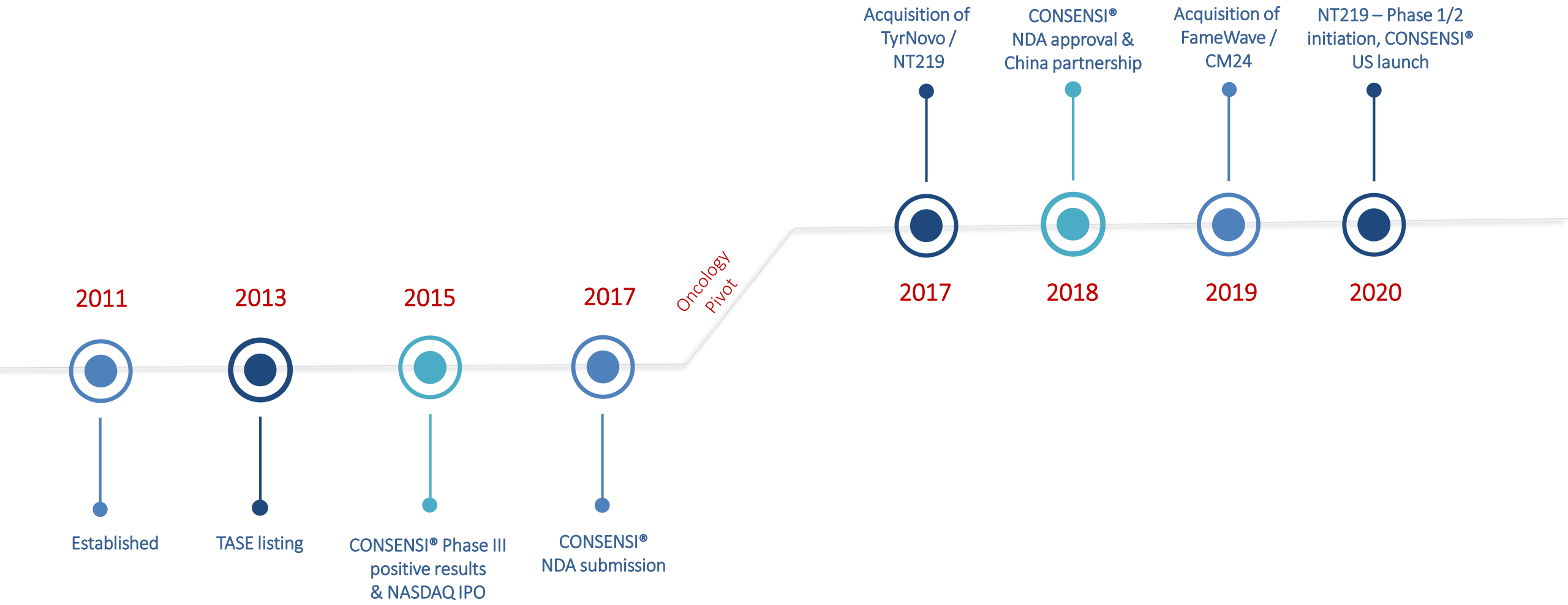
CORPORATE PRESENTATION | NOVEMBER 2020

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 Annual Report on Form 20-F for the year ended December 31, 2019 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.

OUR TRANSFORMATION INTO ONCOLOGY



KITOV PHARMA (NASDAQ/TASE: KTOV)

- ✓ CM24 - First-in-class α-CEACAM1 mAb, 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
  - Market cap. ~$75M*
  - $63M cash as of June 30th, 2020
  - CONSENSI® commercial royalties supports pipeline development
  - ~600K ADSs 3-month avg. trading volume*
  - Current clinical programs fully funded

Advancing clinical-stage novel oncology therapies

* As November 11th , 2020

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EXPANDING PIPELINE

Program	Indication	Preclinical	Phase 1	Phase 2	Phase 3	Partner	Value Drivers
CM24 (CEACAM-1)	Solid tumors (monotherapy)						
	Solid tumors (combination with nivolumab)					Bristol Myers Squibb	Initiation: Q4:20 Topline: H2:21
	NSCLC (combination with nivolumab)						Expansion arms on MTD: Initiation Q4:21
	Pancreatic Cancer (combination with nivolumab and Abraxane)						
NT219 (IRS1/2 & STAT3)	Solid tumors (monotherapy)						On going Topline data: H2:21
	R/M Head and Neck (combination with cetuximab)						Initiation Q1:21

Multiple data read-outs expected in the next 12 months

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Done / Ongoing

Planned

EXPERIENCED LEADERSHIP

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

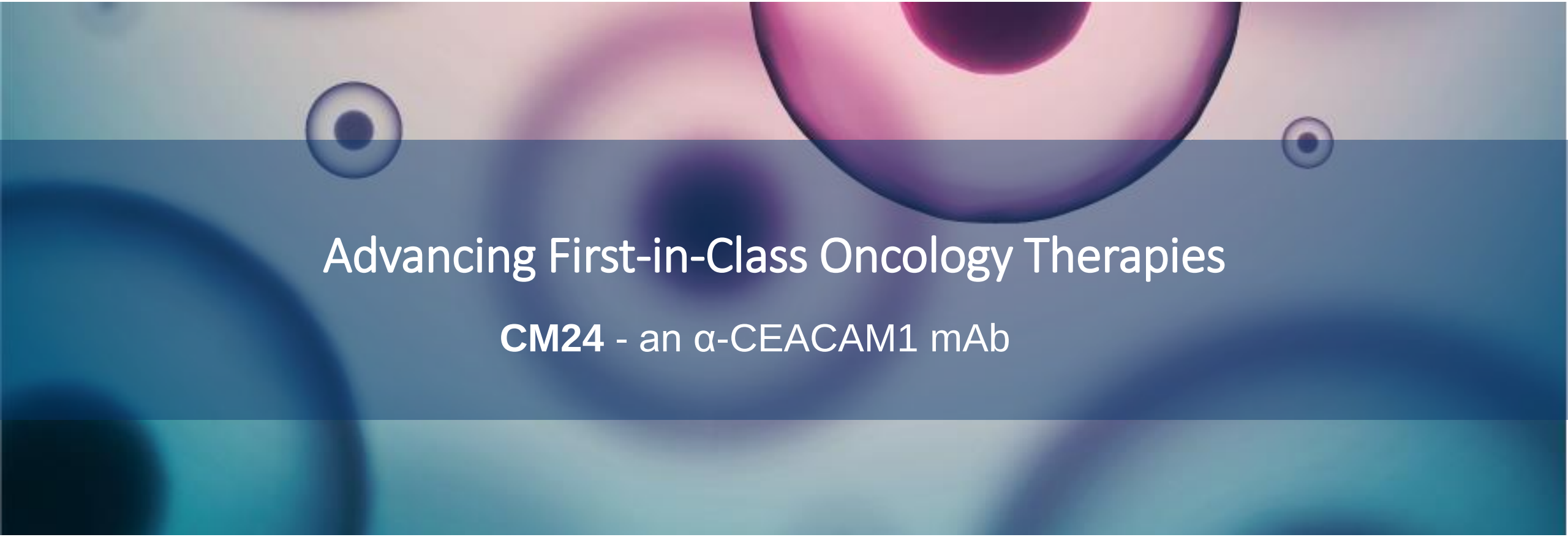
Eric K. Rowinsky, MD
Chairman of the Board
Former CMO at ImClone, Stemline, Board member at Biogen Inc.

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

Hadas Reuveni, Ph.D.
Vice President, Research and Development
Formerly at Keryx (NASDAQ:KERX)

Bertrand Liang, MD, PhD, MBA, AMP
Chief Medical Officer
Formerly at Biogen Idec, Amgen, NCI

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



Advancing First-in-Class Oncology Therapies

CM24 - an α-CEACAM1 mAb

CEACAM1 (Carcinoembryonic Antigen Cell Adhesion Molecule 1):
PLAYS A KEY ROLE IN CANCER BIOLOGY

ADHESION:

Oncogene

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

Horst, 2011

The Journal of Immunology

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

Ferri, 2020

IMMUNE CELLS/IMMUNE EXCLUSION:

Journal of Cancer Science and Research

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

Tsuzuki, 2020

Cancer Biotherapy and Radiopharmaceuticals

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

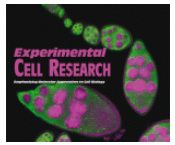
Tsang, 2020

IMMUNO-ONCOLOGY:

nature

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

Bloomberg, 2015



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

Shively, 2013

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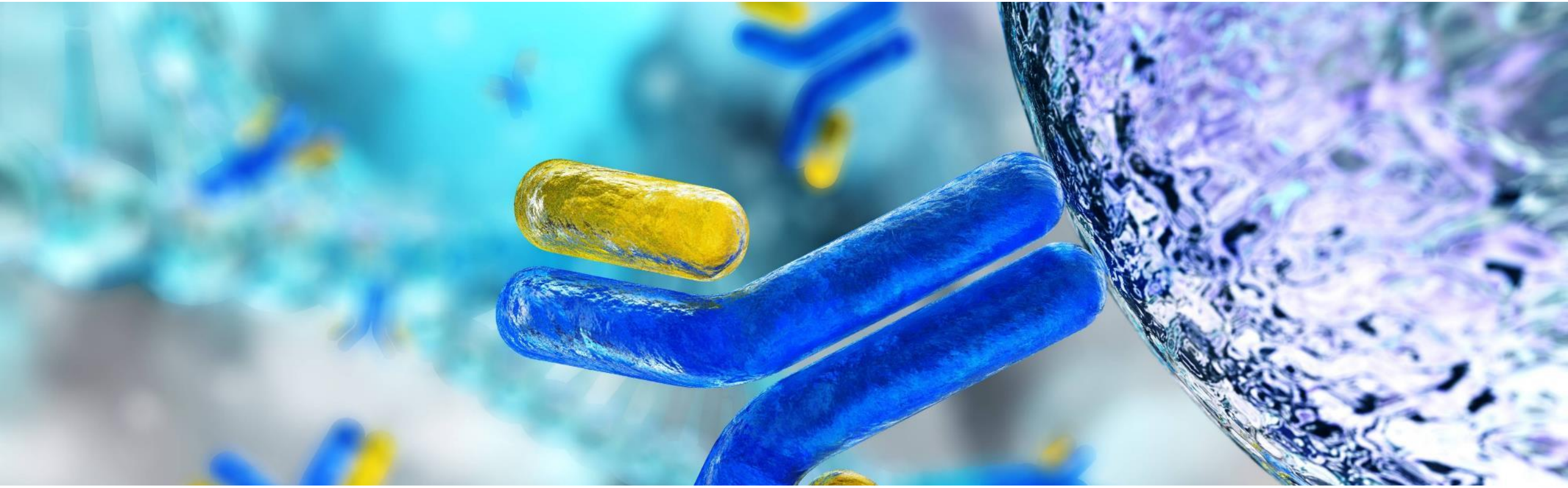
CM24: SELECTIVE BLOCKING OF CEACAM1



- IgG4 humanized monoclonal antibody, similar to anti-PD-1 IO agents (no elicitation of ADCC or CDC)
- Nanomolar efficiency of binding to extracellular domain of CEACAM1
- Anticipated to impact triple mechanisms of action of CEACAM1 in the neoplastic phenotype

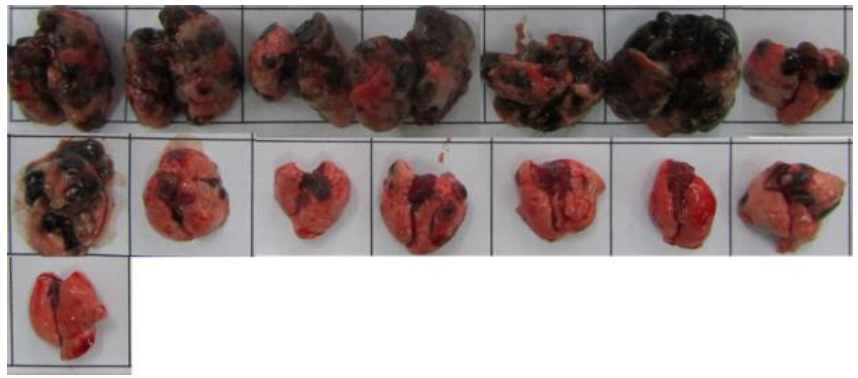
ANTI-CANCER EFFECT FOLLOWING TREATMENT

PRECLINICAL DATA WITH CM24 + TIL AND CM24 + α-PD1

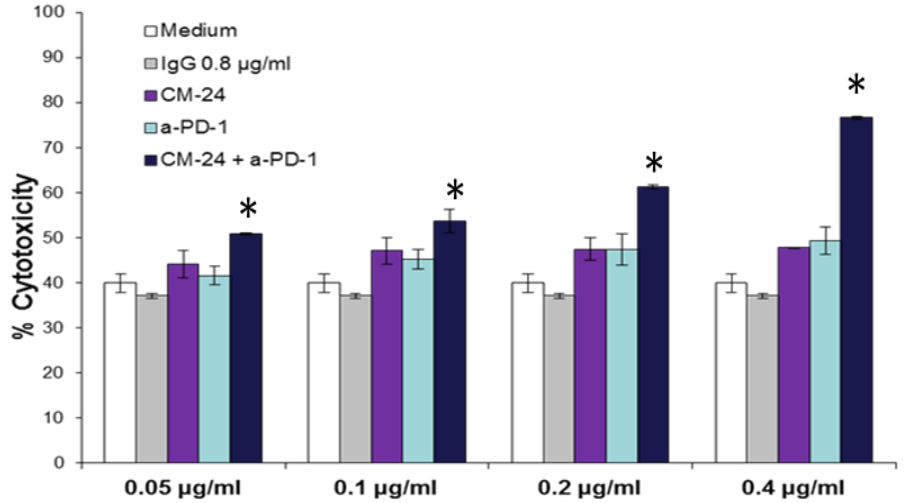
TIL + IgG

TIL + CM24

Naïve



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



Combination index (CI) = 0.15

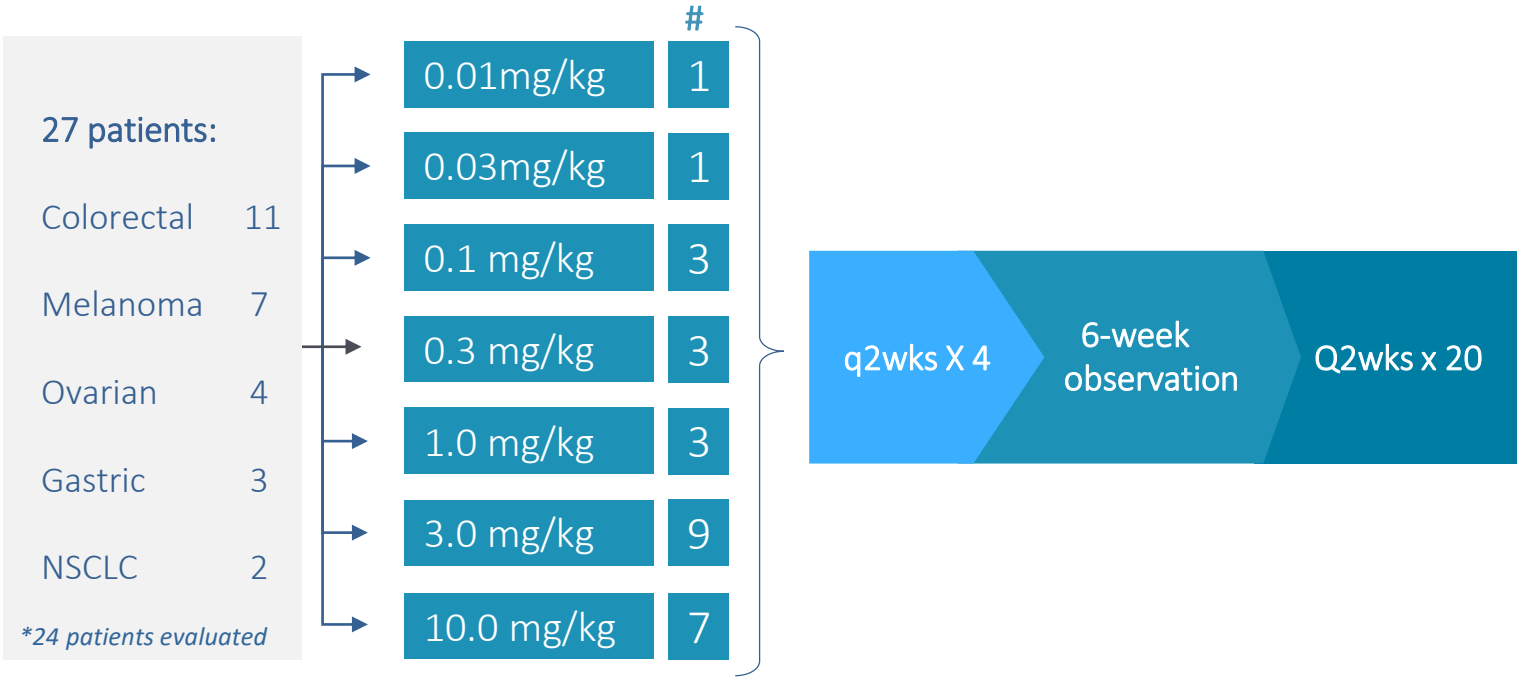
$$CI = \frac{(D)_1}{(D_x)_1} + \frac{(D)_2}{(D_x)_2} < 1 \rightarrow \text{synergy}$$

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 3 prior regimens (range 2-8) and a median of 4 prior regimens at the 3mg/kg & 10mg/kg
- 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



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

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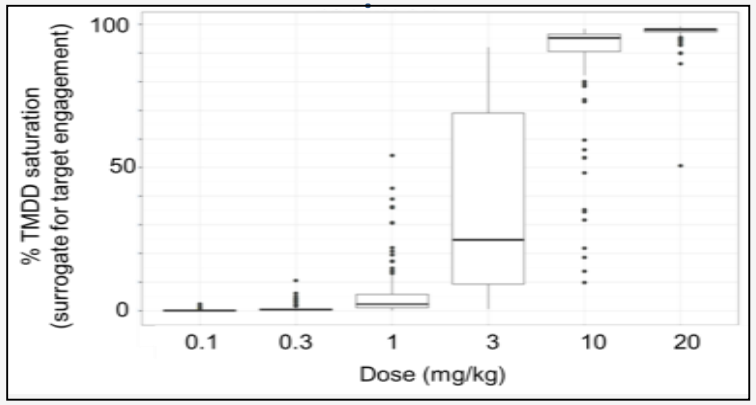
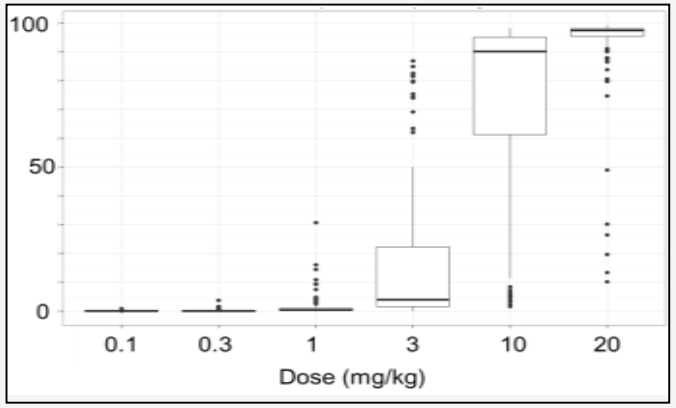
PK/PD MODELING PROVIDES DOSAGE & SCHEDULE GUIDANCE

Simulated TMDD[1] saturation at Ctrough with Q2W



- 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 dose is needed for saturation across population

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

Nivolumab (OPDIVO®[2]) administered Q2W or Q4W, representing good clinical and commercial fit for CM24

[1] *Target-mediated drug disposition*

[2] *OPDIVO® is a registered trademark of Bristol-Myers Squibb.*

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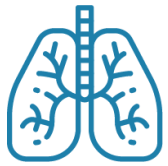
LARGE MARKET OPPORTUNITY | NSCLC & PANCREATIC CANCER

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 supports significant synergy
- Receptor saturation with CM24 is better achieved with a Q2W regimen, aligning with the nivolumab schedule

Targeting unmet medical needs



- 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 has 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]

[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

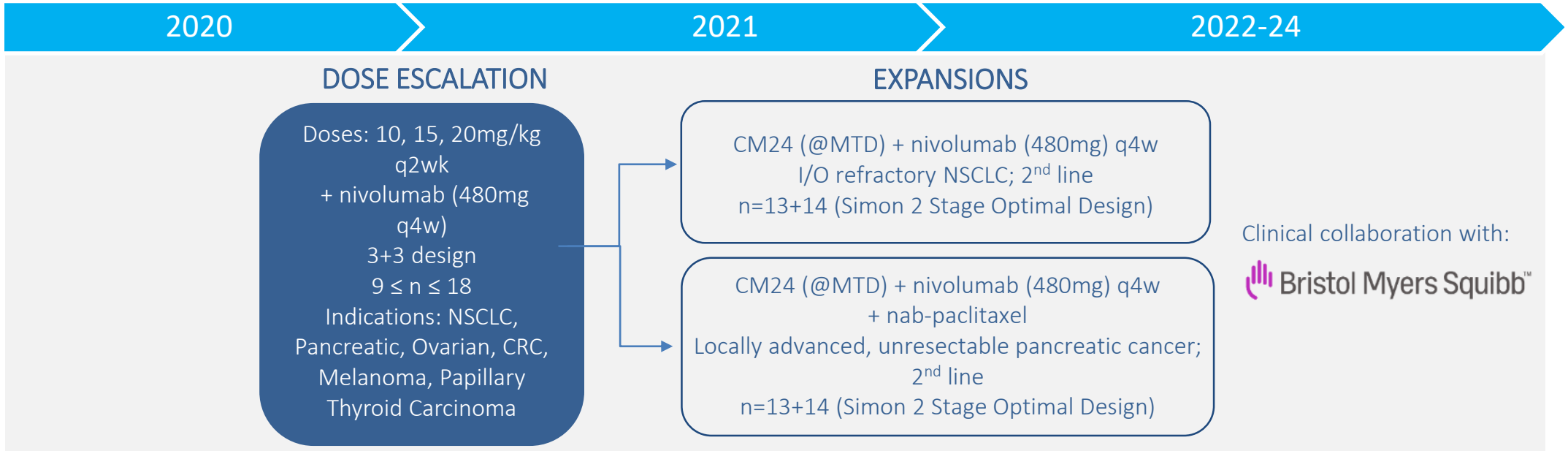
- A Phase 1/2 open label multi center study of CM24 in combination with:
  - Nivolumab in selected cancer indications (Phase 1) and NSCLC (Phase 2)
  - Nivolumab and nab-paclitaxel in Pancreatic cancer (Phase 2)
- Measurement of CEACAM1 based bio-marker

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

- Exploring further studies in other tumor types as well as monotherapy



Advancing First-in-Class Oncology Therapies

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

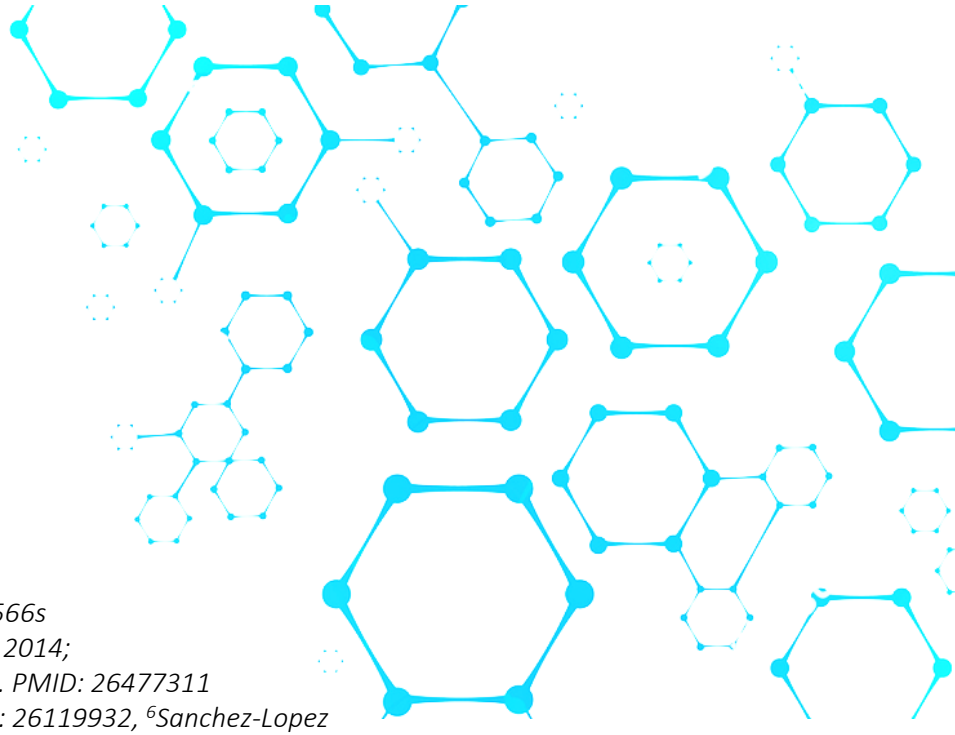
NT219 - DUAL INHIBITOR OF IRS1/2 & STAT3

IRS1/2

- Scaffold proteins, mediating mitogenic, metastatic, angiogenic and anti-apoptotic signals from IGF1R, IR, IL4R and other oncogenes, overexpressed in multiple tumor types[1]
- Regulates major survival pathways such as the PI3K/AKT, MEK/ERK and Wnt/β-catenin[2]
- Activated as a feedback response to anti-cancer therapies[3]

STAT3

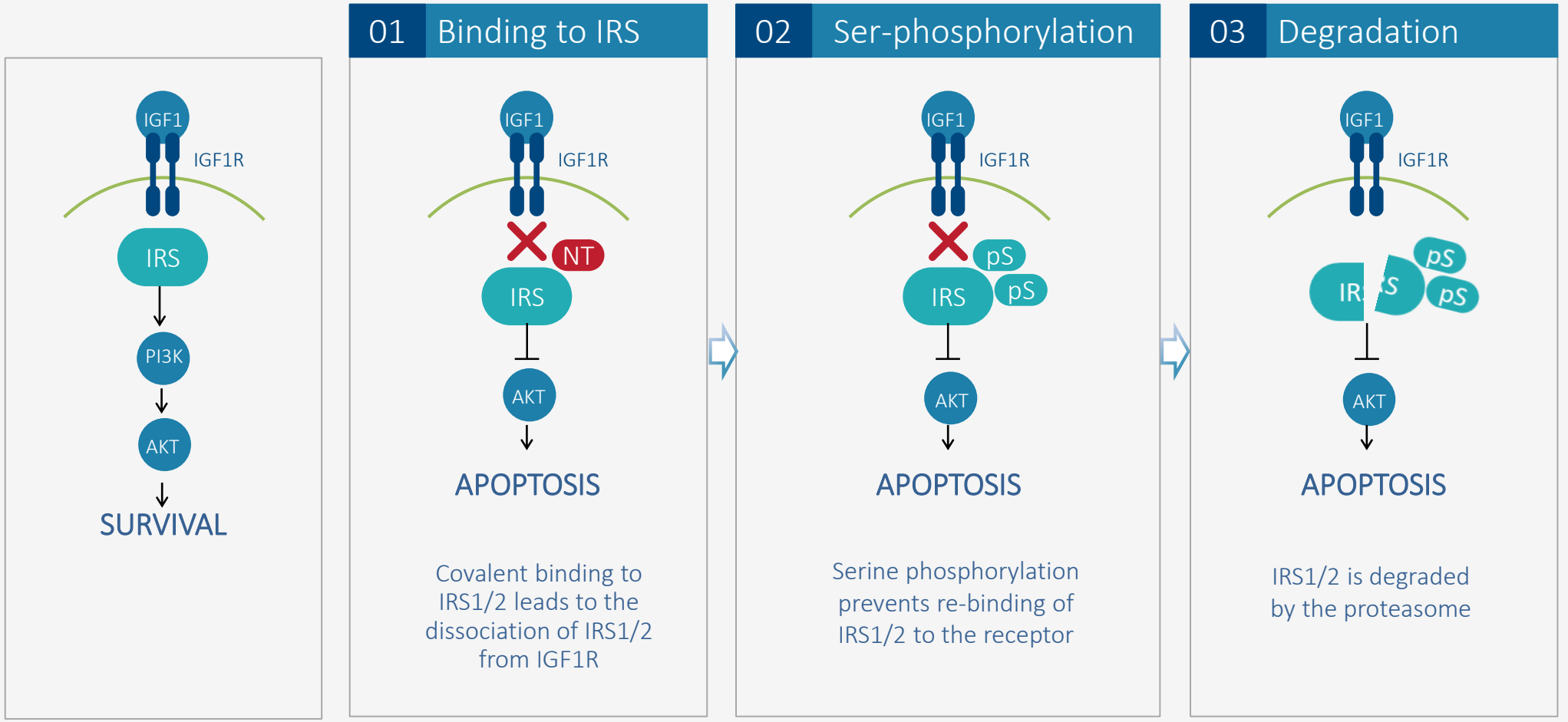
- Well-established transcription factor associated with the tumorigenic phenotype[4]
- Provides a crucial axis to support cell proliferation and survival[5]
- Active in tumor JAK/STAT3 and TGF beta resistance mechanisms[6]



[1]Hadas Reuveni et al.; Cancer Res 2013;73:4383-4394. 2013; [2]Machado-Neto, et al. 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
[5,6]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.; ohnson, 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; 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

NOVEL MOA: IRS DEGRADATION BY NT219

BLOCKING IGF1R-AKT PATHWAY[1]



[1]Reuveni et al. Cancer Res 2013

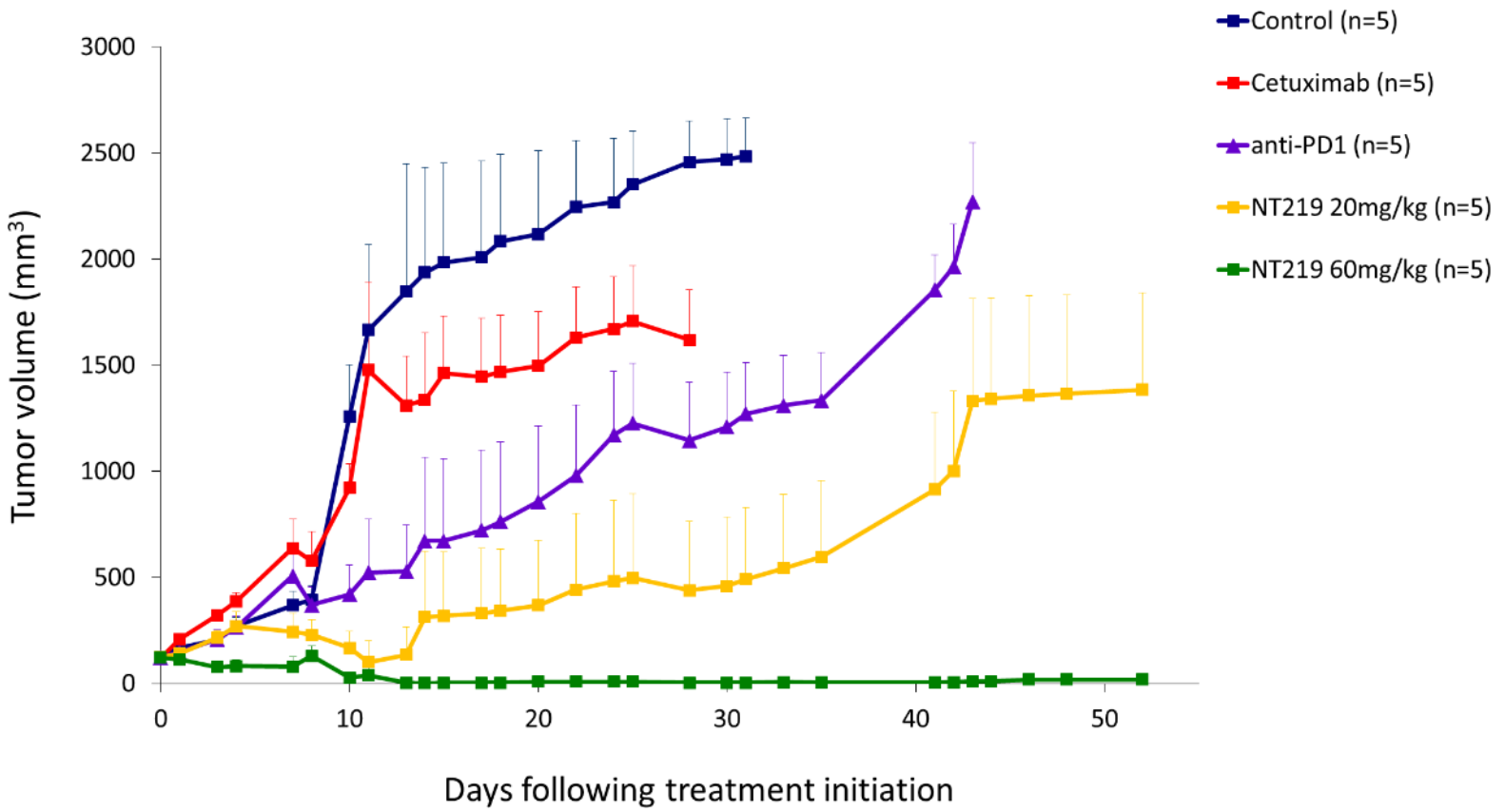
NT219 | EFFICACY AS MONOTHERAPY

Animal model

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

Drugs

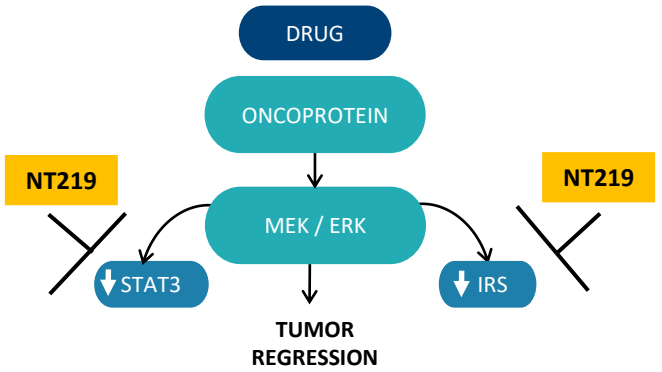
α-PD1
Cetuximab (Erbitux®)
NT219 20mg/kg
NT219 60mg/kg



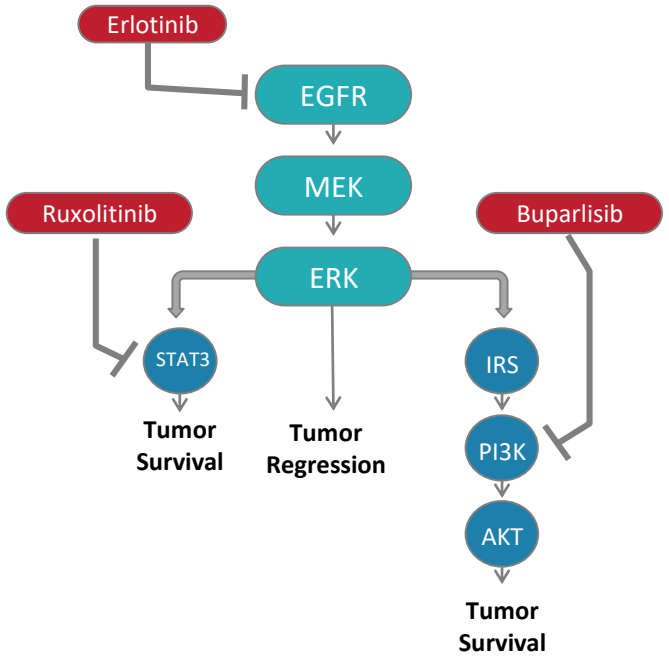
[1] NSG mice were injected SC with SCC-9 cells. PBMCs ($18*10^6$ cells per mouse) administered 4 weeks prior to first treatment. NT219, α-PD1, and cetuximab were administered IV (NT219) and IP (α-PD1 and cetuximab) twice a week for 4 weeks. Graph reflects relevant data adapted from 2020 Multidisciplinary Head and Neck Cancers Symposium Poster presentation

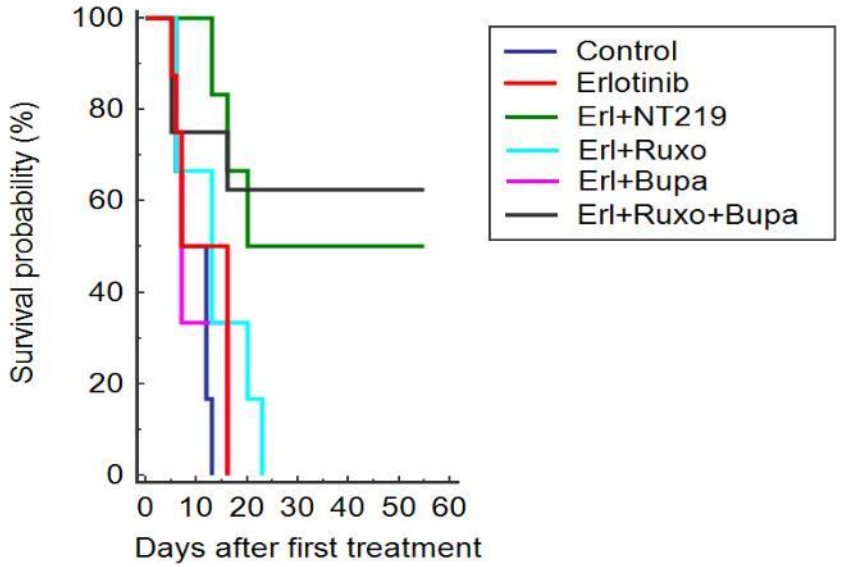
STAT3 AND IRS ARE ESSENTIAL IN THERAPEUTIC RESISTANCE

Blocking survival pathways



Proof of Concept: PDX model of Head and Neck Cancer



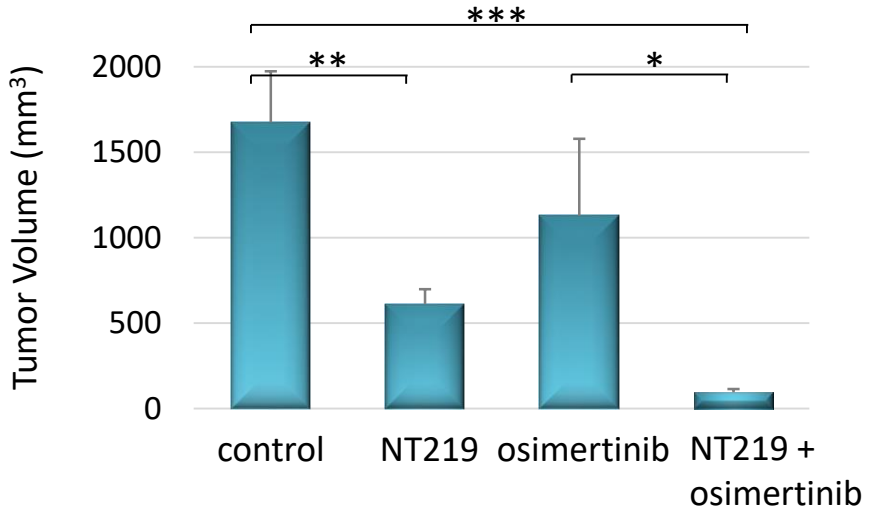


By blocking <u>both</u> STAT3 and IRS pathways, NT219 prevents tumor resistance and re-sensitizes tumors to anti-cancer therapies

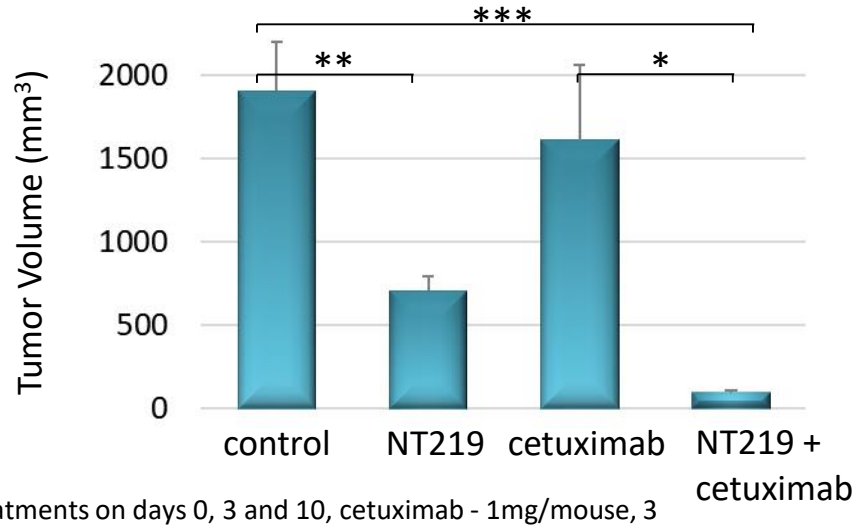
NT219 + TARGETED THERAPIES ESTABLISHED EFFICACY IN PDX MODELS

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

R/M HNSCC 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, 5 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

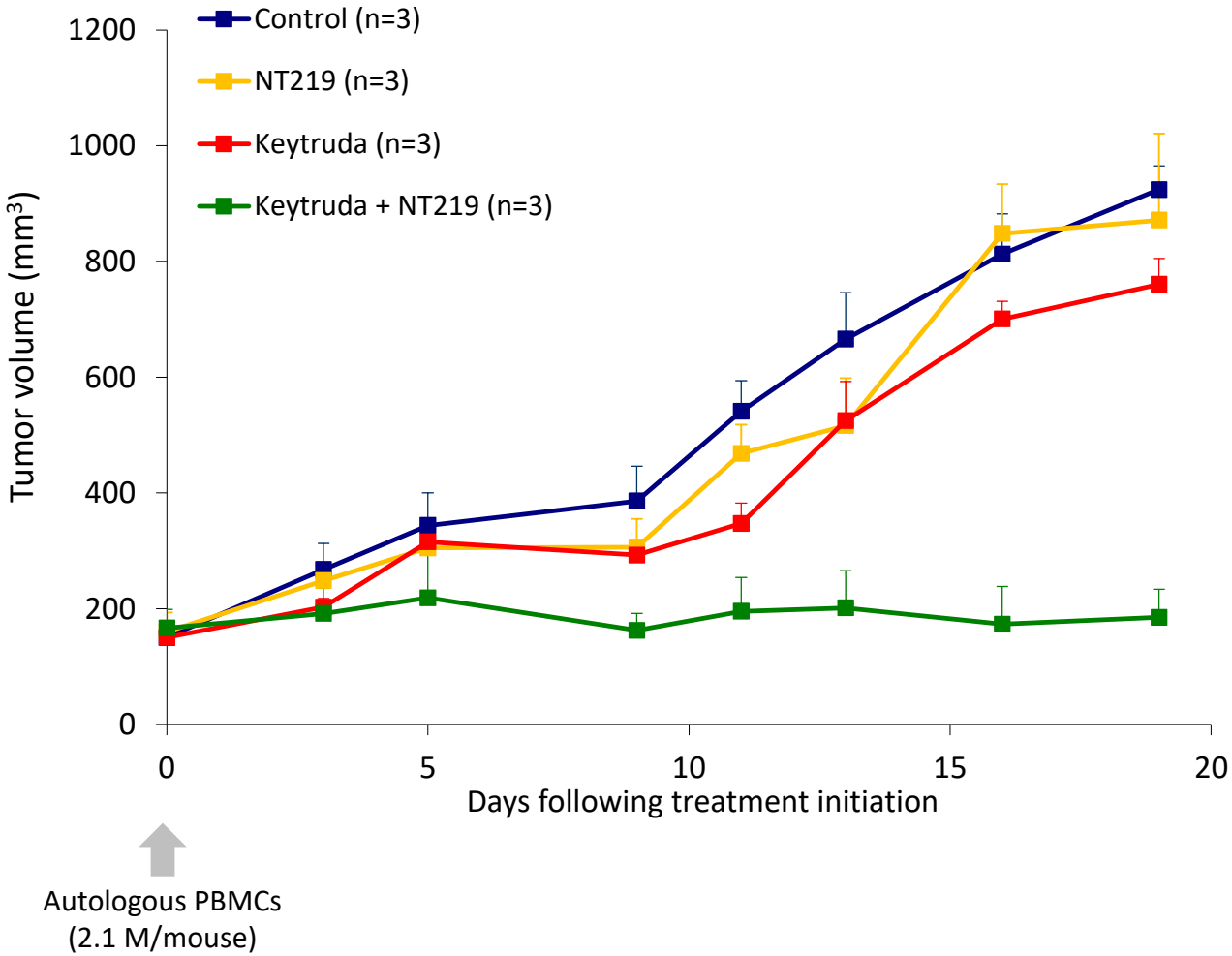
NT219 + α-PD1 RE-SENSITIZES TO REFRACTORY α-PD1 TUMORS

PDX model

Humanized PDX of Esophagus Cancer (refractory to pembrolizumab)

Drug

Pembrolizumab (Keytruda®)



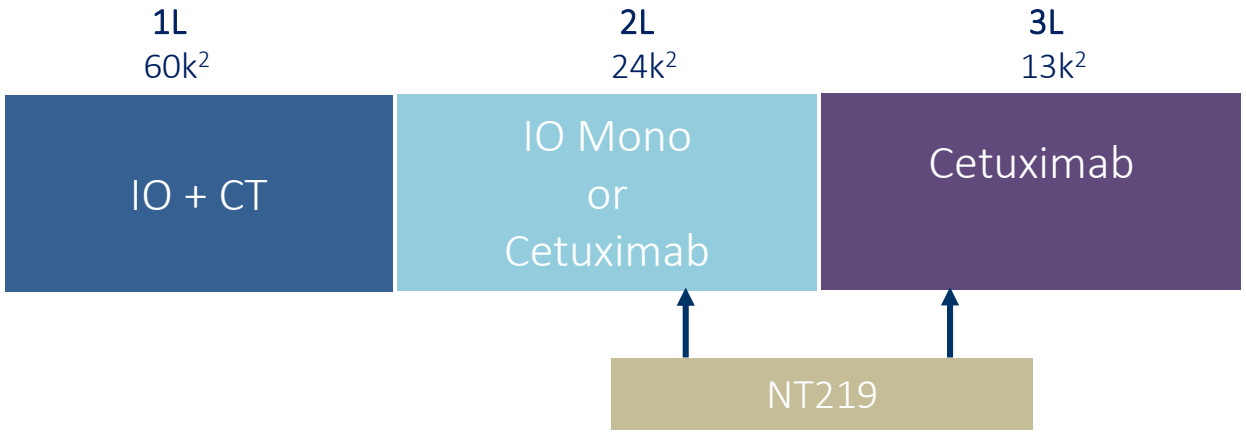
FIRST MARKET OPPORTUNITY | RECURRENT OR METASTATIC SQUAMOUS CELL CARCINOMA OF HEAD AND NECK (SCCHN)

Rationale for combining Cetuximab + NT219

- EGFR and PD(L)-1 are the only clinically validated targets in SCCHN
- Cetuximab inhibits EGFR signaling and promotes ADCC in EGFR expressing tumors
- STAT3 and IRS-to-AKT activation contributes to resistance to cetuximab in HNSCC

Targeting the unmet medical need

- 1L Standard of care has shifted from chemotherapy towards immuno-oncology + chemotherapy
- Only < 20% of R/M SCCHN patients respond to α-PD1s
- 175k new cases/year are expected by 2024[1]



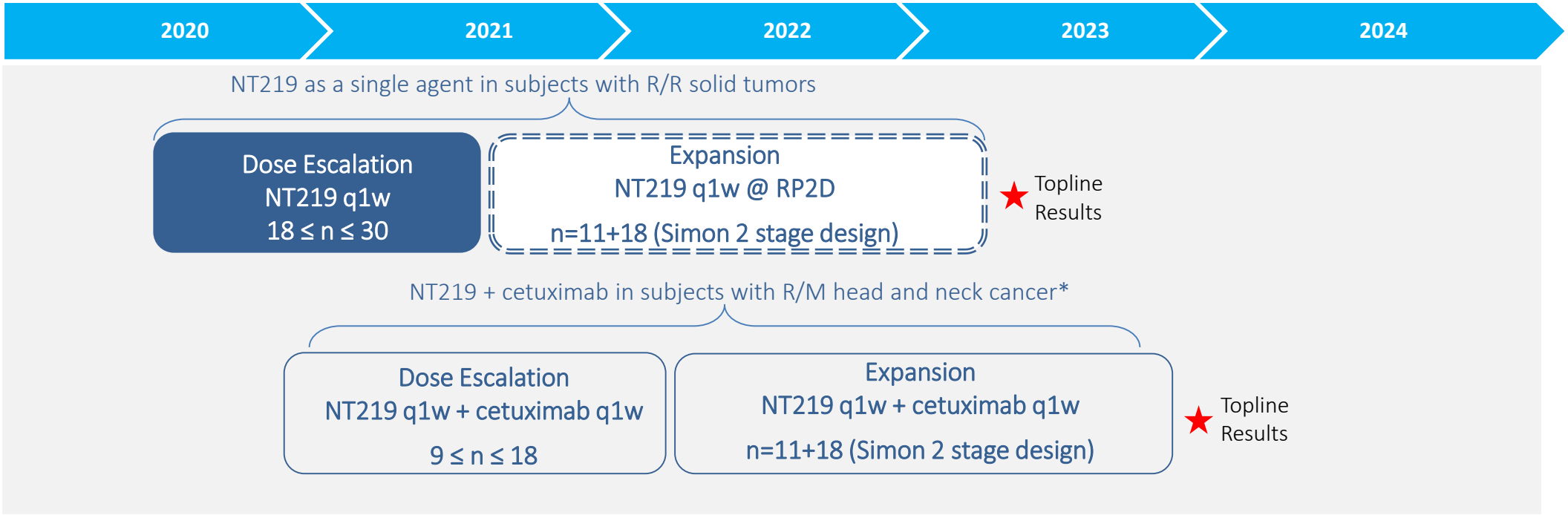
NT219 + cetuximab has the potential to become an attractive 2-3L therapy

[1] Global Data 2018: Head and Neck Squamous Cell Carcinoma: Opportunity Analysis and Forecasts to 2026
[2] Internal best current estimates of patient numbers based on external research, 5 major global territories

NT219 MONOTHERAPY AND COMBINATION PHASE 1/2 STUDY DESIGN

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

- **Primary endpoints**: Safety, pharmacokinetics and to determine the MTD
- **Secondary endpoints**: Obtain preliminary efficacy data



* Colorectal Adenocarcinoma pts will be recruited in the Dose Escalation phase

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

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Advancing clinical-stage novel oncology therapies

* As November 11th , 2020

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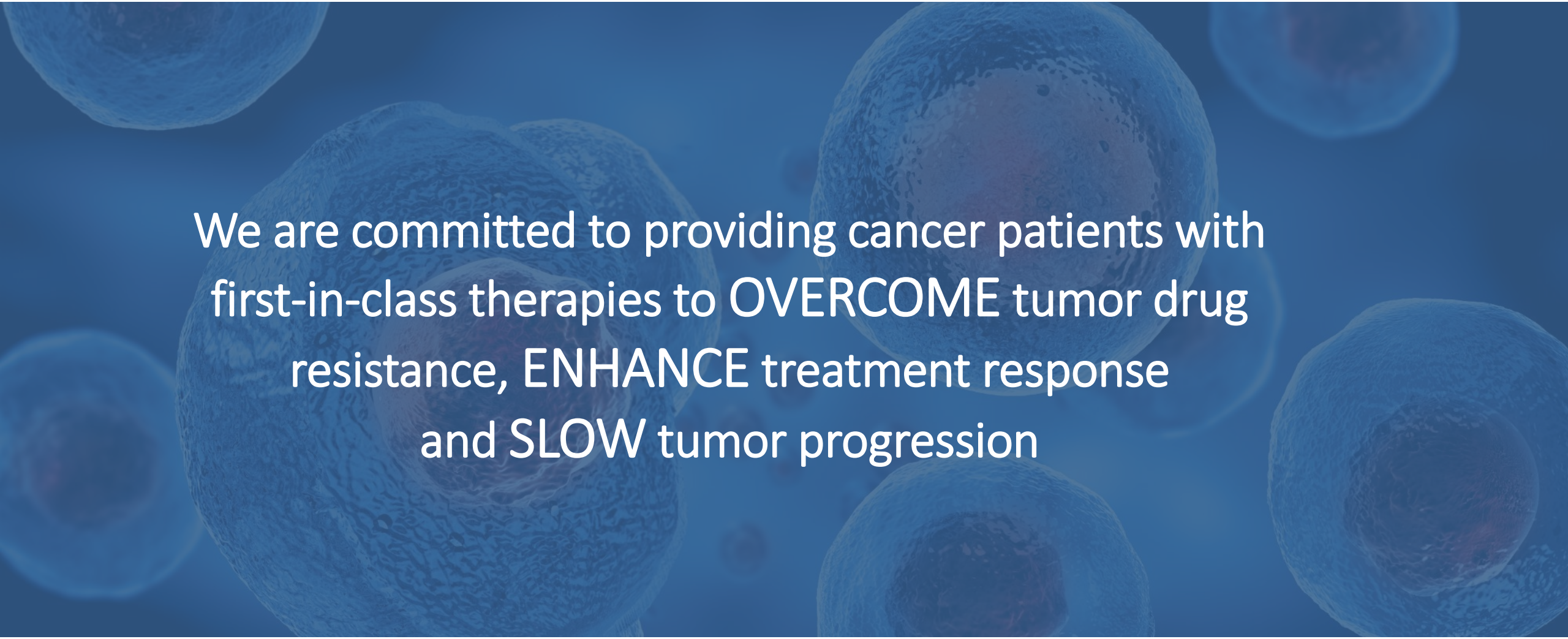
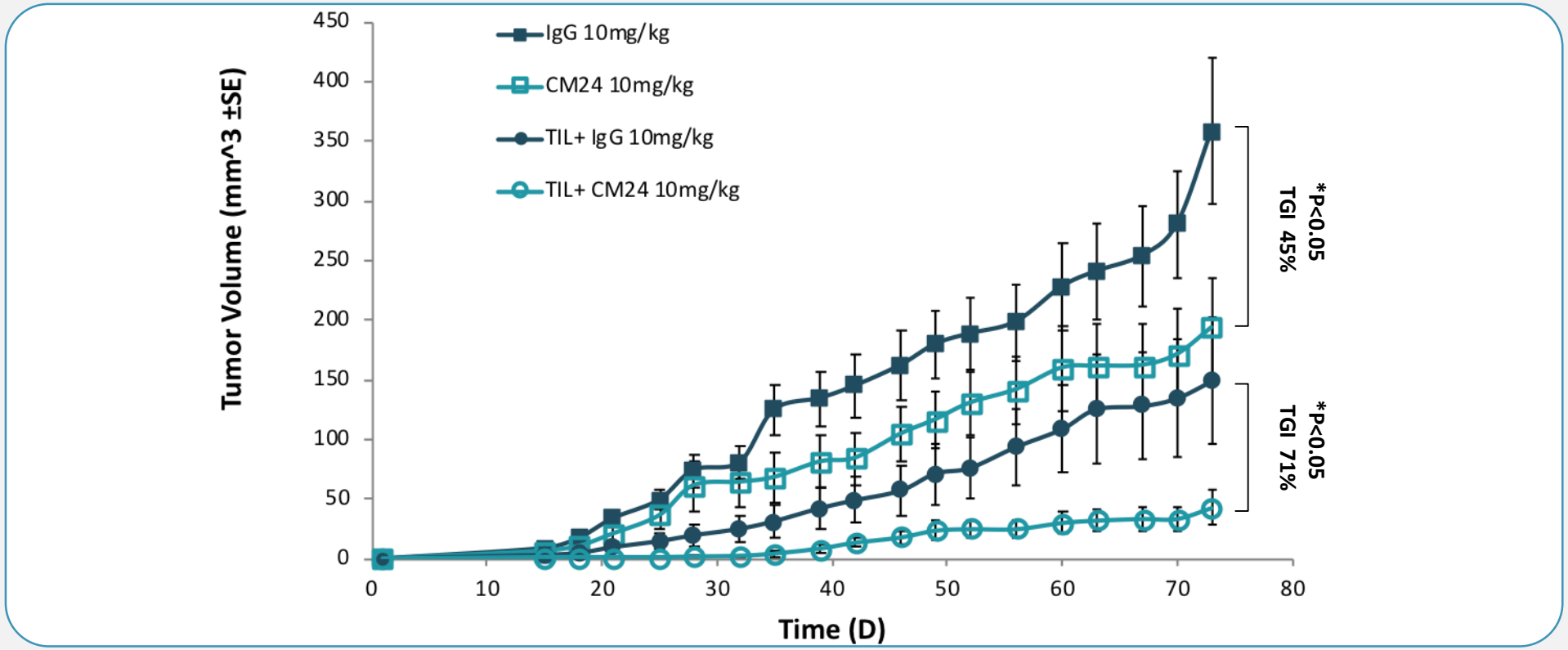

We are committed to providing cancer patients with
first-in-class therapies to OVERCOME tumor drug
resistance, ENHANCE treatment response
and SLOW tumor progression

Appendix A - CM24

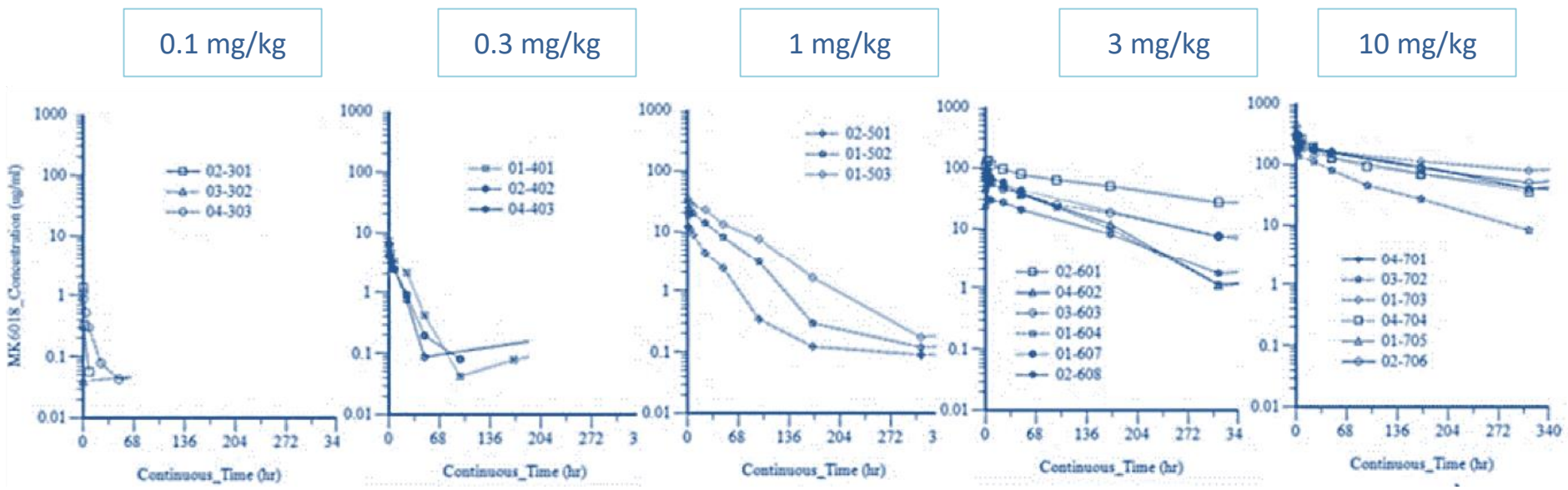
INHIBITION OF MELANOMA GROWTH FOLLOWING CM24 AND CM24 + TIL TREATMENT

CM24 ACTIVITY IS DEMONSTRATED AS SINGLE AGENT AND IN COMBINATION WITH TILS



PHASE 1 PK DATA

SATURATION WAS NOT REACHED WITH DOSES UP TO 10MG/KG



Slower clearance with increasing dose

Higher half-life with increasing dose

Appendix B - NT219

SELECTED PUBLICATIONS

UC San Diego School of Medicine

Michael Karin

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www.nature.com/onc

ORIGINAL ARTICLE

Targeting colorectal cancer via its microenv
inhibiting IGF-1 receptor-insulin receptor s
STAT3 signaling

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

האוניברסיטה העברית בירושלים
THE HEBREW UNIVERSITY OF JERUSALEM

Alexander Levitzki

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

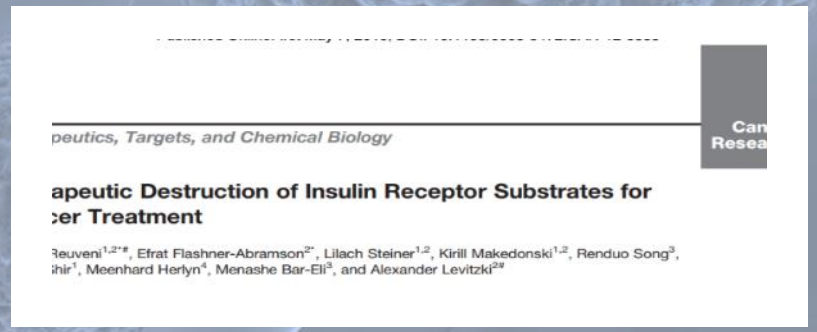
SHORT COMMUNICATION

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



peutics, Targets, and Chemical Biology

apeutic Destruction of Insulin Receptor Substrates for
cer Treatment

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

UBC THE UNIVERSITY OF BRITISH COLUMBIA

Michael Cox

Molecule Therapeutics

Tyrphostin NT157 Suppresses Insulin Receptor
trates and Augments Therapeutic Response of Prostate
cer

u Ibuki[1,2], Mazyar Ghaffari[1,3], Hadas Reuveni[4,5], Mitali Pandey[1], Ladan Fazli[1], Haruhito Azuma[2],

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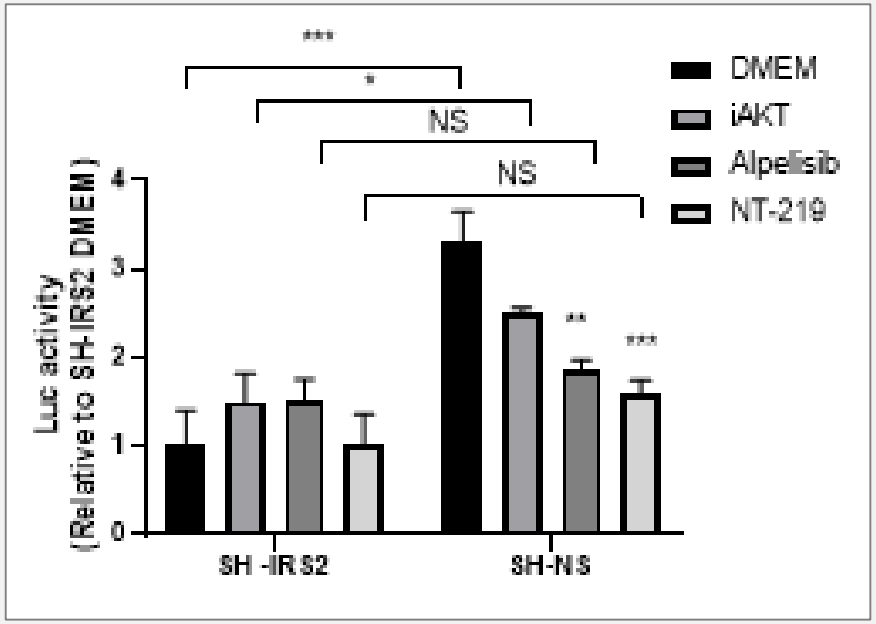
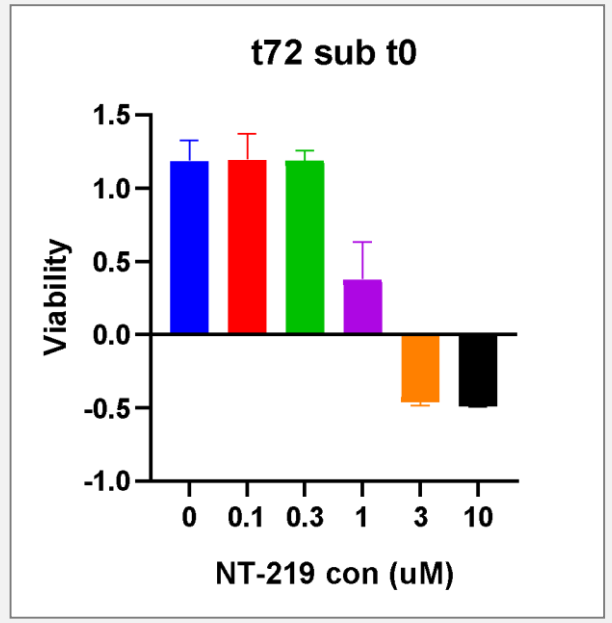
NT219 | SUPPRESSES β-CATENIN ACTIVITY IN CRC CELLS





The colon cancer SW403 cells, where IRS2 is overexpressed, demonstrate enhanced b-catenin activity, IRS1/2 inhibitor NT219, as well as the PI3K inhibitor Alpelisib, suppress the increased b-catenin activity and inhibit SW-403 cell viability

AACR Virtual Special Conference on Epigenetics and Metabolism, Oct 2020

Prof. Ido Wolf, Head of Oncology Division, Tel Aviv Sourasky Medical Center

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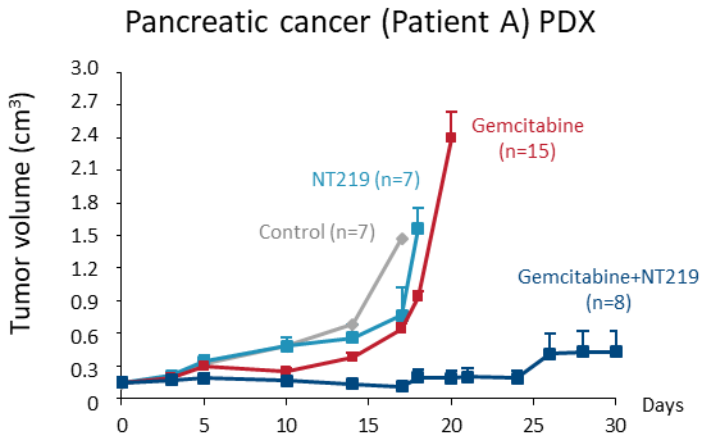
NT219 | PANCREATIC CANCER IN COMBINATION WITH GEMCITABINE

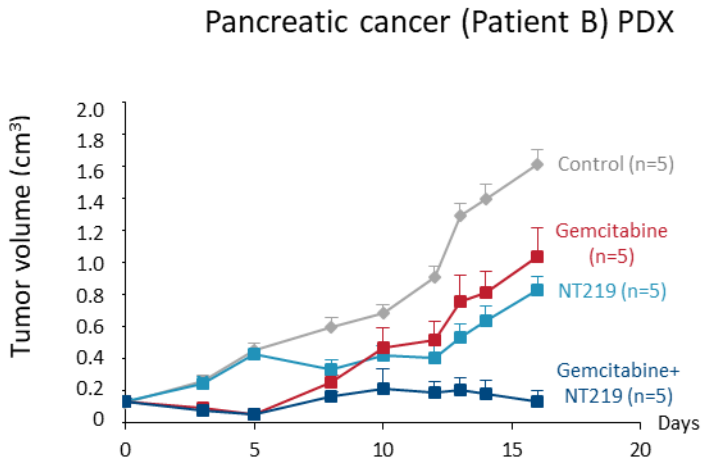
PDX model

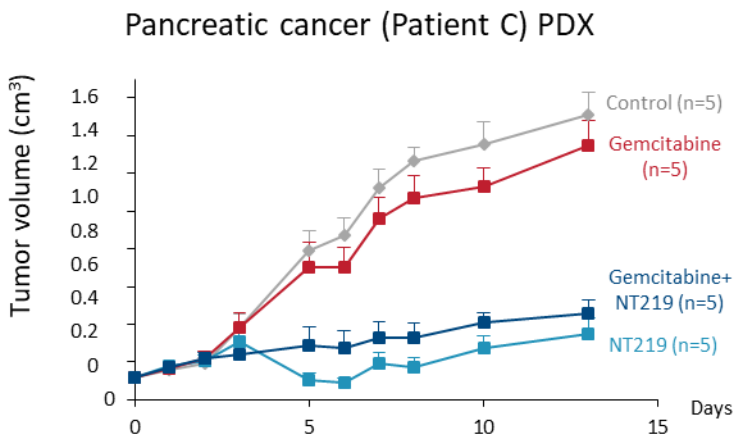
Pancreatic Cancer

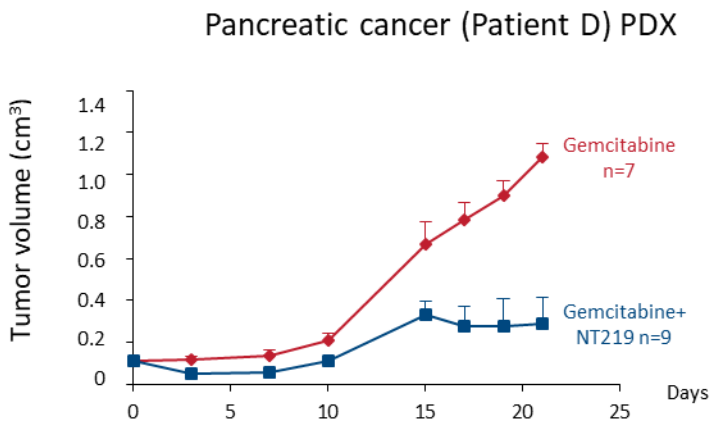
Drug

Gemcitabine (Gemzar®)









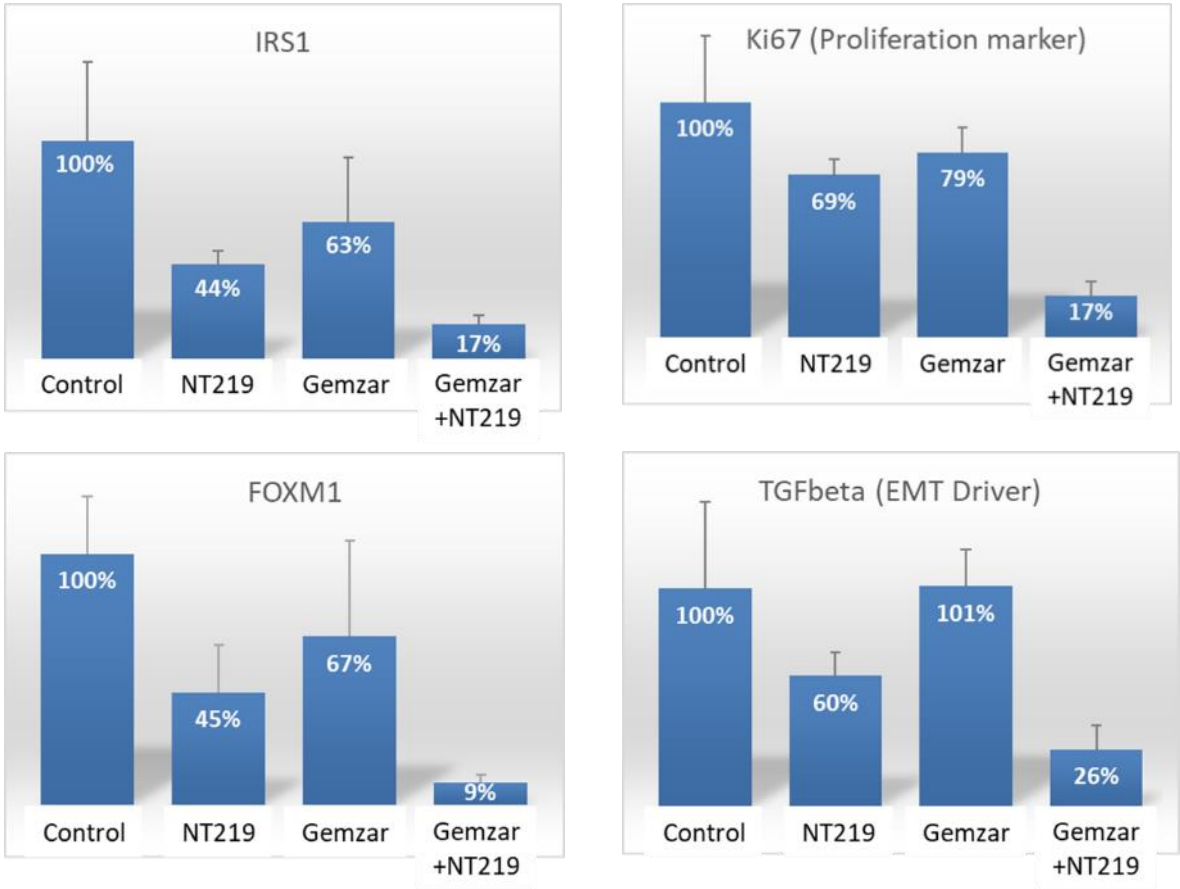
RNA SEQUENCING | ANALYSIS OF TUMORS FOLLOWING TREATMENT

PDX model

Pancreatic Cancer

Drug

Gemcitabine (Gemzar®)



Appendix C - CONSENSI®

CONSENSI® | FROM IND TO THE U.S. MARKET

consensi®
(amlodipine and celecoxib) tablets
2.5mg/200mg, 5mg/200mg, 10mg/200mg

CONSENSI® is the only NSAID whose labeling indicates reduction of blood pressure and consequent risk reduction of heart attack, stroke and death

Full U.S. Prescribing Information is available at http://www.consensi.com

Fixed dose combination of

Celecoxib,
a COX-2 selective NSAID
(the active ingredient in Pfizer's Celebrex®)

+

Amlodipine
a blood pressure-lowering agent (a calcium channel blocker) *(the active ingredient in Pfizer's Norvasc®)*

Launched in the USA- Coeptis Pharmaceuticals

Partnered in China- CSBio

Partnered in S. Korea- Kuhnil Pharmaceutical

Kitov's clinical, regulatory and medical teams developed CONSENSI® internally from IND, through successful Phase III clinical trials, to FDA approval

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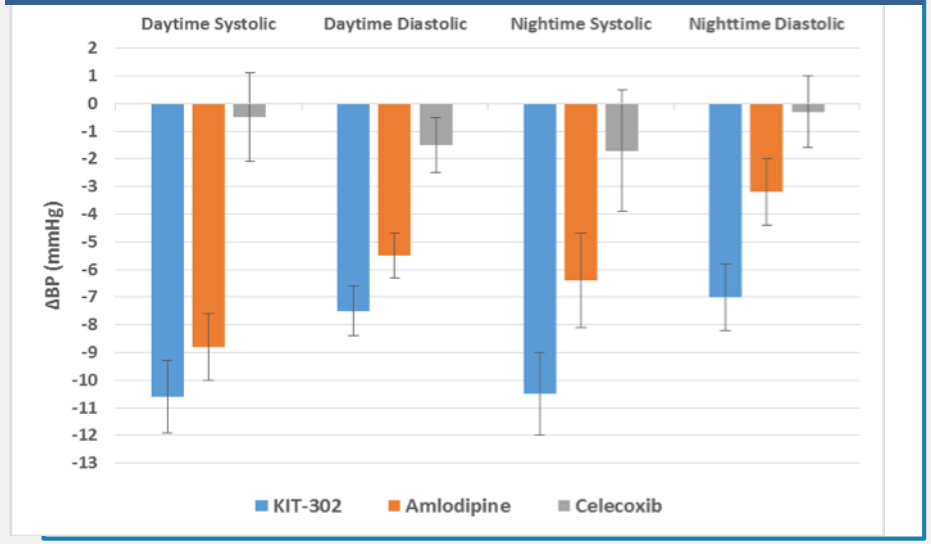
CONSENSI® PHASE III TRIAL RESULTS

Blood Pressure Reduction of Consensi® vs. Amlodipine and Celecoxib*



Consensi® demonstrated even better BP reduction than same amount of amlodipine given without celecoxib

* Error bars – standard error of mean

- Primary efficacy endpoint was successfully achieved (P=0.001)
- Demonstrated 2.5x better blood pressure reduction than FDA requirement (50% of amlodipine arm)
- Demonstrated consistent reduction in all measures of blood pressure
- Observed beneficial renal functions:

Measure	Consensi®	Amlodipine
Creatinine plasma level reduction	-3.22 µmol/L	-2.55 µmol/L
Peripheral edema (% patients)	8.2%	15.6%

- Additional Phase III/IV clinical trial to scientifically validate the renal benefits (not required for NDA submission) was completed. Topline results were announced in October, 2017

KITOV COMMERCIAL DRUG: CONSENSI®

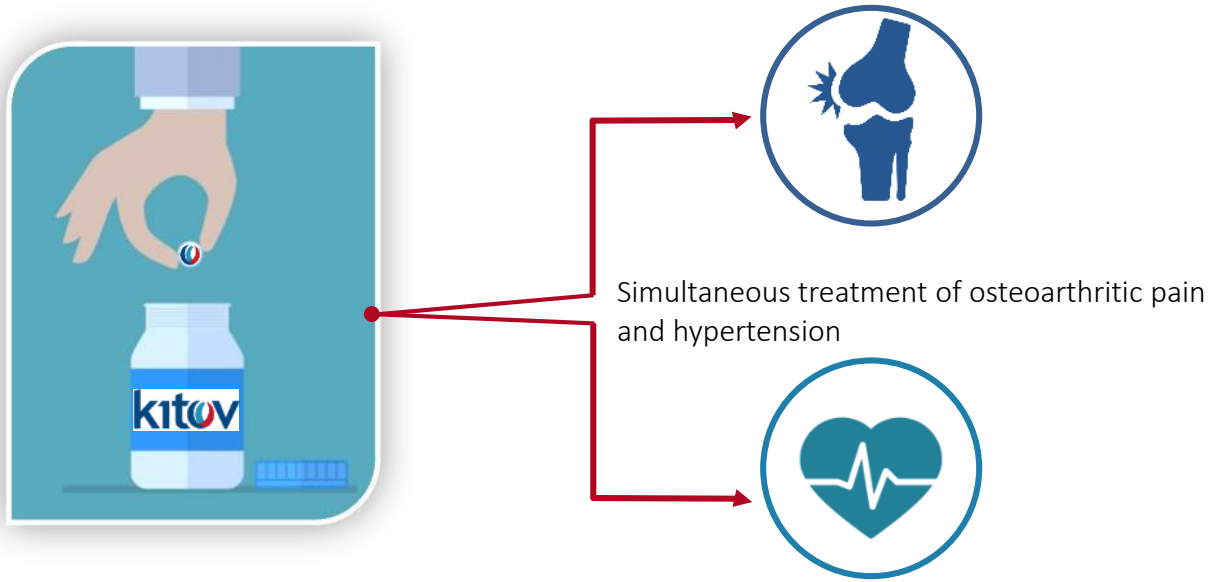
CONSENSI® is the only NSAID whose labeling indicates reduction of blood pressure and consequent risk reduction of heart attack, stroke and death

Full U.S. Prescribing Information is available at: : http://www.consensi.com

Fixed dose combination of **Celecoxib**,
a COX-2 selective NSAID *(the active ingredient in Pfizer's Celebrex®)*

+

Amlodipine,
a blood pressure-lowering agent (a calcium channel blocker) *(the active ingredient in Pfizer's Norvasc®)*



- Approved for marketing by U.S. FDA on May 31, 2018
- Clinical data showed Consensi™ was more effective at lowering blood pressure than amlodipine alone
- Clinical data also demonstrated beneficial renal function measures
- Formulated with 200 mg celecoxib and three different dosages (2.5, 5, 10 mg) of amlodipine
- Manufactured by Dexcel Pharma – Israel's largest private pharmaceutical company

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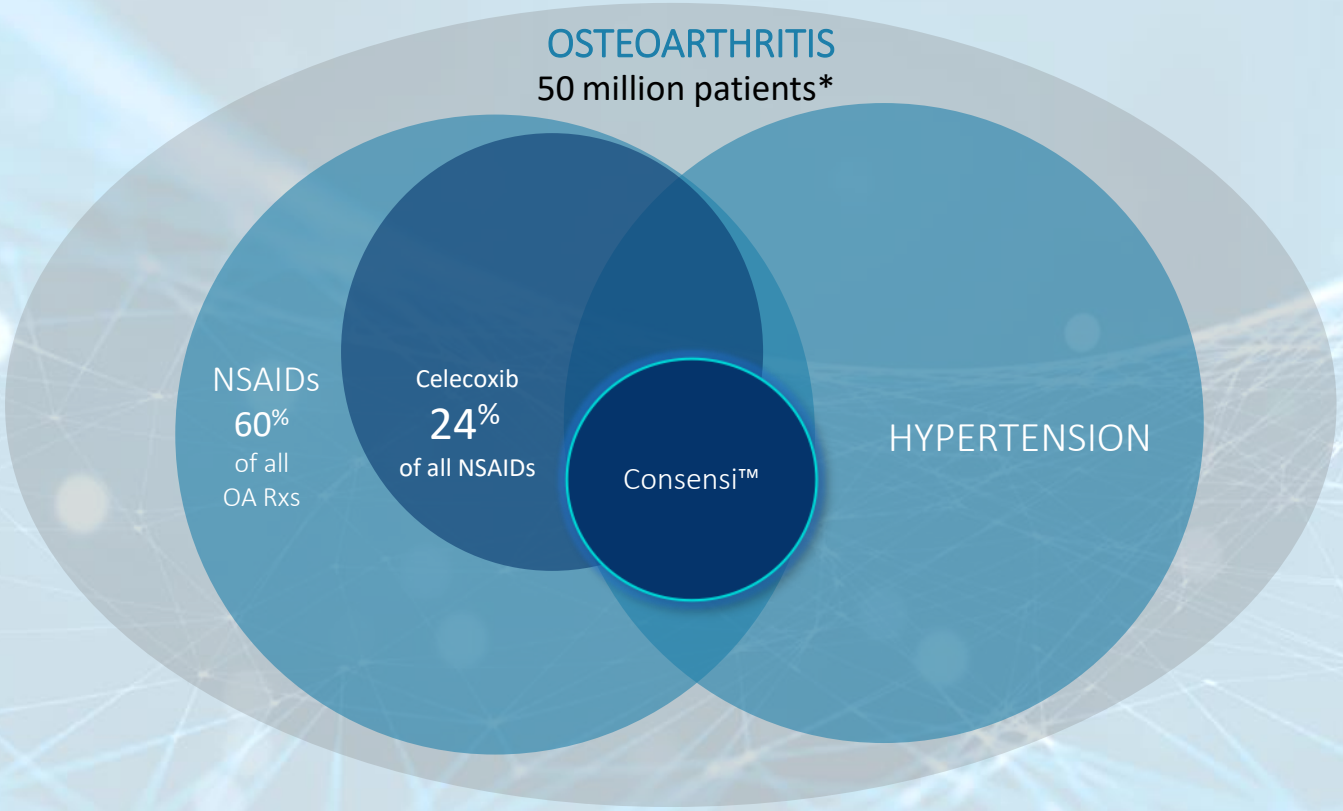
**Celebrex® is a registered trademark of G.D. Searle LLC (a subsidiary of Pfizer Inc.).*
Norvasc® is a registered trademark of Pfizer Inc.

CONSENSI® U.S. TARGET MARKETS

CONSENSI® targets osteoarthritis (OA) patients currently treated with NSAIDs (celecoxib as well as others) who also suffer from existing or newly diagnosed hypertension

ARTHRITIS PREVALENCE*

- More than 50 million adults in the U.S. have doctor-diagnosed osteoarthritis
- 67 million people are expected to have doctor-diagnosed osteoarthritis by 2030



HYPERTENSION PREVALENCE**

- 29% of U.S. adults older than 18
- 65% of U.S. adults older than 60

COMORBIDITIES

- 44% of adults with high blood pressure have osteoarthritis**

** Arthritis Foundation: http://www.arthritis.org/ ** Hypertension Among Adults in the United States: National Health and Nutrition Examination Survey, 2011–2012*

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Thank You