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 2024
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 ☐

On June 4, 2024, Purple Biotech Ltd. (the "Company" or the "Registrant") is announcing that it has made available an updated Company Presentation on its website. A copy of the updated Company Presentation is attached hereto as Exhibit 99.1 and may be viewed at the Company's website at www.purple-biotech.com.

Exhibit	
99.1	Purple Biotech Corporate Presentation June 2024

Incorporation by Reference

This Report on Form 6-K, including all exhibits attached hereto, is hereby incorporated by reference into each of the Registrant's Registration Statement on Form S-8 filed with the Securities and Exchange Commission on May 20, 2016 (Registration file number 333-211478), the Registrant's Registration Statement on Form S-8 filed with the Securities and Exchange Commission on June 6, 2017 (Registration file number 333-218538), the Registrant's Registration Statement on Form F-3, as amended, originally filed with the Securities and Exchange Commission on July 16, 2018 (Registration file number 333-226195), the Registrant's Registration Statement on Form S-8 filed with the Securities and Exchange Commission on March 28, 2019 (Registration file number 333-230584), the Registrant's Registration Statement on Form F-3 filed with the Securities and Exchange Commission on September 16, 2019 (Registration file number 333-233795), the Registrant's Registration Statement on Form F-1 filed with the Securities and Exchange Commission on December 27, 2019 (Registration file number 333-235729), the Registrant's Registration Statement on Form F-3 filed with the Securities and Exchange Commission on May 13, 2020 (Registration file number 333- 238229), the Registrant's Registration Statement on Form S-8 filed with the Securities and Exchange Commission on May 18, 2020 (Registration file number 333-238481), each of the Registrant's Registration Statements on Form F-3 filed with the Securities and Exchange Commission on July 10, 2020 (Registration file numbers 333-239807 and 333-233793), the Registrant's Registration Statement on Form S-8 filed with the Securities and Exchange Commission on April 4, 2022 (Registration file number 333-264107) and the Registrant's Registration Statement on Form F-3 filed with the Securities and Exchange Commission on March 23, 2023 (Registration file number 333-270769), the Registrant's Registration Statement on Form F-3, as amended, originally filed with the Securities and Exchange Commission on December 8, 2022 (Registration file number 333-268710) and the Registrant's Registration Statement on Form F-1 filed with the Securities and Exchange Commission on October 30, 2023 (Registration file number 333-275216), to be a part thereof from the date on which this report is submitted, to the extent not superseded by documents or reports subsequently filed or furnished.

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 4, 2024

PURPLE BIOTECH LTD.

By: */s/ Lior Fhima*
Lior Fhima
Chief Financial Officer

Exhibit 99.1


PURPLE
BIOTECH
CORPORATE PRESENTATION
NASDAQ/TASE: PPBT
June 2024

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, 2023 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.

Corporate highlights

Purple Biotech identifies promising first-in-class drug candidates to treat cancers with high unmet medical need

- Multiple data read-outs expected in 2024
- Two First-in-Class clinical stage drugs
- A preclinical tri-specific immuno-engagers platform
- Lean & global operation
- Cash runway into 1Q25

Purple Biotech (NASDAQ/TASE: PPBT)

As of March 31, 2024

- ADS Outstanding: 26.6 M
- Cash Balance: $10.8 M

Strong position to reach short and mid term value creating clinical data catalysts

A pipeline dedicated to advancing oncology therapies

Project	Target	Indications	Development Stage				Value Drivers
			Pre-Clinical	Phase I	Phase II	Phase III	
CM24	CEACAM1 mAb	Pancreatic Cancer (+nivolumab*+SoC)					❖ Phase 2 interim analysis 2H24 ❖ Phase 2 top line results 2H24
NT219	STAT3xIRS1/2 Dual Inhibitor	Solid tumors (monotherapy)					❖ Initiation of Phase 2 1H24
		Head and Neck & Colorectal Cancer (+Cetuximab)					
IM1240	CD3x5T4xNKG2A Tri-specific Ab	Solid Tumors					

*Clinical collaboration and supply agreement with: Bristol Myers Squibb

Multiple data read-outs expected in the next 12 months

Advancing First-in-Class Oncology Therapies

CM24: an α-CEACAM1* mAb

Lead indication: Pancreatic Ductal Adenocarcinoma (PDAC)

*Carcinoembryonic Antigen Cell Adhesion Molecule

CM24: a new multi-functional immune checkpoint inhibitor

Attractive new target

- CEACAM1 is **overexpressed** on certain **tumor cells and infiltrating immune cells**
- CEACAM1 interacts with **CEACAM1 and CEACAM5** and creates a **tumor-protective environment**

Demonstrated mechanism of action

- CM24 increases **T cell and NK cells cytotoxicity** against tumors
- CM24 shows benefit in combination with immuno-oncology treatments
- CM24 **blocks adhesion** of tumor cells to Neutrophil Extra cellular Traps (NETs)

Signals of clinical efficacy

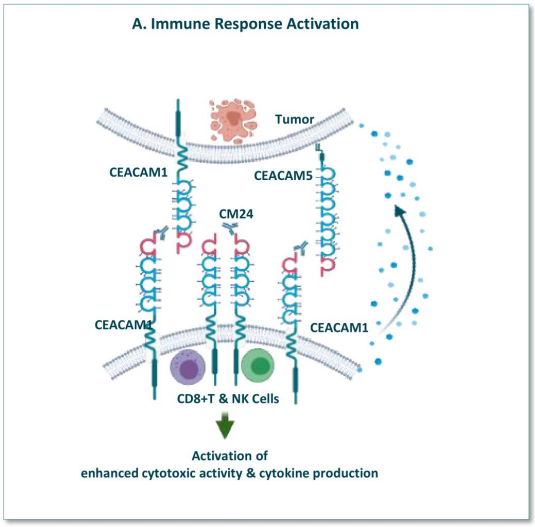
- **Favorable safety profile** in monotherapy and in combination with nivolumab
- **Interim P2** data from the Nal-IRI/5FU/LV sub-study demonstrate a **reduced risk of death or progression, prolongation** of **OS** and **PFS, supported by higher ORR, DCR** and **decreasing CA19-9**
- Potential **biomarkers identified** such as NETs and CEACAM1 levels on TILs
- **Randomized Phase 2 is ongoing, last patient enrolled in Dec-23, top line data 2H24**

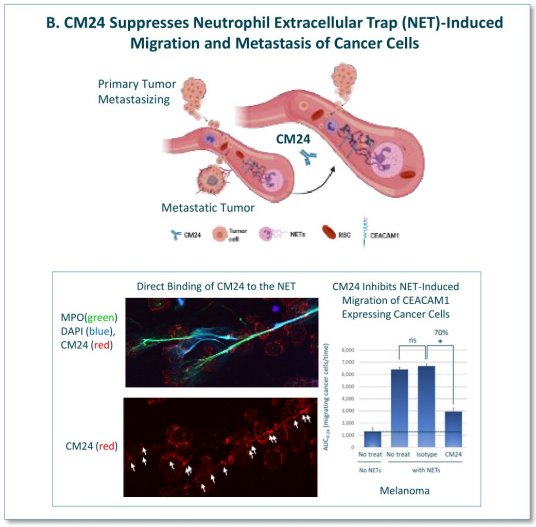
Sizable market potential

- Significant unmet medical need in pancreatic ductal adenocarcinoma (PDAC), most common form of pancreatic cancer
- Strong **IP position** and well **ahead of competitors**
- **Multiple opportunities** to leverage the MoA in other clinical settings

PURPLE biotech

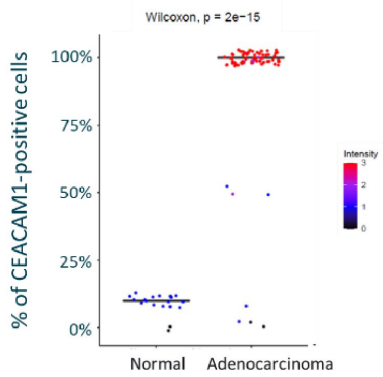
CM24 MOA
Immune check point inhibitor & anti-metastatic activity



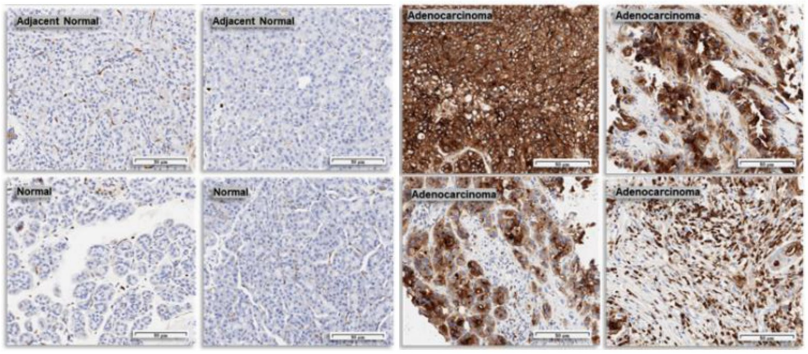


Markel et al, J Immunol 2002, 2006; Immunology, 2008; Cancer Immunol Immunother 2010; Ortenberg et al, Mol Cancer Ther 2012; Zhou, 2009; Li, 2013; Huang, 2015; Acharya N, et al. J Immunotherapy Canc 8:e911-22, 2020.; Rayes RF, et al. Neutrophil Extracellular Trap-Associated CEACAM1 as a Putative Therapeutic Target to Prevent Metastatic Progression of Colon Carcinoma. J Immunol. 2020; Gerstel, D. et al. CEACAM1 creates a pro-angiogenic tumor microenvironment that supports tumor vessel maturation. Oncogene 30, 4275–4288 (2011),

CEACAM1 is over-expressed in pancreatic cancer



Comparison between CEACAM1 staining intensity in pancreatic cancer (38 cases/76 cores) and normal (10 cases/20 cores) tissues



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

Large Market Opportunity in Pancreatic Cancer

- Pancreatic Cancer accounts for ~60K new cases/year in the US alone; with a 5-year relative survival rate of 12%[1]
- Immuno-oncology approaches have been limited to patients with high microsatellite instability (MSI-H) or high tumor mutational burden (TMB-H)
- 5-year overall survival rate with chemotherapy in 2nd line patients is 3%[1]
- **Two main interchangeable regimens are used worldwide in 2nd line:**
  - **Gemcitabine/Nab-paclitaxel[3]: OS 7.9 months, PFS 4.3 months (weighted average)**
  - **Irinotecan (or Nal-IRI)/5FU/LV[4]: OS 6.2 months, PFS 3.1 months**
- CEACAM1 expression correlates with poor prognosis in Pancreatic cancer[2]
- Preclinical data support significant synergy of CM24 with currently marketed immuno-oncology therapies;
- Combining nivolumab with CM24 in a clinical collaboration with Bristol Myers Squibb; **Purple Biotech retains all worldwide rights to CM24**

1. *https://seer.cancer.gov/statfacts/html/pancreas.html*
2. *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*
3. De Jesus VHF, Camandaroba MPG, Calsavara VF, Riechelmann RP. 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

Phase 2 Combination Study Design (NCT04731467)

A study of CM24 in combination with nivolumab plus chemotherapy in patients with PDAC in the 2nd line

The study is conducted in 18 centers in the **US, Spain & Israel**

Primary endpoint :

OS

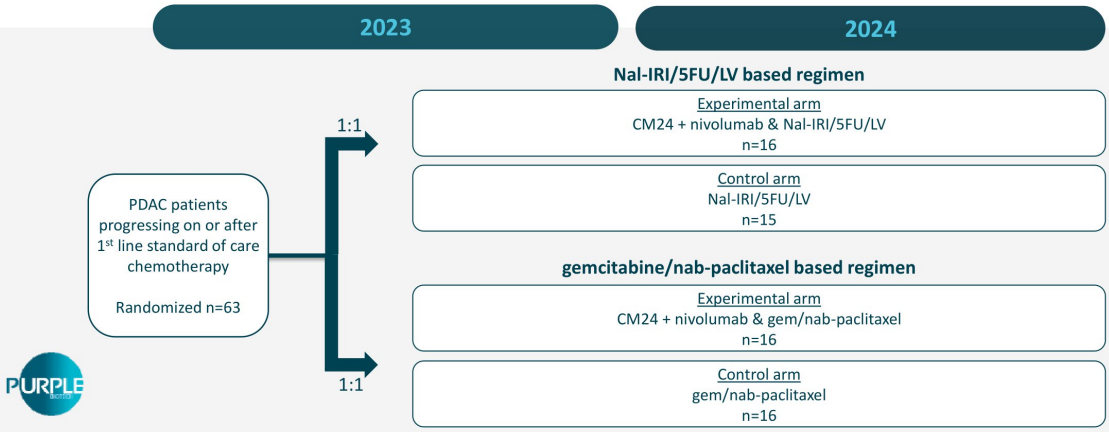
Secondary endpoints:

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

Additional Interim analysis: 2H24

Top line data: 2H24

Measurement of CEACAM1 and other bio-markers is ongoing



Phase 2 interim data (cut off date - May 8, 2024)

CM24+nivolumab+Nal/IRI/5FU/LV sub-study

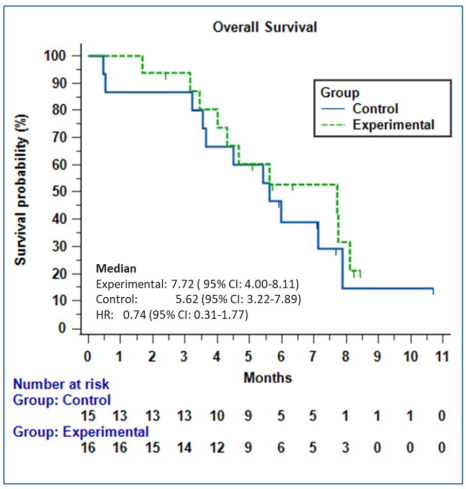
- 31 PDAC patients were randomized to the Nal-IRI/5FU/LV- based study regimen with a median follow up of 8.3 months.
- CM24+nivolumab+Nal/IRI/5FU/LV regimen was well tolerated
  - Most frequent Grade≥3: diarrhea (19%), fatigue (19%) and anemia (6%).
- An analysis of the gemcitabine/nab-paclitaxel study will be performed when the data is sufficiently mature

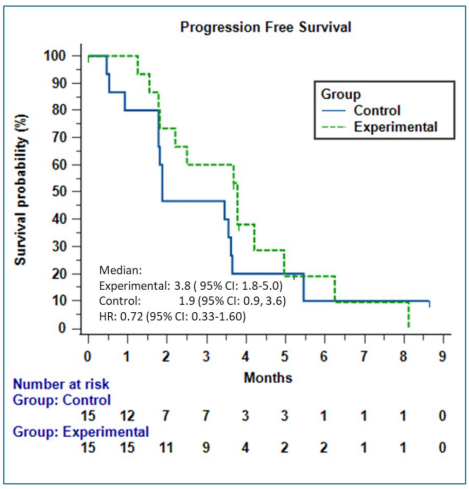
Characteristic		Experimental (n=16)	Control (n=15)
Age (median)		66	68
Age **≥65** (n, %)		8 (50.0)	4 (26.7)
Male (n, %)		10 (62.5)	8 (53.3)
Female (n, %)		6 (37.5)	7 (46.7)
Race/ white (n, %)		15 (93.8)	14 (93.3)
BMI		23.4	23.1
ECOG (n, %)	0	5 (31.3)	3 (20.0)
	1	11 (68.8)	12 (80.0)
Time from initial diagnosis (median, mo)		18	18
Time from most recent disease progression (median, mo)		0.94	0.89
CR/PR/SD to prior line (%)		43.8	60.0

Phase 2 interim data (cut off date - May 8, 2024)

CM24+nivolumab+Nal-IRI/5FU/LV sub-study

- **26% reduction in risk of death (HR=0.74) and 28% reduction in the risk of progression or death (HR=0.72)**
- **Prolongation of 2.1 months in median overall survival and 1.9 months in median progression-free survival**

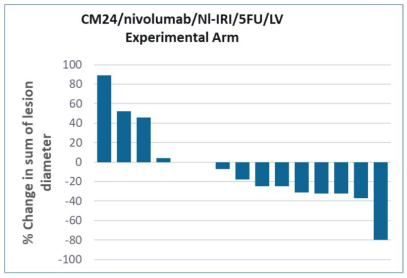


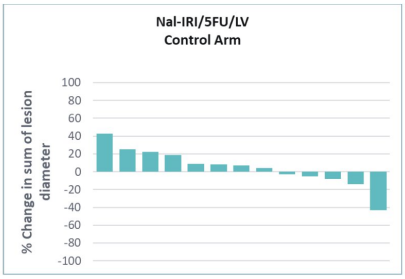


Phase 2 interim data (cut off date - May 8, 2024)
CM24+nivolumab+Nal-IRI/5FU/LV sub-study

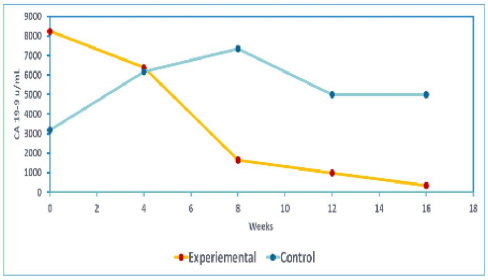
- **Higher objective response rate** (ORR) (25% vs 7%)
- **Higher disease control rate** (DCR) (63% vs 40%)
- **Consistent and continuous decrease in CA19-9** was observed

% change in tumor size





Median CA 19-9 levels



Phase 2 interim data (cut off date - May 8, 2024) Efficacy summary

Parameter	Experimental (n=16)	Control (n=15)
OS (mo, median; 95% CI)	7.72 (4.00-8.11)	5.62 (3.22 -7.89)
OS HR (95% CI)	0.74 (0.31-1.77)	
6 mo OS (%)	52.7	38.9
PFS (mo, median; 95% CI)	3.8 (1.8-5.0)	1.9 (0.9-3.6)
PFS HR (95% CI)	0.72 (0.33-1.60)	
3 mo PFS (%)	60.0	46.7
6 mo PFS (%)	19.0	10.0
ORR (%)	25.0	6.7
DCR (%)	62.5	40.0

Concordant and consistent improvement in the primary and all secondary endpoints

Advancing First-in-Class Oncology Therapies

NT219: A Small Molecule Dual Inhibitor of IRS 1/2 and STAT3

Lead indication: Recurrent/Metastatic Head & Neck Cancer (SCCHN)

NT219, a new solution to improve treatment outcome for cancer patients

Innovative MOA

- NT219 is a **First-in-Class**, small molecule dual inhibitor of **IRS1/2 and STAT3**
- **Covalently** binds to IRS1/2 and leads to their **degradation**
- 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
- Uniquely positioned to **tackle resistance** to cancer treatment such as **EGFRi, MAPKi and ICI**

Clinical Stage

- **No DLTs** in monotherapy or in combination
- **Early clinical activity demonstrated**
- **RP2D determined at 100 mg/kg, Phase 1 concluded. Phase 2 initiation 1H24**

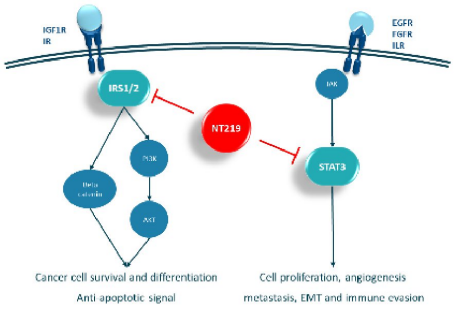
Broad Market Potential

- Opportunity to **establish a Standard of Care** in 2L r/m SCCHN patients
- **Multiple market upsides** in combination with major cancer treatments
- **NT219 is the only IRS inhibitor available** for clinical investigations

NT219 blocks 2 critical signalling pathways at once

IRS1/2

- Scaffold proteins, mediating mitogenic, metastatic, angiogenic and anti-apoptotic signals from IGF1R, IR, IL4R and other oncogenes, overexpressed in multiple tumors
- Regulates major survival pathways such as the PI3K/AKT, MEK/ERK and WNT/β-catenin
- Activated as a feedback response to anti-cancer therapies
- IRS plays an important role in promoting a tumor-protective microenvironment, by mediating upregulation of TAMs and CAFs



STAT3

- Well-established transcription factor associated with the tumorigenic phenotype
- STAT3 is broadly hyperactivated in many cancers, promoting proliferation, survival, angiogenesis and metastasis
- STAT3 pathway is required for TGFβ-induced EMT and cancer cell migration and invasion
- STAT3 is a critical player in tumor immune evasion, suppressing immune stimulators and enhancing immunosuppressive factors

Hadas Reuveni et al. Cancer Res 2013;73:4383-4394; Machado-Neto et al. Clinics 2018; 73,suppl 1 e566s; Naokazu Ibuki, Mazyar Ghaffari, Hadas Reuveni et al. Mol Cancer Ther. 2014; 13(12): 2827-2839; Rampias et al. Oncogene 2016; 35(20):2562-4; Flashner-Abramson, Reuveni Hadas, Levitzki Alexander et al. Oncogene 2016;35(20):2675-80; [6]Sanchez-Lopez et al. Oncogene 2016;35(20):2634-44. Zhao C et al. Trends Pharmacol Sci. 2016;37(1):47-6; Johnson, Daniel E et al. Nature reviews. Clinical oncology 2018; 15(4): 234-248. Zi Ying et al. J Cell Biochem. 2018;119:9419-9432.

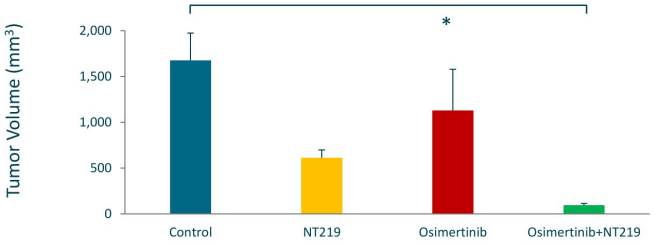
NT219 restores sensitivity to EGFRi in PDX models

Lung Cancer

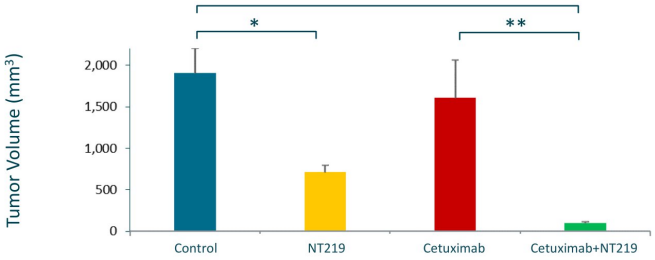
Non-small cell lung cancer (NSCLC)
Exon 19 deletion EGFR and T790M, biopsy of bone marrow metastasis, patient previously progressed on afatinib and osimertinib

Head & Neck Cancer

Recurrent/metastatic squamous cell carcinoma of the head and neck (R/M SCCHN)
metastasis, patient progressed on chemoradiation, several chemotherapies and pembrolizumab



Osimertinib 5 mg/kg, NT219 65 mg/kg, mean tumor volume at the end point, 3 mice/group;

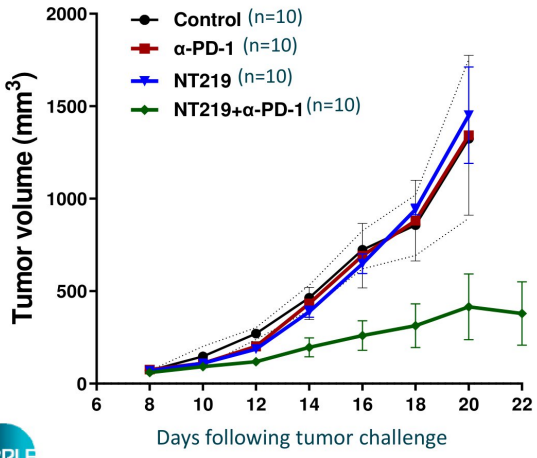


Treatments on days 0, 3 and 10, cetuximab - 1mg/mouse, 3 mice/group; PBMCs (1.4M cells/mouse) were injected on day 6

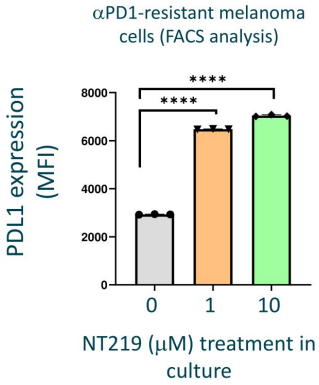
*** p<0.01, * p<0.02 based on one-way ANOVA with post hoc Tukey's HSD test*

NT219 re-sensitizes αPD1-refractory model

NT219+αPD1 reverse resistant tumors



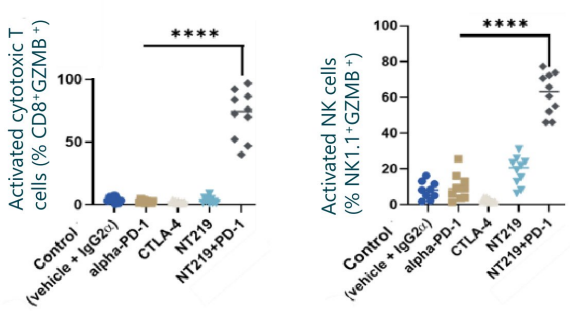
NT219 induces PDL1 expression



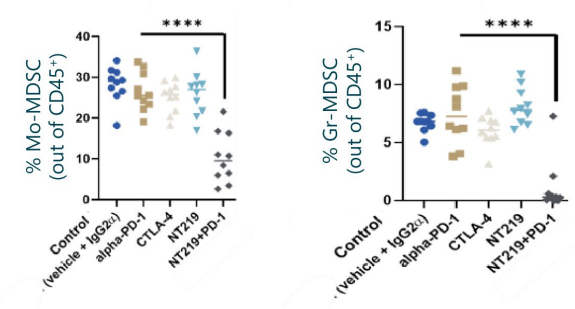
** Collaboration with Prof. Bareli and Prof. Curran, M.D. Anderson cancer center; presented at AACR 2023*

NT219 combination with αPD1 achieves a profound reprograming of the TME

NT219+αPD1 leads to a significant increase in cytotoxic effector cells (T & NK cells)



NT219+αPD1 leads to a significant reduction in myeloid derived suppressor cells (MDSC)



NT219 and αPD1 combination converted immuno-suppressive TME to immuno-reactive

** Collaboration with Prof. Bareli and Prof. Curran, M.D. Anderson cancer center, presented at AACR 2023*

First Market Opportunity

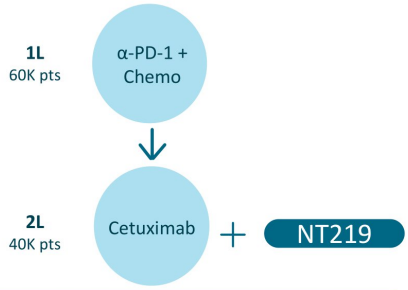
Recurrent or Metastatic Squamous Cell Carcinoma of Head and Neck (SCCHN)

Targeting the unmet medical need

- SCCHN is the 6th most common cancer type ; 175k new cases/year are expected by 2024
- 1L standard of care has shifted from chemotherapy towards immuno-oncology + chemotherapy
- < 20% of R/M SCCHN patients respond to Pembrolizumab
- Market size forecasted to >$5b in 2030

Rationale for combining Cetuximab + NT219

- EGFR and PD(L)-1 are the only clinically validated targets in SCCHN
- < 15% of R/M SCCHN patients respond to Cetuximab
- Cetuximab inhibits EGFR signaling and promotes ADCC in EGFR expressing tumors
- STAT3 and IRS-to-AKT activation contributes to resistance to cetuximab in SCCHN

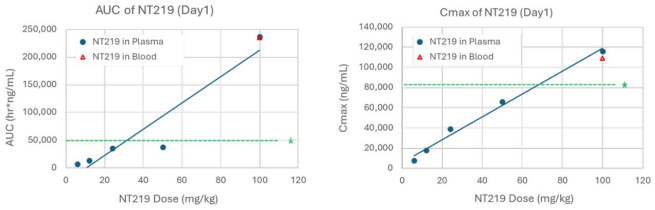


NT219 + Cetuximab has potential to become SOC as 2nd line therapy r/m SCCHN

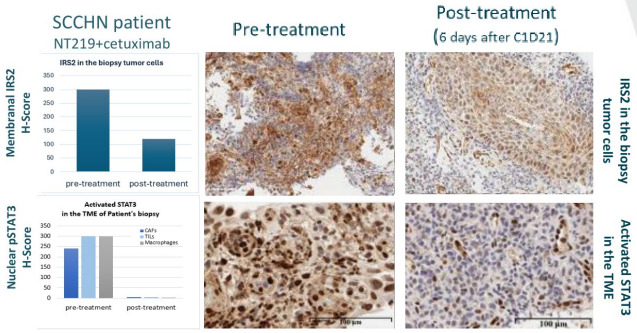
Global Data 2018: Head and Neck Squamous Cell Carcinoma: Opportunity Analysis and Forecasts to 2026 ; Internal best current estimates of patient numbers based on external research,8 major global territories

Phase 1 dose escalation in combination with cetuximab: Well tolerated, target exposure reached, patient's responses observed

- No DLTs reported, NT219 was well tolerated as monotherapy and in combination with cetuximab
- Dose-proportional increase in AUC and Cmax values
- Human Equivalent Dose exposure was reached at 50 mg/kg
- Target engagement demonstrated in patients' biopsies
- RP2D determined at 100 mg/kg



(*) Exposure & Cmax obtained at the effective dose in mice was observed at the ≥50mg/kg dose in human



Phase 1 Dose Escalation (cont.): Anti-tumor activity at target exposure level, 2 confirmed responses in SCCHN patients

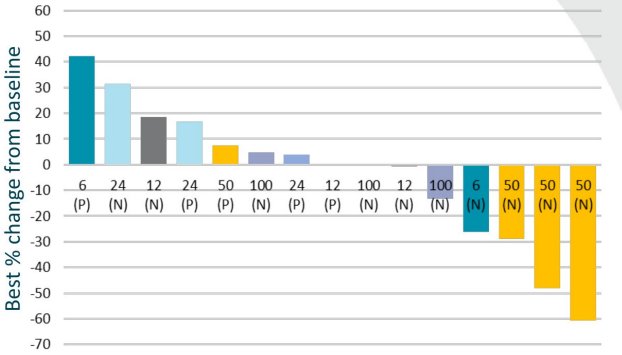
Efficacy overview of monotherapy arm:

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

Efficacy overview of combination arm in SCCHN patients*:

- 15 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 7 treated with 50&100 mg/kg:
  - **2 confirmed PR**
  - **3 SD**
  - ORR:29%, DCR: 71%

** Interim data analysis, cut-off date Jan 25, 2024*



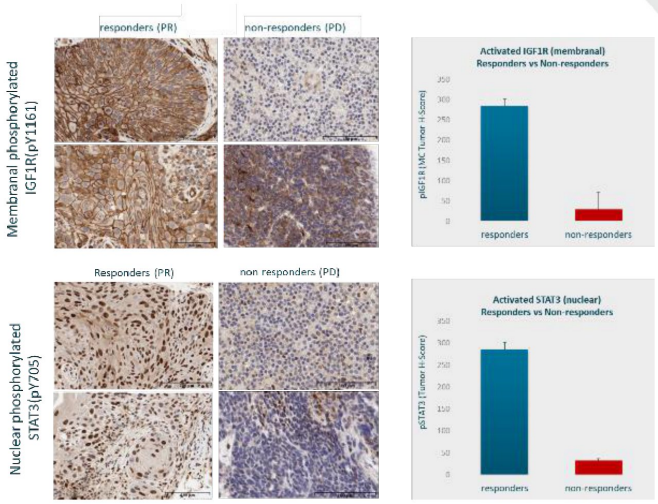
Numbers under horizontal line designate NT219 dose in mg/kg; P = HPV positive; N = HPV negative

In preparation of a phase 2 study of NT219 in combination with cetuximab w/wo chemotherapy in 2L R/M SCCHN

Activated IGF1R and STAT3 as potential predictive biomarkers

Biomarker analysis at the 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)



Advancing First-in-Class Oncology Therapies

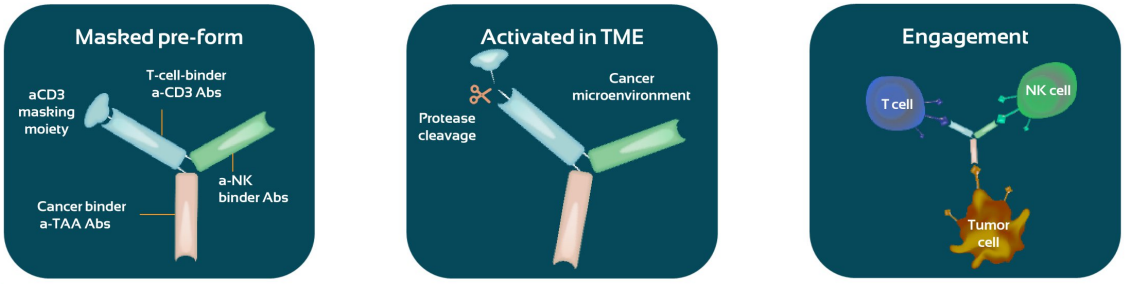
CAPTN-3: Conditionally-Activated Tri-Specific Antibody Platform

Lead candidate: IM1240 (CD3x5T4xNKG2A)

A novel mechanism of action tri-specific antibody

- **Multi-specific biologics is an expanding class** of drugs getting a lot of interest in the industry
- After initial success in hemato-oncology, **new formats are being investigated in solid tumors**

- Technology displays **several distinctive features:**
  - **Dual engagement of T cells and NK cells** to mount an optimal anti-tumoral immune response
  - A tumor-restricted activation through a **cleavable capping system** designed to mitigate cytokine release syndrome and provide a wide therapeutic index
  - Carefully selected Tumor Associated Antigens allowing **patient-centric development**



Removal of T cell engager's masking by proteases of the tumor microenvironment

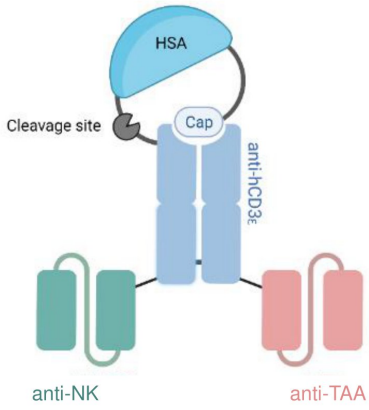
T and NK Cell activation restricted to selected tumors

CAPTN-3 Platform Technology Advantages

CAP / cleavage site:
- **Safety:** Activation only in the TME
- **Efficacy:** better PK

αNK cell arm:
- **NK cell engager**
- **Checkpoint inhibitor**

Enhancement of immune response



Human Serum Albumin (HSA)
- Improved stability in blood circulation

αCD3 arm:
- **T cell engager**
- **T cell activation**

Efficient anti-tumor effect

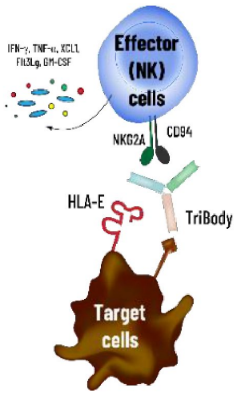
αTAA arm:
- **Tumor Associated Antigen**

Targeted activation against tumor cells

All in one:
- Proximity – increased local concentration
- Synergistic effect
- Molecule size similar to mAb (~170 kDa)

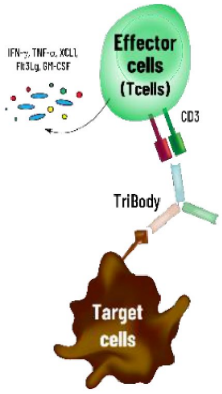
Unleashing both innate and adaptive immune systems

NK cells



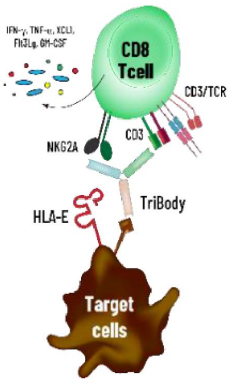
NK cell activation by inhibition of HLAE-NKG2A/CD94 binding

CD4⁺/CD8⁺ T cells



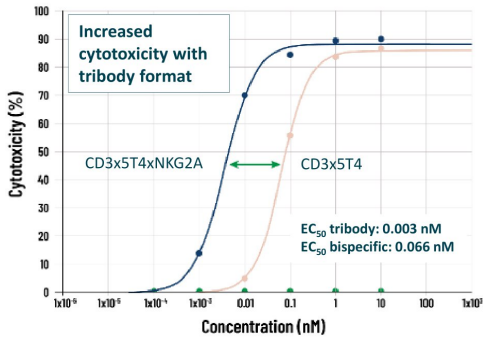
CD4⁺/CD8⁺ T cells activation through CD3 binding

CD8⁺ NKG2A⁺ T cells

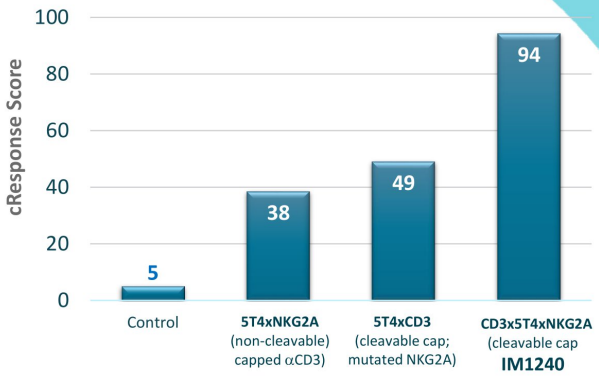


CD8⁺ NKG2A⁺ T cells double activation through CD3 binding and NKG2A-HLA-E axis blocking

POC: αNKG2A arm contributes substantially to tumor cell killing and synergizes with the CD3 Arm



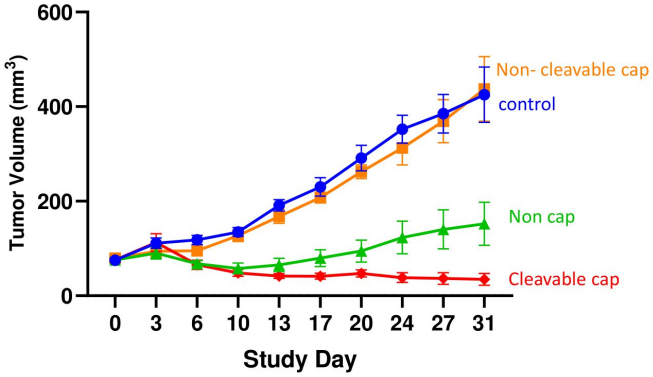
- Tribody induces cytotoxicity at **pM EC_{50}** against NSCLC A549 cells
- **Up to 20-fold more potent** than the bi-specific CD3x5T4
- Cell killing validated on **multiple 5T4⁺ cell lines** (MDA-MB-231, HCT116, NCI-H226)



- Synergistic effect of the αCD3 and αNKG2A arms in suppressing 5T4⁺NCSLC 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).*

Cleavable capping leads to improved in vivo efficacy

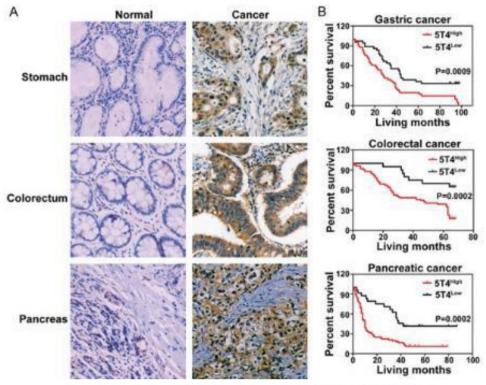


- **Sustained tumor regressions in TNBC xenograft model (MDA-MB-231) in CD34 engrafted humanized mice**
- **The Pro-Tribody, Capped-CD3x5T4xNKG2A, performed better than the uncapped variant**
- **PK analysis in normal mice showed 3-fold higher exposure of the capped tribody compared to the non-capped**
- **No change in body weight**

* Study conditions: dose regimen-0.2mg/kg capped, equimolar 0.1mg/kg non capped, daily IP administration

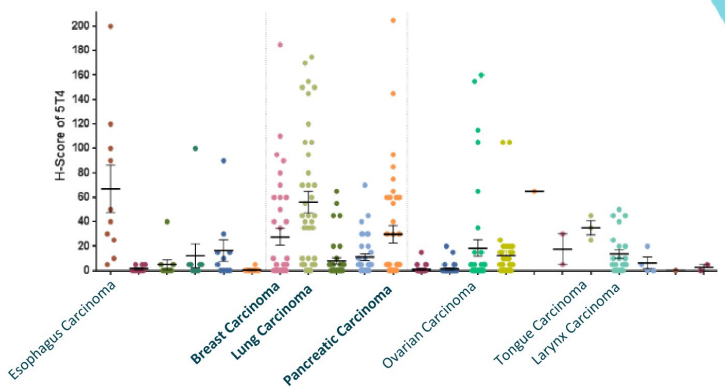
5T4: a Novel Target in Oncology

5T4 is highly expressed on certain tumors and correlates with 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 to several large indications



Opportunity of patient stratification strategy ($5T4^+$)

Corporate highlights

Purple Biotech identifies promising first-in-class drug candidates to treat cancers with high unmet medical need

- Multiple data read-outs expected in 2024
- Two First-in-Class clinical stage drugs
- A preclinical tri-specific immuno-engagers platform
- Lean & global operation
- Cash runway into 1Q25

Purple Biotech (NASDAQ/TASE: PPBT)

As of March 31, 2024

- ADS Outstanding: 26.6 M
- Cash Balance: $10.8 M

Strong position to reach short and mid term value creating clinical data catalysts

PURPLE BIOTECH

THANK YOU

Contact Us:
ir@purple-biotech.com

Appendix A | CM24

CEACAM1 Plays a Key Role in Cancer Biology

01 | ADHESION

Horst, 2011

Oncogene

*"CEACAM1 creates a pro-angiogenic tumor microenvironment that **supports tumor vessel maturation**"*

Ferri, 2020

The Journal of Immunology

*"Neutrophil extracellular trap-associated CEACAM1 as a putative therapeutic target to **prevent metastatic progression** of colon carcinoma"*

02 | IMMUNE CELLS/ IMMUNE EXCLUSION

Tsuzuki, 2020

*"**Immune-checkpoint molecules** on regulatory T-cells as a potential therapeutic target in head and neck squamous cell cancers"*

Tsang, 2020

Cancer Biotherapy & Radiopharmaceuticals

"[Blockade] ***enhances natural killer cell cytotoxicity*** *against tumor cells through blockade of the inhibitory CEACAM1 / CEACAM5 immune checkpoint pathway"*

03 | IMMUNO-ONCOLOGY

Blumberg, 2015

nature

*"CEACAM1 **regulates TIM-3**-mediated tolerance and exhaustion"*

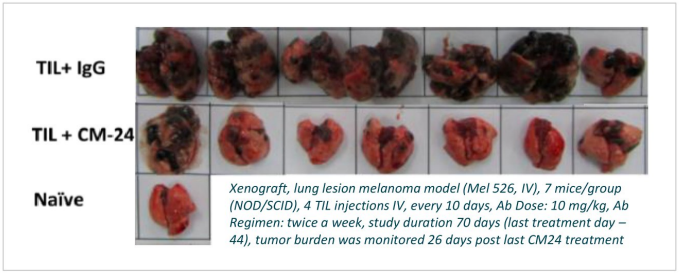
Shively, 2013

Experimental Cell Research

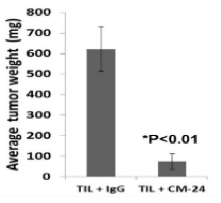
*"CEACAM1 **regulates Fas-mediated apoptosis** in Jurkat T-cells via its interaction with β-catenin"*

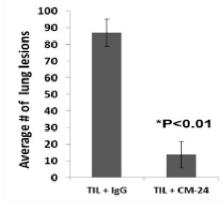
PURPLE Biotech

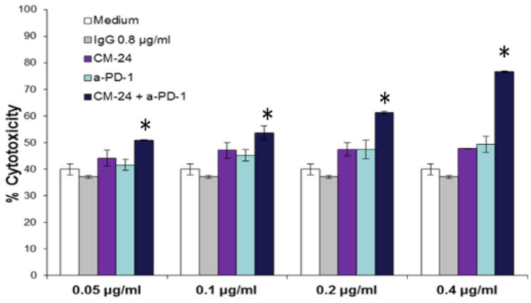
CM24 Reduces Tumor Burden & Synergetic with α-PD-1



Xenograft, lung lesion melanoma model (Mel 526, IV), 7 mice/group (NOD/SCID), 4 TIL injections IV, every 10 days, Ab Dose: 10 mg/kg, Ab Regimen: twice a week, study duration 70 days (last treatment day – 44), tumor burden was monitored 26 days post last CM24 treatment





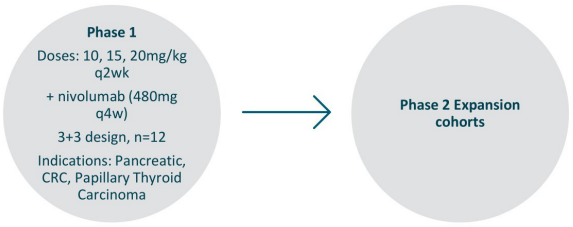


Significant benefits as both single agent and in combination with α-PD-1

Phase 1 Dose Escalation Interim Results
CM24 is Safe and Well Tolerated in Combination with Nivolumab

Study Design

- As of March 8th, 2022, a total of 13 patients were enrolled and 11 patients were evaluable for DLT determination (8 PDAC, 2 CRC and 1 PTC).
- 9 patients had received 2 prior regimens for metastatic disease, 2 patients had one previous line.



Safety

- No DLTs were observed across all dose levels; no Grade 4 AEs or treatment-related deaths have been reported.
- Grade 3 AEs were noted in 6/13 patients (46%).

AE Term	Total	Grade			
		1	2	3	4/5
Diarrhea	5	4		1	
Abdominal pain	4	1	3		
Fever	4	2	2		
Headache	4	3	1		
Fatigue	4	4			
Nausea	3	1	2		
Creatinine increased	3	2	1		
Hypokalemia	2			2	
Dyspnea	2	1		1	
Constipation	2	2			
Cough	2	2			
Abdominal pain aggravated	1			1	
Alkaline phosphatase increase	1			1	
Atrial flutter	1			1	
C-Diff Colitis	1			1	
GI bleed	1			1	
Leukocytosis	1			1	
Small bowel obstruction	1			1	

CM24 Phase 1 Combination Study (NCT04731467)
Demographics

In the Phase 1 part, patients with indicated refractory cancers were administered CM24 at 10, 15, and 20mg/kg q2w and nivolumab 480mg q4w.

- The primary objective of this part was to evaluate safety, tolerability, pharmacokinetics and determine the RP2D
- Safety was assessed according to CTCAE v5 and preliminary anti-tumor activity was assessed by the investigators according to RECISTv1.1 using CT/MRI
- CM24 and CEACAM1 measurements in serum, biopsy specimens, and TILs, as well as tumor and TILs PD-L1 levels are being determined

As of March 8th, 2022, a total of 13 patients were enrolled and 11 patients were evaluable for dose-limiting toxicity (DLT) determination (8 PDAC, 2 CRC and 1 PTC)

- 9 patients had received 2 prior regimens for metastatic disease and 2 patients had one previous line.

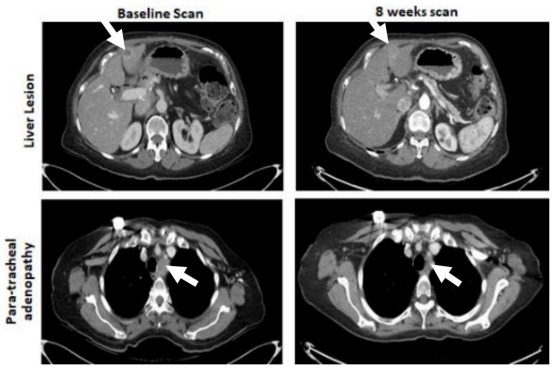
Demographics of patients treated with CM24 (10, 15, 20mg/kg) in combination with nivolumab (480mg)

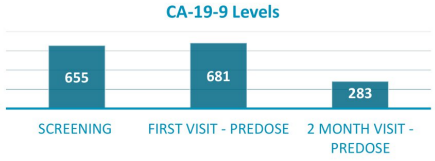
Median age, years (range)	65 (49-76)	Prior Lines of Therapy, n (%)	
Sex, n (%)		1	2 (18%)
Male	5 (45%)	2	9 (82%)
Female	6 (55%)	Diagnosis , n (%)	
Ethnicity, n (%)		Pancreatic cancer	8 (73%)
Not Hispanic or Latino	10 (91%)	Papillary Thyroid cancer	1 (9%)
Hispanic or Latino	1 (9%)	Colorectal cancer	2 (18%)
Race, n (%)		Median Time from Initial Diagnosis months (range)	23 (11-73)
White	10 (91%)	ECOG, n (%)	
Black or African American	1 (9%)	0	7 (64%)
		1	4 (36%)

Confirmed Partial Response in a 3L PDAC Patient

Patient Profile

- 65 y/o female, pancreatic cancer
- 2 prior lines of treatments: FOLFIRINOX and gemcitabine/nab-paclitaxel
- Post Whipple Procedure
- Patient had a germline NF1 VUS, with MSI-S and PDL-1 IHC 2+ and 5% staining
- Confirmed Partial Response: after initial treatment, the patient had a Partial Response of 40%, with a definite reduction of the para-tracheal adenopathy and liver lesions and 58% reduction in CA19-9 levels
- Under treatment for 6 months, still under monitoring.





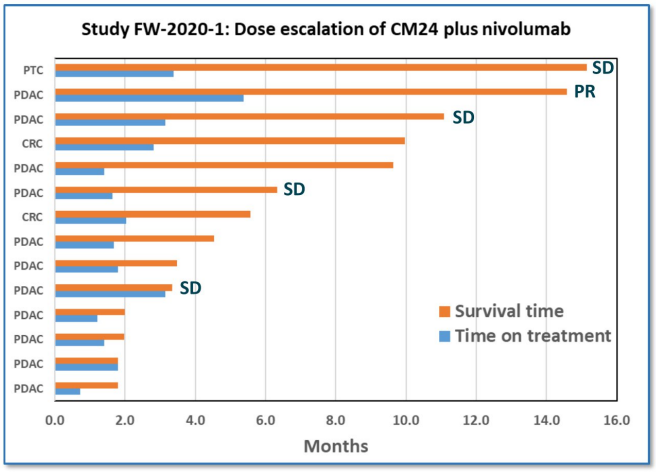
CM24 Phase 1 Dose Escalation Results

Encouraging data in 2L/3L Pancreatic Ductal Adenocarcinoma (PDAC) patients

Study Results

14 patients were evaluable for efficacy:

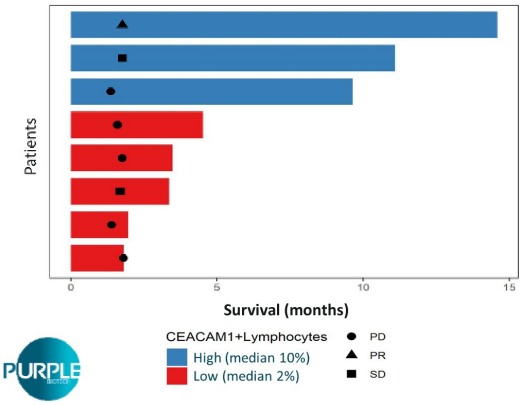
- Best overall response included **1 Partial Response (PR)** (PDAC) and **4 Stable Disease (SD)** (3 PDAC and 1 papillary thyroid cancer (PTC))
- Pharmacokinetic analysis of CM24 shows exposure is dose-proportional across the 3 doses in this study
- Well tolerated with no Dose Limiting Toxicities (DLTs) and no grade ≥ 4 Adverse Events (AEs)
- Median Overall Survival 4.5 months (95% CI 2.0-11.1) for 11 PDAC patients



Phase 1 study results (cont.): Identification of potential exploratory biomarkers supporting CM24's mechanism of action

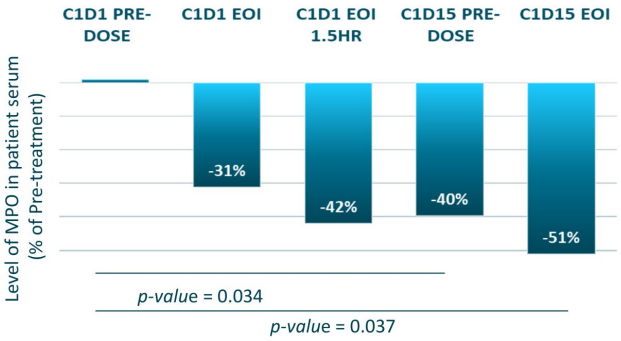
Higher pre-treatment levels of tumor infiltrating lymphocytes that express CEACAM1 are associated with longer survival

- consistent with the CM24 MoA in suppressing the immune evasion
- suggest CEACAM1 expressing lymphocytes as a potential biomarker



CM24 treatment significantly reduced NET marker in serum

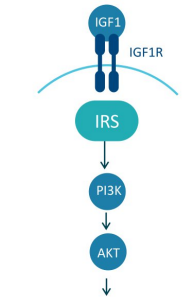
- especially relevant in PDAC patients
- may be used as a pharmacodynamic marker



C1D1 = Cycle 1 Day 1; EOI = End of CM24 Infusion; C1D15 = Cycle 1 Day 15

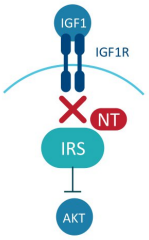
Appendix B | NT219

Novel MOA: IRS Degradation By NT219
Blocking IGF1R-AKT Pathway[1]



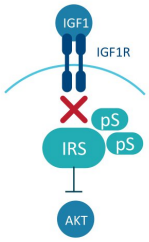
CANCER CELL SURVIVAL

1 Binding to IRS



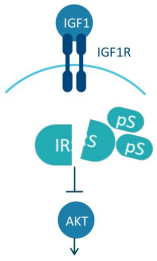
Covalent binding to IRS1/2 leads to the dissociation of IRS1/2 from IGF1R

2 Ser-phosphorylation



Serine phosphorylation prevents re-binding of IRS1/2 to the receptor

3 Degradation



CANCER CELL APOPTOSIS

The proteasome degrades IRS1/2

[1]*Reuveni et al. Cancer Res 2013 ; Ibuki et al. Mol Cancer Ther 2014*

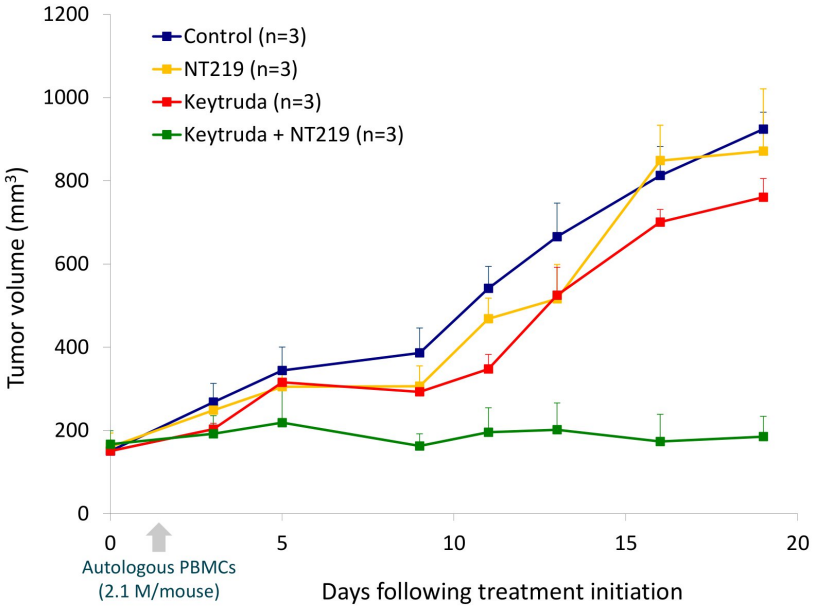
NT219 Re-sensitizes Tumors Refractory to α-PD1

PDX Model

Humanized PDX of GastroEsophageal Junction (GEJ) Cancer (refractory to pembrolizumab)

Drug

Pembrolizumab (Keytruda®)

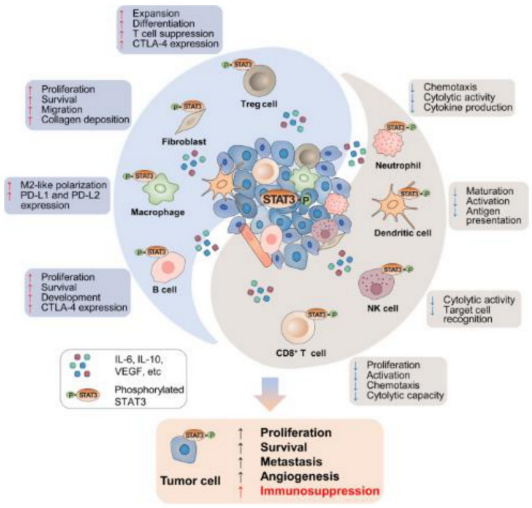


** Double autologous model - Tumors & PBMCs are from the same patient (#RA236) | Keytruda - 6mg/kg IP, NT219 - 60mg/kg IV*

Novel MOA
Signal Transducer and Activator of Transcription 3 (STAT3) Inhibition

- Point of convergence for numerous oncogenic signaling pathways
- Central in regulating the anti-tumor immune response
- Broadly hyperactivated both in cancer and non-cancerous cells within the tumor ecosystem and plays important roles in inhibiting the expression of crucial immune activation regulators and promoting the production of immunosuppressive factors
- Targeting the STAT3 signaling pathway has emerged as a promising therapeutic strategy for numerous cancers

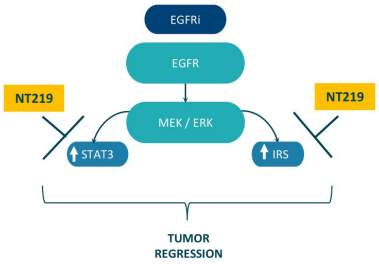
NT219 demonstrates a durable and dose-dependent suppression of STAT3 tyrosine phosphorylation, affecting both the tumor cells and the tumor microenvironment.



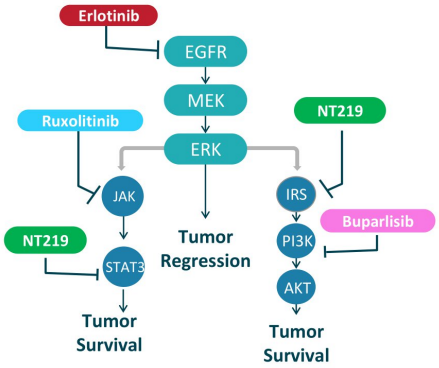
Zou, S., Tong, Q., Liu, B. et al. Targeting STAT3 in Cancer Immunotherapy. Mol Cancer 19, 145 (2020). https://doi.org/10.1186/s12943-020-01258-7

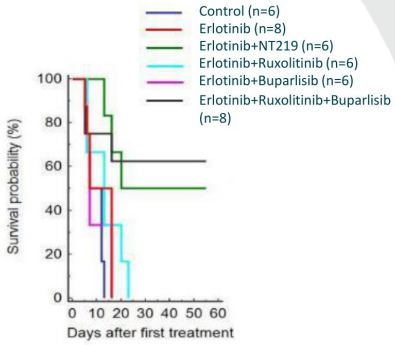
Simultaneous Blockade of STAT3 and AKT Pathways are Required to Overcome Resistance to EGFRi

Overcoming drug resistance



Proof of Concept: PDX model of Head and Neck Cancer





By blocking both STAT3 and IRS resistance pathways, NT219 re-sensitizes tumors to anti-cancer therapies

Selected Publications

UC San Diego School of Medicine

Michael Karin

ORIGINAL ARTICLE

Targeting colorectal cancer via its microenvironment by inhibiting IGF-1 receptor-insulin receptor substrate and STAT3 signaling

האוניברסיטה העברית בירושלים
THE HEBREW UNIVERSITY OF JERUSALEM

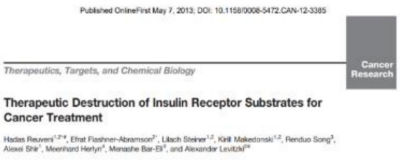
Alexander Levitzki

SHORT COMMUNICATION

Targeting melanoma with NT157 by blocking Stat3 and IGF1R signaling

THE UNIVERSITY OF TEXAS MD Anderson Cancer Center

Menashe Bar-Eli



Published OnlineFirst May 7, 2013; DOI: 10.1158/0008-5472.CAN-12-3385

Therapeutics, Targets, and Chemical Biology

Cancer Research

Therapeutic Destruction of Insulin Receptor Substrates for Cancer Treatment

THE UNIVERSITY OF BRITISH COLUMBIA

Michael Cox

Published OnlineFirst September 29, 2014; DOI: 10.1158/1535-7163.MCT-13-0842

Small Molecule Therapeutics

Molecular Cancer Therapeutics

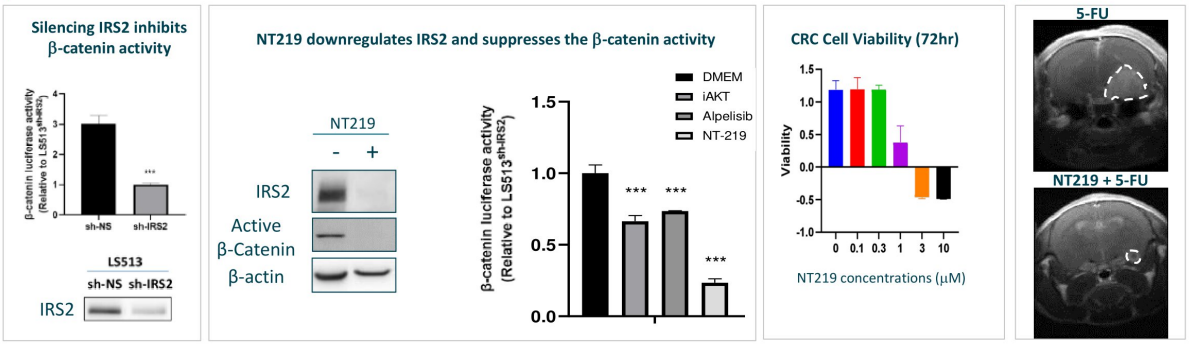
The Tyrphostin NT157 Suppresses Insulin Receptor Substrates and Augments Therapeutic Response of Prostate Cancer

PURPLE BIOTECH

NT219 Suppresses β-Catenin activity in CRC Cells and Inhibited CRC Brain Metastasis

In Vitro

In Vivo



Colon cancer LS-513 cells overexpressing IRS2 demonstrate enhanced β-catenin activity.

Targeted inhibition of IRS2 by NT219 or IRS2-SH RNA, suppresses the increased β-catenin activity and inhibit LS-513 cell viability.

Combination of 5-FU and NT219 significantly inhibited the growth of CRC tumors in brain, using intracranial model and extended mice survival.

NT219 | Pancreatic Cancer in Combination with Gemcitabine

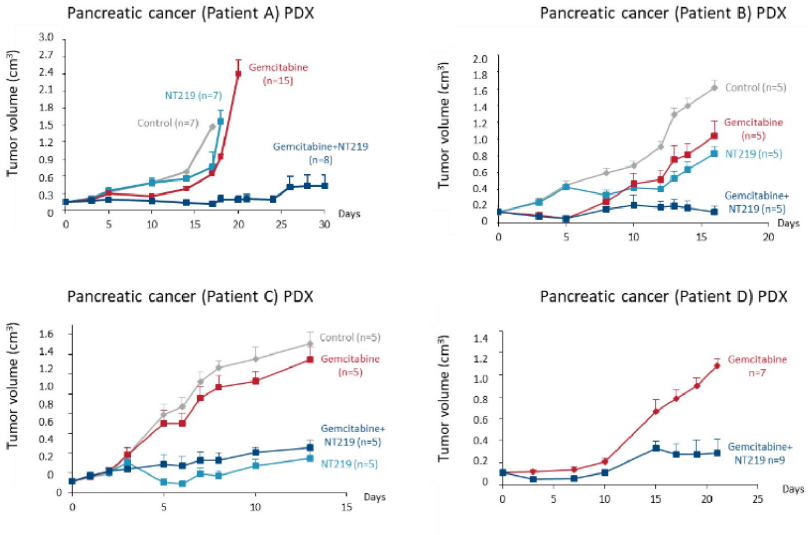
PDX model

Pancreatic Cancer

Drug

Gemcitabine (Gemzar®)

Highly effective anti cancer activity exhibited by NT219 in combination with Gemcitabine



RNA Sequencing | Analysis of Tumors Following Treatment

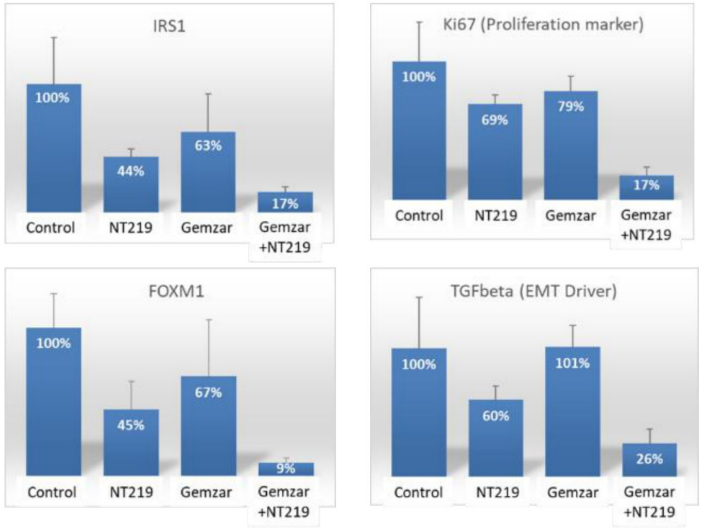
PDX model

Pancreatic Cancer

Drug

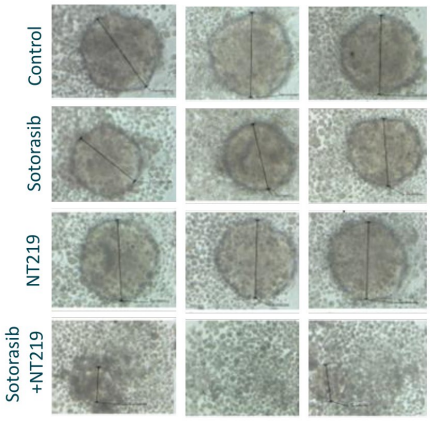
Gemcitabine (Gemzar®)

Reduced expression of IRS1, Ki67, FOXM1 & TGFb is exhibited by pancreatic cancer treated with NT219 alone and in combination with gemcitabine

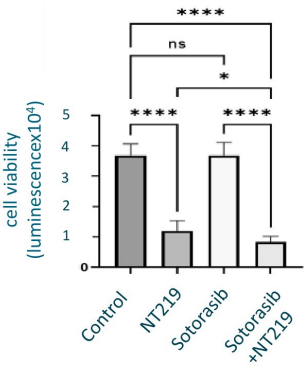


NT219 suppresses cancer stem cell (CSC)-mediated resistance to $KRAS^{G12C}$ inhibitors and synergizes with sotorasib to combat NSCLC

NT219+sotorasib leads to disaggregation of established NSCLC spheroids

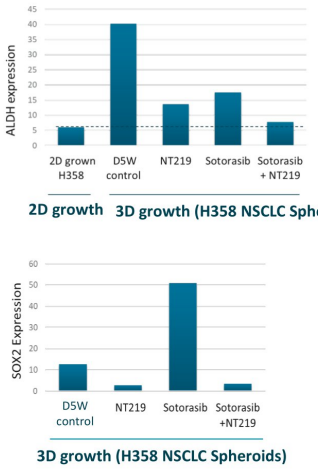


Effect on CSC viability



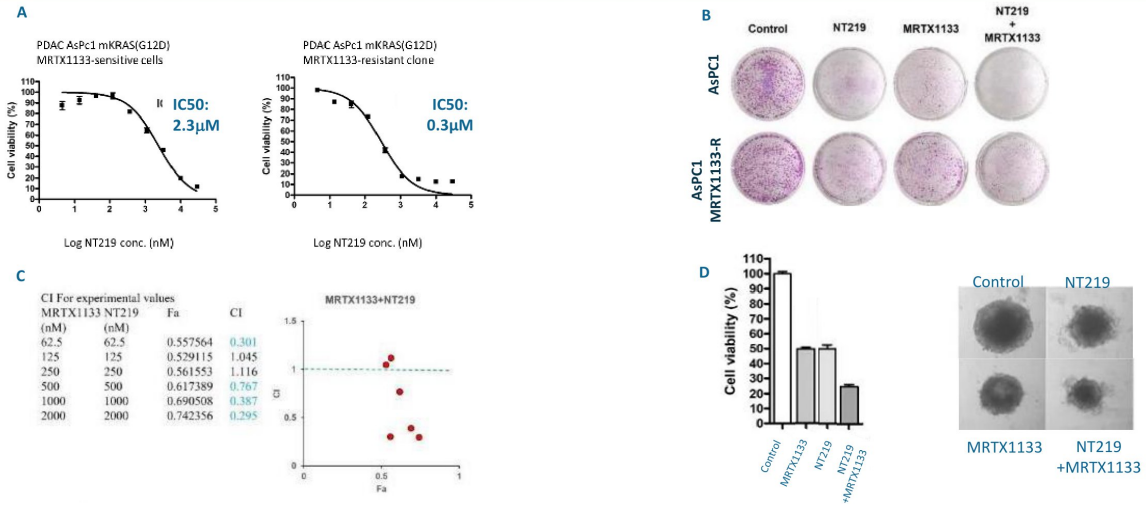
Enrichment of CSC population in established spheroid is suppressed by NT219

Effect on cancer stemness markers



Presented at AACR Annual Meeting 2024

NT219 overcomes resistance to KRAS^{G12D} inhibitors and synergizes with MRTX1133 to combat pancreatic cancer



Higher sensitivity of mKRAS(G12D)i resistant PDAC and synergy of NT219 with MRTX1133. a MRTX1133-resistant PDAC clone was developed. Inhibition of mKRAS G12Di-sensitive (AsPC-1) and resistant (AsPC1-MRTX1133-R) by NT219 (2D cell proliferation) shows 8-fold lower IC50 for the resistant cell line (**A**). NT219 is effective both as monotherapy and in combination with MRTX1133 in colony formation assay of sensitive and resistant PDAC cell lines (**B**). Synergistic effect (CI<1) of NT219 and MRTX1133 was demonstrated in 2D growth (**C**) and in spheroid 3D growth (**D**) of HPAC PDAC cells.

Collaborationn with Dr. Azmi, Karmanos. Presented at AACR Annual Meeting 2024