Investor Presentation

Building a powerful new future in cellular IO

August 2020

Bellicum

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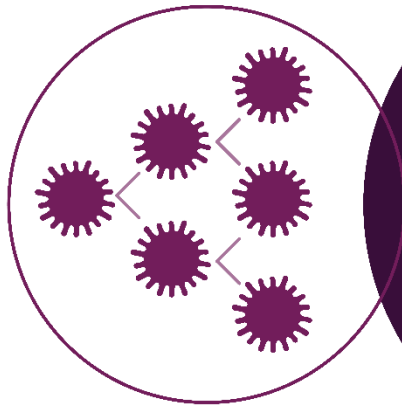
Forward Looking Statement

This presentation contains estimates, projections and other forward-looking statements, concerning, among other things: our research and development activities relating to our GoCAR™ (incorporating “iMC”), GoCAR-T® CaspaCIDe® (“iC9”), and related technologies; our product candidates including BPX-601, BPX-603, OTS GoCAR-NK, and rimiducid; the timing and success of our current and planned clinical trials, including the timing of receipt of data from such clinical trials and the timing of our reports of such data; the possible range of applications of our cell therapy programs and potential curative effects and safety in the treatment of diseases, including as compared to other treatment options and competitive therapies; and the success of our collaborations with academic and commercial partners, including with respect to our manufacturing facility. Our estimates, projections and other forward-looking statements are based on our management's current assumptions and expectations of future events and trends, which affect or may affect our business, strategy, operations or financial performance. Although we believe that these estimates, projections and other forward-looking statements are based upon reasonable assumptions, they are subject to numerous known and unknown risks and uncertainties and are made in light of information currently available to us. Many important factors, in addition to the factors described in this presentation, may adversely and materially affect our results as indicated in forward-looking statements. All statements other than statements of historical fact are forward-looking statements.

Estimates, projections and other forward-looking statements speak only as of the date they were made, and, except to the extent required by law, we undertake no obligation to update any forward-looking statement. These statements are also subject to a number of material risks and uncertainties that are described more fully in Bellicum’s filings with the Securities and Exchange Commission, including without limitation our annual report on Form 10-K for the year ended December 31, 2019 and our quarterly report on Form 10-Q for the period ended June 30, 2020.

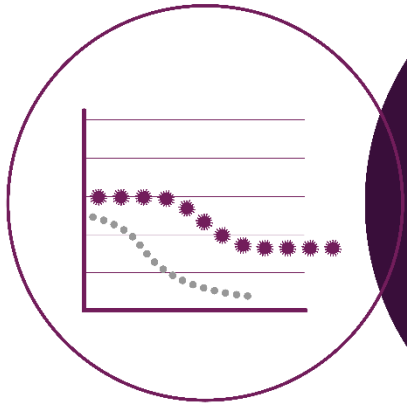
Building a Powerful New Future in Cellular IO

Our GoCAR platform is engineered to break through the limitations of current cell therapies



Proliferation

Boosts effector cell proliferation and extends survival, potentially leading to more durable responses

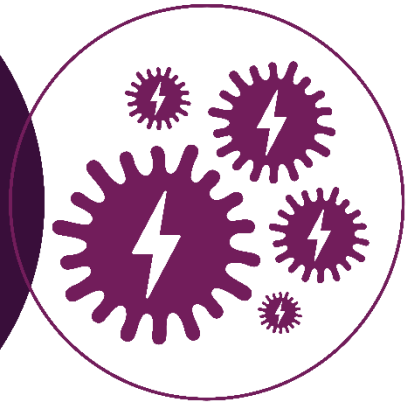


Persistence

Enhances effector cell functional persistence by resisting exhaustion and inhibitory signals from the tumor environment

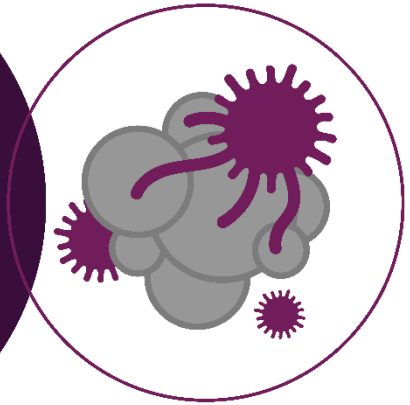
Power

Re-ignites the host immune response, unleashing the power to combat tumor tolerance and intensify tumor killing



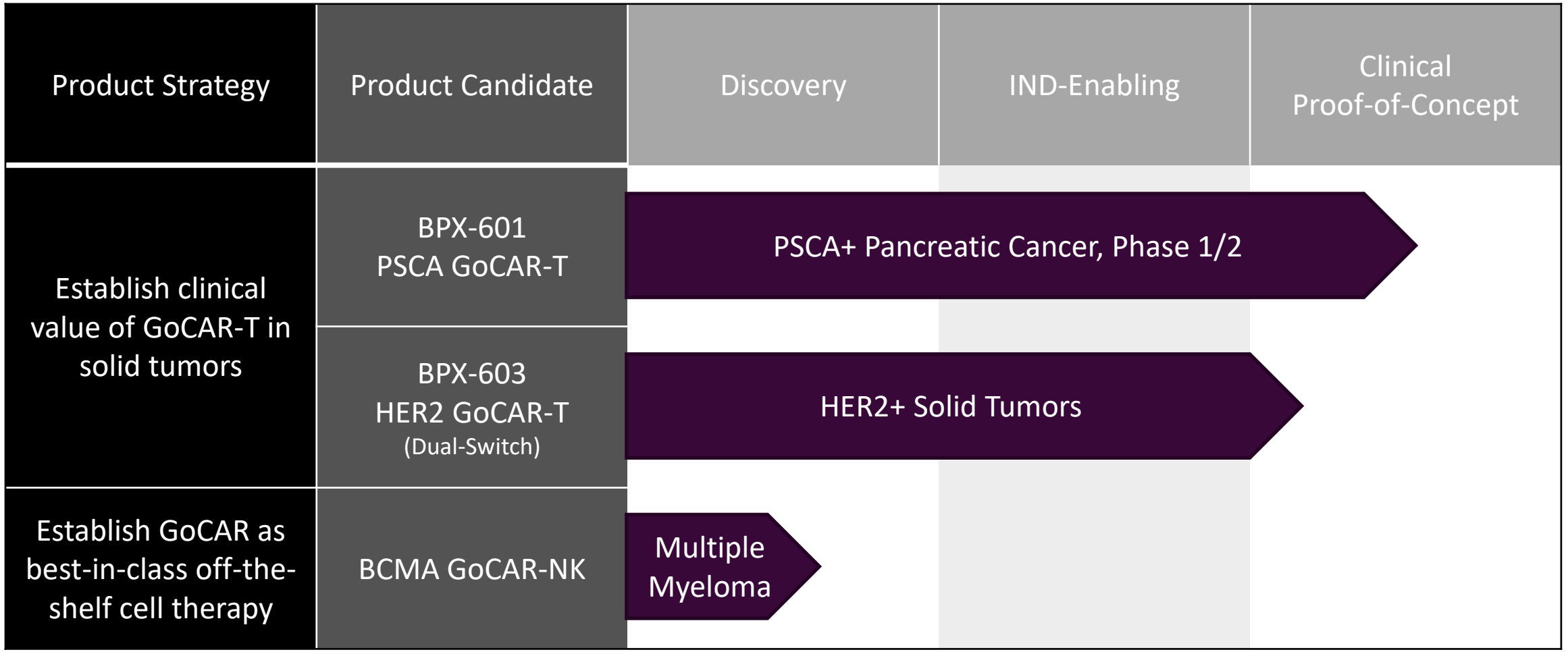
Performance

Molecular switch technology enables superior control over GoCAR cells



Product Pipeline

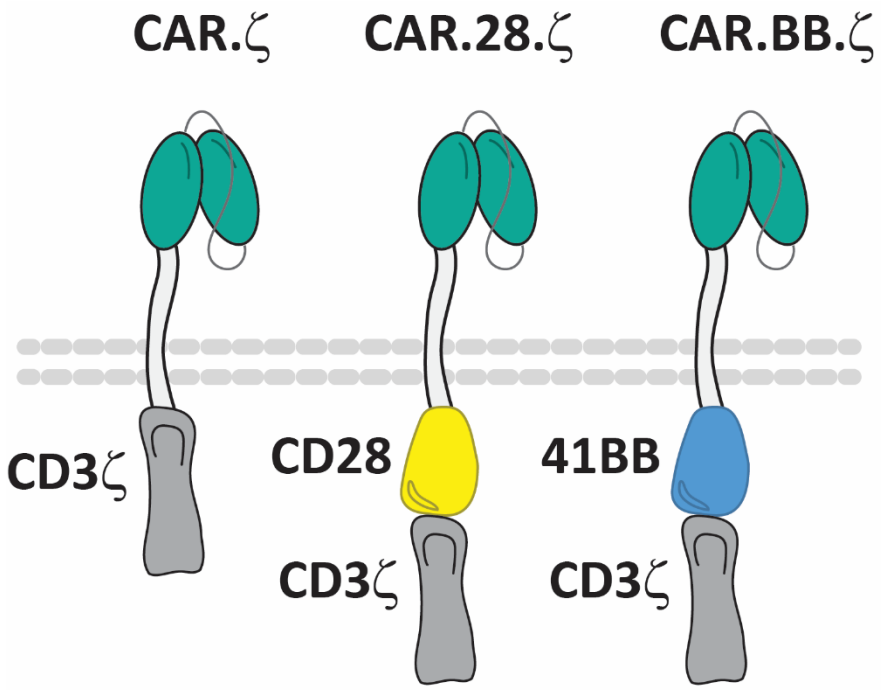
Leveraging the GoCAR platform to propel cellular IO forward



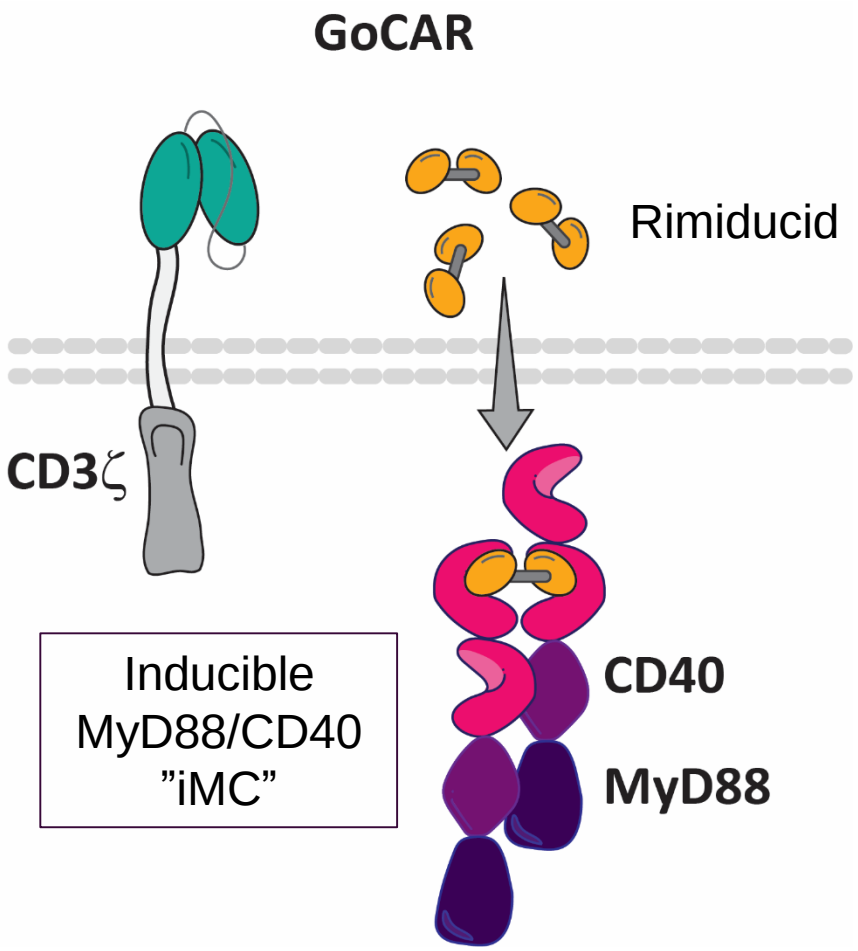
Technology Overview

GoCAR: Differentiated Technology Platform

Current Generation CAR Technology

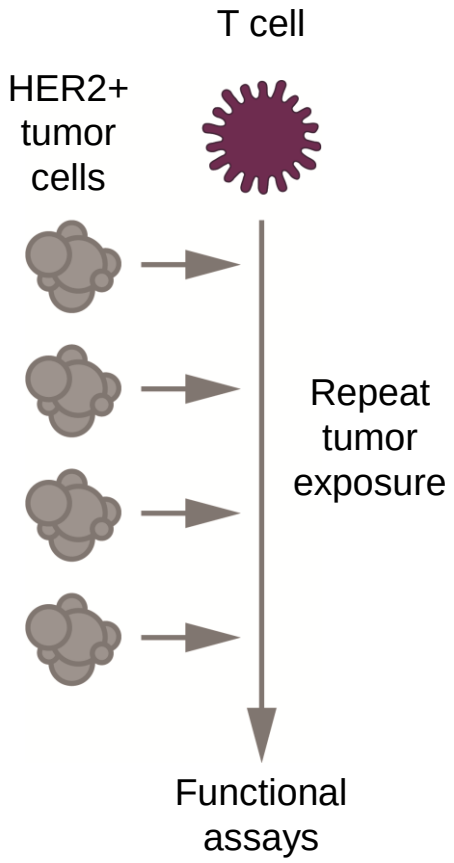


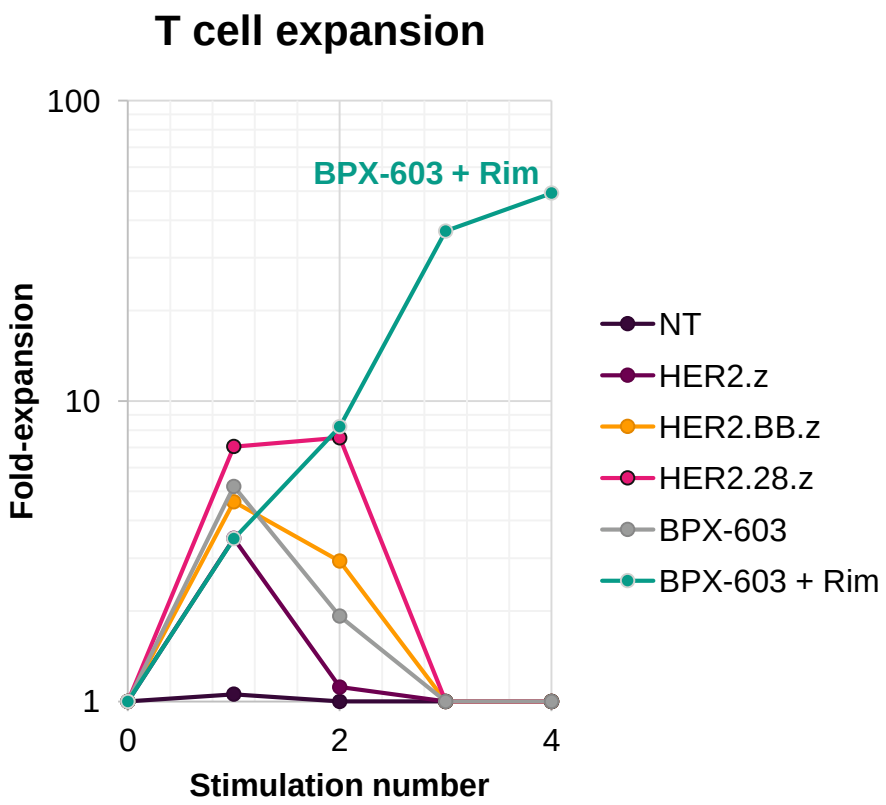
Next Generation GoCAR Technology

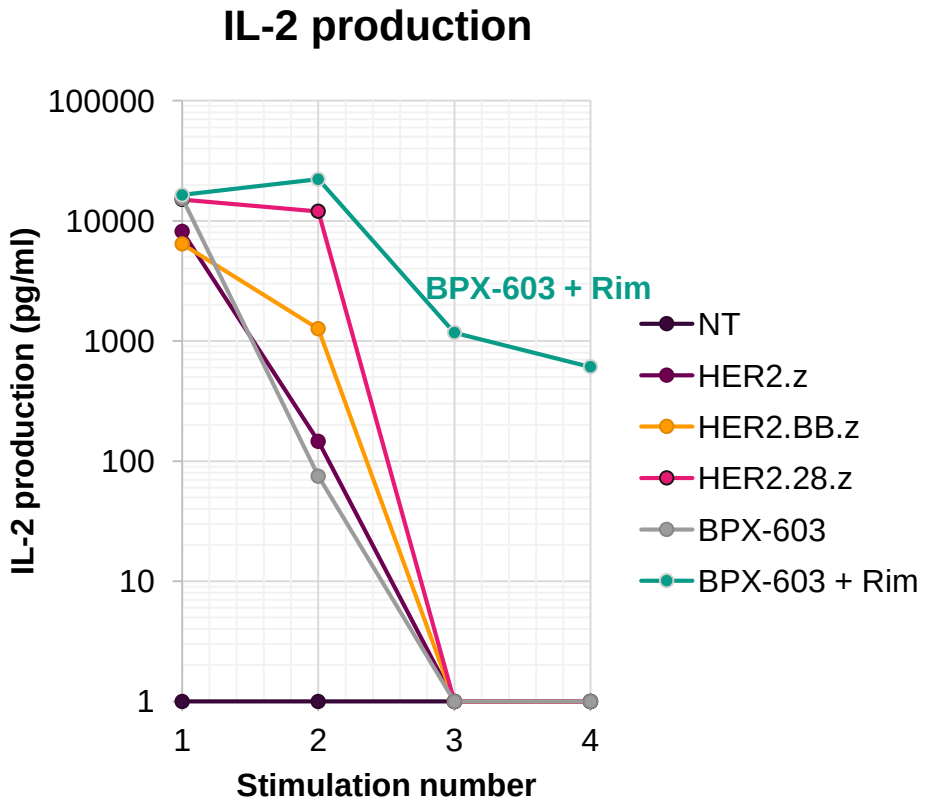


GoCAR Proliferation: Superior Expansion and Resistance to T Cell Exhaustion

iMC activation limits T cell dysfunction in repeat tumor stimulation exhaustion assay

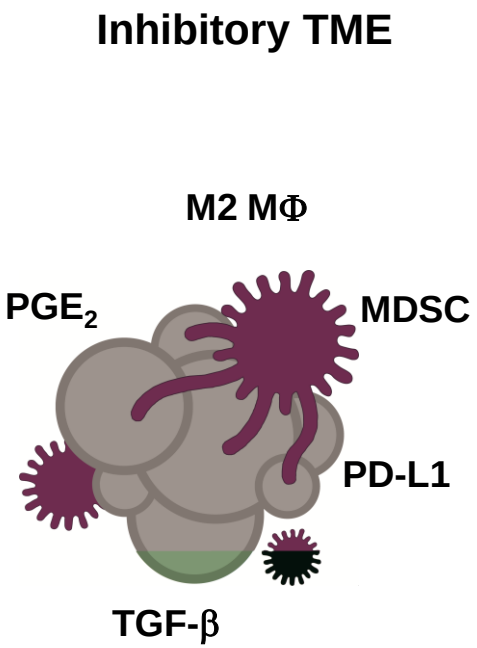


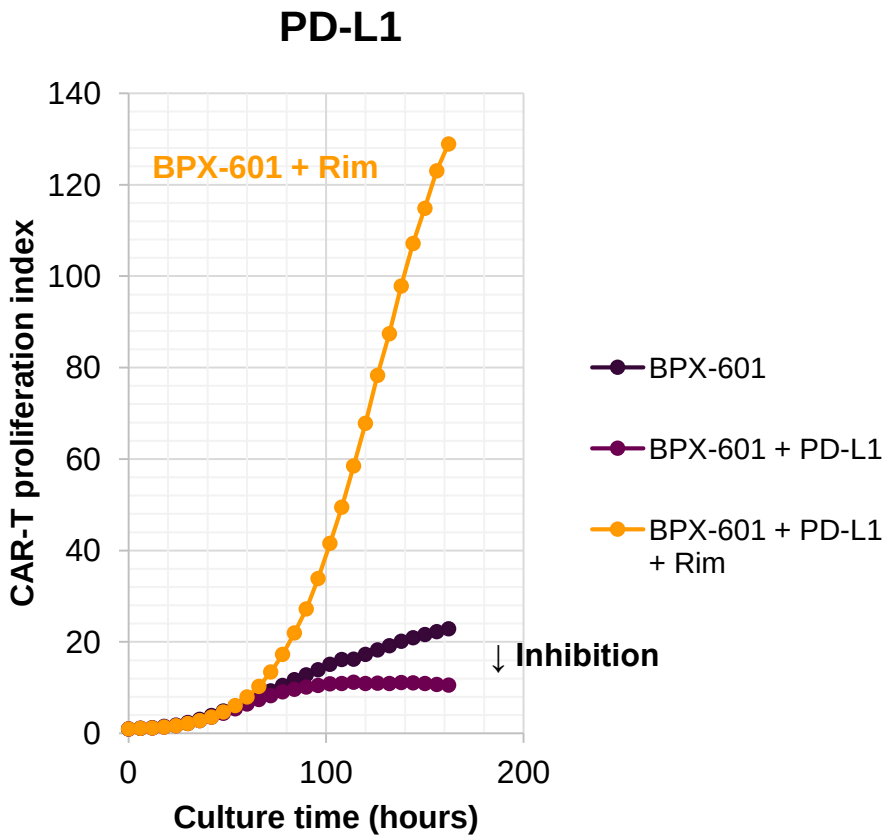


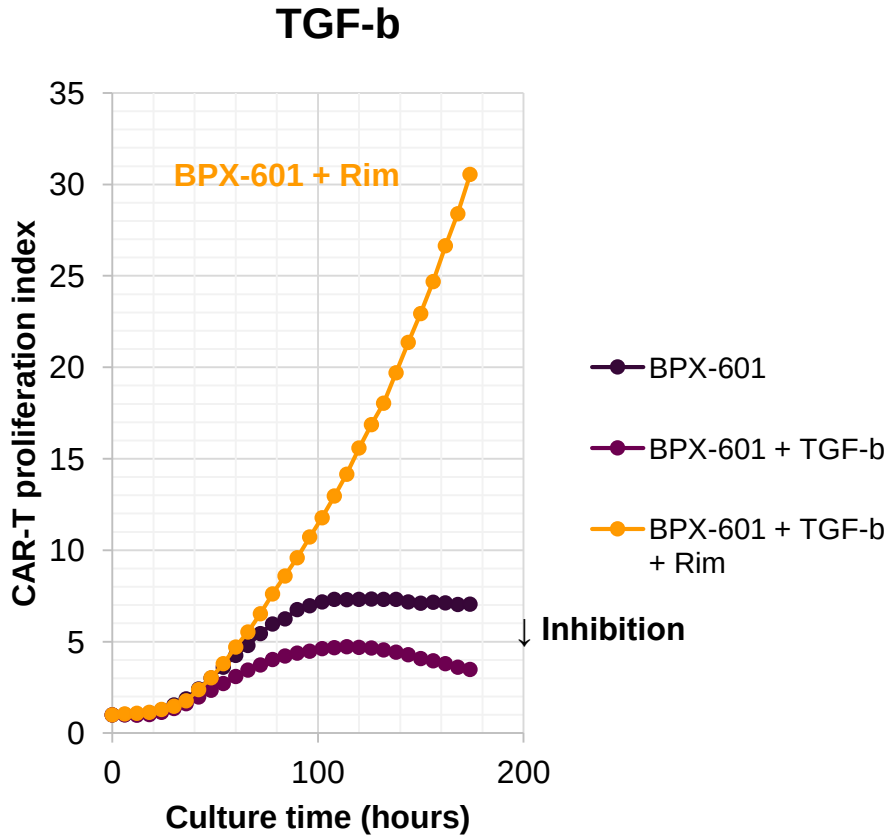


GoCAR Persistence: Resistance to Immune Suppressive TME

iMC overrides common inhibitory molecules in the tumor microenvironment







TME – tumor microenvironment

BPX-601
PSCA GoCAR-T

BPX-601 GoCAR-T Targets Solid Tumors Expressing PSCA

Product Profile Summary

- Attractive first-in-class solid tumor CAR-T opportunity
- First-in-human experience with iMC
- Phase 1 results presented at ASCO 2019 and ASCO GI 2020 demonstrate manageable safety, iMC-driven T cell activation and persistence, modulation of the tumor micro-environment, and biologic activity
- Phase 1 enrollment ongoing; data update expected Q4 2020

Unmet Need

High unmet need in solid tumors expressing prostate stem cell antigen (PSCA)

	Annual Incidence (U.S.)	Annual Deaths (U.S.)	% Expressing PSCA
Pancreatic	55k	44k	~50%
Prostate	165k	29k	75-90%

Incidence and annual deaths: Noone AM, Howlader N, Krapcho M, Miller D, Brest A, Yu M, Ruhl J, Tatalovich Z, Mariotto A, Lewis DR, Chen HS, Feuer EJ, Cronin KA (eds). SEER Cancer Statistics Review, 1975-2015, National Cancer Institute. Bethesda, MD, https://seer.cancer.gov/csr/1975_2015/, based on November 2017 SEER data submission, posted to the SEER web site, April 2018.
PSCA expression: Argani et al, Cancer Res 2001; Reiter et al., PNAS 1998; Abate-Daga et al, HGT 2014; Data on file

BPX-601: Phase 1 Trial Enrolling Cohort 5C

BP-012 trial in relapsed/refractory pancreatic cancer

	Lead-in (Cohort 0)	Dose Escalation (Cohorts 3, 4, 5A)	Standard Conditioning (Cohort 5B)	Repeat Rimiducid (Cohort 5C)
Pancreatic Patient Population	2L to 6L	2L to 6L	2L	2L
BPX-601 Dose *x10^6 cells/kg @ Day 0*	1.25	**1.25, 2.5, 5.0**	5.0	5.0
Conditioning	Cytoxan 1g/m^2 @ Day -3	Cytoxan 1g/m^2 @ Day -3	**Cytoxan 0.5g/m^2 Fludarabine 30mg/m^2 @ Days -5, -4, -3**	Cytoxan 0.5g/m^2 Fludarabine 30mg/m^2 @ Days -5, -4, -3
Rimiducid Dose	None	Single dose Day 7	Single dose Day 7	**Repeat dosing starting at Day 7**
Enrollment Status	Completed			Enrolling

Lead-In & Dose Escalation

Conservatively designed to evaluate safety

- Lead-in cohort with cells only
- Partial conditioning with Cytoxan monotherapy
- Single dose of rimiducid to activate iMC

Standard Conditioning Cohort (5B)

- Evaluated safety of standard Flu/Cy regimen with GoCAR-T
- Single dose of rimiducid to activate iMC

Repeat Rimiducid Cohort (5C)

- First POC using iMC repeatedly as designed
- Clinical data expected by end of 2020

ClinicalTrials.gov Identifier: NCT02744287

BPX-601: No Dose Limiting Toxicities Observed

Data presented at ASCO 2019

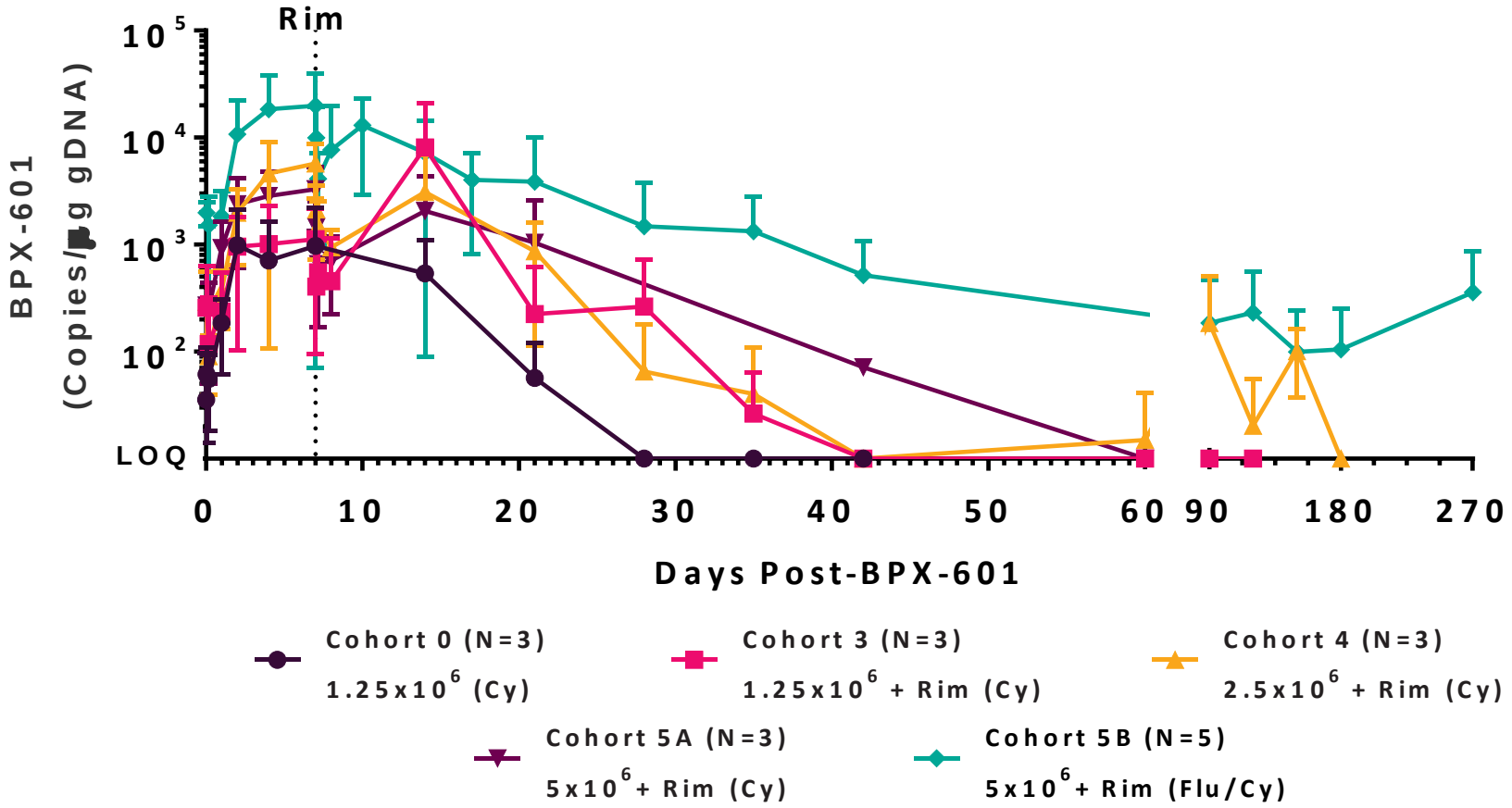
Patients, n (%)	Cohort 0 n = 3	Cohort 3 n = 3	Cohort 4 n = 3	Cohort 5A n = 4	Cohort 5B n = 5	All Patients N = 18
Any AE	3 (100)	3 (100)	3 (100)	4 (100)	5 (100)	18 (100)
Any SAE	2 (67)	1 (33)	0	3 (75)	4 (80)	10 (56)
Grade 3 & 4 TRAEs	0	0	0	0	1 (20)	1 (<1)
AEs in >15% of all patients, n (%)						
Febrile neutropenia	0	0	0	2 (50)	4 (80)	6 (33)
Fatigue	2 (67)	1 (33)	0	2 (50)	0	5 (28)
Neutropenia	0	0	0	1 (25)	4 (80)	5 (28)
Pyrexia	0	0	1 (33)	2 (50)	2 (40)	5 (28)
Dysuria	0	0	0	0	4 (80)	4 (22)
Hematuria	0	0	0	0	4 (80)	4 (22)
Nausea	2 (67)	0	0	0	2 (40)	4 (22)
Abdominal pain	1 (33)	1 (33)	0	0	1 (20)	3 (17)
Abdominal pain upper	0	1 (33)	1 (33)	1 (25)	0	3 (17)
Anemia	0	0	0	1 (25)	2 (40)	3 (17)
Back pain	1 (33)	1 (33)	0	1 (25)	0	3 (17)
Blood bilirubin increased	0	0	0	1 (25)	2 (40)	3 (17)
Hypotension	0	0	2 (67)	1 (25)	0	3 (17)

Becerra et al, ASCO 2019

- No dose limiting toxicities observed
- Adverse events (AEs) were generally consistent with cytotoxic chemotherapy or other cancer immunotherapies
- AEs related to BPX-601/rimiducid included:
  - One case of Grade 2 cytokine release syndrome (CRS)
  - One case of Grade 2 encephalopathy
  - Four cases of Grade 1-3 urologic toxicity (dysuria, hematuria, cystitis)

BPX-601: iMC-Driven T Cell Proliferation & Persistence

Flu/Cy lymphodepletion results in increased BPX-601 cell proliferation and persistence



- Peak expansion 4.9-fold higher in Flu/Cy cohort vs Cy alone cohorts
- Persistence improves with:
  - Administration of rimiducid to activate iMC
  - Higher cell dose
  - Lymphodepletion with Flu/Cy

Shaw et al, ASCO GI 2020

Data points represent the mean log VCN for each cohort and the dotted black line represents rimiducid administration at Day 7; † Patient 3 in cohort 5A did not have data for time points beyond Day 4 and thus is not included in the summary of cell persistence.

Cy, cyclophosphamide; Flu, fludarabine; LOQ, limit of quantitation; pts, patients; Rim, rimiducid.

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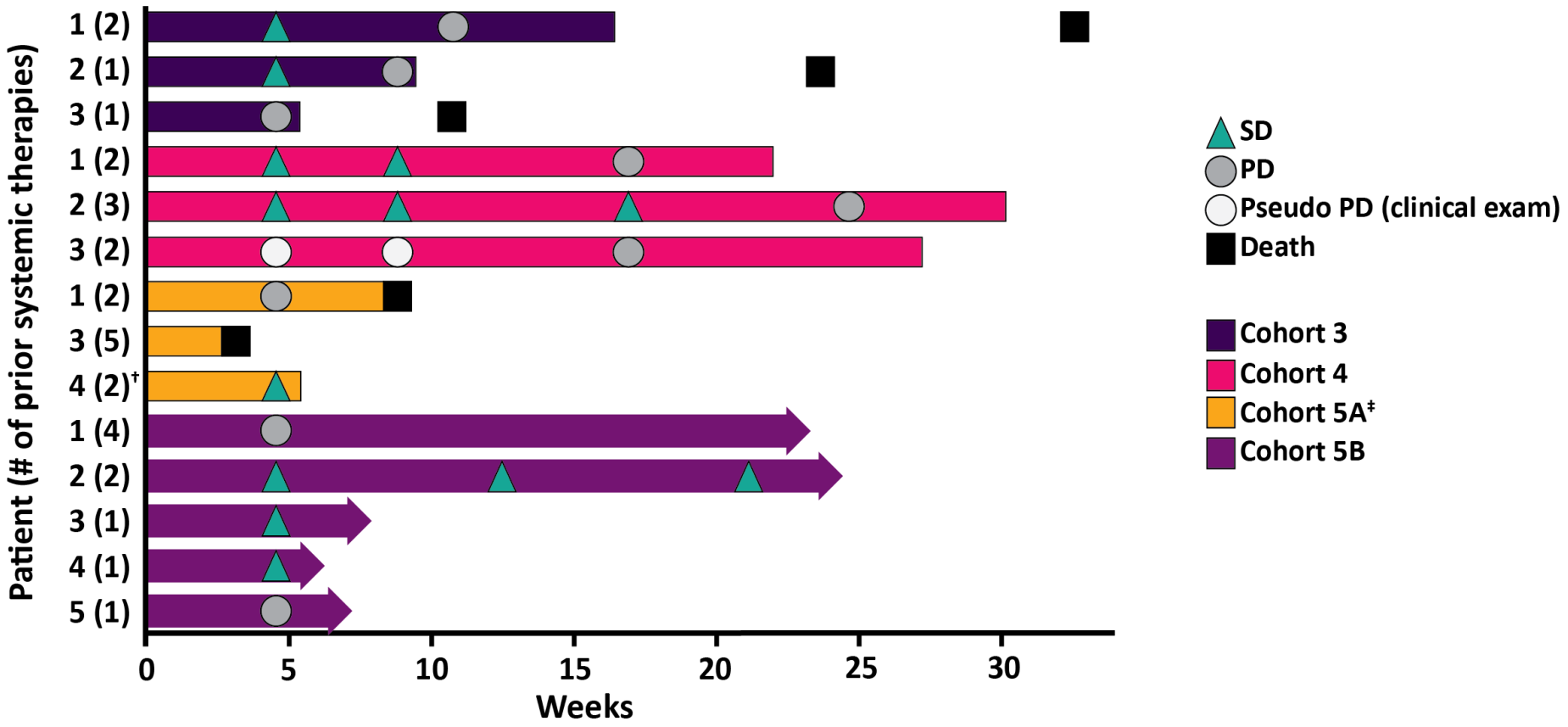
BPX-601: Evidence of Anti-tumor Activity



- 8 (62%) of 13 evaluable patients treated with BPX-601 and single-dose rim achieved stable disease; 3 had tumor shrinkage of 10-24%
- With 9.1 weeks median follow-up (range: 2.9-30.3), median time to next cancer therapy in patients that received subsequent treatment was 16.6 weeks (range 5.6-30.3)
- In Flu/Cy cohort, 2 patients with >median follow-up had time to next treatment >22 weeks (ongoing)

* Right arrow cap indicates ongoing treatment-free interval; † Patient withdrew consent for further follow-up; ‡ Patient 2 was not efficacy evaluable due to non-measurable disease at baseline.
PD, progressive disease; pseudo, pseudoprogression; SD, stable disease.

Becerra et al, ASCO 2019

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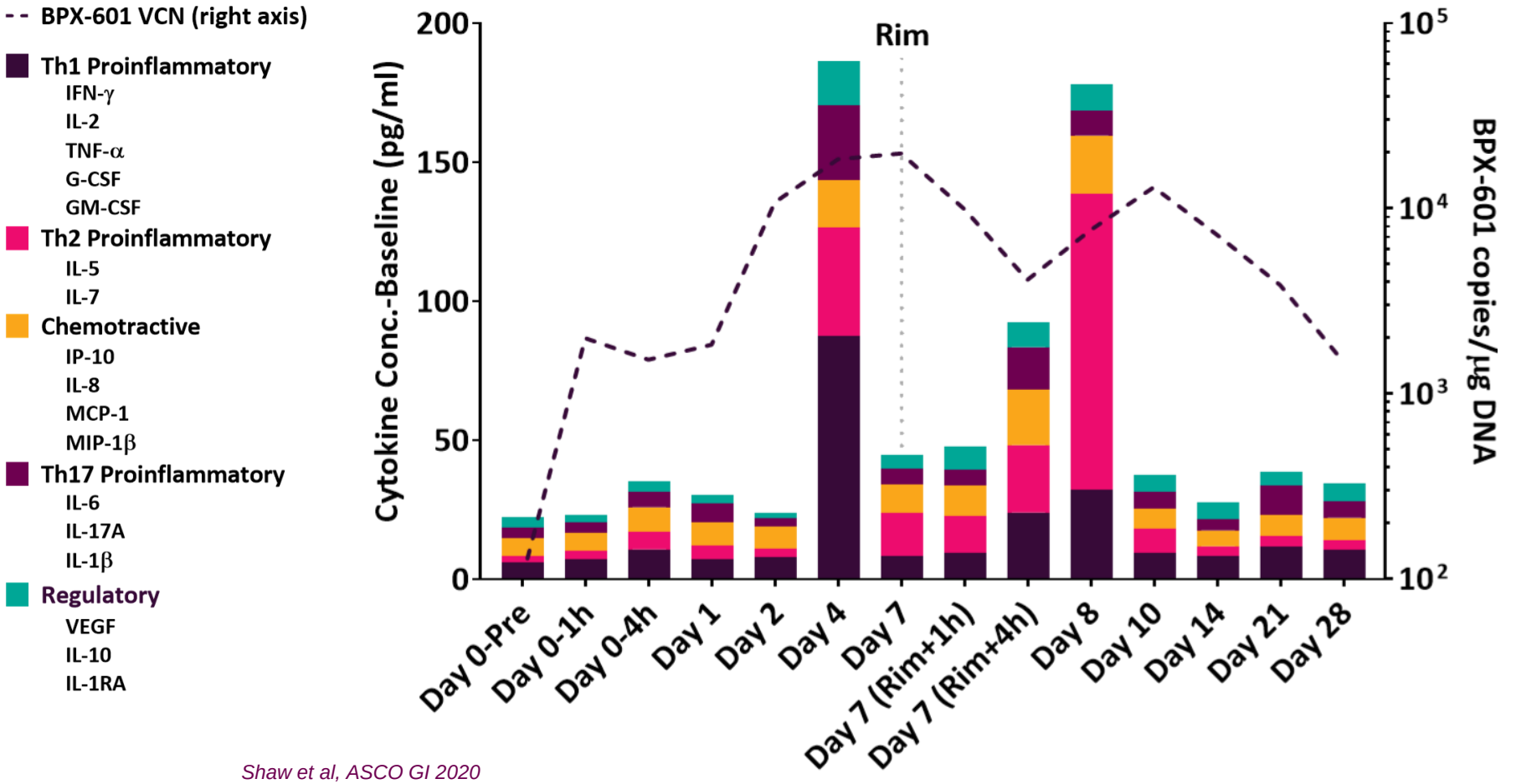
BPX-601: GoCAR-T Increased Immunomodulatory Cytokines

Infusion of BPX-601 and activation with rimiducid increased immunomodulatory cytokines



- Increases in Th1 and Th2 cytokines were observed with:
  - Administration of BPX-601 GoCAR-T cells
  - GoCAR-T activation with rimiducid

Shaw et al, ASCO GI 2020

Stacked bars represent the summed mean fold-change in concentration of cytokines in each category in patients from Cohort 5B (n=5). Black dotted line represents the mean VCN for Cohort 5B. Gray dotted line represented rimiducid administration on Day 7.
Conc., concentration; Rim, rimiducid.

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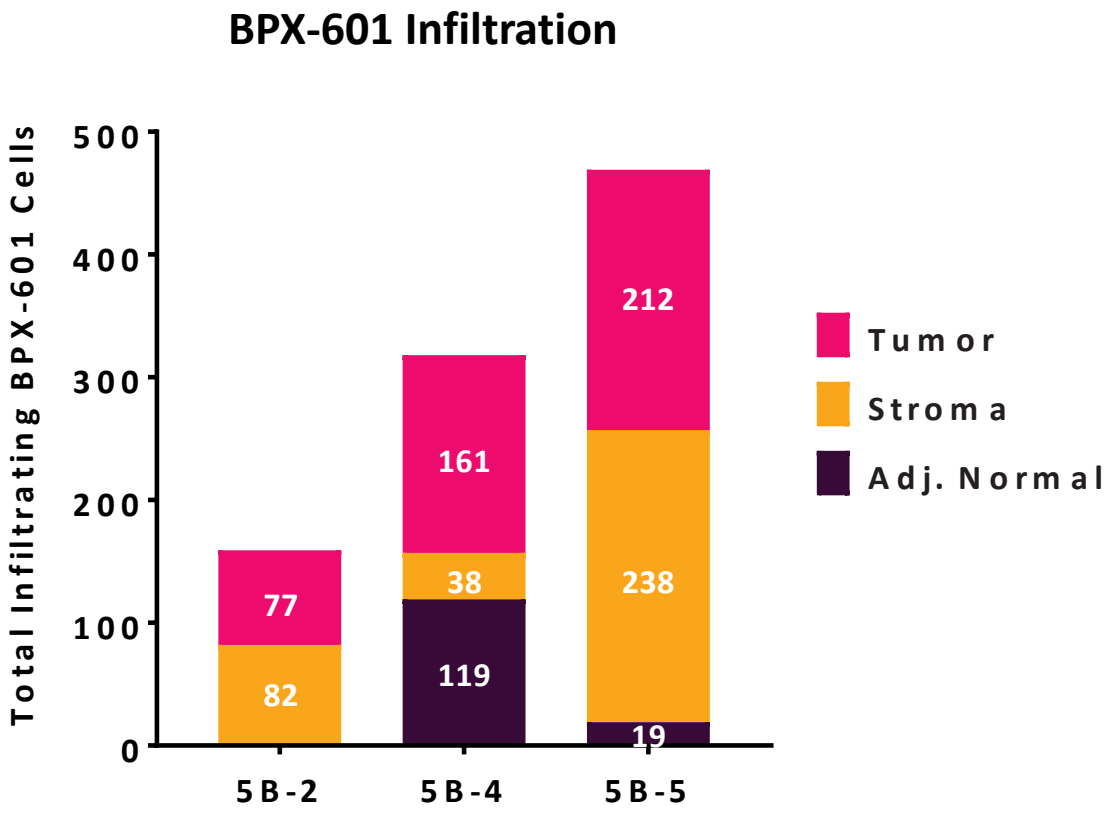
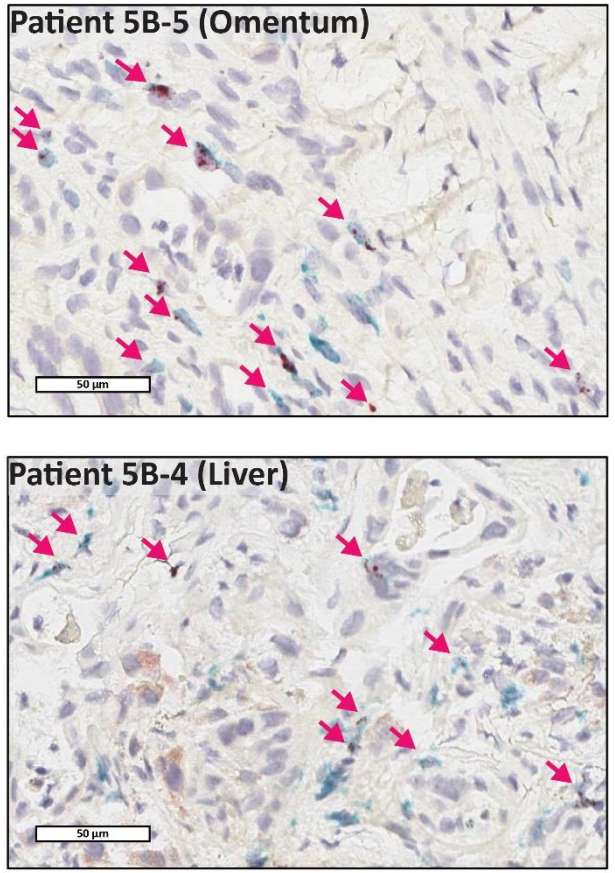
BPX-601: GoCAR-T Infiltrated Metastatic Pancreatic Tumors

On-treatment biopsies taken from metastatic lesions show BPX-601 tumor infiltration





CD3 = Blue; BPX-601 = Red, arrows

- Analysis of tumor metastases from patients showed:
  - Infiltration of BPX-601 GoCAR-T cells
  - BPX-601 effectively localized to tumor

Shaw et al, ASCO GI 2020

(Left) Stacked bars represent the total number of BPX-601 cells quantified in ISH stained tissue sections of available (n=3) biopsies from metastatic lesions of Cohort 5B patients. White numbers in bars indicate the number of BPX-601 cells measured within each ROI.
(Right) Representative images of CD3 (IHC) and BPX-601 (ISH) stained tissue sections of available (n=3) biopsies from metastatic lesions of Cohort 5B patients. Red arrows indicate BPX-601 GoCAR-T cells
Adj. normal, adjacent normal; ROI, region of interest.

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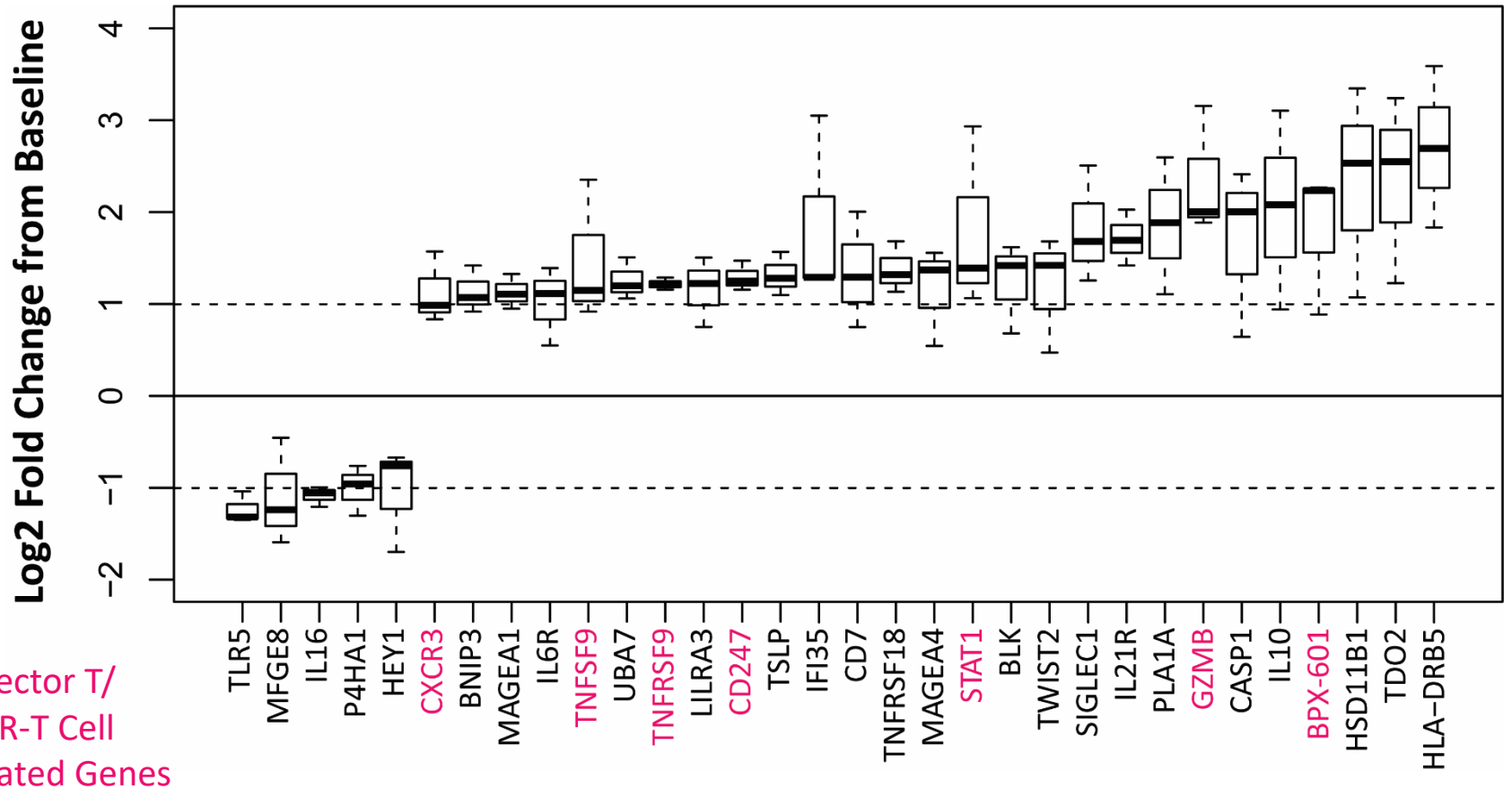
BPX-601: Modulation of Tumor Microenvironment

Changes in gene expression consistent with productive T cell immune responses

Differentially Expressed Genes in Tumor Metastases After BPX-601 + Rim

(Cohort 5B, n=3)



Effector T/ CAR-T Cell Associated Genes

- Upregulation of T/CAR-T cell associated genes including:
  - GZMB – Target cell killing by cytotoxic T cells
  - CXCR3 – Activated T cell trafficking
  - 41BB(TNFSF9) / 41BBL(TNFRSF9) – T cell costimulation
  - CD3Z (CD247) – TCR Signaling
  - STAT1 – Interferon signaling
  - BPX-601 – Infiltrating GoCAR-T cells

Shaw et al, ASCO GI 2020

Box and whisker plots indicate log2 fold change of genes with altered expression (upregulation or downregulation) while on-treatment (Day 14-21) from paired baseline sample (p-value < 10%). nCounter data using NanoString PanCan IO360 panel.

BPX-603
HER-2 GoCAR-T

BPX-603 Dual Switch GoCAR-T Targeting HER2

Product Profile Summary

- HER2 is a validated tumor antigen expressed on numerous solid tumors with high unmet need
- Historical HER2 CAR-T studies have shown modest overall activity and off-tumor / on-target toxicity
- BPX-603 designed to potentially address limitations of previous efforts:
  - Moderate affinity scFv to enhance target engagement and activity
  - MC signaling to increase cell proliferation & persistence, modulate the TME, and enhance host immunity
  - Bellicum switch technology designed to time and manage CAR-T activation and enable mitigation of acute toxicities

Unmet Need

Indication	Incidence[1]	HER2+	5-year OS (Stage IV)[1]
Gastric	28,000	10-30%[3]	<20%
Colorectal	145,000	10%[4]	<15%
Ovarian	22,000	20-30%[5]	<30%
Uterine/ Endometrial	61,000	50-80%[6]	14-69%
Glioblastoma	12,000	20-30%[2]	<20%
Breast	271,000	16%[7]	90%

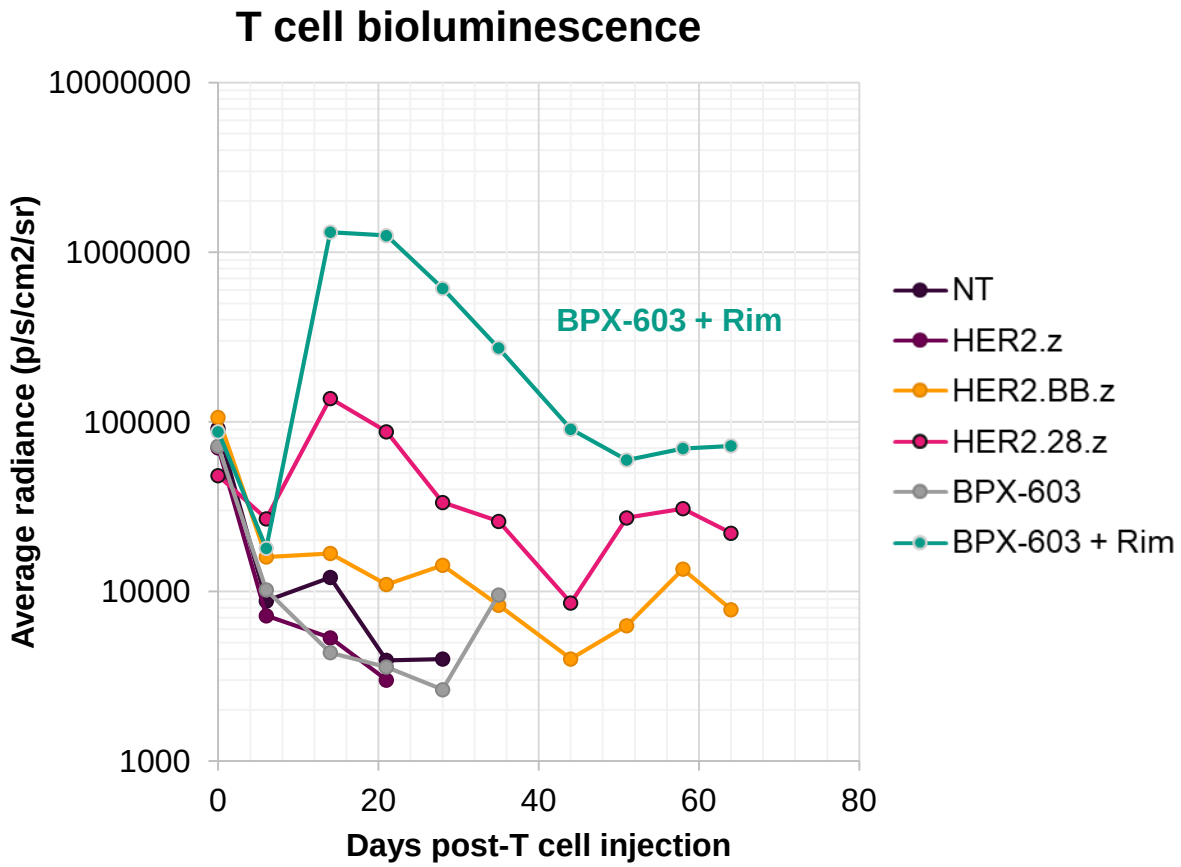
[1]National Cancer Database, American Cancer Society, https://www.cancer.org, accessed 21 December 2018; [2]Liu et al., Cancer Res 2004; [3]Gravalos et al., Annals Oncol 2008; [4]Tu et al., Exp Ther Med 2018; [5]Berchuck et al., Cancer Res 1990, Bartlett et al., Brit J Cancer 1996; [6]Grushko et al., Gynecologic Oncol 2008, (7) Cronin et al, Cancer Invest. 2010

Historical HER2 Studies: Modest Clinical Outcomes

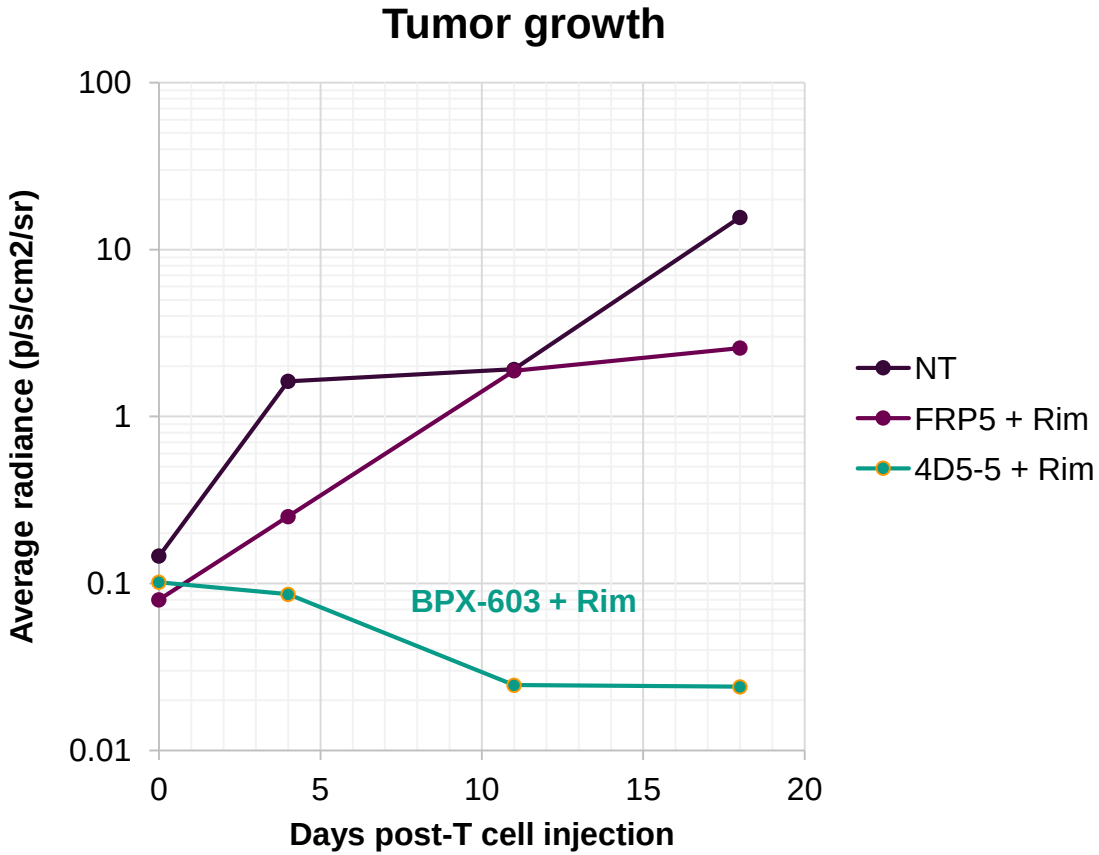
Study Properties	Morgan, 2010	Ahmed, 2015	Feng, 2017	Ahmed, 2017	Hegde, 2019
Construct	4D5-28-BB-z	FRP5-28-z	Her2-BB-z	FRP5-28-z	FRP5-28-z
Indication(s)	Metastatic colon	Sarcomas	CCA and PCa	GBM	Sarcomas
Patient number	1	19	11	17	10
HER2 expression	≥2+ (IHC)	≥1+ (IHC)	>50% positive	≥1+ (IHC)	≥1+ (IHC)
CAR-T dose	10^{10}	10^{4} - 10^{8}	10^{6}	10^{6} - 10^{8}	10^{8}
CAR-T expansion	NE	Negligible	>1,000 copies	Negligible	>10,000 copies
Toxicity	Lung reactivity	No DLTs	Mild AEs	Mild AEs	Mild AEs
Outcome	Grade 5 toxicity	1 PR	1 PR	1 PR	2 CR
Total Responses	2 CR, 3 PR, 5/58 (8.6% ORR)				

BPX-603: Compelling Preclinical Evidence

iMC co-activation enhances cell proliferation relative to current CAR-T standards



Moderate affinity scFv enhances anti-tumor effect relative to low affinity FRP5



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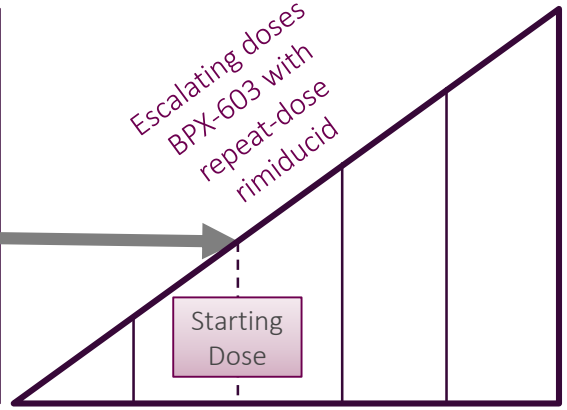
BPX-603 Phase 1/2 Trial Design

Two-Part Safety/Activity Study of HER2-Targeted Dual Switch GoCAR-T Cells in Previously Treated HER2+ Solid Tumors

Phase 1: 3+3 Dose Escalation

Locally advanced or metastatic HER2+ solid tumors[1] where standard treatment is no longer effective, does not exist, or subject is ineligible[2]



- Sequential patient enrollment
  - ≥28 days for cohort 1
  - ≥14 days for subsequent cohorts
- First subject in each dose level receives cells only without rimiducid

Phase 2: Multi-Arm Dose Expansion in Select Tumor Types

Cohort 1: Gastric

Cohort 2: Breast

If ≥1 response

- Cohort 3: Ovarian
- Cohort 4: Colorectal
- Cohort 5: GBM[3]
- Cohort 6: Uterine/Endometrial

- Expansion cohorts 10 patients each
- Ability to expand each cohort based on clinical response

[1] GBM excluded from Phase 1

[2] Must include approved HER2-targeted therapy for breast/gastric cancers

[3] Subjects with GBM will be dosed at recommended dose for expansion (RDE) -1

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Off-the-Shelf Program BCMA GoCAR-NK

GoCAR-NK Powers Potential of NK Cell Therapies

NK Cells Have Therapeutic Advantages

- Innate ability to kill tumor cells through multiple mechanisms
- Favorable safety profile following adoptive transfer
- Potential off-the-shelf cell therapy given low propensity to cause GvHD

Other NK Cell Features Limit Therapeutic Utility

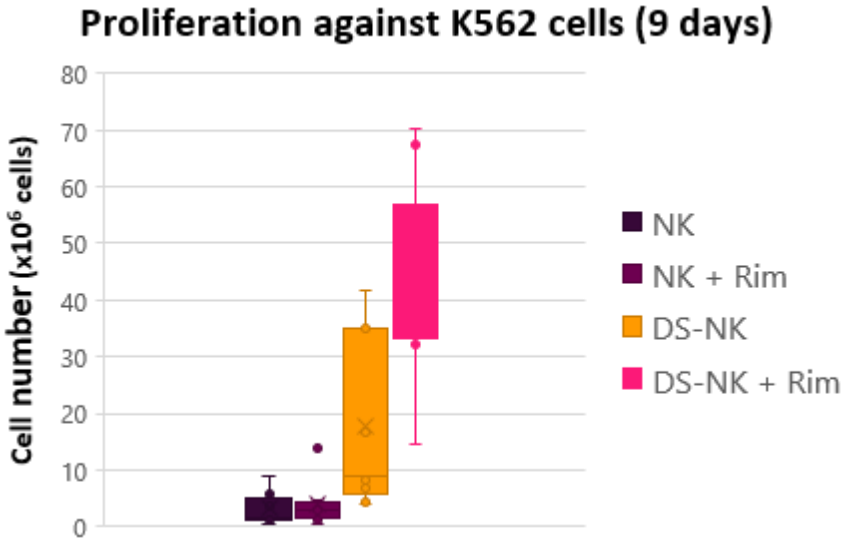
- Unmodified NK cells show limited in vivo expansion and persistence (7-14 days)
- Tumors can develop defense mechanisms to limit NK cell cytotoxicity and cytokine production

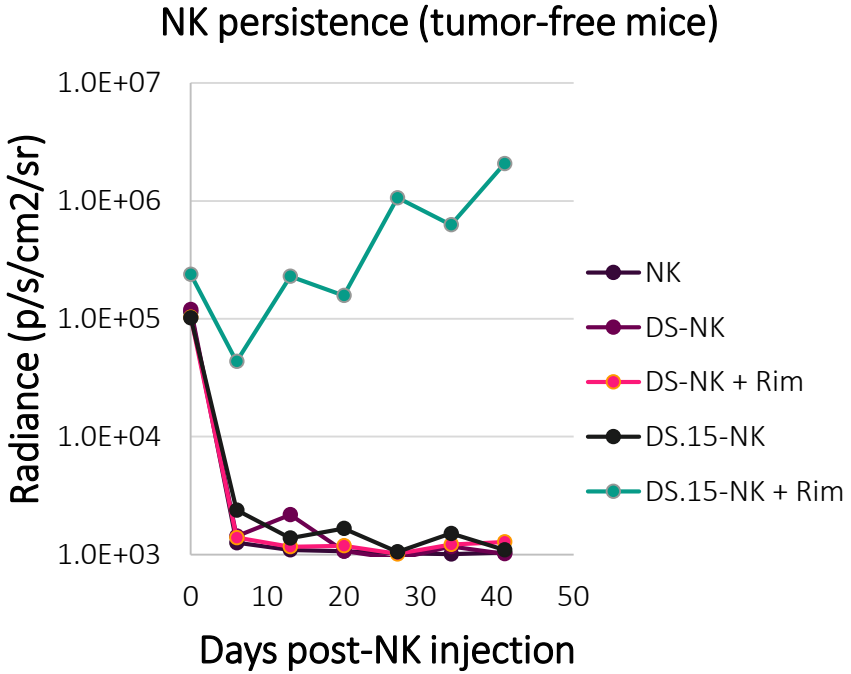
Preclinical Data Support GoCAR-NK Advantages

- MC improves proliferation and survival of NK cells
- MC signaling enhances innate cytotoxicity of NK cells
- MC synergizes with IL-15 to further increase anti-tumor potency
- iMC, IL-15 and tumor-specific CAR transgene expression result in superior anti-tumor effects in multiple tumor models

iMC Drives NK Cell Proliferation

iMC and IL-15 synergize to promote NK cell survival and persistence





- NK cell infusions frequently suffer from poor growth and survival
- iMC activation enhances NK cell expansion
- IL-15 is a potent growth and survival factor for T and NK cells, but not T_{reg}
- iMC activation synergizes with autocrine expression of IL-15 to promote NK cell persistence in vivo

*DS: Dual switch that includes iMC and CaspaCIDe switches;
DS.15: Dual switch and express IL-15

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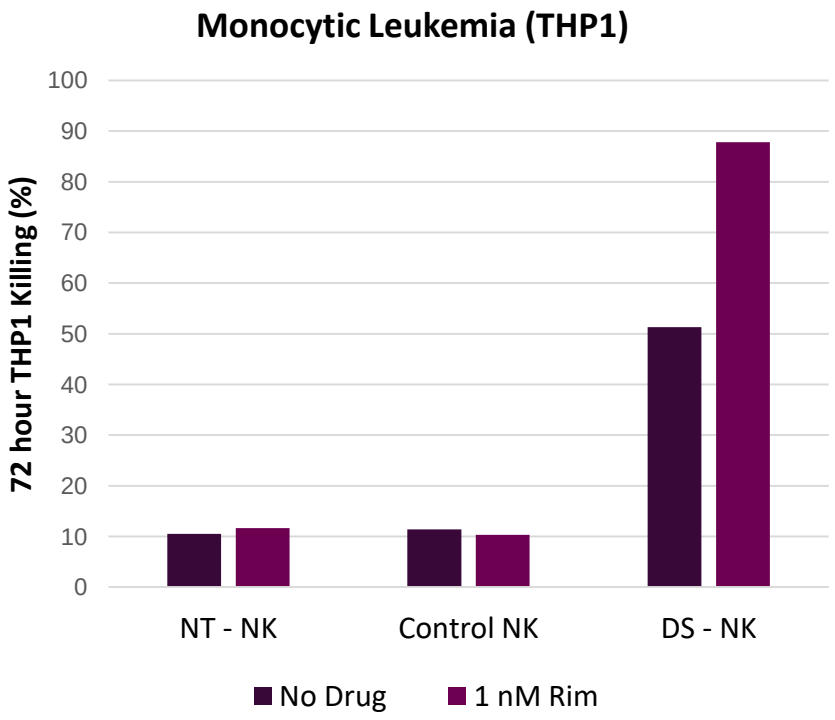
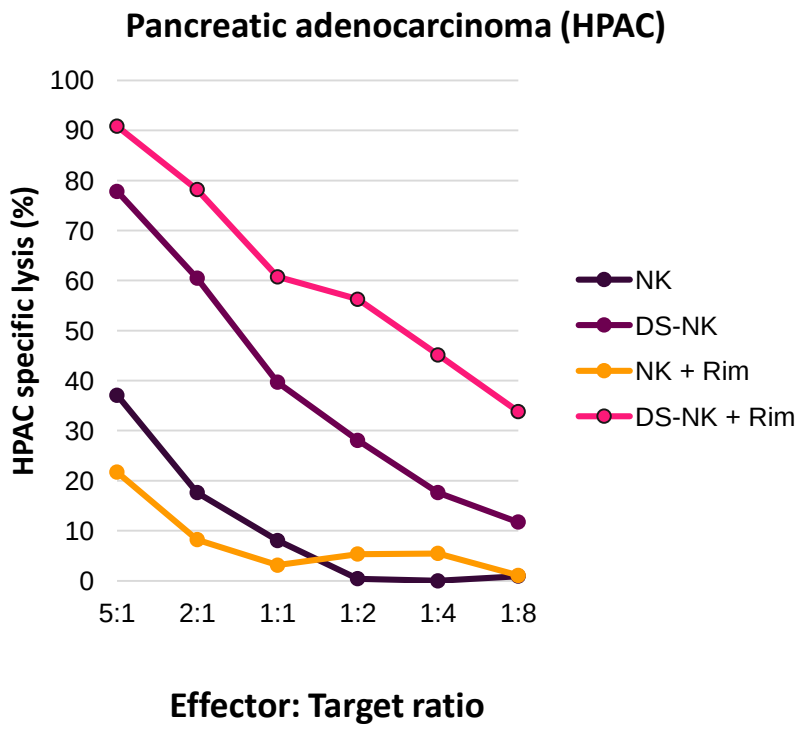
iMC Increases Innate Cytotoxicity of NK Cells



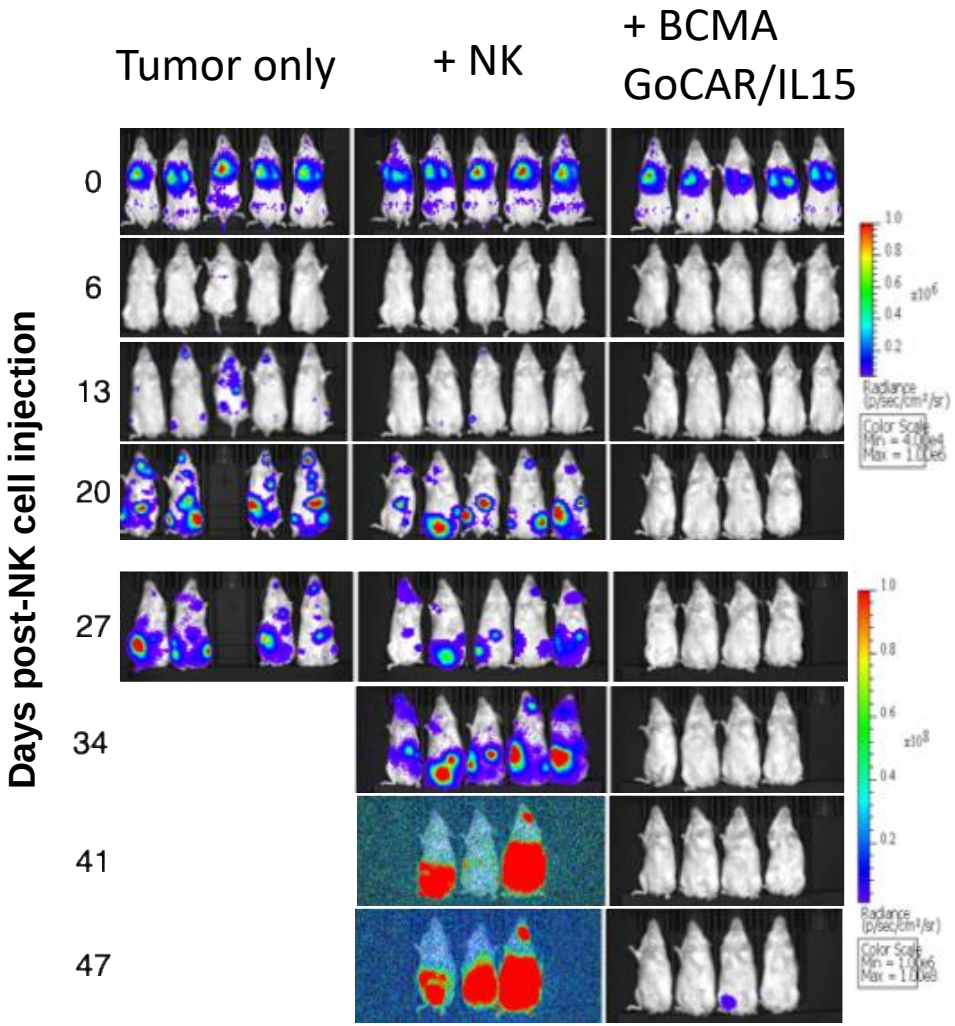


- CAR-based cell therapies frequently suffer from tumor relapse due to loss of the CAR antigen's expression
- NK cells possess mechanisms to direct innate anti-tumor cytotoxicity
- iMC expression and activation enhances innate NK cell cytotoxicity against tumor targets with high and low MHC-I expression
- iMC enhanced CAR-NK therapy may reduce the risk of tumor-antigen escape

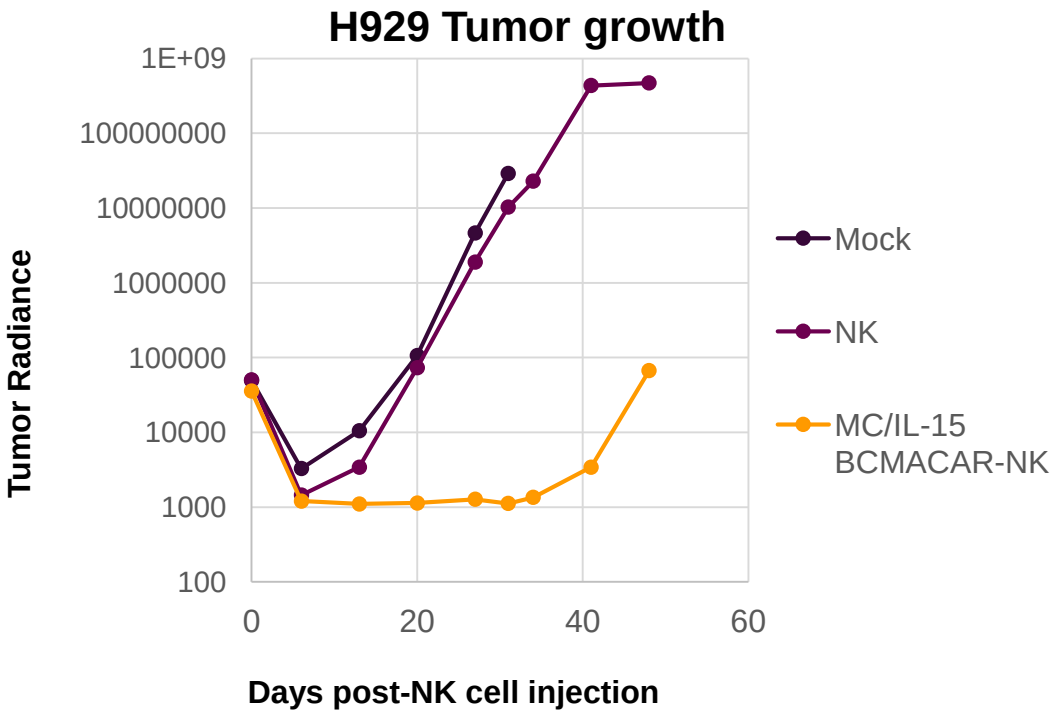
*DS: Dual switch that includes iMC and CaspaCIDe switches

MC/IL-15 BCMA CAR-NK Cells - Anti-tumor Activity

CAR-NK persistence with rimiducid stimulation extends past eight weeks



*Tumor cell bioluminescence



Bellicum

BCMA - Attractive Target for OTS GoCAR-NK

- BCMA is a well validated target for autologous CAR-T therapy
- GoCAR-NK has the opportunity to improve durability of allogeneic cell therapy
  - GoCAR enhances NK cell persistence and cytotoxicity
  - GoCAR enhances innate NK cell anti-tumor activity against myeloma cells that may compensate for antigen loss
  - Potential to improve durability using healthy patient donor cells[2,3]
- OTS GoCAR-NK cells expected to have added advantages of shorter time to treatment and lower cost of goods
- Additional preclinical data to be presented by end of 2020

[1]BMS and Bluebird joint ASH2019 press release, NCT03361748 KarMMa topline data
[2] Graham et al. Cells 2018;
[3] June et al. NEJM 2018

Summary

Anticipated Key Program Goals & Milestones

	Goals & Milestones	Planned Timing
BPX-601	Initial Phase 1 cohort 5C data (repeat rimiducid) – Pancreatic Cancer	Q4'20
	Phase 1/2 data update – Pancreatic Cancer	2H'21
	Initial Phase 1/2 data – Prostate Cancer	2022
BPX-603	Initiate Phase 1/2 trial	2H'20
	Initial Phase 1 data	2H'21
	Phase 1 data update	2022
OTS GoCAR-NK	Preclinical data update	Q4'20
	Preclinical data update	2H'21
	IND Clearance	2022

Investment Summary

Building a next generation cell therapy pipeline around the GoCAR platform

GoCAR Platform

Differentiated co-activation domain (MyD88/CD40) and switch technology drive greater proliferation, persistence, power, and performance

BPX-601

- Autologous GoCAR-T targeting PSCA in pancreatic & prostate cancers
- Phase 1/2 enrolling
- Data update planned 4Q 2020

BPX-603

- Autologous GoCAR-T targeting HER2 in HER2+ solid tumors
- Initiate Phase 1/2 trial 2H 2020

GoCAR-NK Program

- First off-the-shelf (OTS) GoCAR program targeting BCMA
- Preclinical data update planned 4Q 2020

Cash runway extends into 2H'21

- $68.0M cash as of June 30, 2020