CORPORATE PRESENTATION

NASDAQ/TASE: PPBT
September 2021

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, 2020 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

CM24 - First-in-class α-CEACAM1 mAb, validating clinical collaboration with Bristol Myers Squibb

NT219 - First-in-class, small molecule, dual inhibitor of IRS 1/2 and STAT3

H2:21 - Two phase 1 study readouts

Strong balance sheet and cash position

Purple Biotech (NASDAQ/TASE: PPBT)

ADSs outstanding: 17.5M

$53M cash as of June 30st, 2021

Cash runway into 2024

Advancing Clinical-stage Novel Oncology Therapies

Program	Indication	Preclinical	Phase 1	Phase 2	Phase 3	Partner	Value Drivers
CM24 (CEACAM-1)	Solid tumors (monotherapy) (Completed) Solid tumors (combination with nivolumab) NSCLC (combination with nivolumab) Pancreatic Cancer (combination with nivolumab and nab-paclitaxale)					Bristol Myers Squibb™	Expansion arms on RP2D: Initiation Q4:21 **Topline data: H2:21**
NT219 (IRS1/2 & STAT3)	Solid tumors (monotherapy) R/M SCCHN & CRC (dose escalation with cetuximab); R/M SCCHN (expansion - combination with cetuximab on RP2D)						Expansion arms on RP2D: Initiation Q4:21 **Topline data: H2:21**

Expansion | Done / Ongoing | Planned study

Multiple data read-outs expected in the next 12 months

Experienced Leadership

Isaac Israel
Chief Executive Officer
Former CEO of BeeContact Ltd. (TASE:BCNT). NextGen Biomed (TASE: NXGN)

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

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

Hadas Reuveni, Ph.D
Vice President, R&D
Formerly at Keryx (NASDAQ:KERX)

Bertrand Liang, MD, Ph.D, MBA/AMP, FAAN
Chief Medical Officer
Formerly at Biogen Idec, Amgen, NCI

Michael Schickler, Ph.D
Head of Clinical & Regulatory Affairs
Formerly at Hoffmann-La Roche, CEO at CureTech

Advancing First-in-Class Oncology Therapies

CM24 - an α-CEACAM1 mAb

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

Journal of Cancer Science and Research ISSN: 2576-1447

*"**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

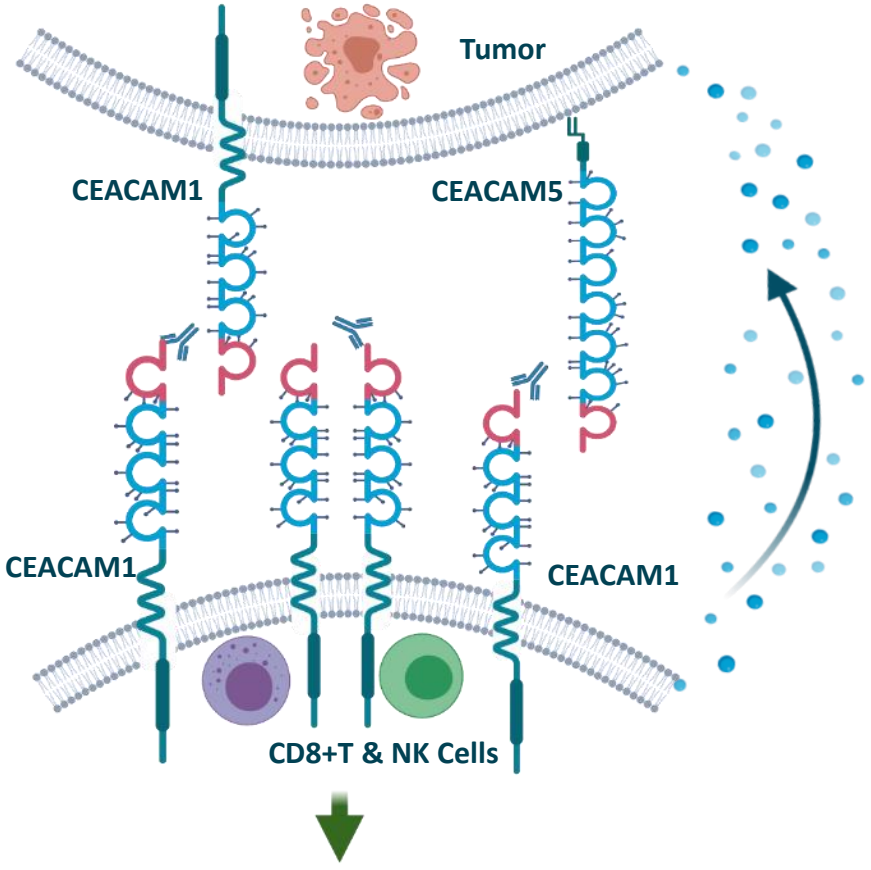
Experimental Cell Research

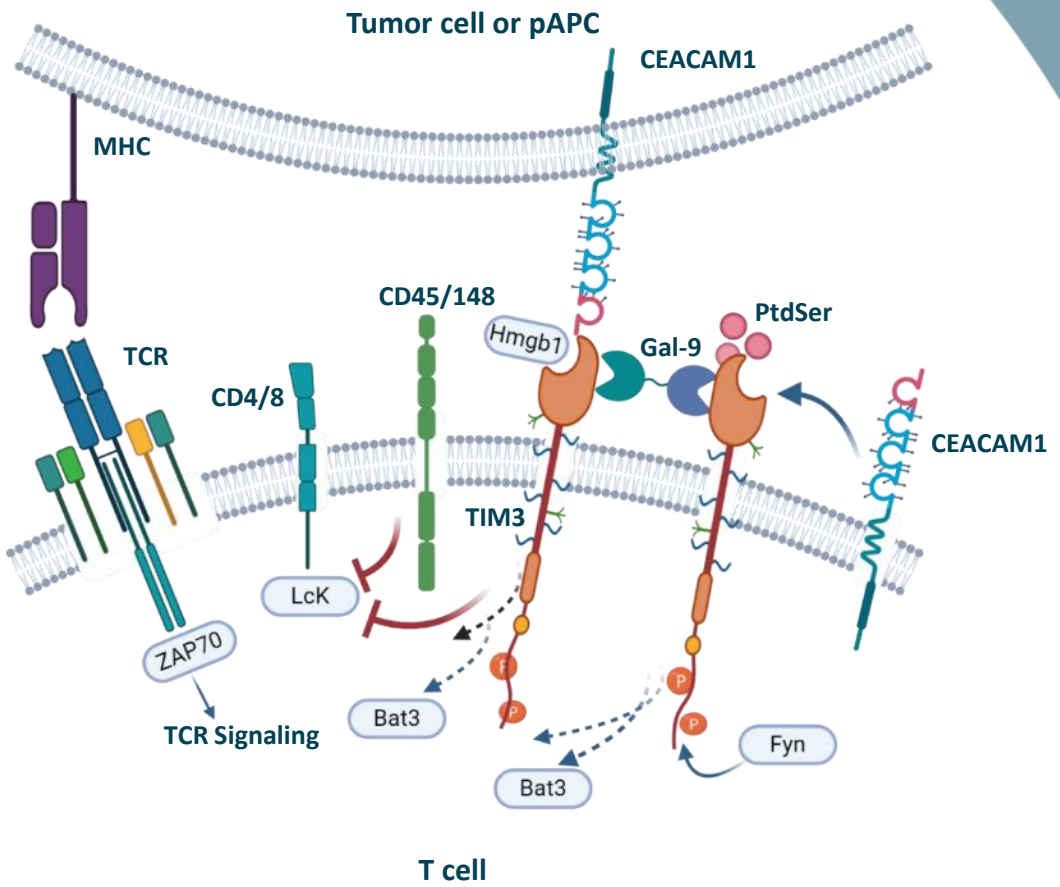
*"CEACAM1 **regulates Fas-mediated apoptosis** in Jurkat T-cells via its interaction with β-catenin"*

PURPLE BIOTECH

*Carcinoembryonic Antigen Cell Adhesion Molecule

CM24 MOA | Immuno-oncology

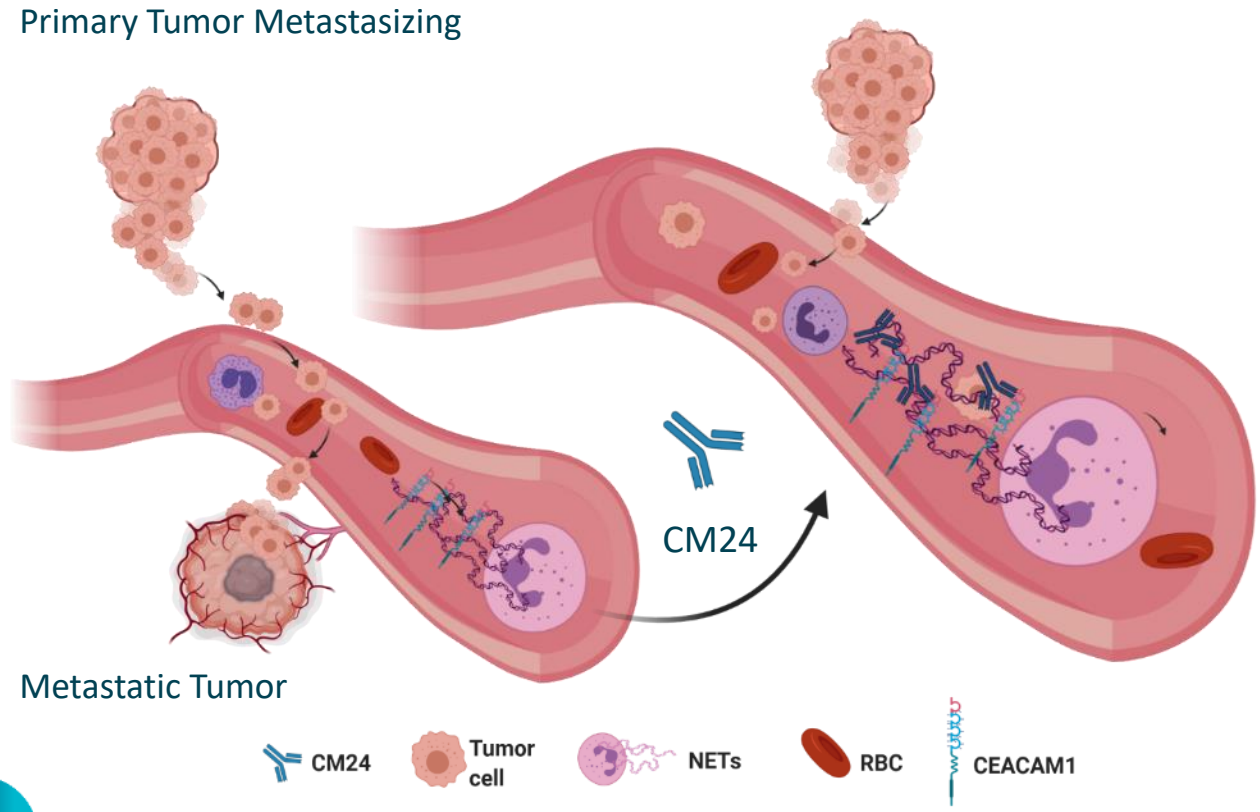


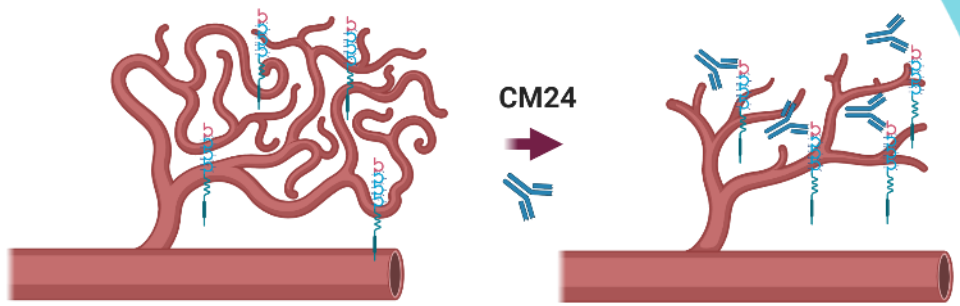


Markel et al, J Immunol 2002, 2006; Immunology, 2008; Cancer Immunol Immunother 2010; Ortenberg et al, Mol Cancer Ther 2012; Zhou, 2009; Li, 2013; Huang, 2015; Acharya N, et al. J Immunotherapy Canc 8:e911-22, 2020.

CM24 MOA | Adhesion

Neutrophil extracellular trap-associated CEACAM1 as a putative therapeutic target to prevent metastatic progression:





CEACAM1 creates a pro-angiogenic tumor microenvironment that supports tumor vessel maturation. CM24 is proposed to attenuate/block tumor growth and metastatic processes.

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)

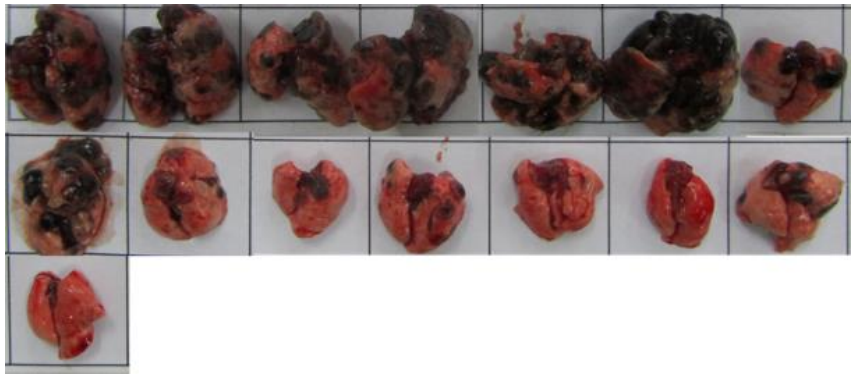
Anti-cancer Effect Following Treatment

Preclinical Data With CM24 + TIL and CM24 + α-PD1

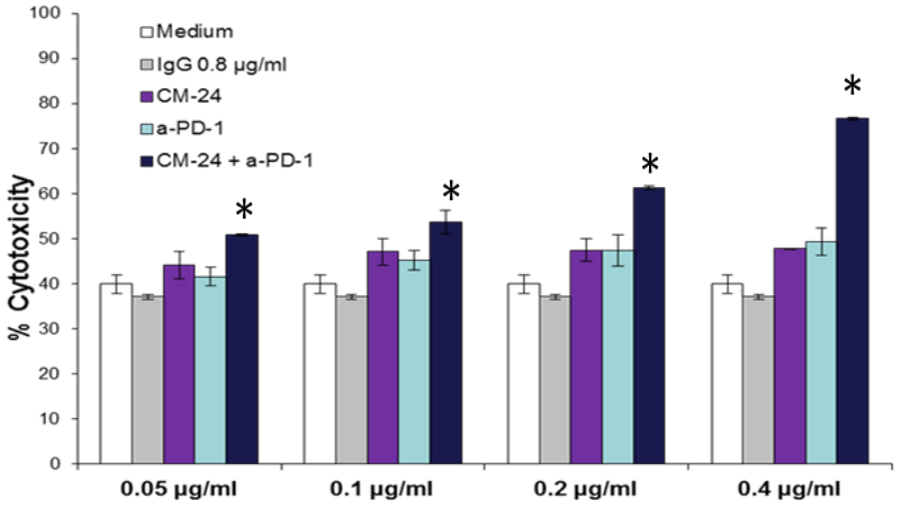
TIL + IgG

TIL + CM24

Naïve



- Xenograft, lung lesion, Mel 526 (IV)
- Tumor burden was monitored 26 days post last CM24 treatment



Combination index (CI) = 0.15

$$CI = \frac{(D)_1}{(D_x)_1} + \frac{(D)_2}{(D_x)_2} < 1 \rightarrow \text{synergy}$$

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

CM24 Phase 1 Monotherapy Trial

UCLA YALE-NEW HAVEN HOSPITAL

- Open-label, multi-dose escalation monotherapy study to assess safety and tolerability
- Heavily pre-treated patients with a median of 4 prior regimens (range 2-8) Overall, treatment was well tolerated
- Most of the responding patients were treated with the highest dose levels of 3mg/kg & 10mg/kg
- PK analysis revealed non-linearity, modeling recommended to continue administration of higher doses to reach saturation

27 patients:

Colorectal	11
Melanoma	7
Ovarian	4
Gastric	3
NSCLC	2

**24 patients evaluated*

	# pts
0.01mg/kg	1
0.03mg/kg	1
0.1 mg/kg	3
0.3 mg/kg	3
1.0 mg/kg	3
3.0 mg/kg	9
10.0 mg/kg	7

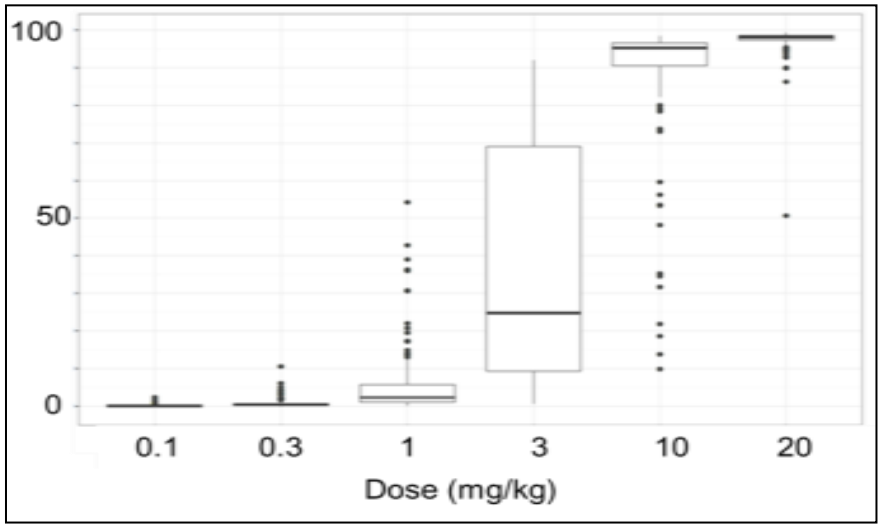
PURPLE BIOTECH

No DLTs up to 10 mg/kg	No discontinuation of study drug due to an AE	No drug related mortalities	33.3% SD (RECIST)

PK/PD Modeling Provides Dosage & Schedule Guidance

- Consistent with observed PK showing high clearance at doses <10 mg/kg, plot shows low TMDD saturation at low doses
- 10 mg/kg has a broad range of saturation, and >10 mg/kg, Q2W dose is needed for saturation across population
- Nivolumab administered Q2W or Q4W, representing good clinical and commercial fit for CM24

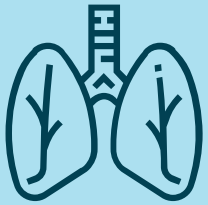
Simulated TMDD[1] saturation at Ctrough with Q2W regimen



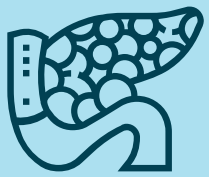
Phase 1b/2a study will continue escalating the CM24 dose above 10mg/kg q2wk, in combination with nivolumab

[1]Target-mediated drug disposition. [2] OPDIVO® is a registered trademark of Bristol-Myers Squibb. Ctrough is the drug concentration reached by CM24 before the next dose is administered

Large Market Opportunity in NSCLC & Pancreatic Cancer



- NSCLC accounts for ~200K new cases/year in the US; with a 5-year relative survival rate of 23%[2]
- Immunotherapy is now recommended as first line therapy in all patients with NSCLC and no driver mutations[3]
- 5-year overall survival rates with chemotherapy in 2L is 2.6% and with I/O Opdivo® is 13.4%[4]



- Pancreatic Cancer accounts for ~60K new cases/year in the US; with a 5-year relative survival rate of 10%[2]
- 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 rates with chemotherapy in 2L is 3%[2]

Combining nivolumab with CM24 in a clinical collaboration with

Bristol Myers Squibb™

- CEACAM1 expression correlates with poor prognosis in NSCLC and Pancreatic cancer[1]
- Preclinical data support significant synergy
- Receptor saturation with CM24 is better achieved with a Q2W regimen, aligning with the nivolumab regimen

1 Dango et al, Lung Cancer 2008; 60:426 & Calinescu et al, Journal of Immunology Research 2018: 7169081.
2 American Cancer Society, Cancer Facts & Figures 2019, and the ACS website, https://seer.cancer.gov/statfacts/html/pancreas.html
3 Economopoulou P, Mountzios G. The emerging treatment landscape of advanced non-small cell lung cancer. Ann Transl Med. 2018;6(8):138. doi:10.21037/atm.2017.11.07
4 Gettinger S, et al "Five-year outcomes from the randomized, phase III trials CheckMate 017/057: Nivolumab vs docetaxel in previously treated NSCLC" WCLC 2019; Abstract OA14.04.

CM24 Phase 1/2 Combination Study Design (NCT04731467)

A Phase 1/2 open label multi center study of CM24 in combination with:

- Nivolumab in selected cancer indications (Phase 1) and NSCLC (Phase 2)
- Nivolumab and nab-paclitaxel in pancreatic cancer (Phase 2)

Primary endpoints:

Phase 1: Evaluate the safety, PK and the MTD/RP2 dose

Phase 2: Evaluate preliminary efficacy in 2nd line NSCLC and pancreatic cancer

Measurement of CEACAM1 based bio-marker.

Exploring further studies in other tumor types as well as monotherapy

2021	2022	2023-24

Dose Escalation

Doses: 10, 15, 20mg/kg q2wk
+ nivolumab (480mg q4w)
3+3 design
$9 \leq n \leq 15$

Indications: NSCLC, Pancreatic, Ovarian, CRC, Melanoma, Papillary Thyroid Carcinoma

Expansions

CM24 (@RP2 dose) + nivolumab (480mg) q4w
I/O refractory NSCLC; 2nd line
n=13+14 (Simon 2 Stage Design)

CM24 (@RP2 dose) + nivolumab (480mg) q4w
+ nab-paclitaxel
Locally advanced, unresectable pancreatic cancer; 2nd line
n=13+14 (Simon 2 Stage Design)

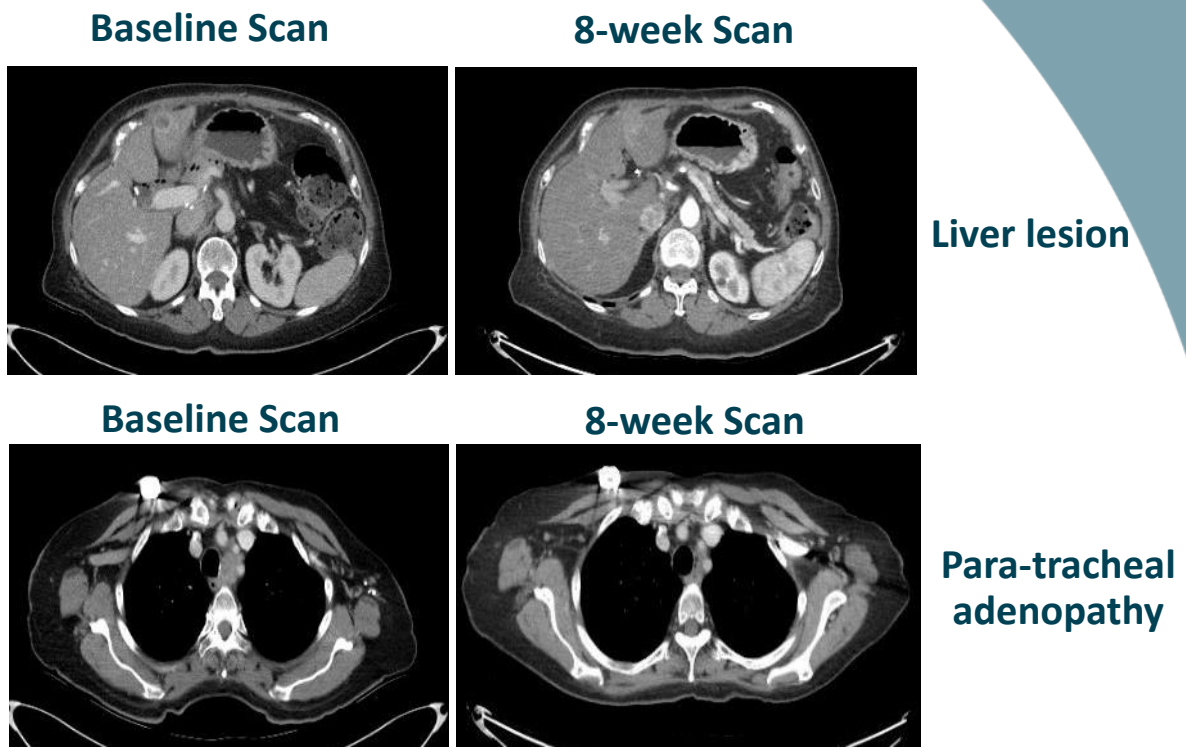
Clinical collaboration with:

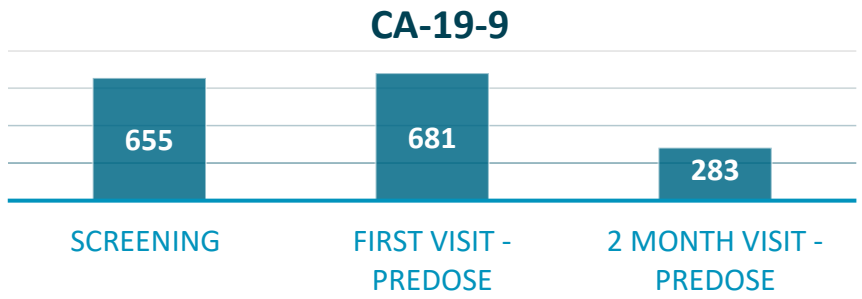
Bristol Myers Squibb™

1st Cohort Analysis – SAFETY and RESPONSE

10mg/kg Dose Level in combination with nivolumab

- The administration of CM24 at 10mg/kg q2wks in combination with nivolumab at 480mg q4wks was well tolerated with no SAEs in patients with refractory PDAC
- Three patients were enrolled into the first dose cohort, all with PDAC. Two patients progressed after 1.5 and 2 months of treatment
- The third patient, previously treated with FOLFIRINOX and gemcitabine/nab-paclitaxel, had a germline NF1 VUS, with MSI-S and PDL-1 IHC 2+ and 5% staining
- After initial treatment, the patient had a Partial Response of 40%, with a definite reduction of the para-tracheal adenopathy and liver lesions and 56% reduction in CA19-9 levels





Advancing First-in-Class Oncology Therapies

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

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 tumor types
- Regulates major survival pathways such as the PI3K/AKT, MEK/ERK and WNT/β-catenin
- Activated as a feedback response to anti-cancer therapies

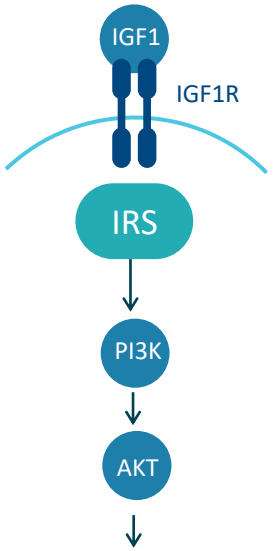
STAT3

- Well-established transcription factor associated with the tumorigenic phenotype
- Provides a crucial axis to support cell proliferation and survival
- Active in tumor JAK/STAT3 and TGF-β resistance mechanisms

[1]Hadas Reuveni et al.; Cancer Res 2013;73:4383-4394. 2013 , [2]Machado-Neto, et al. Clinics (Sao Paulo, Brazil) vol. 73,suppl 1 e566s. 11 Oct. 2018, doi:10.6061/clinics/2018/e566s
[3]Naokazu Ibuki1,2, Mazyar Ghaffari1,3, Hadas Reuveni4 et al. DOI: 10.1158/1535-7163.MCT-13-0842 Published December 2014; [4]Rampias T, Favicchio R, Stebbing J, Giamas G. 2016 May 19;35(20):2562-4. doi: 10.1038/onc.2015.392. Epub 2015 Oct 19. PMID: 26477311
[5]Flashner-Abramsonet al.. Oncogene. 2016 May 19;35(20):2675-80. doi: 10.1038/onc.2015.229. Epub 2015 Jun 29. PMID: 26119932, [6]Sanchez-Lopez E,. Oncogene. 2016 May 19;35(20):2634-44. doi: 10.1038/onc.2015.326. Epub 2015 Sep 14. PMID: 26364612; PMCID: PMC4791217.
[7]Zhao C, et al. Trends Pharmacol Sci. 2016 Jan;37(1):47-61. doi: 10.1016/j.tips.2015.10.001. Epub 2015 Nov 12. PMID: 26576830, [8] Johnson, Daniel E et al. "Targeting the IL-6/JAK/STAT3 signaling axis in cancer." Nature reviews. Clinical oncology vol. 15,4 (2018): 234-248. doi:10.1038/nrclinonc.2018.8

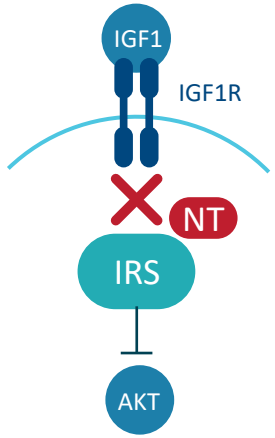
Novel MOA: IRS Degradation By NT219

Blocking IGF1R-AKT Pathway[1]



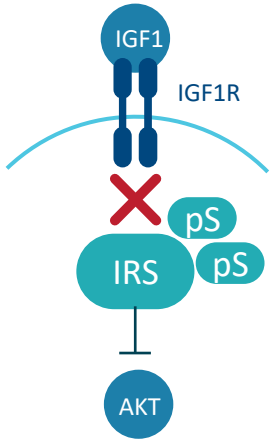
SURVIVAL

1 Binding to IRS



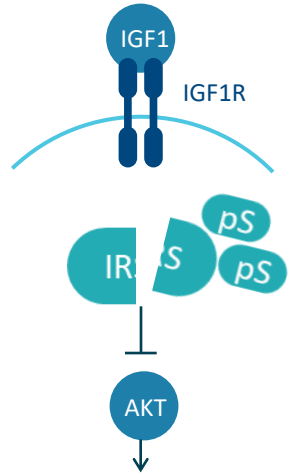
Covalent binding to IRS1/2 leads to the dissociation of IRS1/2 from IGF1R

2 Ser-phosphorylation



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

3 Degradation



APOPTOSIS

IRS1/2 is degraded by the proteasome

[1]*Reuveni et al. Cancer Res 2013*

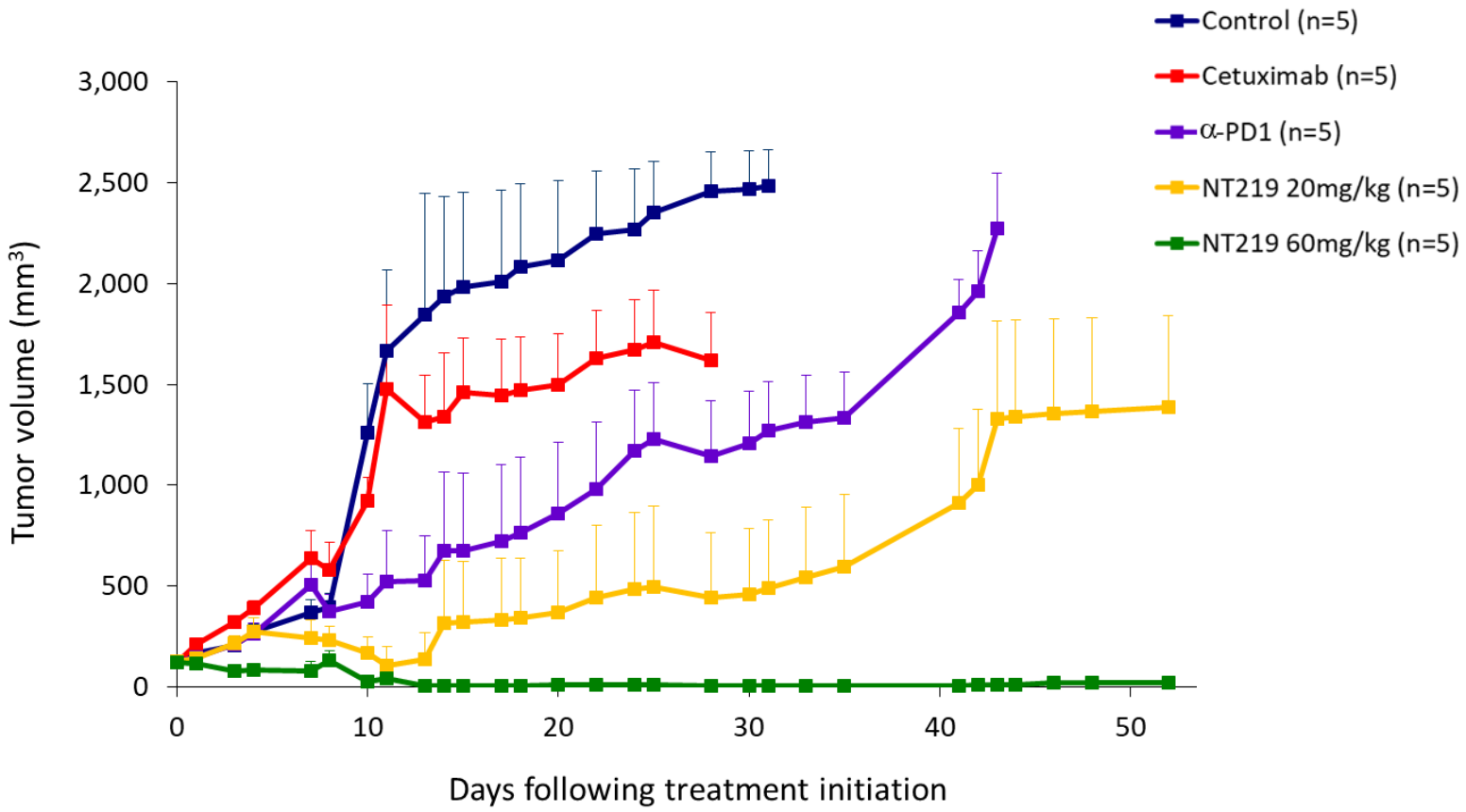
NT219 Efficacy as Monotherapy

Animal model

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

Drugs

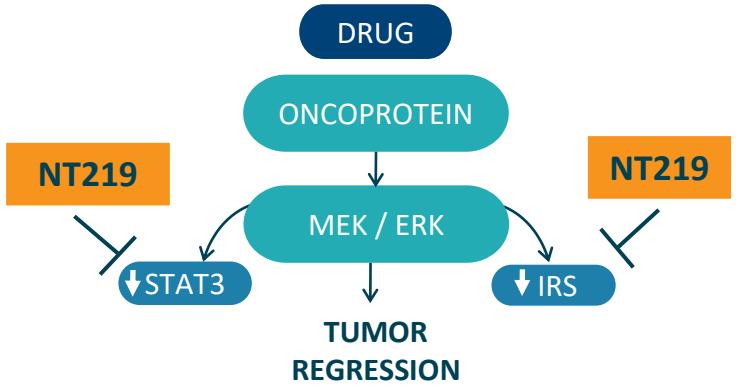
α-PD1
Cetuximab (Erbitux®)
NT219 20mg/kg
NT219 60mg/kg



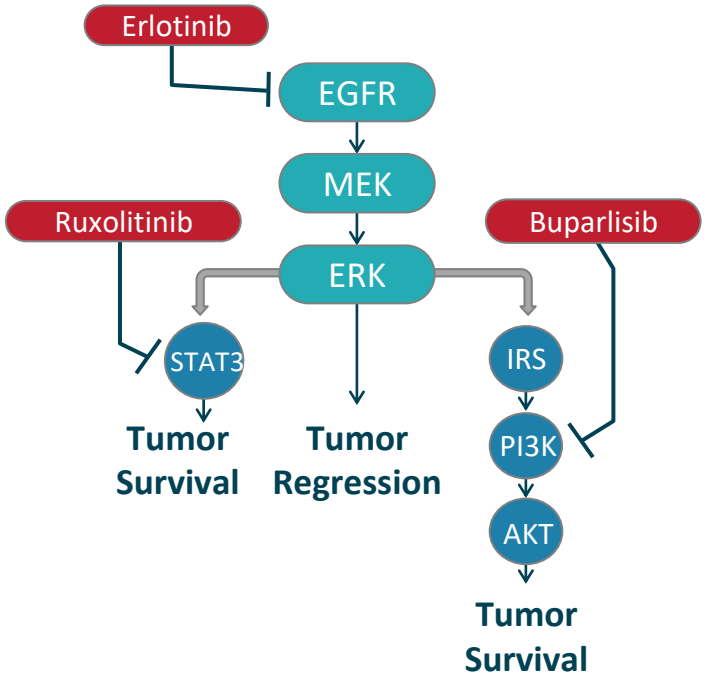
*1 NSG mice were injected SC with SCC-9 cells. PBMCs (18*10⁶ cells per mouse) administered 4 weeks prior to first treatment. NT219, α-PD1, and cetuximab were administered IV (NT219) and IP (α-PD1 and cetuximab) twice a week for 4 weeks. Graph reflects relevant data adapted from 2020 Multidisciplinary Head and Neck Cancers Symposium Poster presentation*

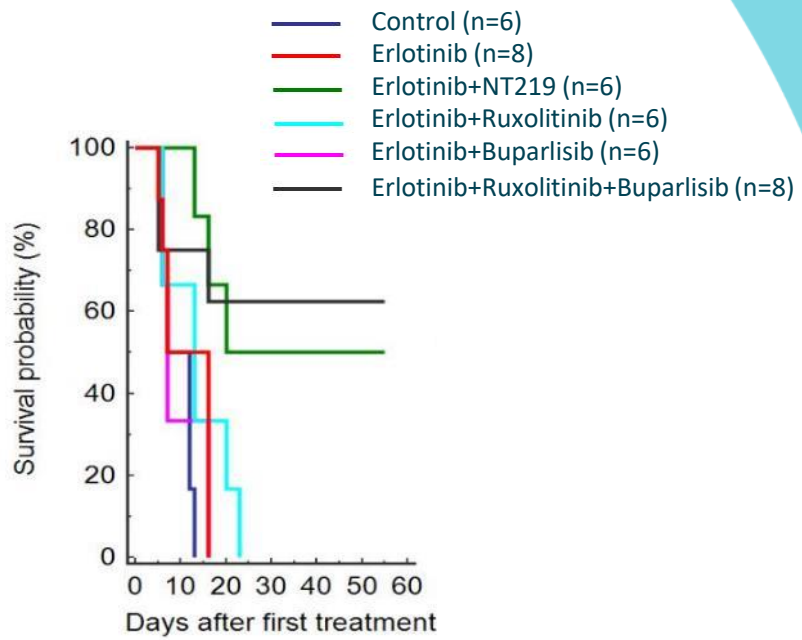
STAT3 and IRS are Essential in Therapeutic Resistance

Blocking survival pathways



Proof of Concept: PDX model of Head and Neck Cancer





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

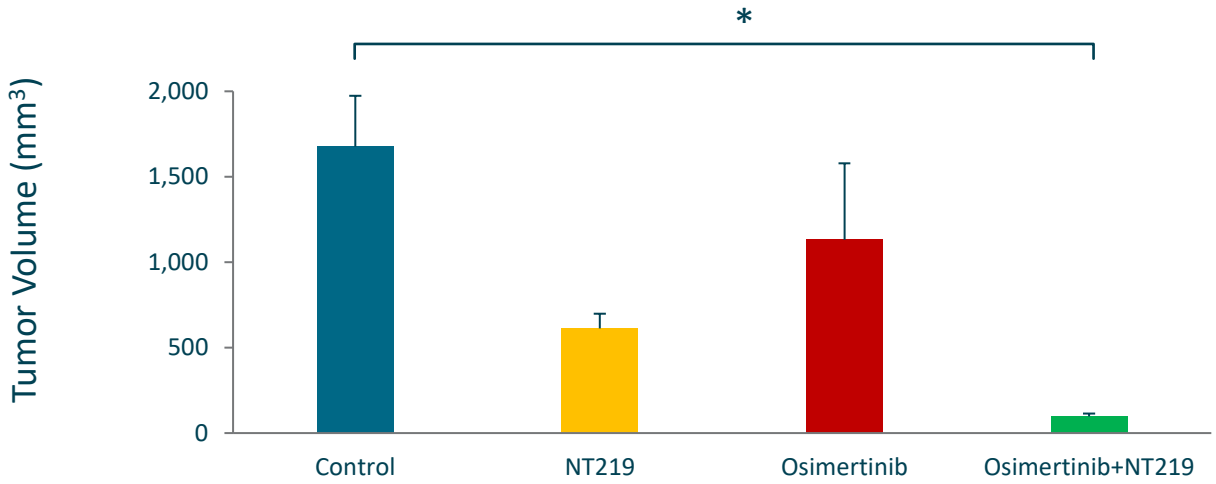
NT219 + Targeted Therapies Established Efficacy in PDX Models

NSCLC

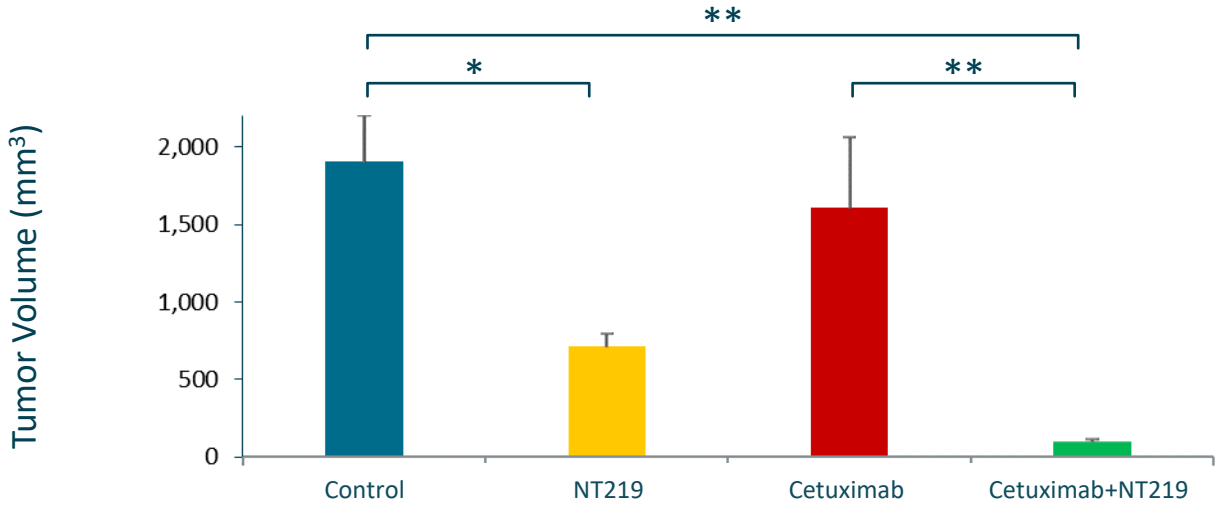
Exon 19 deletion EGFR and T790M, biopsy of bone marrow metastasis, patient previously progressed on afatinib and osimertinib

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*

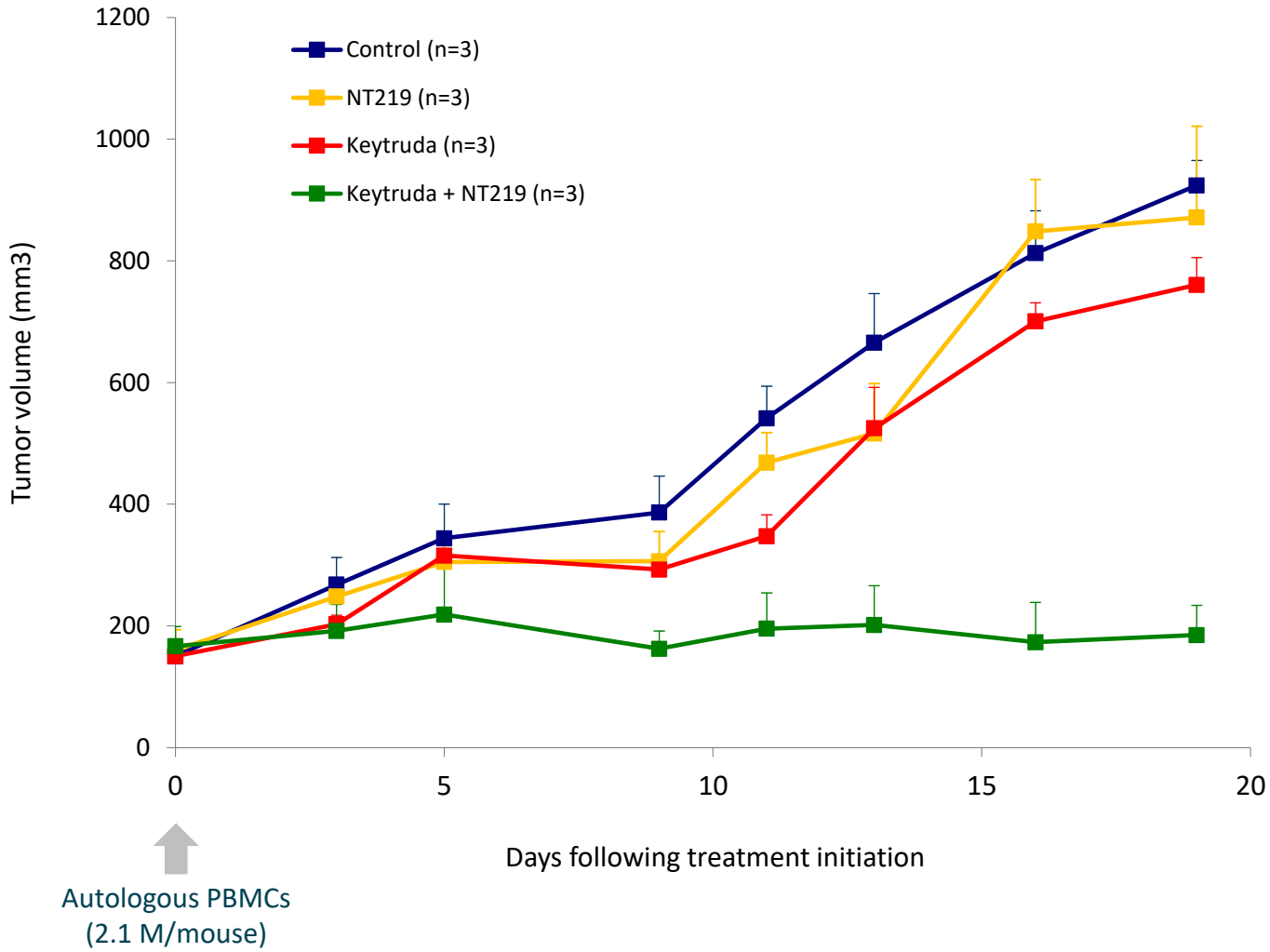
NT219 + α-PD1 Re-sensitizes to Refractory α-PD1 Tumors

PDX Model

Humanized PDX of Esophagus 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*

First Market Opportunity

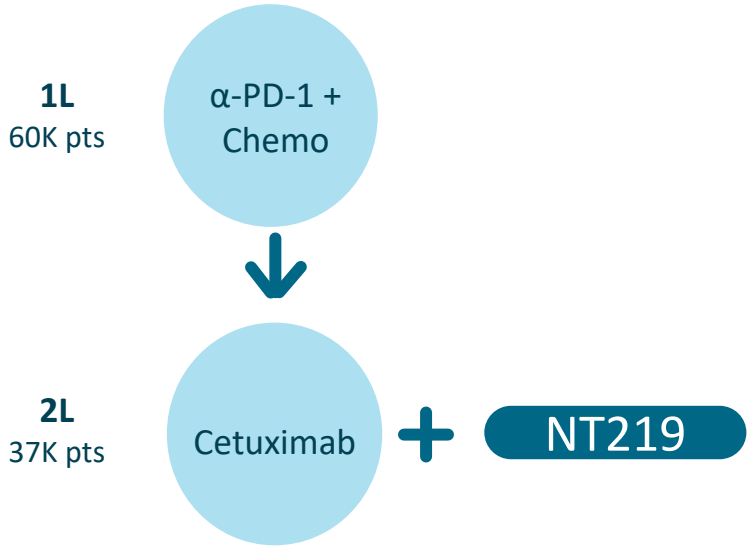
Recurrent or Metastatic Squamous Cell Carcinoma of Head and Neck (SCCHN)

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 α-PD1s
- 175k new cases/year are expected by 2024

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 an attractive 2nd line therapy

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

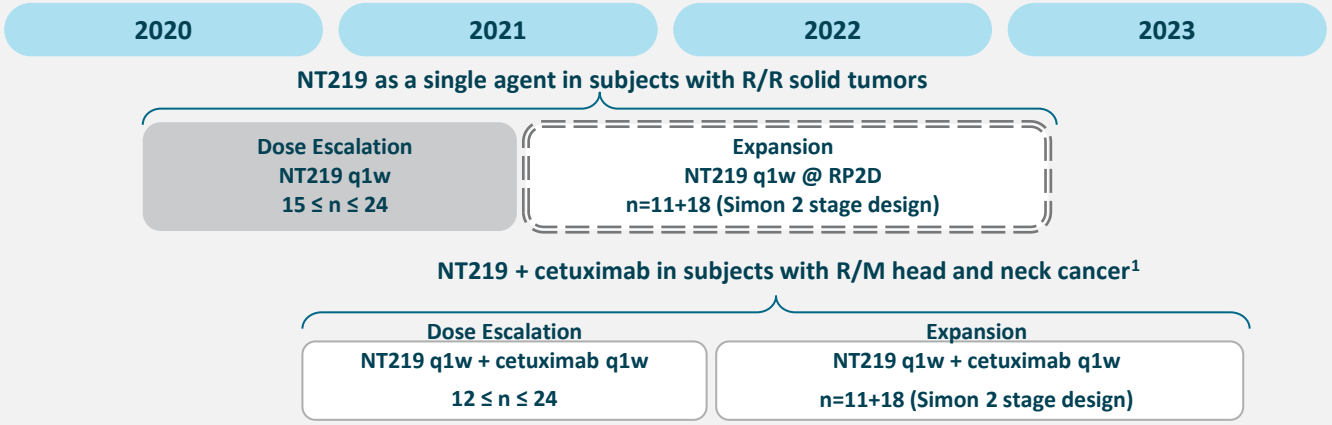
Primary endpoints:

Safety, pharmacokinetics and to determine the MTD

Secondary endpoints:

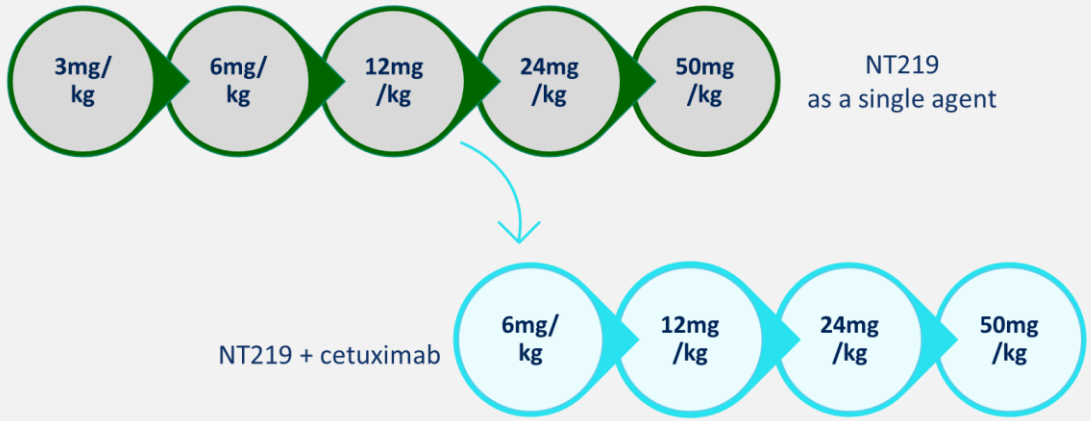
Obtain preliminary efficacy data

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



Interim Analysis – SAFETY and RESPONSE

3 mg/kg Dose Level as a Single Agent

- NT219 has been found to be well-tolerated with minimal adverse events
- In a patient with refractory GE junction disease, NT219 administration was associated with a partial response (PR):
  - ✓ Complete remission at the largest target lesion and one non-target central mesenteric lymph node
  - ✓ Stable Disease at the other non-target lesion pre-carinal lymph node

Response Analysis

	Cancer Type	Prior Lines of Therapy	Treatment Duration(Weeks)	Best Response*	
NT219 3mg/kg	Pancreatic Cancer	3	8	PD	
	GE Junction Cancer	4	22	**PR**	Target lesion: **Absent**
					Non target lesion 1: **Absent**
					Non target lesion 2: **Stable**
	Breast Cancer	11	8	PD	

*Interim data

Business Highlights

CM24 - First-in-class α-CEACAM1 mAb, validating clinical collaboration with Bristol Myers Squibb

NT219 - First-in-class, small molecule, dual inhibitor of IRS 1/2 and STAT3

H2:21 - Two phase 1 study readouts

Strong balance sheet and cash position

Purple Biotech (NASDAQ/TASE: PPBT)

ADSs outstanding: 17.5M

$53M cash as of June 30st, 2021

Cash runway into 2024

We are committed

to providing cancer patients with first-in-class therapies to OVERCOME tumor drug resistance, ENHANCE treatment response and SLOW tumor progression

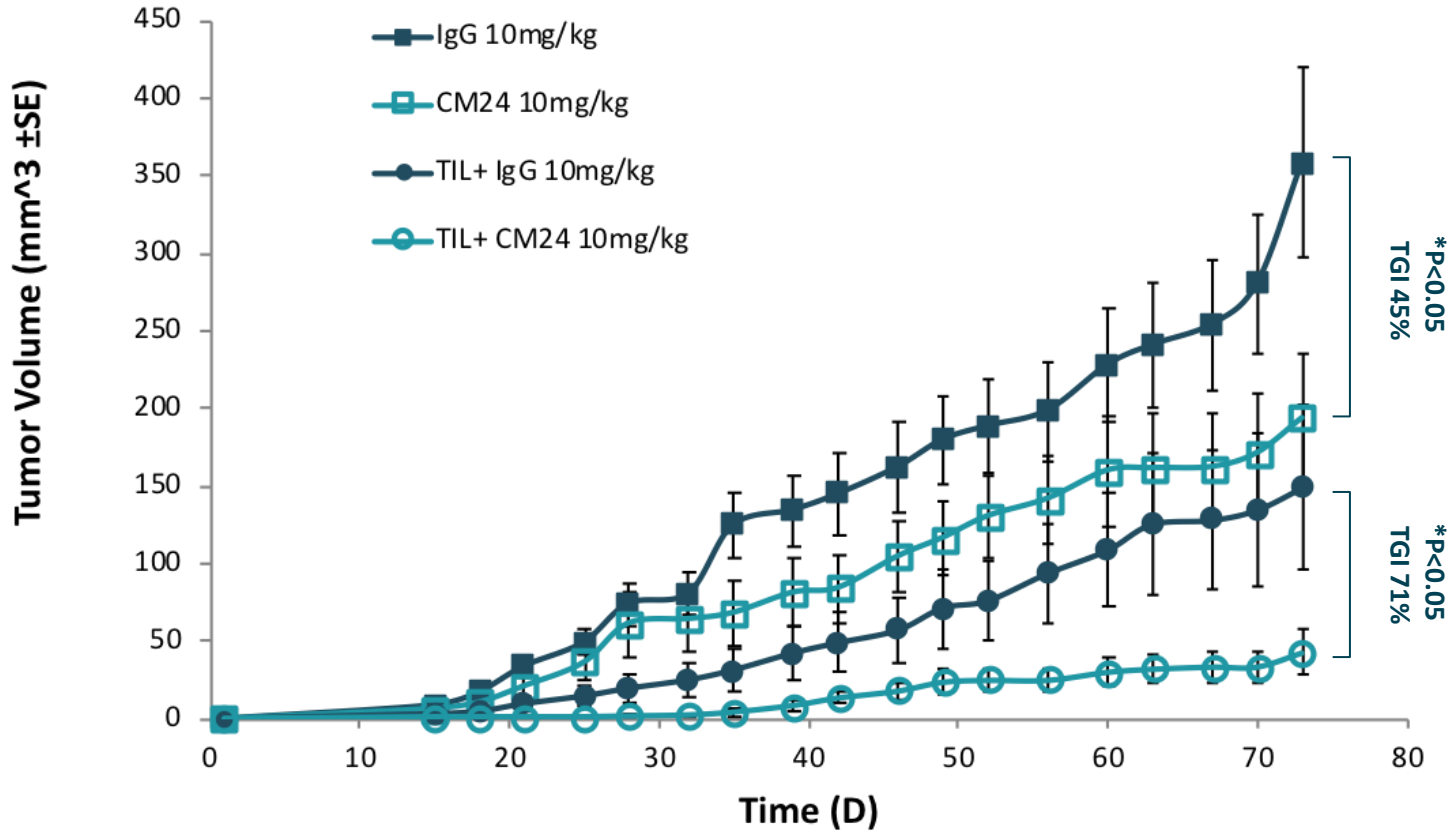
THANK YOU

Contact Us:
ir@purple-biotech.com

Appendix A - CM24

Inhibition of Melanoma Growth Following CM24 and CM24 + TIL Treatment

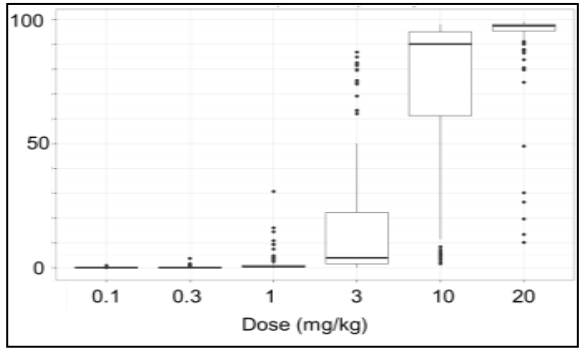
CM24 activity is Demonstrated as Single Agent and in Combination with TILS



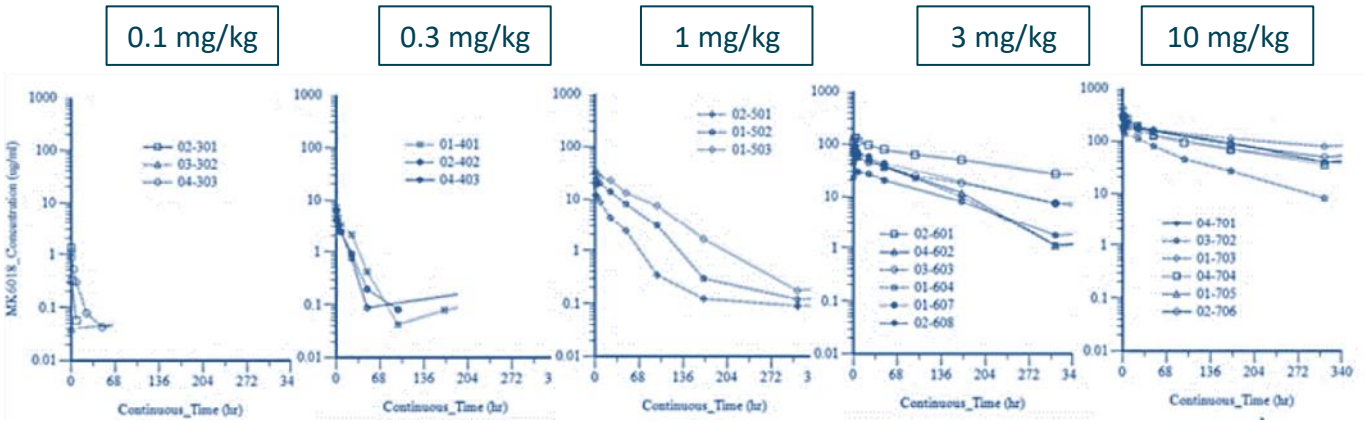
PHASE 1 PK DATA

Target saturation was not reached with doses up to 10mg/kg

Predictions with Q3W regimen



- Modeling with increased interval between doses demonstrates lower TMDD at 10mg/kg dose
- With Q3W, 20mg/kg dosing is not sufficient for saturation across population



Slower clearance with increasing dose

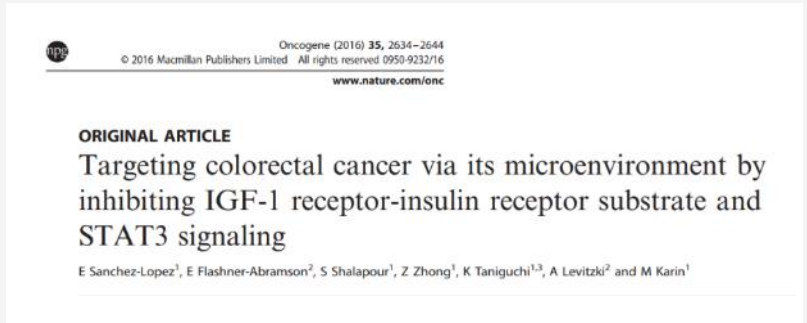
Higher half-life with increasing dose

Appendix B - NT219

Selected Publications

UC San Diego School of Medicine

Michael Karin



Oncogene (2016) 35, 2634–2644
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www.nature.com/onc

ORIGINAL ARTICLE

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

E Sanchez-Lopez[3], E Flashner-Abramson[2], S Shalapour[1], Z Zhong[1], K Taniguchi[1,3], A Levitzki[2] and M Karin[1]

האוניברסיטה העברית בירושלים
THE HEBREW UNIVERSITY OF JERUSALEM

Alexander Levitzki

Oncogene (2016) 35, 2675–2680
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www.nature.com/onc

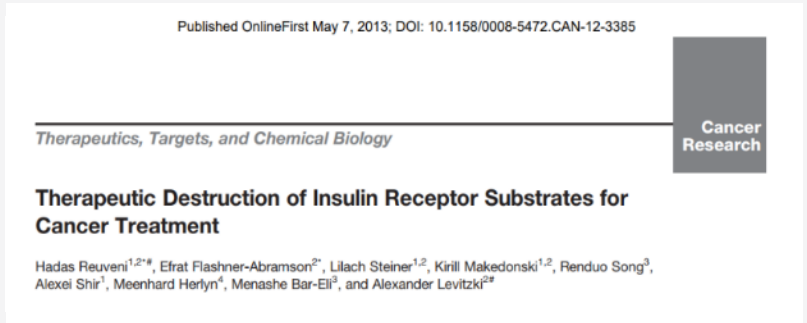
SHORT COMMUNICATION

Targeting melanoma with NT157 by blocking Stat3 and IGF1R signaling

E Flashner-Abramson[1], S Klein[1], G Mullin[1], E Shoshan[2], R Song[2], A Shir[1], Y Langut[1], M Bar-Eli[2], H Reuveni[1,3,4,5] and A Levitzki[1,4]

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

Hadas Reuveni[1,2,*,#], Efrat Flashner-Abramson[2*], Lilach Steiner[1,2], Kirill Makedonski[1,2], Renduo Song[3], Alexei Shir[1], Meenhard Herlyn[4], Menashe Bar-Eli[3], and Alexander Levitzki[2#]

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

Naokazu Ibuki[1,2], Mazyar Ghaffari[1,3], Hadas Reuveni[4,5], Mitali Pandey[1], Ladan Fazli[1], Haruhito Azuma[2], Martin E. Gleave[1,6], Alexander Levitzki[5], and Michael E. Cox[1,6]

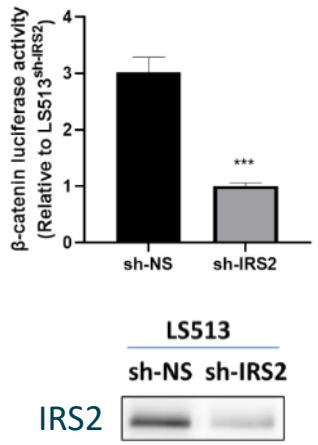
PURPLE BIOTECH

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

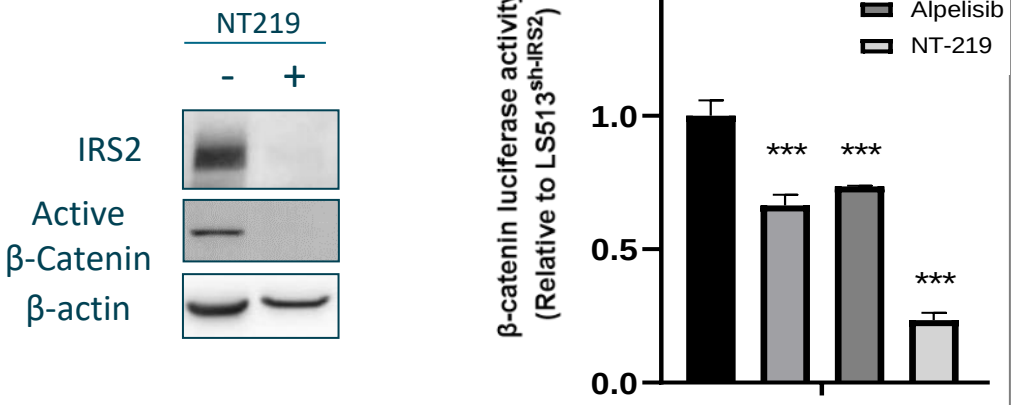
In Vitro

In Vivo

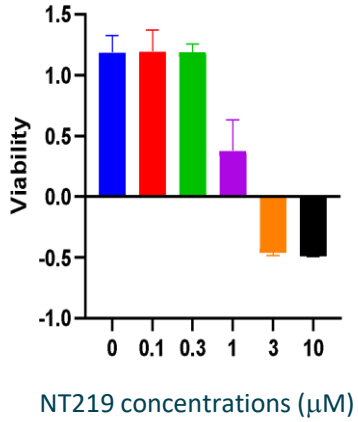
Silencing IRS2 inhibits β-catenin activity



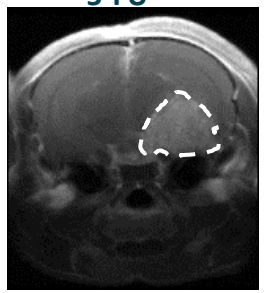
NT219 downregulates IRS2 and suppresses the β-catenin activity



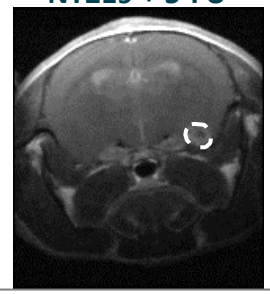
CRC Cell Viability (72hr)



5-FU



NT219 + 5-FU



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

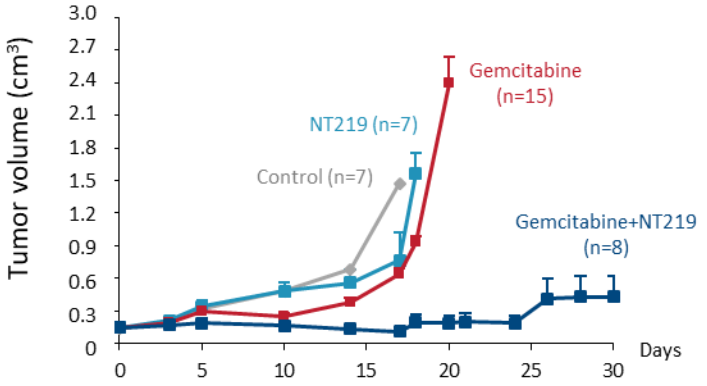
Pancreatic Cancer

Drug

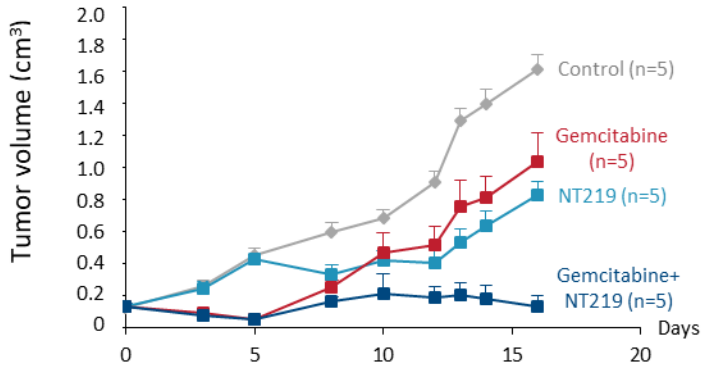
Gemcitabine (Gemzar®)

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

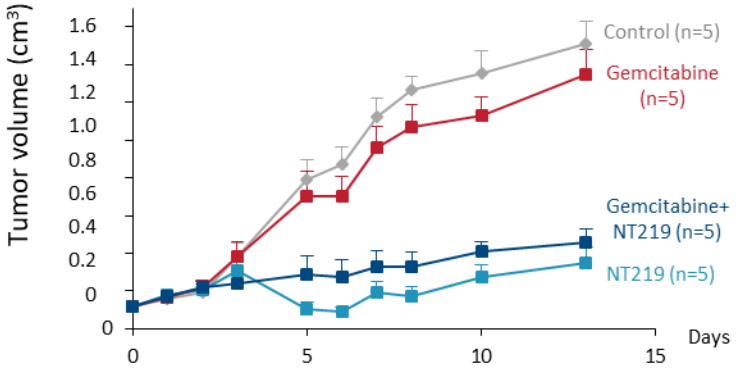
Pancreatic cancer (Patient A) PDX



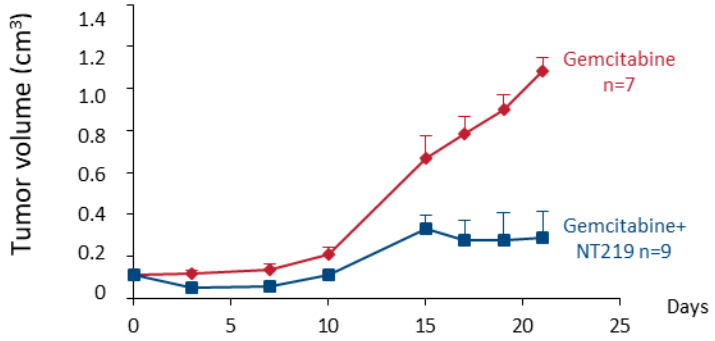
Pancreatic cancer (Patient B) PDX



Pancreatic cancer (Patient C) PDX



Pancreatic cancer (Patient D) PDX



RNA Sequencing | Analysis of Tumors Following Treatment

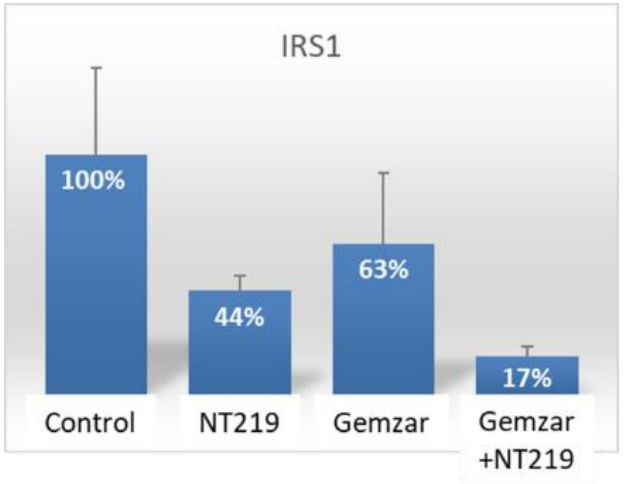
PDX model

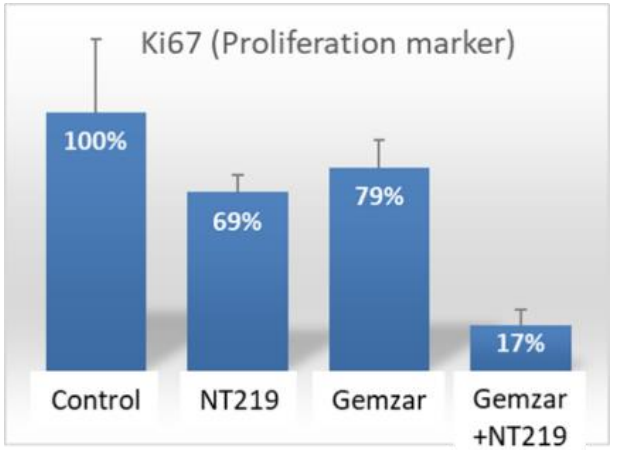
Pancreatic Cancer

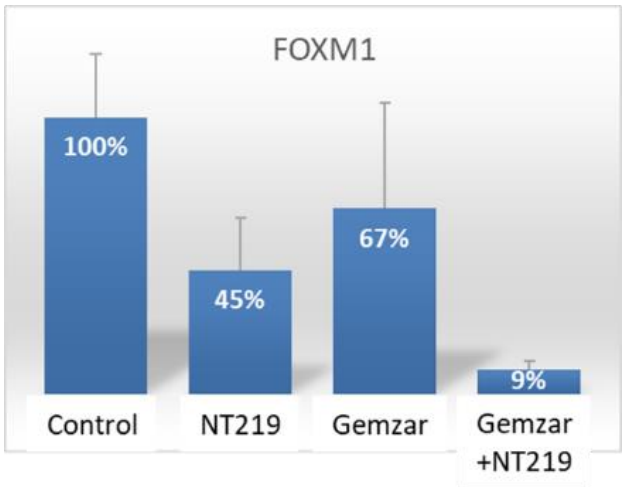
Drug

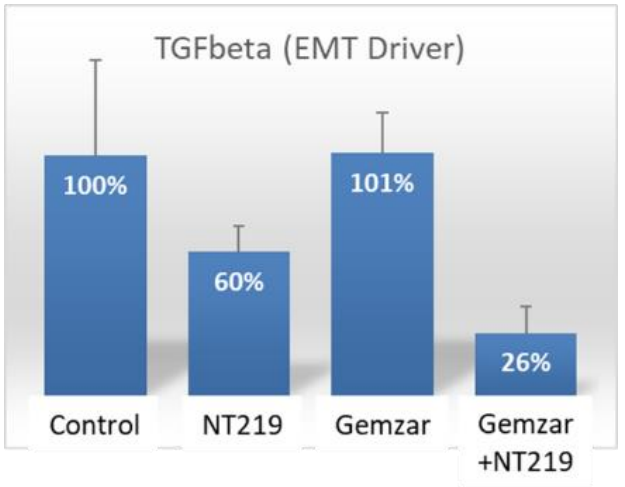
Gemcitabine (Gemzar®)

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









NT219 – DEMOGRAPHICS & SAFETY
3 mg/kg Dose Level as a Single Agent

Patients Demographics

Demographics of Patients treated with NT219 3mg/kg (n=3)	
Median age (range)	74 (69-79)
Male/Female, n (%)	2(66.6%)/1(33.3%)
Race	
White n (%)	3 (100%)
Prior Lines of Therapy	
3 n (%)	1 (33.3%)
4, n (%)	1 (33.3%)
11, n (%)	1 (33.3%)
Diagnosis, n	
Pancreatic Cancer	1
Gastroesophageal Junction Cancer	1
Breast Cancer	1
ECOG, n (%)	
1	3 (100%)
Median time from initial Diagnosis, Months (range)	62 (22-90)

Summary of Adverse Events

Adverse Event Description	Grade 1 n (#Events)	Grade 2 n (#Events)	Grade 3 n (#Events)
Abdominal Pain	1(2)	1(1)	
Alanine Aminotransferase Increased	1(1)		
Alkaline Phosphatase Increased			1(1)*
Aspartate Aminotransferase Increased	1(1)		
Back Pain		1(1)	
Belching	2(2)		
Cellulitis Left Foot		1(1)	
Dyspnea	2(2)		
Fatigue	1(1)		
Nausea	1(1)	1(1)	
Rash Pustular		1(1)	
Retching	1(2)		
Toxic Encephalopathy			1(1)**

*Transient- G2 after 2 weeks, **Transient- less than 24h

Presented at the ASCO annual meeting June 2021