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 November 2022
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 ☐

Indicate by check mark if the Registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(1): ☐

Indicate by check mark if the Registrant is submitting the Form 6-K in paper as permitted by Regulation S-T Rule 101(b)(7): ☐

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	Company Presentation – November 2022

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-3 filed with the Securities and Exchange Commission on December 2, 2019 (Registration file number 333-235327), 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) and 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), 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.

November 9, 2022

PURPLE BIOTECH LTD.

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

Exhibit 99.1


PURPLE
BIOTECH
CORPORATE PRESENTATION
NASDAQ/TASE: PPBT
November 2022

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. 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 and CM24; the process by which early stage therapeutic candidates such as NT219 and CM24 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 lack of sufficient funding to finance the 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 attained 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, and other factors that are discussed in our in our Annual Report on Form 20-F for the year ended December 31, 2021 and in our other filings with the U.S. Securities and Exchange Commission (the "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 presentation 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, http://www.sec.gov.

Business Highlights

We are a clinical-stage company committed to the development of first-in-class, effective therapies by harnessing the power of the tumor microenvironment (TME) to overcome drug resistance and improve treatment outcomes for cancer patients

- Two first-in-class clinical stage drugs
- Multiple Phase II studies ongoing and planned
- Lean & global operation
- BD activity to grow our pipeline with innovative assets
- Extended cash runway to end of 2024

Strong balance sheet and cash position

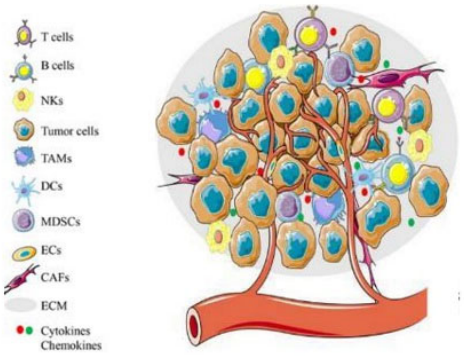
Purple Biotech (NASDAQ/TASE: PPBT)

ADSs outstanding: 18.4M

$35.7M cash as of September 30, 2022

Multiple data read-outs expected in the next 12 months

Harnessing the Power of the TME



- Hammering cancer cells is not sufficient (i.e, chemotherapy, radiotherapy)
- Tumors rely on a favorable environment to strive and escape the Immune surveillance
- Multiple resistance mechanisms prevent long-term survival
- Concept fully validated with the rise of Immuno-oncology treatments
- The current focus is largely on lymphoid cells while the “myeloid” component is largely overlooked

We focus on MOAs that impact both tumor cells and TME

Shi, Rongchen et al. “Metabolism in tumor microenvironment: Implications for cancer immunotherapy.” MedComm vol. 1,1 47-68. 3 Jun. 2020, doi:10.1002/mco2.6

A Pipeline Dedicated to Advancing Oncology Therapies

Program	Indication	Phase 1	Phase 2	Phase 3	Value Drivers
CM24 (CEACAM-1)	Solid tumors (monotherapy)	Completed			√ RP2D in combination: 20 mg/kg √ Initiation of phase II in 2L PDAC Follow up OS data from P1
	Solid tumors (combination with nivolumab*)				Interim analysis P2 2H23 Top line P2 2H24
	Pancreatic Cancer (combination with nivolumab+chemo*)				
	Combination with SOC, undisclosed indication				Investigation in a second indication
NT219 (IRS1/2 & STAT3)	Solid tumors (monotherapy)				RP2D monotherapy & combination 1H23
	R/M SCCHN & CRC (dose escalation + P2 with cetuximab)				Initiation of P2 combination with cetuximab 2Q23
	Combination with SOC, undisclosed indication				Investigation in a second indication

Ongoing | Planned study

*Clinical collaboration and supply agreement with: Bristol Myers Squibb

Advancing First-in-Class Oncology Therapies

CM24: an α-CEACAM1* mAb

*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 MOA

- CM24 inhibits the **immunosuppressive effect of CEACAM1**
- CM24 increases **T cell and NK cells cytotoxicity** against tumors
- CM24 is a **new Immune Checkpoint Inhibitor** to be combined with IO treatments

Signals of clinical efficacy

- **Favorable safety profile** in monotherapy and in combination with nivolumab
- **1 PR and 3 SD** in dose escalation study with nivolumab
- P2 at **RP2D initiated in Q2 2022**

Sizable market potential

- Significant unmet medical need in **PDAC**
- Strong **IP position** and well **ahead of competitors**
- Multiple opportunities to leverage the **synergy with approved IO** or chemotherapy

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"*

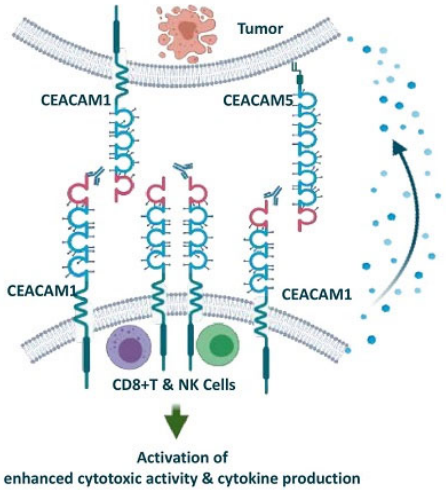
Shively, 2013

Experimental Cell Research

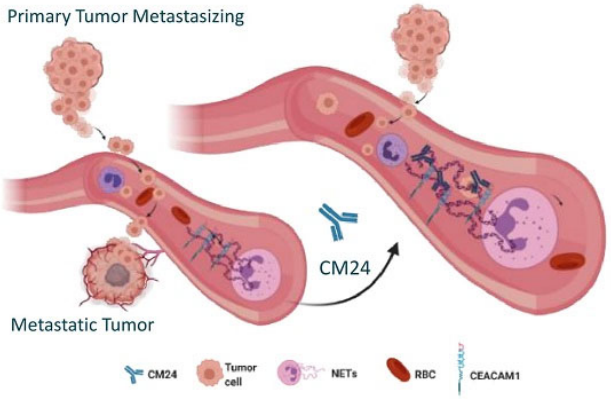
*"CEACAM1 **regulates Fas-mediated apoptosis** in Jurkat T-cells via its interaction with β-catenin"*

PURPLE

CM24 MOA | Immuno-Oncology & Adhesion

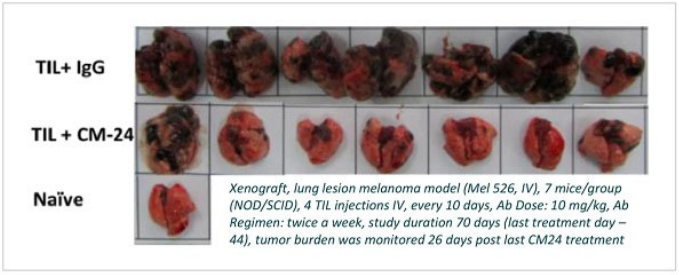


Neutrophil Extracellular Trap (NET)-associated CEACAM1 as a putative therapeutic target to prevent metastatic progression:

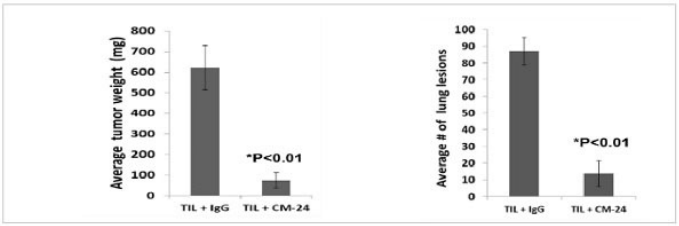


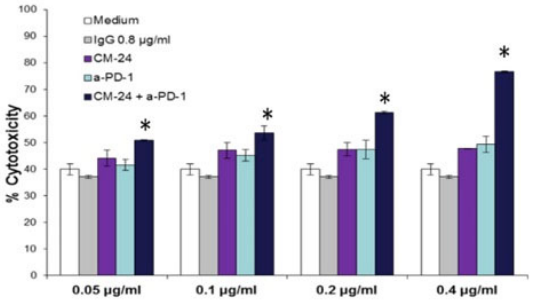
Markel et al, J Immunol 2002, 2006; Immunology, 2008; Cancer Immunol Immunother 2010; Ortenberg et al, Mol Cancer Ther 2012; Zhou, 2009; Li, 2013; Huang, 2015; Acharya N, et al. J Immunotherapy Canc 8:e911-22, 2020.; Rayes RF, et al. Neutrophil Extracellular Trap-Associated CEACAM1 as a Putative Therapeutic 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)

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



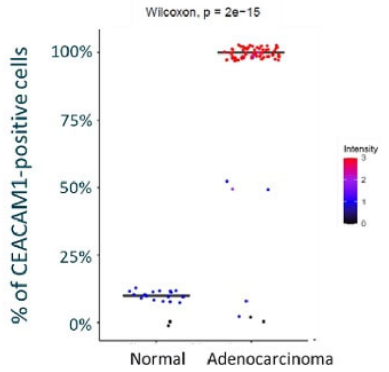
Xenograft, lung lesion melanoma model (Mel 526, IV), 7 mice/group (NOD/SCID), 4 TIL injections IV, every 10 days, Ab Dose: 10 mg/kg, Ab Regimen: twice a week, study duration 70 days (last treatment day – 44), tumor burden was monitored 26 days post last CM24 treatment



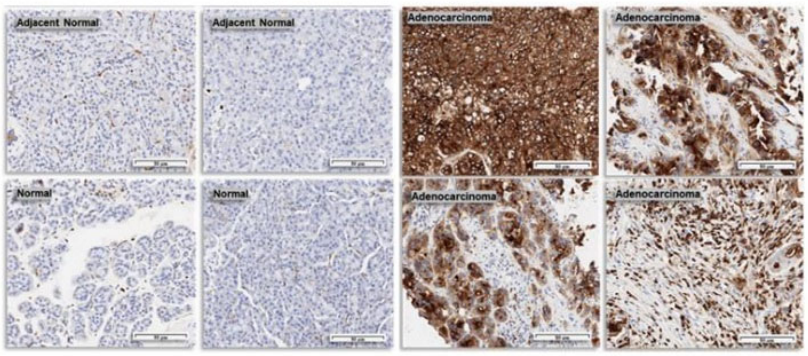


Significant benefits as both single agent and in combination with α-PD-1

CEACAM1 is Over-Expressed in PDAC



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

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	

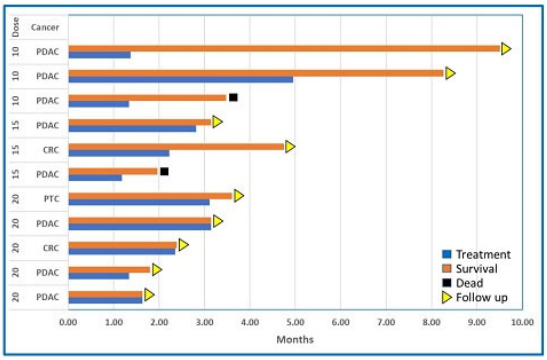
CM24 Phase 1 Dose Escalation Interim Results (cont.)
Sustained Clinical Benefit Even After Treatment Cessation

For the 11 evaluable patients as of March 8th, 2022, best overall response included **1 confirmed PR** (PDAC patient**) 3 SD** (2 PDAC and 1 PTC) for a disease control rate of 36%

- 9/11 of the evaluable patients are in study follow-up
- Pharmacokinetic analysis of CM24 shows exposure is dose-proportional across the 3 doses in this study with a complete receptor occupancy of peripheral CEACAM1 receptors on T cells and neutrophils at CM24 doses of 15 or 20mg/kg
- Median overall survival has not yet been reached

The Phase 2 portions of the study has been initiated at the conclusion of this dose-escalation part.

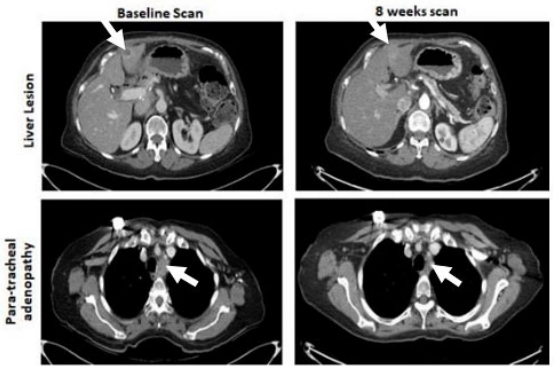
Overall Survival



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.



Large Market Opportunity in Pancreatic Cancer

- Pancreatic Cancer accounts for ~60K new cases/year in the US; with a 5-year relative survival rate of 11.5%[1]
- I/O 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 2L is 3%[1]
- 2L SoC regimens efficacy data: Gemcitabine/Nab-paclitaxel[3]: OS 7.9 months, 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

Combining nivolumab with CM24 in a clinical collaboration with Bristol Myers Squibb

Bristol Myers Squibb™

1. *American Cancer Society, Cancer Facts & Figures 2019, and the ACS website,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

CM24 Phase 2 Combination Study Design (NCT04731467)

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

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

Primary efficacy endpoint of the randomized sub-study:

OS

Secondary endpoints:

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

Interim analysis:

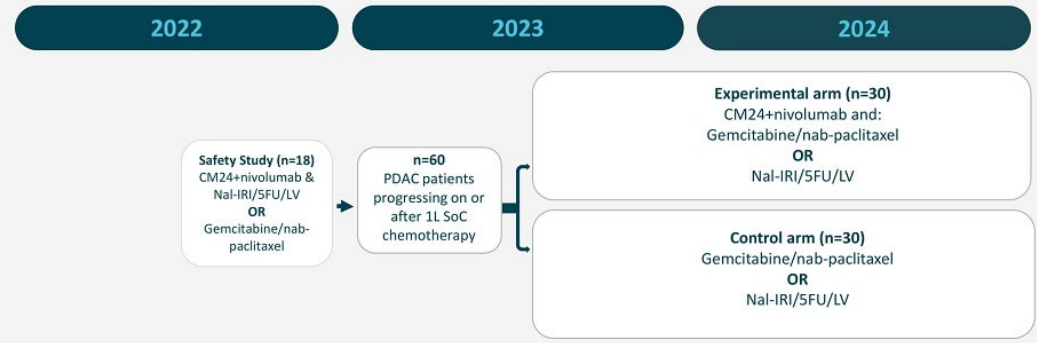
- PFS rate @ 6M planned in 2H23

Top line data:

- planned in 2H24

Measurement of CEACAM1 and other bio-markers is ongoing

Planning of further studies in other tumor types is ongoing



Advancing First-in-Class Oncology Therapies

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

NT219 - A Novel Approach to
Overcome Resistance to EGFRi and Beyond

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
- Modulation of the **tumor microenvironment**
- Uniquely suited to **tackle resistance to EGFRi and other agents**

Clinical Stage

- Early signs of clinical **efficacy as single agent**
- **No DLTs** observed to date, RP2D has not been determined yet

Broad Market Potential

- **Short path to registration** in 2L r/mSCCHN and **multiple market upsides**

NT219 - Dual Inhibitor of IRS1/2 & STAT3

IRS1/2

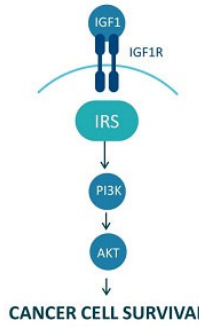
- Scaffold proteins, mediating mitogenic, metastatic, angiogenic and anti-apoptotic signals from IGF1R, IR, IL4R and other oncogenes, overexpressed in multiple tumors
- Regulates major survival pathways such as the PI3K/AKT, MEK/ERK and WNT/β-catenin
- Activated as a feedback response to anti-cancer 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

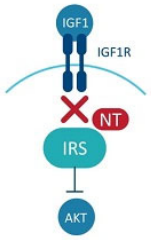
Hadas Reuveni et al. Cancer Res 2013;73:4383-4394; Machado-Neto et al. Clinics 2018; 73,suppl 1 e566s; Naokazu Ibuki, Mazyar Ghaffari, Hadas Reuveni et al. Mol Cancer Ther. 2014; 13(12): 2827-2839; Rampias et al. Oncogene 2016; 35(20):2562-4; Flashner-Abramson, Reuveni Hadas, Levitzki Alexander et al. Oncogene 2016;35(20):2675-80; [5]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.

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



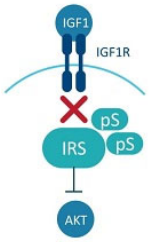
CANCER CELL SURVIVAL

1 Binding to IRS



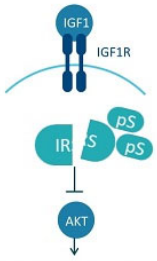
Covalent binding to IRS1/2 leads to the dissociation of IRS1/2 from IGF1R

2 Ser-phosphorylation



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

3 Degradation



CANCER CELL APOPTOSIS

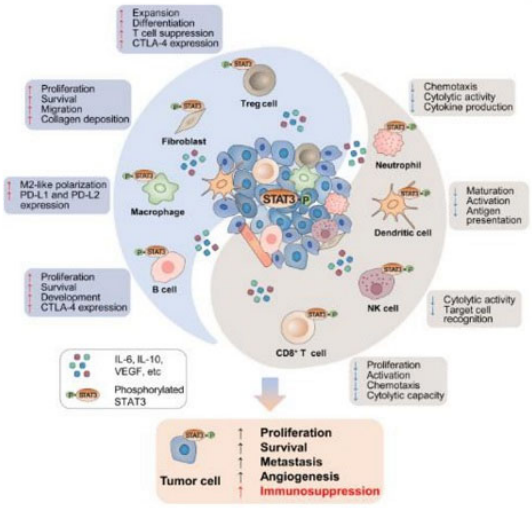
The proteasome degrades IRS1/2

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

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

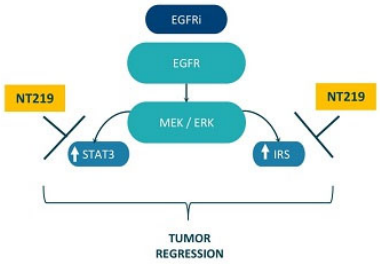
NT219 demonstrates a durable and dose-dependent suppression of STAT3 tyrosine phosphorylation, affecting both the tumor cells and the tumor microenvironment.



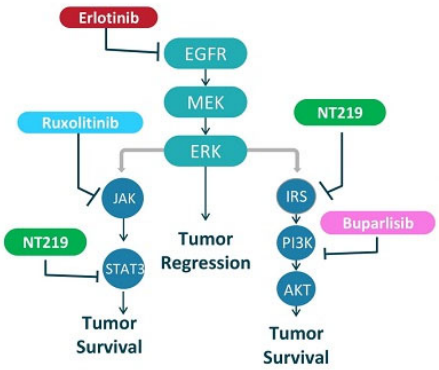
Zou, S., Tong, Q., Liu, B. et al. Targeting STAT3 in Cancer Immunotherapy. Mol Cancer 19, 145 (2020). https://doi.org/10.1186/s12943-020-01258-7

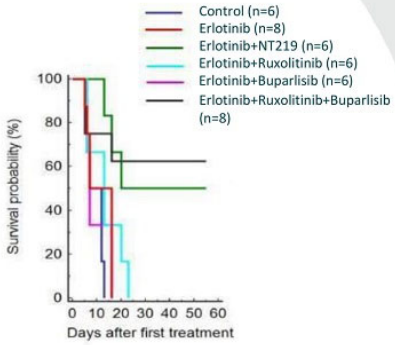
Simultaneous Blockade of STAT3 and AKT Pathways are Required to Overcome Resistance to EGFRi

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

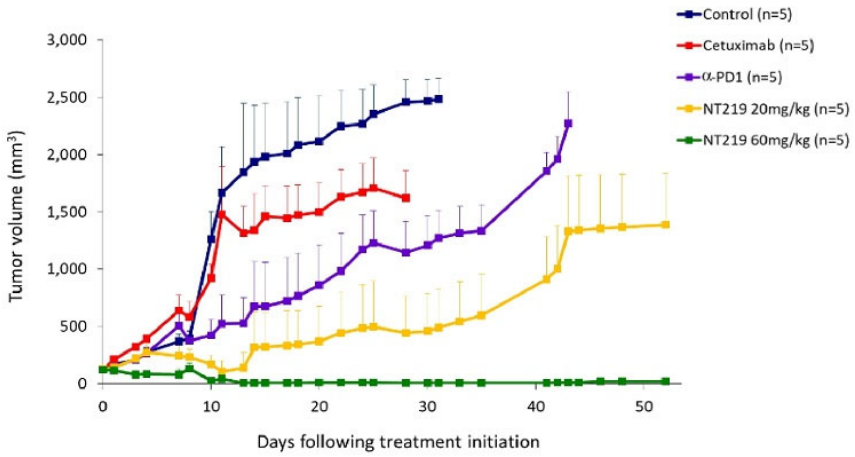
NT219 is Effective as Monotherapy

Animal Model

Head & Neck Cancer (SCC-9) NSG™, PBMCs-injected[1]

Drugs

α-PD1
Cetuximab (Erbitux®)
NT219 20mg/kg
NT219 60mg/kg



1 NSG mice were injected SC with SCC-9 cells. PBMCs were injected to the mice (18M cells per mouse) and treatments initiated when tumors were established. (NT219) and IP (α-PD1 and cetuximab) twice a week for 4 weeks. Graph reflects relevant data adapted from 2020 Multidisciplinary Head and Neck Cancers Symposium Poster presentation

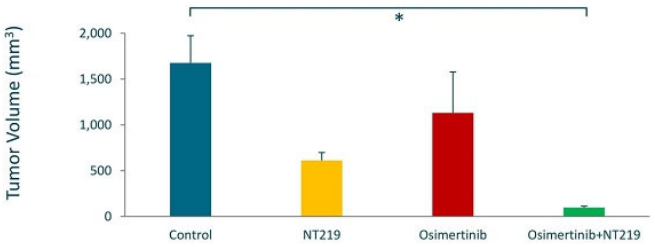
NT219 Restores Sensitivity to EGFRi in PDX Models

NSCLC

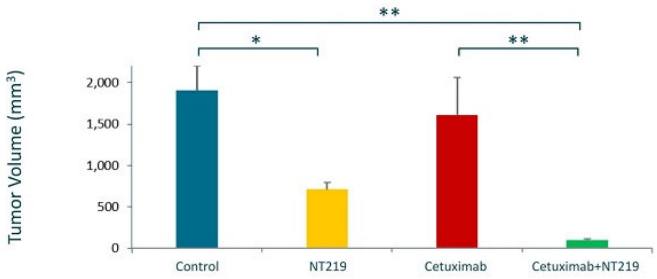
Exon 19 deletion EGFR and T790M, biopsy of bone marrow metastasis, patient previously progressed on afatinib and osimertinib

R/M SCCHN

metastasis, patient previously 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*

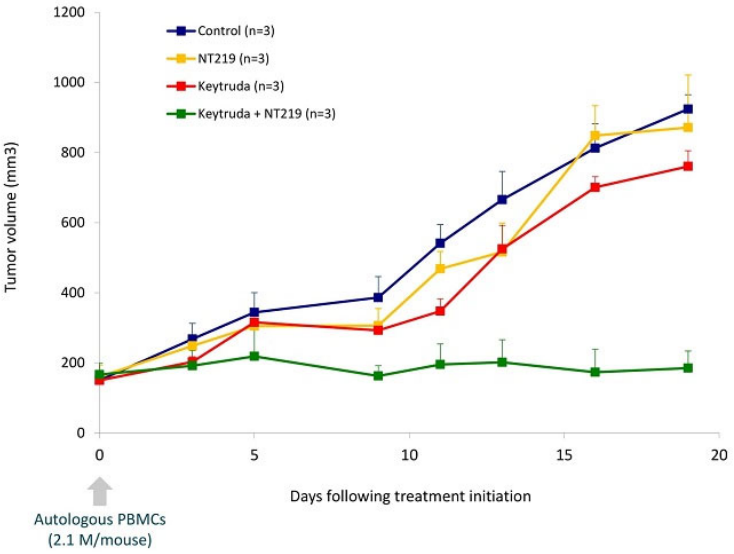
NT219 Re-sensitizes Tumors Refractory to α-PD1

PDX Model

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

Drug

Pembrolizumab (Keytruda®)

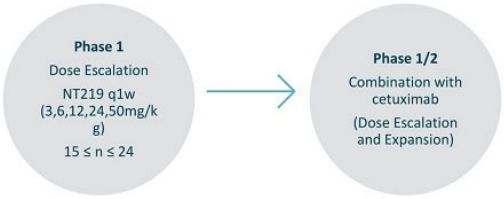


** Double autologous model - Tumors & PBMCs are from the same patient (#RA236) | Keytruda - 6mg/kg IP, NT219 - 60mg/kg IV*

NT219 Phase 1 Dose Escalation Monotherapy
Interim Results

Study Design

- As of data cutoff date of May 12th, 2022, a total of 14 patients were enrolled and 12 patients were evaluable for DLT determination (4 CRC, 3 pancreatic cancer, 2 breast cancer, 1 GEJ, 1 esophageal, and 1 appendiceal cancer)
- Median number of prior treatment regimens for metastatic disease was 4 (2-11).



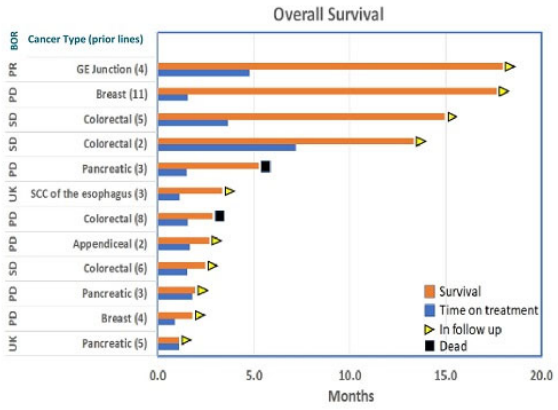
Safety

- No DLTs were observed across all dose levels.
- Nine Grade 3 adverse events (AEs) were observed, two of which possibly related to NT219

AE Term	Total	Grade			
		1	2	3	4/5
Fatigue	6	6			
Constipation	4	4			
ALP increased	3	2		1	
ALT increased	3	1	2		
Anemia	3	1	2		
AST increased	3	1	1	1	
Diarrhea	3	2	1		
Headache	3	3			
Nausea	3	2	1		
Abdominal pain	2	1	1		
Belching	2	2			
Cough	2	2			
Dizziness	2	2			
Dyspnea	2	2			
Edema limbs	2	2			
Fever	2	2			
Hot flashes	2	2			
Hyperhidrosis	2	2			
Urinary tract infection	2		2		
Closed displaced fracture of right femoral neck	1			1	
Intractable right hip pain	1			1	
Malignant hypercalcemia	1			1	
Toxic Encephalopathy	1			1	
Worsening back pain	1			1	
Abdominopelvic Ascites	1			1	

NT219 Phase 1 Dose Escalation Monotherapy
Interim Results: Encouraging Initial Efficacy Signals

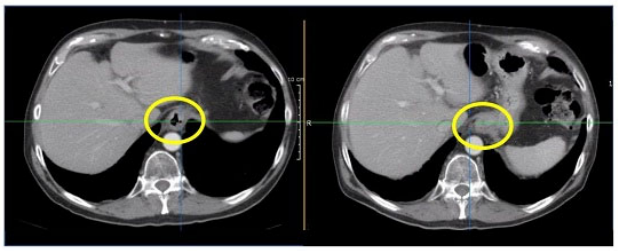
- For the 12 evaluable patients, best overall response included **one confirmed PR** (GEJ patient, > 5.5 months duration of response following end of treatment), and **3 SD** with one patient awaiting follow up MRI/CT scans
- As of the cutoff date (May 12th , 2022), **10/12 patients are either on treatment or in follow up** (range 1.1 to 18 months).



Durable PR in a GEJ patient and SDs in 3 out of 4 mutated KRAS CRC patients

Confirmed PR as Single Agent in a GEJ Cancer Patient

- In a patient with refractory GE junction disease (mutated KRAS, TP53), NT219 administration (3mg/kg as a single agent) was associated with a confirmed partial response (PR):
  - Complete remission at the largest target lesion (right)
  - Complete resolution of all non-target lesions (two lymph nodes) has also been demonstrated
  - The patient remained on treatment for nearly 6 months.



GEJ tumor at baseline screening

CT imaging of the GEJ tumor after 5 months of treatment with NT219

First Market Opportunity

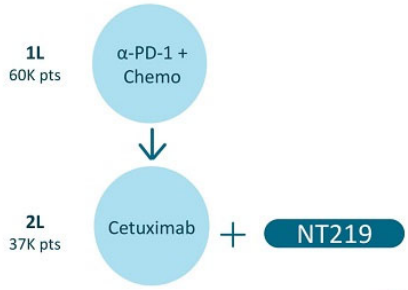
Recurrent or Metastatic Squamous Cell Carcinoma of Head and Neck (SCCHN)

Targeting the unmet medical need

- 1L Standard of care has shifted from chemotherapy towards immuno-oncology + chemotherapy
- < 20% of R/M SCCHN patients respond to Pembrolizumab
- SCCHN is the 6th most common cancer type ; 175k new cases/year are expected by 2024
- 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 the potential to become the SOC in 2L 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, 5 major global territories

NT219 Monotherapy and Combination Phase 1/2 Study Design (NCT04474470)

Study Title

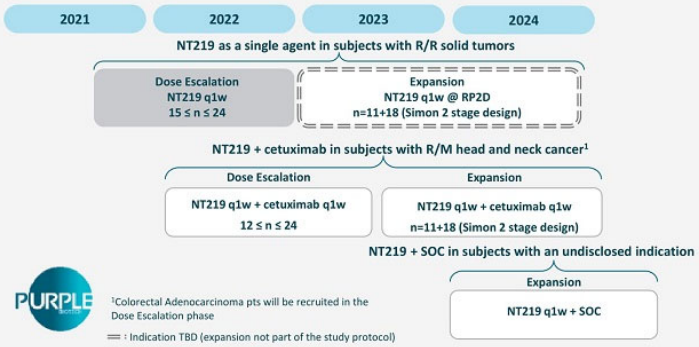
A phase 1/2 study with open-label, dose escalation phase followed by single-arm expansion to assess the safety, tolerability, PK, PD and efficacy of NT219, alone in adults with recurrent or metastatic solid tumors and in combination with Erbitux® (cetuximab) in Head and Neck cancer

Endpoints

Primary endpoints:
Safety, pharmacokinetics and to determine the MTD

Secondary endpoints:
Obtain preliminary efficacy data

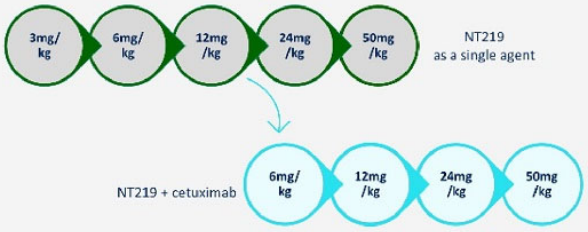
Study Design



[1]Colorectal Adenocarcinoma pts will be recruited in the Dose Escalation phase

= : Indication TBD (expansion not part of the study protocol)

Dose Escalation Design



- Currently treating 5th dose level cohort as single agent (50mg/kg) and 3rd dose level cohort (24mg/kg) in combination with cetuximab (CRC & SCCHN)
- No DLTs, no significant safety concerns have been observed to date

Business Highlights

We are a clinical-stage company committed to the development of first-in-class, effective therapies by harnessing the power of the tumor microenvironment (TME) to overcome drug resistance and improve treatment outcomes for cancer patients

- Two first-in-class clinical stage drugs
- Multiple Phase II studies ongoing and planned
- Lean & global operation
- BD activity to grow our pipeline with innovative assets
- Extended cash runway to end of 2024

Strong balance sheet and cash position

Purple Biotech (NASDAQ/TASE: PPBT)

ADSs outstanding: 18.4M

$35.7M cash as of September 30, 2022

Multiple data read-outs expected in the next 12 months

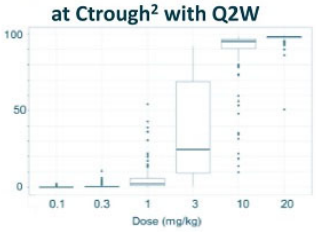

PURPLE
BIOTECH
THANK YOU
We are committed
to provide cancer patients with first-in-class
therapies to OVERCOME
tumor drug resistance,
ENHANCE treatment response
and SLOW tumor progression
Contact Us:
ir@purple-biotech.com

Appendix A | CM24

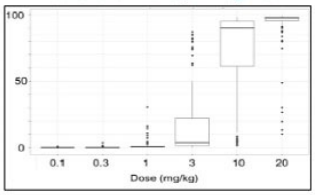
PK/PD Modeling Provides Dosage & Schedule Guidance

- CM24 completed Phase 1 monotherapy open-label, dose-escalation study to assess safety and tolerability
- Heavily pre-treated 24 evaluable patients with a median of 4 prior regimens
- Overall, treatment was well tolerated, no DLTs
- 33% SD (RECIST 1.0), mostly at the highest dose levels of 3mg/kg & 10mg/kg
- PK analysis revealed non-linearity, modeling recommended continuing administration of higher doses to reach saturation, consistent with observed PK showing high clearance at doses <10 mg/kg
- 10 mg/kg has a broad range of saturation
- Q2W regimen preferable to Q3W

Simulated TMDD[1] saturation at Ctrough[2] with Q2W



Predictions with Q3W regimen



[1]Target-mediated drug disposition. [2]Ctrough is the drug concentration reached by CM24 before the next dose is administered

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)

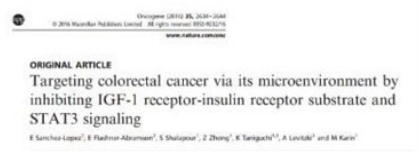
Median age, years (range)	65 (49-76)	Prior Lines of Therapy, n (%)	
Sex, n (%)		1	2 (18%)
Male	5 (45%)	2	9 (82%)
Female	6 (55%)	Diagnosis , n (%)	
Ethnicity, n (%)		Pancreatic cancer	8 (73%)
Not Hispanic or Latino	10 (91%)	Papillary Thyroid cancer	1 (9%)
Hispanic or Latino	1 (9%)	Colorectal cancer	2 (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%)

Appendix B | NT219

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

Therapeutics, Targets, and Chemical Biology

Therapeutic Destruction of Insulin Receptor Substrates for Cancer Treatment

Cancer Research

THE UNIVERSITY OF BRITISH COLUMBIA

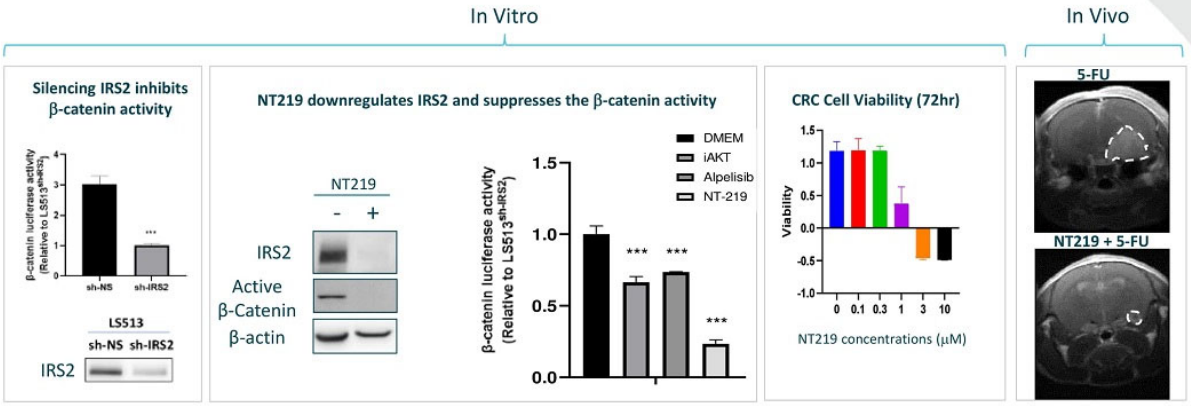
Michael Cox

Small Molecule Therapeutics

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

Molecular Cancer Therapeutics

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



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.

AACR Annual Meeting, April 2021, AACR Virtual Special Conference on Epigenetics and Metabolism, Oct 2020, Ido Wolf, MD, Head of Oncology Division, Tel Aviv Sourasky Medical Center

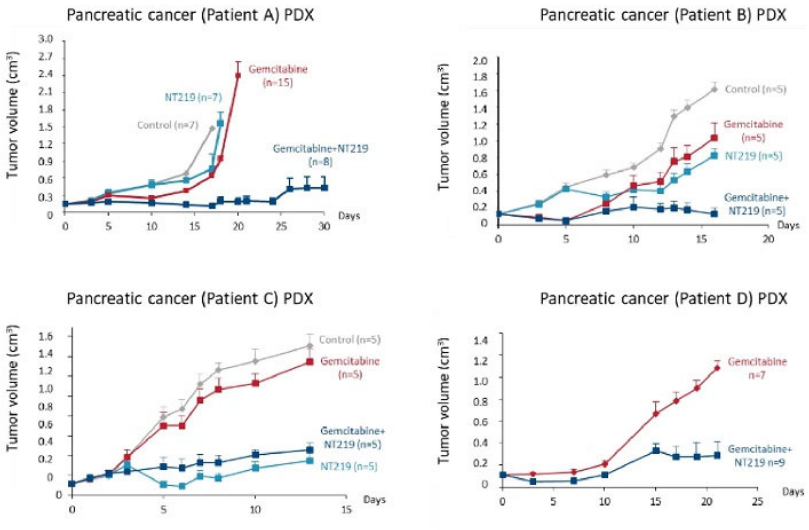
NT219 |
Pancreatic Cancer in Combination with Gemcitabine

PDX model
Pancreatic Cancer

Drug
Gemcitabine (Gemzar®)

PURPLE

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



RNA Sequencing | Analysis of Tumors Following Treatment

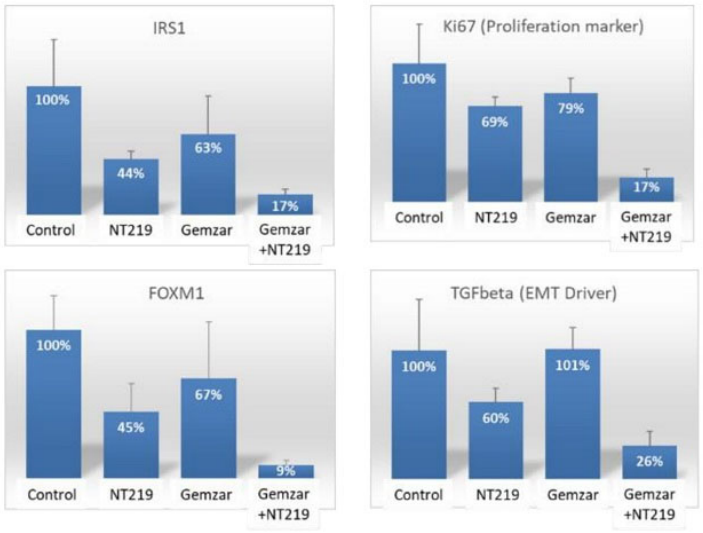
PDX model

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 Phase 1 Dose Escalation Monotherapy
Demographics

As of data cutoff date of May 12th , 2022, a total of 14 patients were enrolled to 4 NT219 dose levels (3 - 24mg/kg)

- 12 patients were evaluable for DLT determination including 4 CRC, 3 pancreatic cancer, 2 breast cancer, and one of each of the following cancers: GEJ, esophageal and appendiceal cancer
- Median number of prior treatment regimens for metastatic disease was 4 (2-11)

Demographics of patients treated with NT219 (3, 6, 12, 24mg/kg)			
Median age, years (range)	67 (39-79)	Diagnosis , n (%)	
Sex, n (%)		Pancreatic	3(25%)
Male	4(33%)	GE Junction	1(8%)
Female	8 (67%)	Breast	2(17%)
Ethnicity, n (%)		Colorectal	4(33%)
Not Hispanic or Latino	11 (92%)	Appendiceal	1(8%)
Hispanic or Latino	1 (8%)	SCC of the esophagus	1(8%)
Race, n (%)		Prior Lines of Therapy, n (%)	
White	10 (83%)	2	2 (17%)
Black or African American	2 (17%)	3	3 (25%)
ECOG, n (%)		4	2(17%)
0	5 (42%)	5	2(17%)
1	7 (58%)	6	1(8%)
Median Time from Initial Diagnosis months (range)	36(10-153)	8	1(8%)
		11	1(8%)