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 March 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 ☐

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 March 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.

March 6, 2024

PURPLE BIOTECH LTD.

By: */s/ Lior Fhima*

Lior Fhima
Chief Financial Officer

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Exhibit 99.1

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 1H25

Purple Biotech (NASDAQ/TASE: PPBT)

As of December 31, 2023

- ADS Outstanding: 25.2 M
- Cash Balance: $15.3 M

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

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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 1H24 ❖ 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

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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
- **Partial response and stable disease** in dose escalation study with nivolumab
- 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

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A. Immune Response Activation

Tumor

CEACAM1

CEACAM5

CM24

CEACAM1

CEACAM1

CD8+T & NK Cells

Activation of
enhanced cytotoxic activity & cytokine production

B. CM24 Suppresses Neutrophil Extracellular Trap (NET)-Induced Migration and Metastasis of Cancer Cells

Primary Tumor
Metastasizing

CM24

Metastatic Tumor

CM24   Tumor cell   NETs   RBC   CEACAM1

Direct Binding of CM24 to the NET

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

CM24 (red)

CM24 Inhibits NET-Induced Migration of CEACAM1 Expressing Cancer Cells

ns   70%

8,000
7,000
6,000
5,000
4,000
3,000
2,000
1,000
0

AUC_{0-8} (migrating cancer cells/time)

No treat   No treat   Isotype   CM24

No NETs   with NETs

Melanoma

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),

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CEACAM1 is Over-Expressed in Pancreatic Cancer
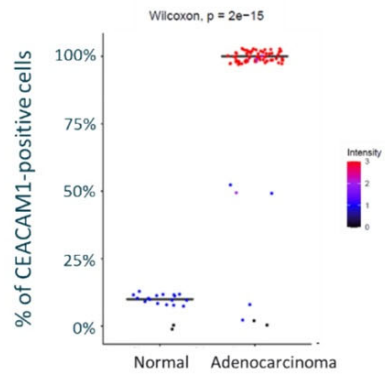
Comparison between CEACAM1 staining intensity in pancreatic cancer (38 cases/76 cores) and normal (10 cases/20 cores) tissues
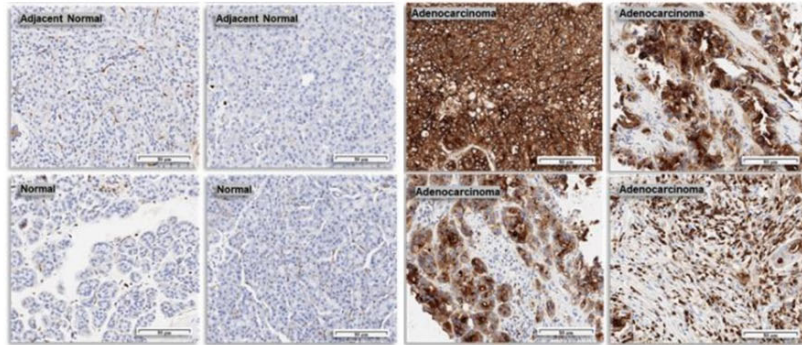
Representative examples of CEACAM1 immunohistochemical images of pancreatic adenocarcinoma and normal tissues

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]
- 3L standard of care regimens efficacy data: patients treated without chemotherapy: Overall Survival (OS) 2 months, OS of 3-4 months with chemotherapy
- 2L standard of care regimens efficacy data: Gemcitabine/Nab-paclitaxel[3]: OS 7.9 months, Progression Free Survival (PFS) 4.3 months 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

Bristol Myers Squibb™

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
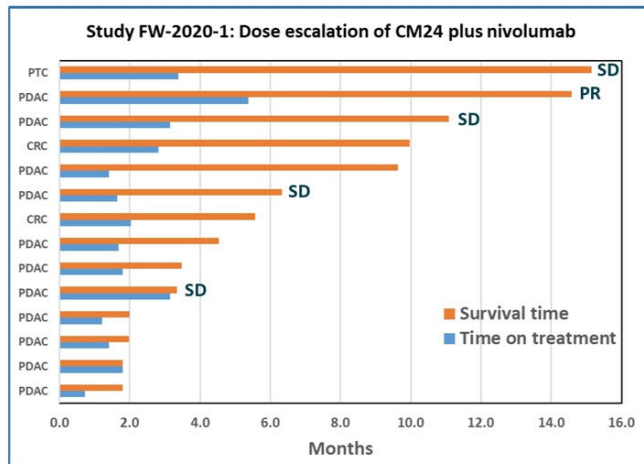
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CM24 Phase 1 Dose Escalation Results
Encouraging data in 2L/3L Pancreatic Ductal Adenocarcinoma (PDAC) patients

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

Study FW-2020-1: Dose escalation of CM24 plus nivolumab

Legend:
- ■ Survival time
- ■ Time on treatment

x-axis: Months (0.0, 2.0, 4.0, 6.0, 8.0, 10.0, 12.0, 14.0, 16.0)

Patient labels (top to bottom): PTC (SD), PDAC (PR), PDAC (SD), CRC, PDAC, PDAC (SD), CRC, PDAC, PDAC, PDAC (SD), PDAC, PDAC, PDAC, PDAC

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
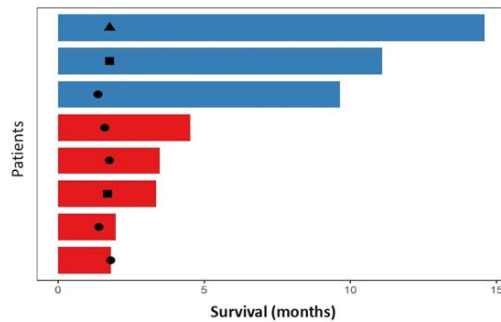
CM24 treatment significantly reduced NET marker in serum

- especially relevant in PDAC patients
- may be used as a pharmacodynamic marker
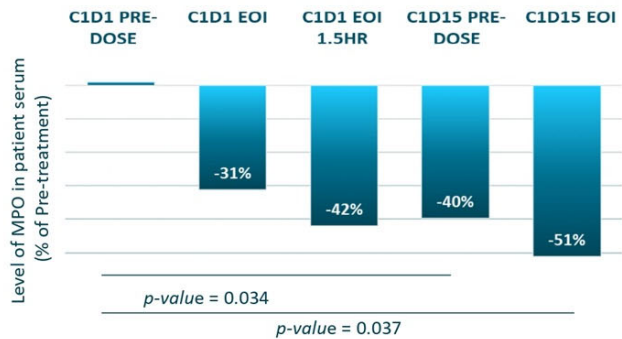
C1D1 = Cycle 1 Day 1; EOI = End of CM24 Infusion; C1D15 = Cycle 1 Day 15

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Phase 2 Combination Study Design (NCT04731467)

A study of CM24 in combination with nivolumab plus chemotherapy in PDAC patients in 2L treatment

18 centers are currently active in **US, EU & Israel**

Primary endpoint :
OS

Secondary endpoints:
PFS, OS rate @ 6 & 12 months,
PFS rate @ 3 & 6 months, ORR

Interim analysis: 1H24
Top line data: 2H24

Measurement of CEACAM1 and other bio-markers is ongoing

2023	2024

n=60
PDAC patients progressing on or after 1L SoC Chemotherapy

Safety Study (n=16)
CM24+nivolumab
&
Nal-IRI/5FU/LV
OR
Gemcitabine/nab-paclitaxel

Completed

Experimental arms (n=30)
CM24+nivolumab and
Gemcitabine/nab-paclitaxel
OR
Nal-IRI/5FU/LV

Control arms (n=30)
Gemcitabine/nab-paclitaxel
OR
Nal-IRI/5FU/LV

Topline (OS)

Ongoing- enrollment completed

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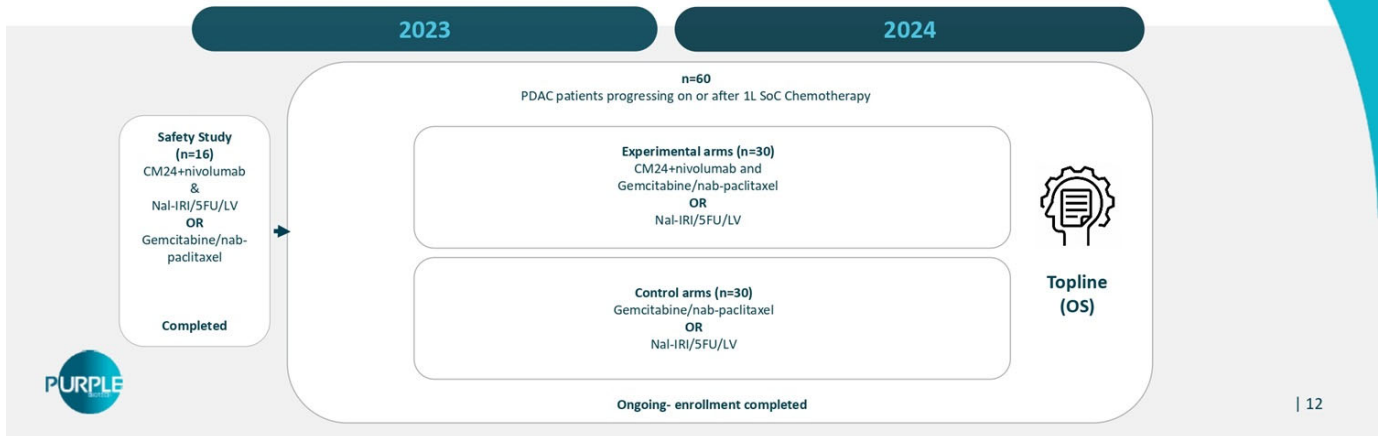
Advancing First-in-Class Oncology Therapies

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**

Robust preclinical package
- **Outstanding efficacy** in various PDX models in monotherapy and in combination
- Uniquely positioned to **tackle resistance** to **EGFRi, MAPKi and ICI**

Clinical Stage
- **No DLTs** in monotherapy or in combination
- **Early activity demonstrated**
- **RP2D determined at 100 mg/kg, Phase 1 concluded**

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

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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

IGF1R
IR

EGFR
FGFR
ILR

IRS1/2

NT219

JAK

PI3K

STAT3

Beta catenin

AKT

Cancer cell survival and differentiation
Anti-apoptotic signal

Cell proliferation, angiogenesis
metastasis, EMT and immune evasion
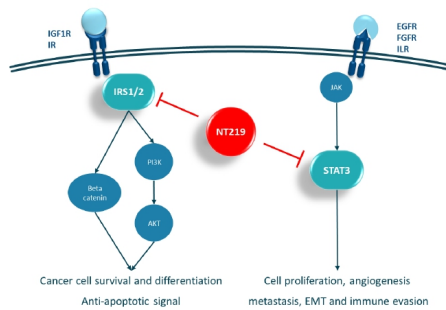
STAT3

- 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; &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.

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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
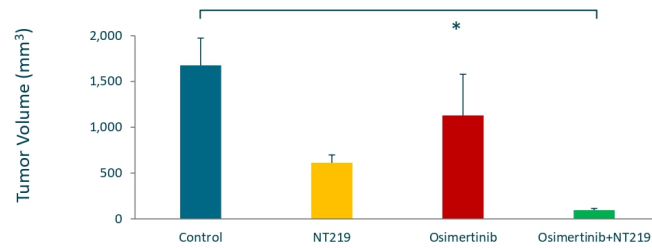
Osimertinib 5 mg/kg, NT219 65 mg/kg, mean tumor volume at the end point, 3 mice/group;
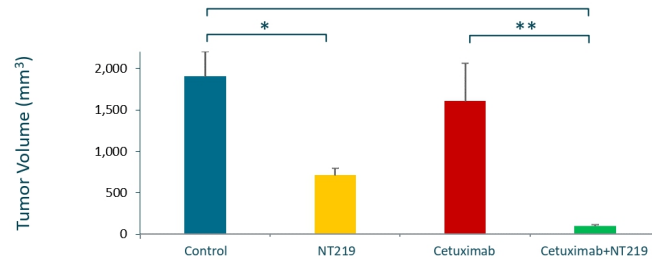
Treatments on days 0, 3 and 10, cetuximab - 1mg/mouse, 3 mice/group; PBMCs (1.4M cells/mouse) were injected on day 6

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

NT219 induces PDL1 and re-sensitizes aPD1-refractory model

NT219+αPD1 reverse resistant tumors

Legend:
- Control (n=10)
- α-PD-1 (n=10)
- NT219 (n=10)
- NT219+α-PD-1 (n=10)

Y-axis: Tumor volume (mm^3)
X-axis: Days following tumor challenge
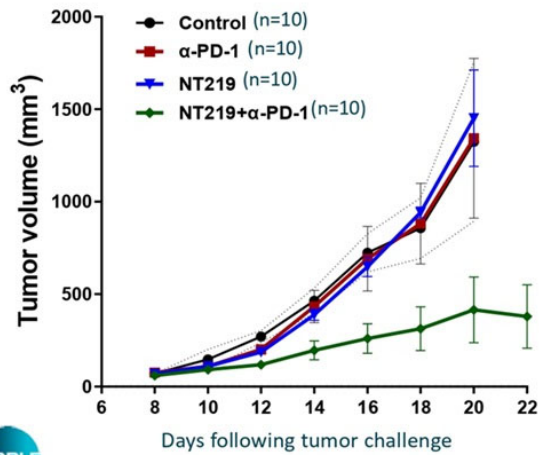
NT219 induces PDL1 expression

αPD1-resistant melanoma cells (FACS analysis)

Y-axis: PDL1 expression (MFI)
X-axis: NT219 (μM) treatment in culture

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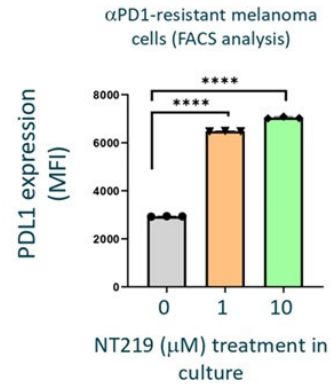
Collaboration with Prof. Bareli and Prof. Curran, M.D. Anderson cancer center; presented at AACR 2023

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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)
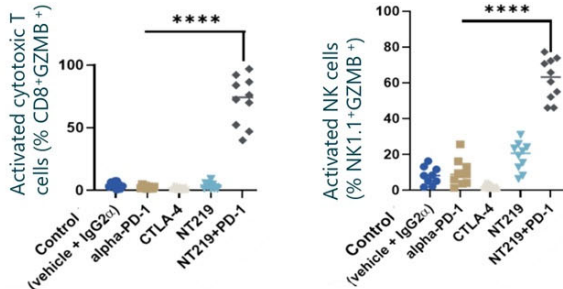
NT219+αPD1 leads to a significant reduction in myeloid derived suppressor cells (MDSC)
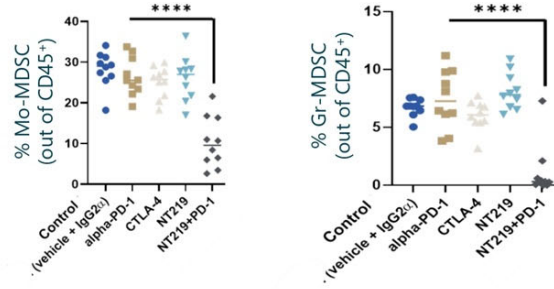
NT219 and aPD1 combination converted immuno-suppressive TME to immuno-reactive

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

1L
60K pts

α-PD-1 + Chemo

↓

2L
40K pts
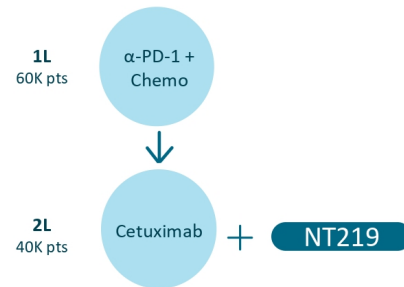
Cetuximab + NT219

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

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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

SCCHN patient
50 mg/kg NT219 + cetuximab

AUC vs Dose (D0)

Cmax vs Dose (D0)
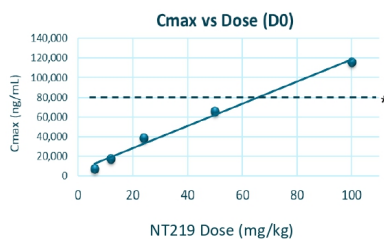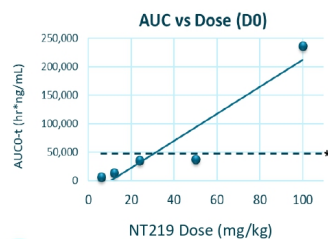
(*) Exposure & Cmax obtained at the effective dose in mice was observed at the ≥50mg/kg dose in human
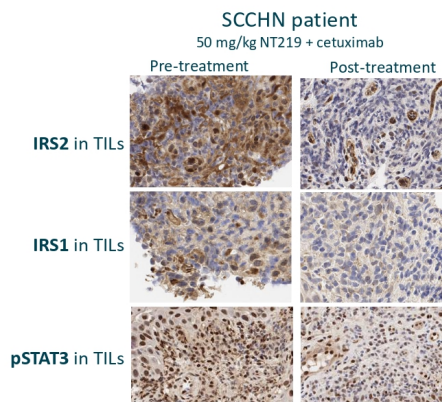
Efficacy overview of monotherapy arm:

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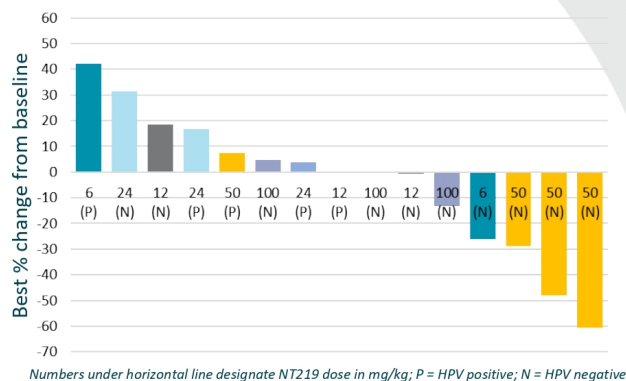
In preparation of a phase 2 study of NT219 in combination with cetuximab w/wo chemotherapy in 2L R/M SCCHN

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Advancing First-in-Class Oncology Therapies

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 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
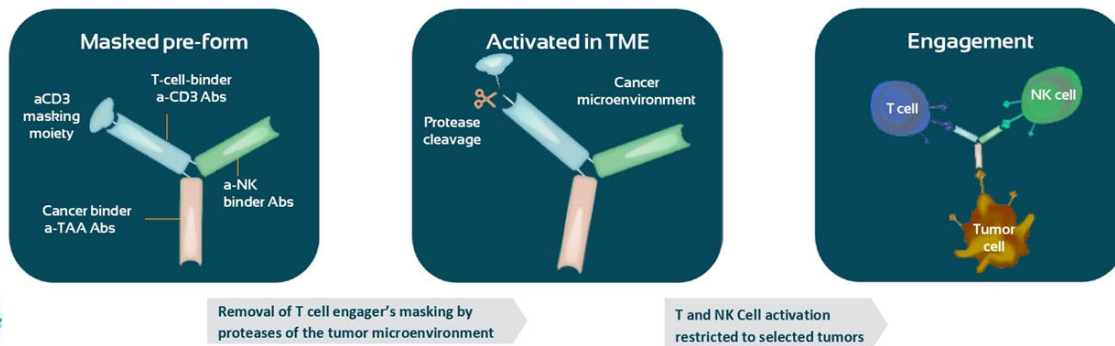
Unleashing both Innate and Adaptive Immune Systems

NK-cells	CD4⁺-T-cells	CD8⁺-NKG2A⁺-T-cells

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

CD4⁺-T-cells activation through CD3 binding

CD8⁺-NKG2A⁺ T-cells double activation through CD3 binding and NKG2A-HLA-E axis blocking
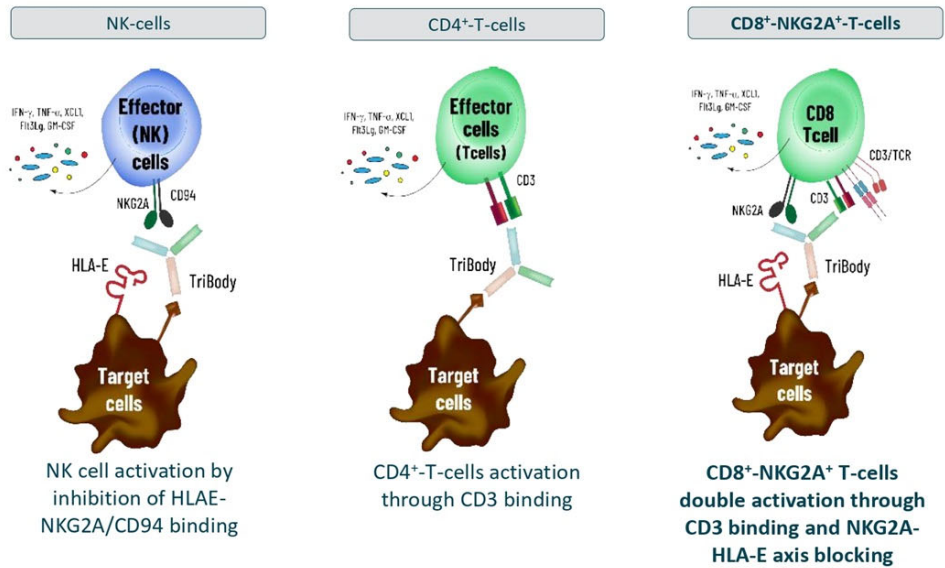
Promising Proof of Concept Data

Increased cytotoxicity with tribody format

CD3x5T4xNKG2A   CD3x5T4

EC_{50} tribody: 0.003 nM
EC_{50} bispecific: 0.066 nM
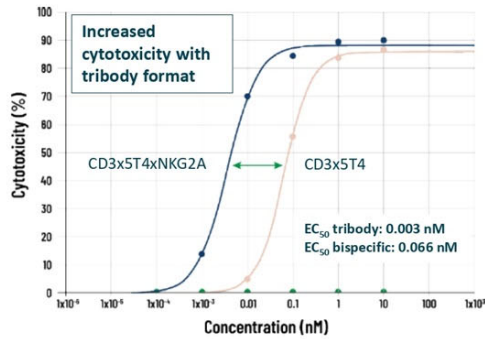
- Tribody induces **pM EC_{50}** against A549 cells
- **20-fold more potent** than CD3x5T4 variant
- Cell killing validated on **multiple 5T4+ cell lines** (MDA-MB-231, HCT116, NCI-H226)

Cleavable capping leads to improved in vivo efficacy

Control
Non-cleavable Cap

No Cap

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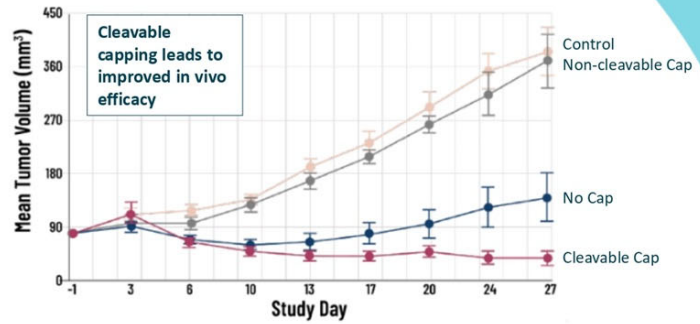
- **Sustained tumor regressions in Breast Cancer** xenograft **humanized** model (MDA-MB-231)
- **The Pro-Tribody Capped-CD3x5T4xNKG2A performed better** than the uncapped variant
- **No change in body weight**
- PK analysis in normal mice shows **3-fold higher exposure of the capped tribody compared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

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5T4: a Novel Target in Oncology

5T4 is highly expressed on certain tumors and correlates with poor prognosis
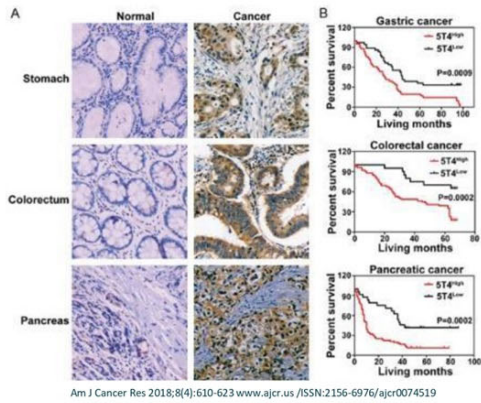
5T4 is a Tumor Associated Antigen prevalent to several large indications
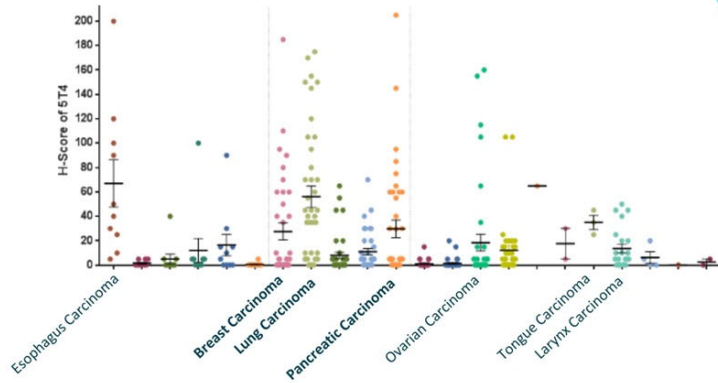
Opportunity of patient stratification strategy (5T4⁺)

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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 1H25

Purple Biotech (NASDAQ/TASE: PPBT)

As of December 31, 2023

- ADS Outstanding: 25.2 M

- Cash Balance: $15.3 M

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

THANK YOU

PURPLE BIOTECH

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 and 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

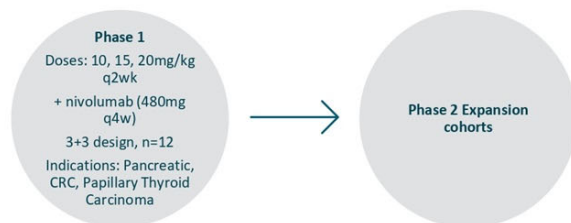# CM24 Reduces Tumor Burden & Synergetic with α-PD-1

TIL+ IgG

TIL + CM-24

Naïve

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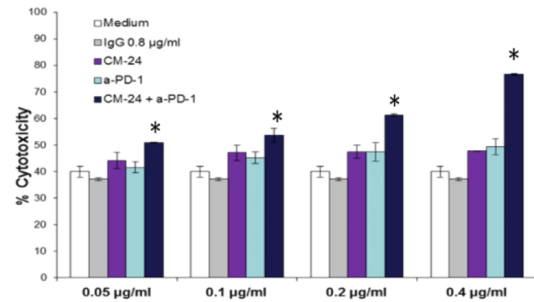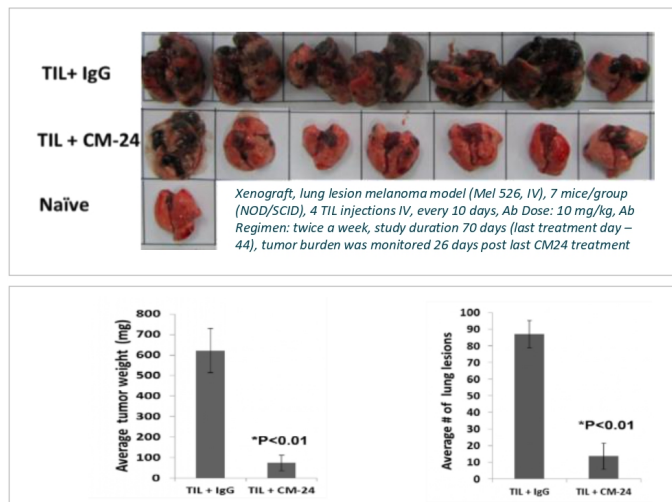
Significant benefits as both single agent and in combination with α-PD-1

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.

Phase 1

Doses: 10, 15, 20mg/kg q2wk

+ nivolumab (480mg q4w)

3+3 design, n=12

Indications: Pancreatic, CRC, Papillary Thyroid Carcinoma

→ **Phase 2 Expansion cohorts**

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	

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CM24 Phase 1 Combination Study (NCTO4731467)
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.

CA-19-9 Levels

SCREENING	FIRST VISIT - PREDOSE	2 MONTH VISIT - PREDOSE
655	681	283
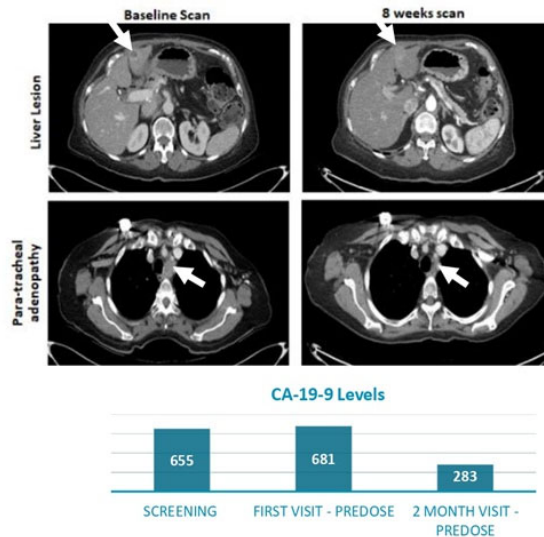
Novel MOA: IRS Degradation By NT219
Blocking IGF1R-AKT Pathway[1]

CANCER CELL SURVIVAL
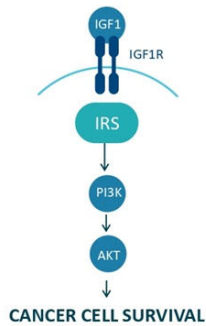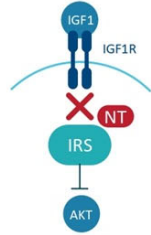
1 Binding to IRS

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

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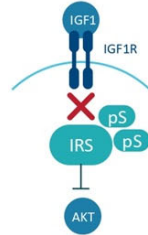
Serine phosphorylation prevents re-binding of IRS1/2 to the receptor

3 Degradation
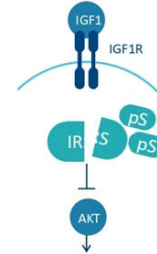
CANCER CELL APOPTOSIS

The proteasome degrades IRS1/2

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

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NT219 Re-sensitizes Tumors Refractory to α-PD1

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

Drug
Pembrolizumab (Keytruda®)

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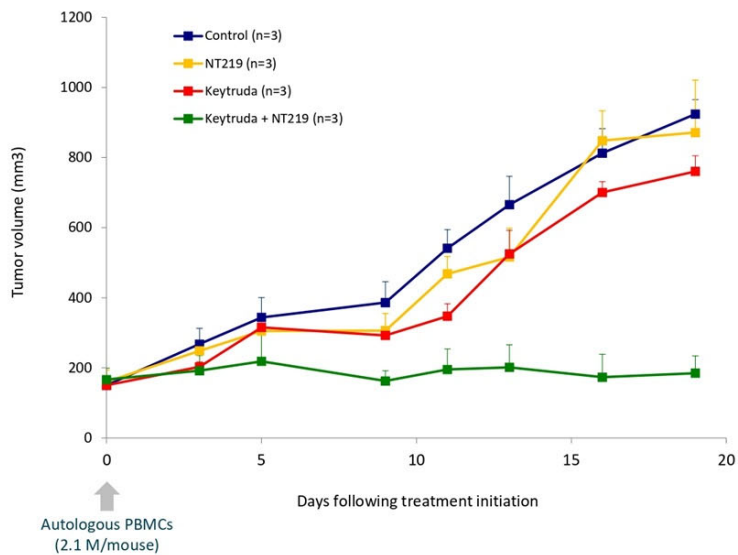
* Double autologous model - Tumors & PBMCs are from the same patient (#RA236) | Keytruda - 6mg/kg IP, NT219 - 60mg/kg IV

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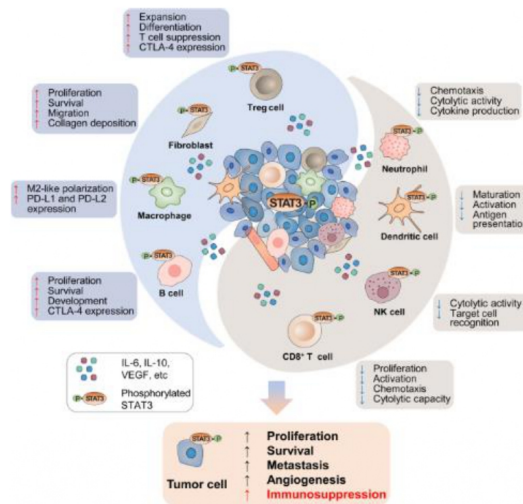
Simultaneous Blockade of STAT3 and AKT
Pathways are Required to Overcome Resistance to EGFRi

Overcoming drug resistance
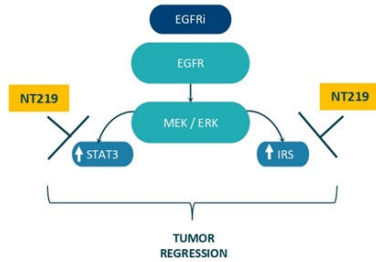
Proof of Concept: PDX model of Head and Neck Cancer
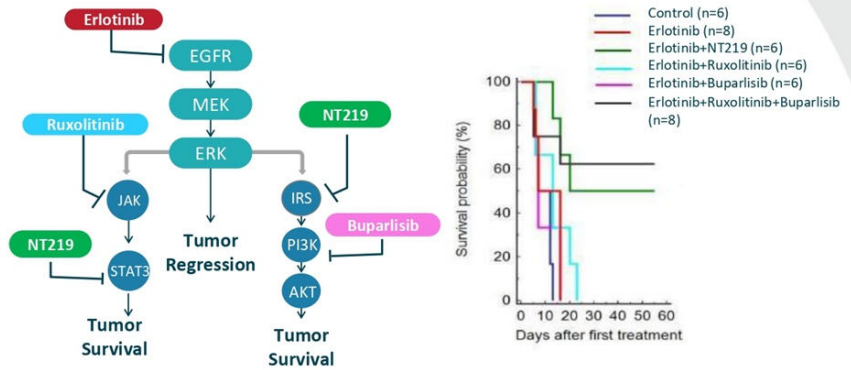
By blocking both STAT3 and IRS resistance pathways,
NT219 re-sensitizes tumors to anti-cancer therapies

Selected Publications

Michael Karin

ORIGINAL ARTICLE

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

Alexander Levitzki

SHORT COMMUNICATION

Targeting melanoma with NT157 by blocking Stat3 and IGF1R signaling

Menashe Bar-Eli

Therapeutics, Targets, and Chemical Biology

Therapeutic Destruction of Insulin Receptor Substrates for Cancer Treatment

Michael Cox

Small Molecule Therapeutics

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

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

In Vitro

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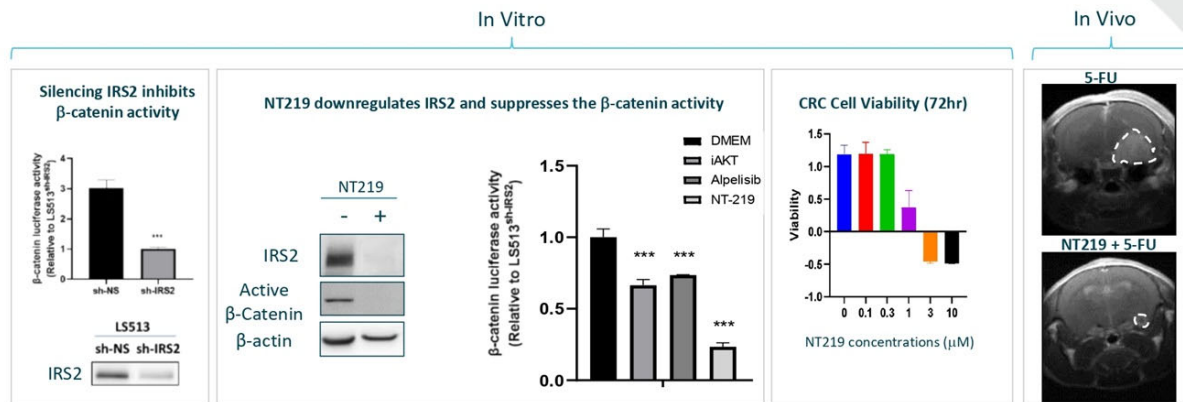
Colon cancer LS-513 cells overexpressing IRS2 demonstrate enhanced β-catenin activity.

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 |
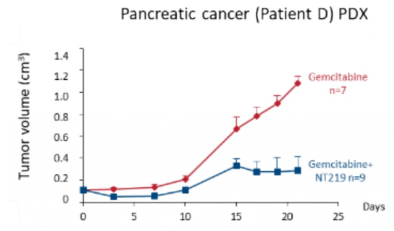## Pancreatic Cancer in Combination with Gemcitabine

PDX model
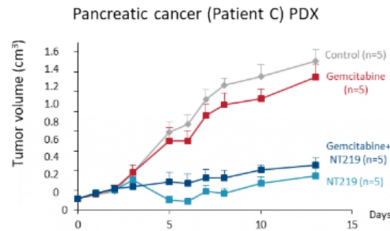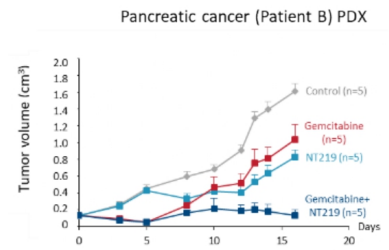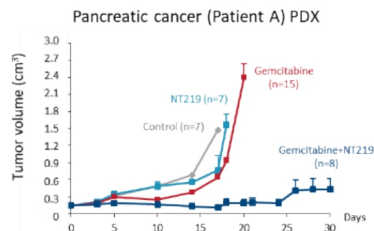**Pancreatic Cancer**

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

IRS1

Control	100%
NT219	44%
Gemzar	63%
Gemzar +NT219	17%

Ki67 (Proliferation marker)

Control	100%
NT219	69%
Gemzar	79%
Gemzar +NT219	17%

FOXM1

Control	100%
NT219	45%
Gemzar	67%
Gemzar +NT219	9%

TGFbeta (EMT Driver)

Control	100%
NT219	60%
Gemzar	101%
Gemzar +NT219	26%