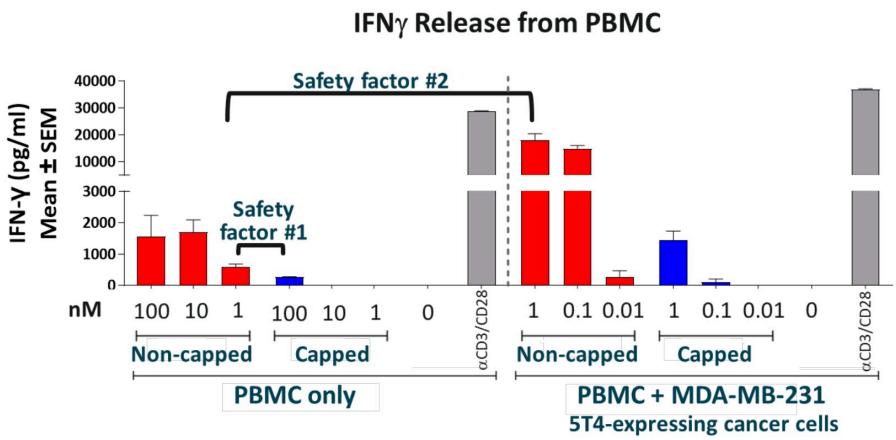


IM1222	IM1322	IM1353
CD3x5T4xNKG2A	CD3x5T4xNKG2Amutated	CD3mutatedx5T4x NKG2A
EC_{50} (IM1222) ~ 70pM	EC_{50} (IM1322) ~ 240pM	

NKG2A is often upregulated in exhausted $CD8^+$ T cells in the TME, and these cells often co-express other inhibitory receptors such as PD-1, LAG-3, or TIM-3. The above demonstrates:

- Upregulation of T cell exhaustion markers following repeated stimulation every 2 days with CD3/CD28 beads.
- Exhausted T cells co-cultured with TNBC MDA-MB-231 cells for 24hr with increasing concentrations of IM1222 showed a significant cytotoxic effect (pM range EC50), similar to fresh T cells.
- Synergistic effect of the arms is observed, demonstrating loss of efficacy by mutating each of the CD3 or NKG2A arms

Cytokine release is 5T4-dependent and suppressed by the cap for improved safety



Two safety factors through the design of the tri-specific Ab: the TCE capping and the advantage of its TAA arm:

- **Safety factor #1**: The capped variant vs the non capped, showed a decreased IFNγ release > 150-fold
- **Safety factor #2**: In the absence of 5T4-expressing cancer cells, the non-capped variant showed a decreased IFNγ release ~50-fold

CM24: an α-CEACAM1* mAb

Significant opportunity in multiple large indications with unmet medical need

Clinical POC achieved in PDAC

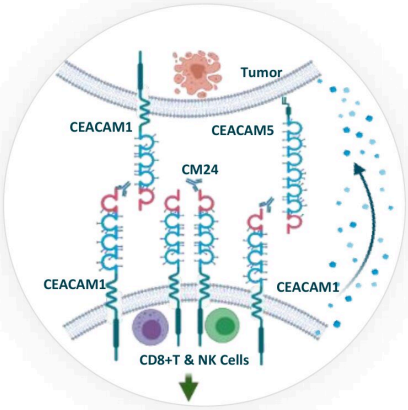
*Carcinoembryonic Antigen Cell Adhesion Molecule

CM24: Targeting CEACAM1 expressing tumors

Attractive new target	• CEACAM1 is **overexpressed** in >90% of colon, pancreatic and bladder cancers and in >70% in lung, gastric and biliary tract cancers • CEACAM1 is a part of the **Neutrophil Extracellular Traps (NETs) structure**
Demonstrated mechanism of action	• CM24 increases **T cell and NK cell-mediated cytotoxicity** against tumors • CM24 **binds** to CEACAM1 on NETs and **inhibits NET-related activities** • CM24 shows benefits in combination with immuno-oncology treatments
PoC Clinical efficacy	• 19% reduction in risk of death (HR=0.81) and 25% reduction in the risk of progression or death (HR=0.75) and 25% ORR, in **metastatic PDAC** as second-line treatment • **Potential biomarkers:** Serum CEACAM1 and MPO, CEACAM1^{+} tumor cells and CPS
Sizable market potential	• **Large opportunities** to leverage the MoA in multiple indications (lung, colon, GI etc.) • Significant unmet medical need in pancreatic ductal adenocarcinoma (PDAC), the most common form of pancreatic cancer

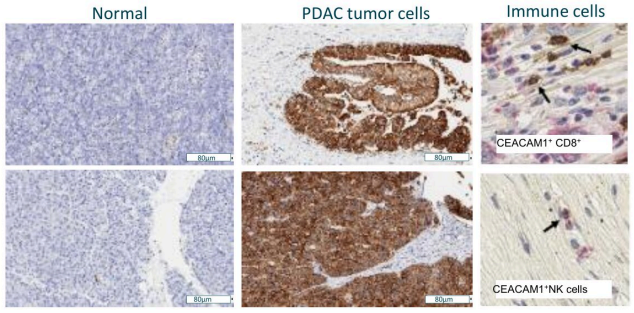
CM24: Immune modulation of CEACAM1 expressing TMEs

Activating the immune response

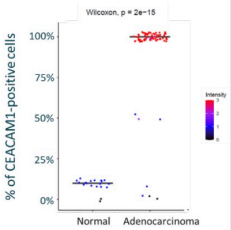


Making the tumor visible

CEACAM1 expression on tumor and tumor-infiltrating immune cells in PDAC



Representative examples of CEACAM1 immunohistochemical images of pancreatic adenocarcinoma and normal tissues



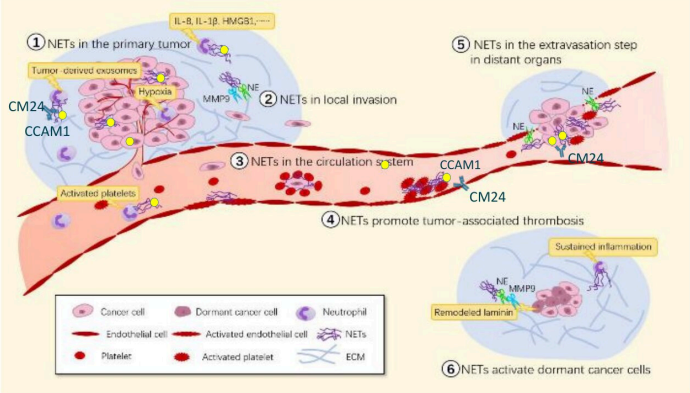
CEACAM1 scoring in PDAC TMA vs normal tissues

TME- Tumor Microenvironment

*Markel et al, J Immunol 2002, 2006; Immunology, 2008; Cancer Immunol Immunother 2010; Ortenberg et al, Mol Cancer Ther 2012; Zhou, 2009; Li, 2013; Huang, 2015; Acharya N, et al. J Immunotherapy Canc 8:e911-22, 2020.; Gerstel, D. et al. CEACAM1 creates a pro-angiogenic tumor microenvironment that supports tumor vessel maturation. Oncogene 30, 4275–4288 (2011), *Beauchemin N, Cancer Metastasis Rev 32, 643-671 (2013)*

CM24 blocks NET-related activities by CEACAM1 inhibition

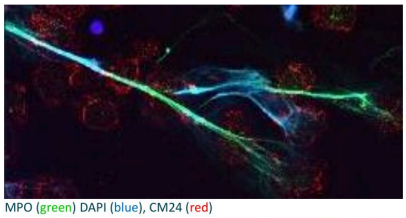
CEACAM1 is a part of the **Neutrophil extracellular traps (NET)** structure



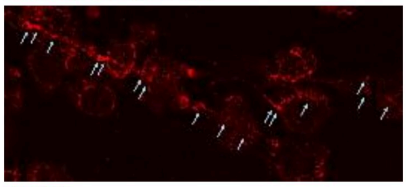
NETs are web-like structures involved in:

- Tumor immune evasion
- Tumor progression
- Metastasis
- Cancer-associated thrombosis

CM24 binds to CEACAM1 on NETs



MPO (green) DAPI (blue), CM24 (red)



CM24 (red)

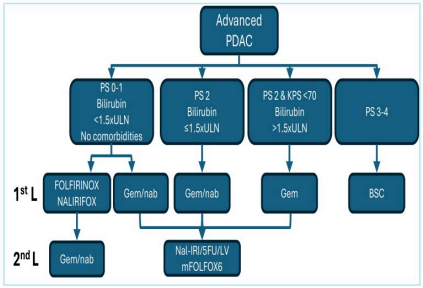
(adapted from 'Chen, Q et al. Cancers 2021, 13, 2832'); *Rayes RF, et al. Neutrophil Extracellular Trap-Associated CEACAM1 as a Putative Therapeutic Target to Prevent Metastatic Progression of Colon Carcinoma. J Immunol. 2020; NETs Primed intercellular communication in cancer progression as a promising therapeutic target [Shang et al. Biomarker Research (2023) 11:24]*

Large market opportunity based on CEACAM1 expression in PDAC

Key PDAC Statistics

- ~60K new cases/year in the US[1]; 140K new cases in EU in 2023
- 5-year relative survival rate of 12%[1]
- 2nd line chemo has 5-year overall survival rate of 3%[1]
- 2nd line Overall Survival of 6 to 8 months[2,3,4]

Limited Treatment Options



pan-RAS inhibitor advancing in Phase 3 as second-line treatment

CM24 Opportunity

- CEACAM1 expression correlates with poor prognosis in pancreatic cancer[5]
- Clinical and preclinical data support targeting patients based on CEACAM1 levels
- Synergy of CM24 with currently marketed IO therapies

Market opportunity in additional indications based on CEACAM1 expression

1.https://seer.cancer.gov/statfacts/html/pancreas.html , 2. De Jesus VHF, Camandaroba MPG, Calsavara VF, Riechelmann RP. 3. Systematic review and meta-analysis of gemcitabine-based chemotherapy after FOLFIRINOX in advanced pancreatic cancer. Therapeutic Advances in Medical Oncology. 2020;12. doi:10.1177/1758835920905408 4. Wang-Gillam A, Hubner RA, Siveke JT, et al. NAPOLI-1 phase 3 study of liposomal irinotecan in metastatic pancreatic cancer: Final overall survival analysis and characteristics of long-term survivors. Eur J Cancer. 2019;108:78-87. doi:10.1016/j.ejca.2018.12.007 .5. Calinescu et al, Journal of Immunology Research 2018: 7169081; Carcinoembryonic antigen-related cell adhesion molecules (CEACAM) 1, 5 and 6 as biomarkers in pancreatic cancer, DOI:10.1371/journal.pone.0113023

Completed Randomized Phase 2 Combination Study (NCT04731467)

A randomized Bayesian study of CM24 in combination with nivolumab plus SoC chemotherapy in 2nd line PDAC patients*

Primary endpoint : OS

Secondary endpoints: PFS, ORR, DCR

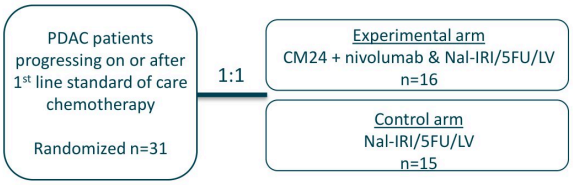
OS rate @ 6 & 12 months, PFS rate @ 3 & 6 months

Exploratory biomarkers:

Serum & tumor CEACAM1,

NET marker (Myeloperoxidase-MPO),

PDL1



Demographics and patient characteristics (ITT)

	Nal-IRI/5FU/LV	
Characteristic	Experimental (n=16)	Control (n=15)
Age (median)	66.0	68.0
Male (n, %)	10 (62.5)	8 (53.3)
Female (n, %)	6 (37.5)	7 (46.7)
Race/ white (n, %)	15 (93.8)	14 (93.3)
BMI (median)	23.4	23.1
ECOG (n, %) 0	5 (31.3)	3 (20.0)
1	11 (68.8)	12 (80.0)
Time from initial diagnosis (median, m)	17.8	17.6
Time from most recent disease progression (median, m)	1.0	1.0
BOR CR/PR to prior line (%)	6.3	33.3
BOR SD to prior line (%)	37.5	26.7
BOR CR/PR/SD to prior line (%)	43.8	60.0
Tumor M1 stage at study entry: N (%)	14 (87.5)	14 (93.3)
Pancreaticoduodenectomy	0 (0.0)	1 (6.7)

Safety- well tolerated

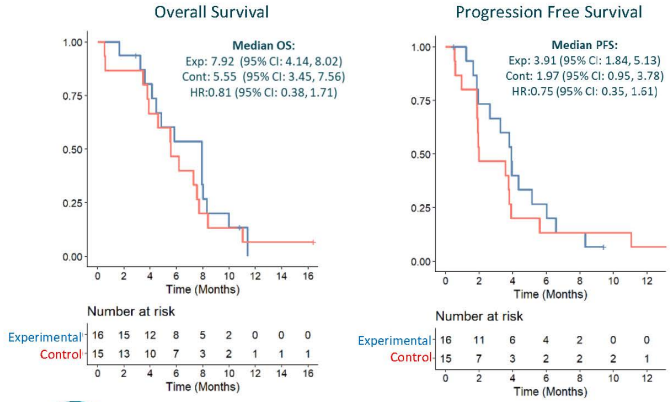
Grade ≥3 TEAE	Nal-IRI/5FU/LV	
	Experimental (n=16) N (%)	Control (n=15) N (%)
Neutropenia	2 (12.5)	0 (0.0)
Diarrhea	4 (25.0)	1 (6.7)
Fatigue	2 (12.5)	0 (0.0)
Anaemia	0 (0.0)	0 (0.0)
Nausea	1 (6.3)	0 (0.0)
Vomiting	1 (6.3)	0 (0.0)
Thrombocytopenia	0 (0.0)	0 (0.0)
White blood cell count decreased	0 (0.0)	0 (0.0)

*The other part of the study with gemcitabine/nab-paclitaxel regimen (n=32) was affected by informative censoring hence non interpretable

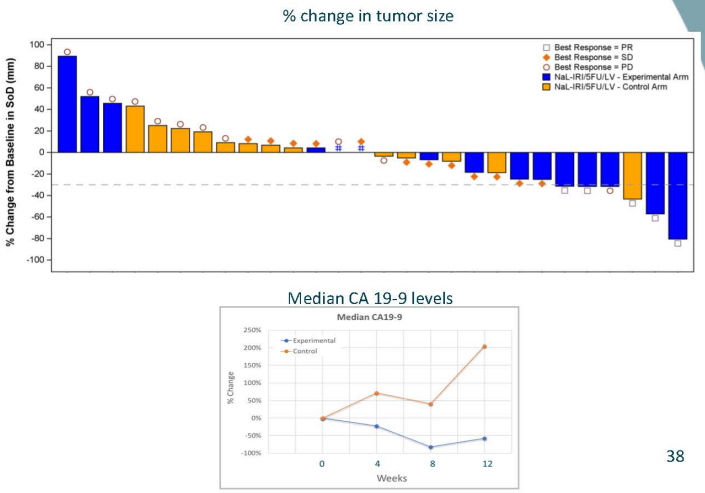
Phase 2 Efficacy

Proof of Concept demonstrated: Consistent efficacy across all endpoints

- **19% reduction** in risk of death **(HR=0.81**) and **25% reduction** in the risk of progression or death (**HR=0.75**)
- **Prolongation of 2.3** months in median overall survival **and 1.9** months in median progression-free survival



- **Higher** objective response rate (**ORR) (25% vs 6.7%**)
- **Higher** disease control rate (**DCR) (62.5% vs 46.7%)**
- Consistent and continuous **decrease of CA19-9**

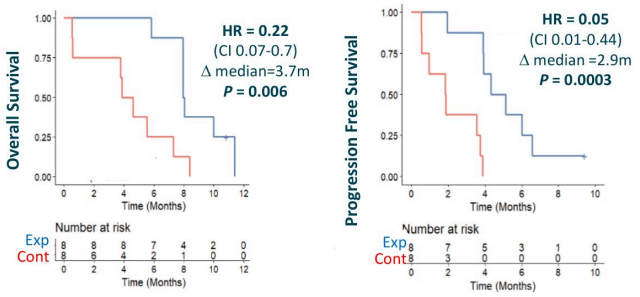


Phase 2 Biomarkers analysis in serum or tumor

Significantly improved OS, PFS and ORR

Serum/tumor CEACAM1 enriched patients

CEACAM1+Tumor cell H score 115-275 or Serum CEACAM1 6K-15K pg/mL

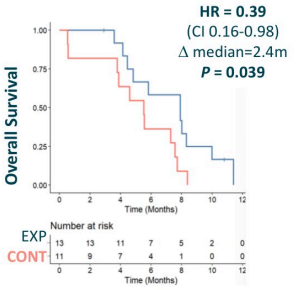


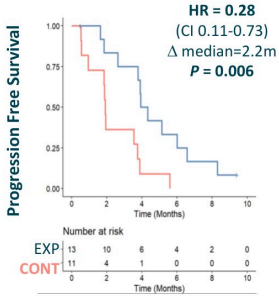
Statistically significant results in patients with pre-treatment **serum CEACAM1 levels (6K-15K pg/mL) or tumor CEACAM1 expression (H-score 115-275)**

- **78% reduction** in risk of death (**HR 0.22**, p 0.006) and **95% reduction** in the risk of progression or death (**HR 0.05**, p 0.0003)
- **Prolongation of 3.7 months** in median overall survival **and 2.9 months** in median progression-free survival
- **Higher** objective response rate (**ORR) (37.5% vs 0%**)

serum CEACAM1 or MPO enriched patients

6K < sCCAM1 < 15K pg/mL or 200 < MPO < 600 ng/mL





Statistically significant results in patients with baseline **serum CEACAM1 or serum MPO levels representing 80% of patients in the study:**

- **61% reduction** in risk of death (**HR = 0.39,** p 0.039) and **72% reduction** in the risk of progression or death (**HR=0.28,** p 0.006)
- **Prolongation of 2.4 months** in median overall survival and **2.2 months** in median progression-free survival
- **Higher** objective response rate (**ORR) (30.7% vs 0%**)

NT219: Small Molecule Degrader of IRS 1/2 and Blocker of STAT3

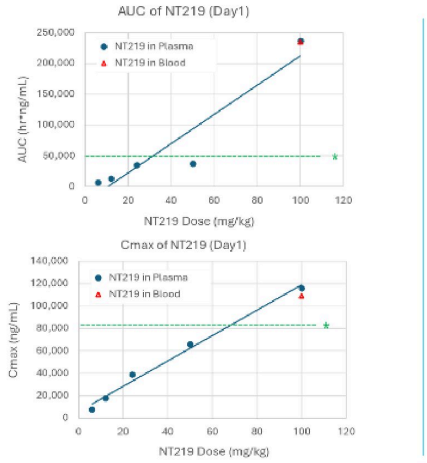
Recurrent/Metastatic Head & Neck Cancer (R/M SCCHN)

NT219: Novel IRS1/2 and STAT3 dual inhibitor for multiple indications

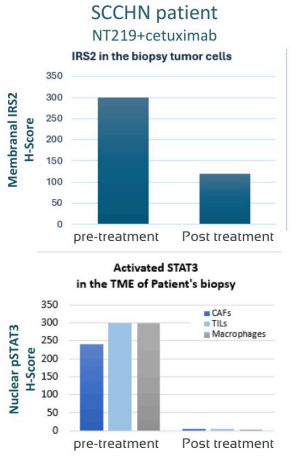
Innovative MOA	• **Covalently** binds to Insulin Receptor Substrate **IRS1/2** and leads to their **degradation** • Dual inhibitor of **STAT3 & IRS1/2**, both required to overcome drug resistance • Affects both the **tumor and the TME** • Suppresses **cancer stem cells**
Robust preclinical package	• **Outstanding efficacy** in various PDX models in monotherapy and in combination • Potential in **EGFRi, MAPKi and ICI resistant cancers**
Clinical Stage	• **No DLTs** in monotherapy or in combination • **Early clinical proof-of-mechanism** • Activated IGF1R and STAT3 identified as potential **predictive biomarkers** • **RP2D determined at 100 mg/kg, Phase 1 concluded. Phase 2 initiated**
Broad Market Potential	• Opportunity to **establish a Standard of Care** in 2L r/m SCCHN patients • **Multiple market upsides** in combination with approved cancer treatments • **NT219 is the only IRS degrader available** for clinical investigation

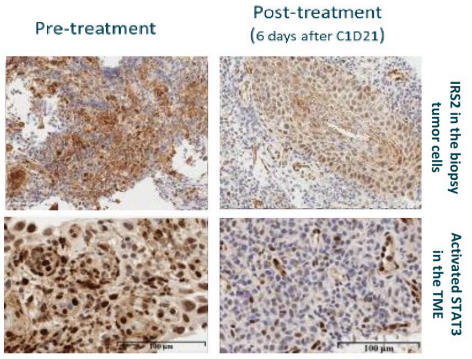
Dose escalation in combination with cetuximab:
Well tolerated, target exposure reached, responses observed

Dose-proportional increase in AUC and Cmax values



Target engagement demonstrated in patients' biopsies





- NT219 was well tolerated as monotherapy and in combination with cetuximab; No DLTs reported
- Human Equivalent Dose exposure was reached at 50 mg/kg
- RP2D determined at 100 mg/kg

NT219: Activity in mono and combo dose escalation

Monotherapy efficacy:

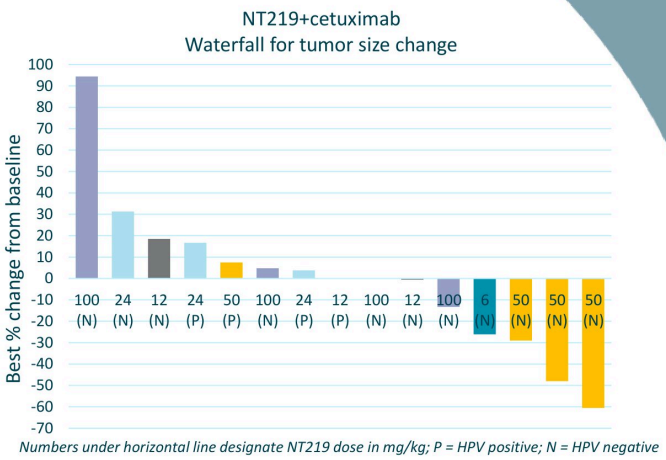
- 20 evaluable patients (all doses): 2 PR (confirmed-GEJ, unconfirmed-PDAC), 5 SD

Efficacy overview of combination arm in SCCHN patients:

- 16 evaluable patients (all doses 6, 12, 24, 50, 100 mg/kg)
- **Median follow-up of 9.4 months (95% CI: 3.4-10.0)**
- Out of 8 treated with active doses (50 and 100 mg/kg):
  - **2 confirmed PRs; 3 SD**
  - ORR:25%, DCR: 62.5%

Biomarker analysis at 50mg/kg dose of NT219 with cetuximab:

- Significant differences in the activated pIGF1R and pSTAT3 were revealed in the 2 responders (PR) compared to the 2 non-responders (PD), verified in the 100mg/kg cohort.

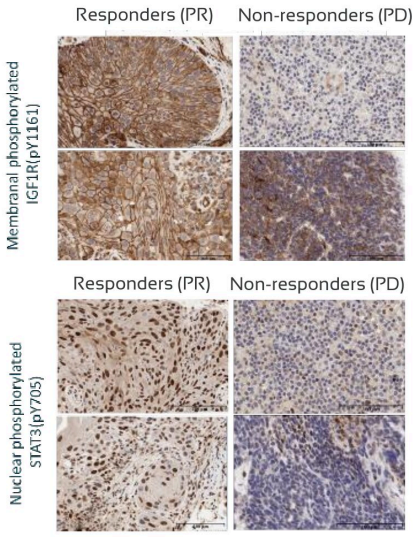


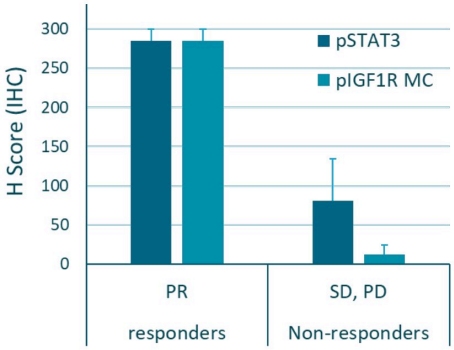
Numbers under horizontal line designate NT219 dose in mg/kg; P = HPV positive; N = HPV negative

Activated pIGF1R and STAT3, NT219 targets, as potential predictive biomarkers

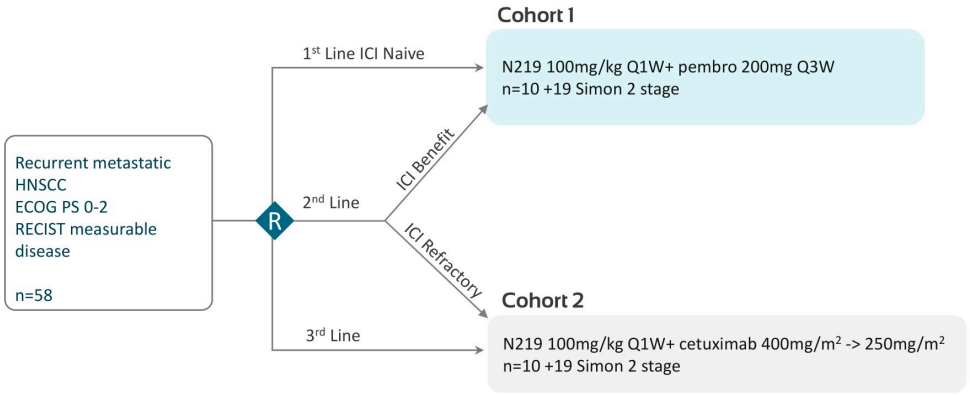
Biomarker analysis at the effective doses of NT219 (50&100mg/kg) with cetuximab:

- Significant differences in the pretreatment levels of activated pIGF1R and pSTAT3 were revealed in the responders (PR) compared to the non-responders (PD,SD)
- The findings at the 50mg/kg were verified at the 100mg/kg dosed patients





Phase 2: NT219 + pembro or cetuximab in patients with R/M HNSCC



Phase 2 study initiated June 2025

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