Corporate Presentation

December 18, 2024

BrightPath Biotherapeutics

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BrightPath Bio (Tokyo Stock Exchange Growth 4594)

- BrightPath Bio is a clinical-stage biopharmaceutical company focused on developing immuno-oncology products
- Three focused modalities: cell therapy, immune modulatory antibody, and cancer vaccine
- BP2202 originates from a novel allogeneic CAR-T platform utilizing iPS cell-derived NKT cells

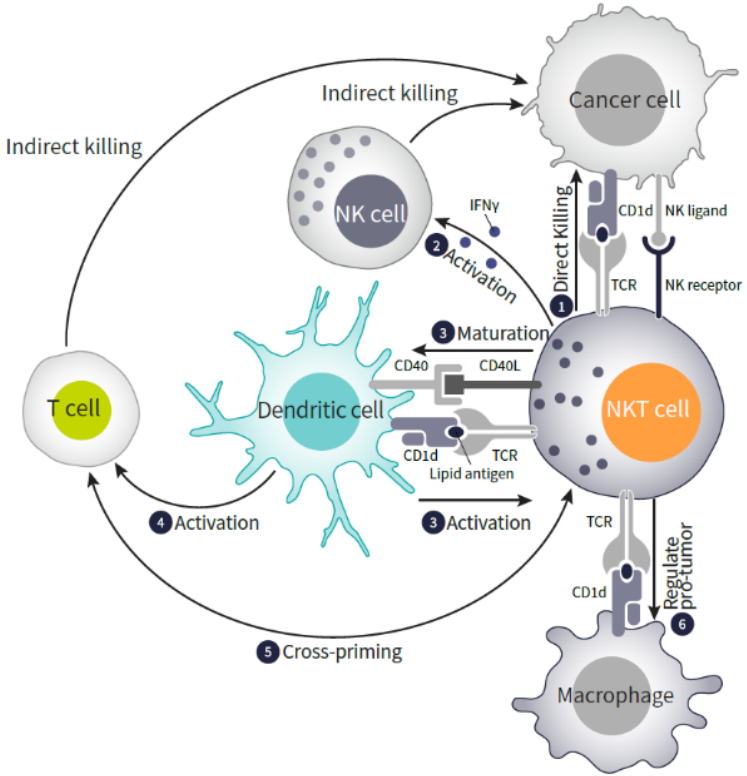
Developed product	Mechanism/target	Cancer type	Discovery	Preclinical	PI	PII
Cell Therapy						
BP2201	iPS cell-derived NKT cells	HNSCC				
BP2202	BCMA CAR-iPSNKT	Multiple Myeloma				
BP2301	HER2 CAR-T	Sarcoma Gynecological Tumors				
Antibody						
BP1200	CD73					
BP1202	CD39					
BP1210	TIM-3					
BP1212	CD39 × TIM-3					
BP1223	CD39 × CD3	Acute Myeloid Leukemia				
Cancer vaccines						
BP1209	Personalized neoantigen	Solid Tumor				

Cell Therapy

BP2202 (iPSC-derived BCMA CAR-iNKT)

A novel allogenic CAR-T platform utilizing iPS cell-derived NKT cells as effector

- **Invariant natural killer T (iNKT)** cell is a rare subset of T lymphocytes that has not only direct but indirect anti-tumor activity by **priming CD8⁺ T cells** and other immune cells
- Allogeneic iPSC-derived CAR-iNKT cells retains the native iNKT cells' function of inducing host CD8⁺ T cells
  - The enhanced fitness and the spread antigens of the **induced host CD8⁺ T cell** are expected to prolong the durability of clinical response
- **Induced pluripotent stem (iPS)** cell technology enables clinical-scale manufacturing of iNKT cells, which are naturally rare in human blood, while preserving their original functions

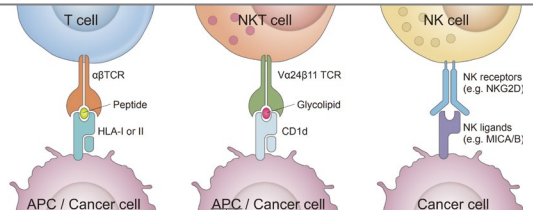


BP2202 (cont'd)

Differentiation factors: iPS cell-derived iNKT cells induce host endogenous CD8+ T cells with memory phenotype, which is expected to provide durability of clinical responses

	allo NKT	alo αβT	allo γδT	allo NK
Innate - adaptive immunity bridging				
DC cross-talking	✓	Possible source of durability		
CD8+ T cross-priming	✓			
Myeloid cell (TAM, MDSC) reprogram[1]	✓			
Innate anti-tumor response	✓		✓	✓
HLA independency				
No need to TCR gene editing[2]	✓		✓	n.a.
Low GvHD risk	✓		✓	✓
Proliferating capacity	✓	✓	✓	

1 iNKT cells kill tumor-associated macrophage / myeloid-derived suppressive cell thorough TCR/CD1d.
2 TCR gene editing is not necessary to avoid the risk of GvHD. Thus, proliferative capacity is not dampened

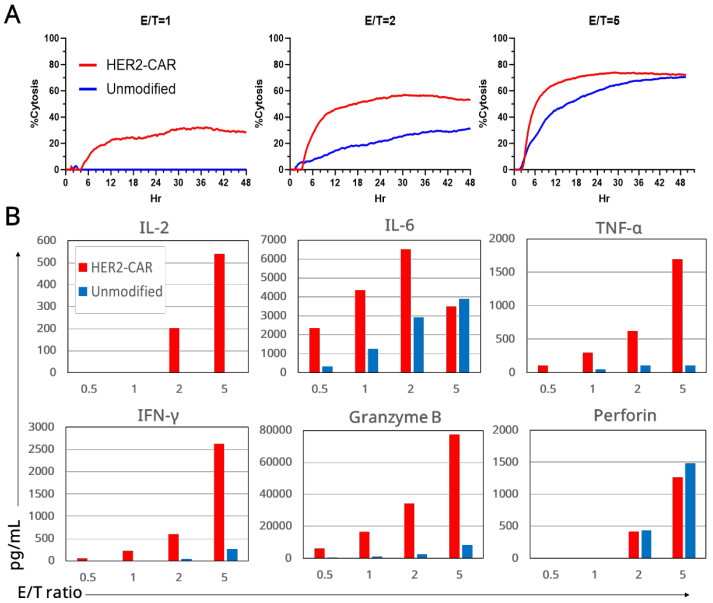


- NKT cell is a rare subset of innate lymphocytes representing less than 1% of the total lymphocyte
  - ➢ Rationale to use iPSC as cell source for clinical scale manufacturing of functional iNKT cells
- Express a semi-invariant TCR recognizing glycolipids presented by the monomorphic MHC like molecule CD1d
  - ➢ HLA independency provides low GvHD risk

BP2202 (cont'd)

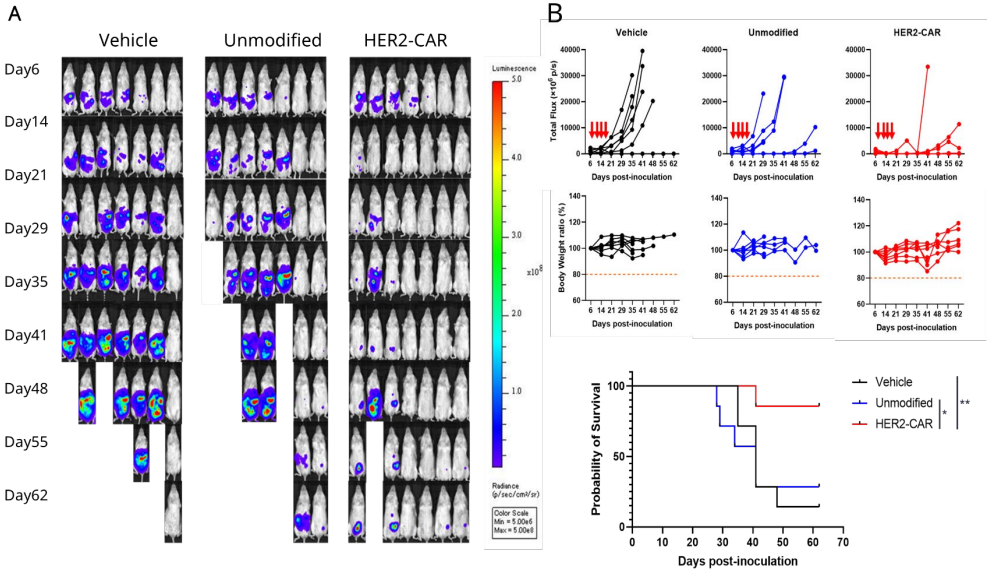
Prototype iPSC-derived HER2-CAR iNKT showed target-specific anti tumor effects

- Cytotoxic activity (A) and Cytokine secretion (B) (HER-2 expressing tumor cell line SK-OV-3)



BrightPath SITC2022

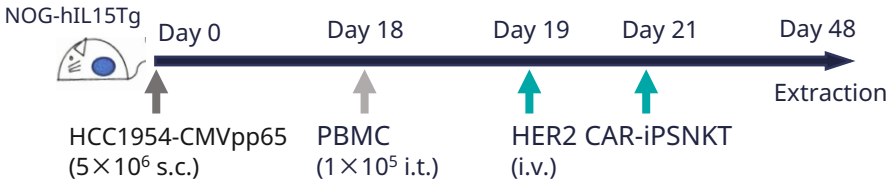
- Tumor burden of SK-OV-3-luc bearing mice (A), Spider plot of total flux and body weight ratio (B) and Survival rate (C)

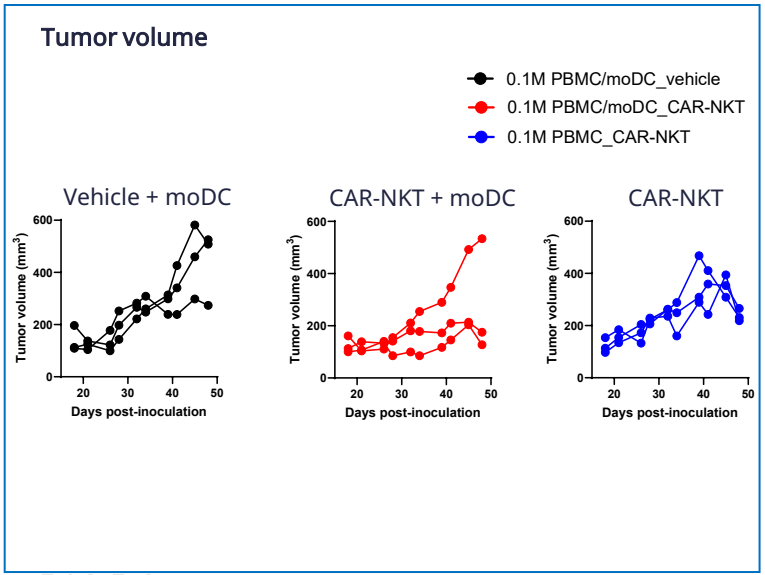


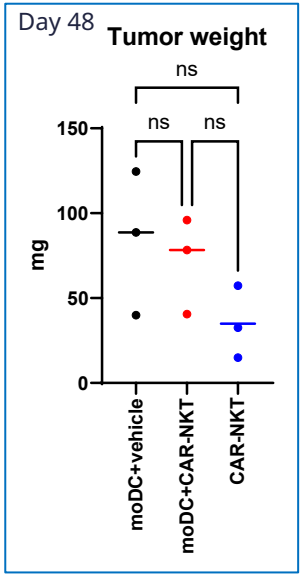
BrightPath SITC2023

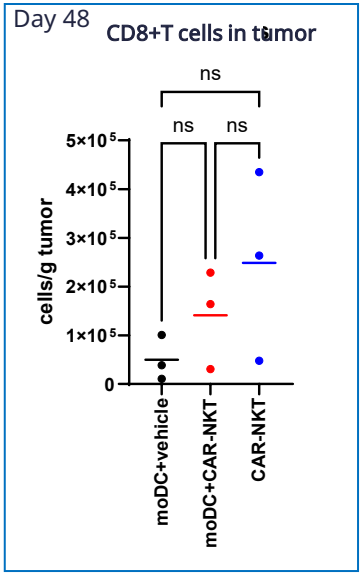
BP2202 (cont'd)

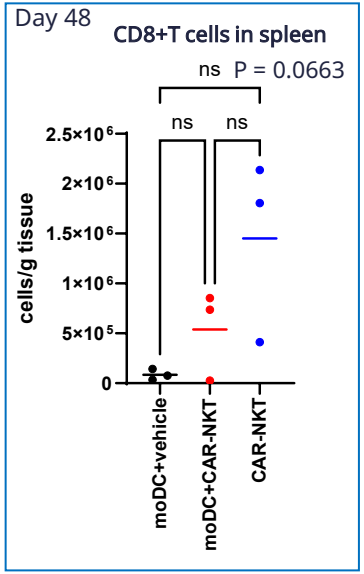
Prototype iPSC-derived HER2 CAR-iNKT demonstrated the ability to activate CD8+ T cells in vivo, leading to a reduction in tumor burden











BP2202 (cont'd)

Early clinical activities of unmodified iPS-NKT has been confirmed in 2023

Target (IND filing year)	Company	unmodified
iPS-**NKT**	BrightPath Biotherapeutics	2020 (RIKEN)

- The first-in-human Phase I study of the iPS cell-derived NKT cells was conducted in patients with r/r HNSCC under the primary endpoint of tolerability and safety from mid 2020
- Some patients at high-dose experienced some level of tendency of tumor shrinkage, which has demonstrated encouraging **early clinical activity of the iPS cell-derived NKT cells**

- These initial safety and efficacy results of the first-in-human study are encouraging and provide preliminary evidence that using iPS-NKT as effector cells for a novel allogeneic CAR-T platform might be an effective cancer treatment strategy

  - In this study, iPS-NKT cells were administered at a low-dose ($3x10^7$cells/m^2) and high-dose ($1x10^8$cells/ m^2) in multiple dosing, through the tumor artery as monotherapy without prior lymphodepletion to exert its most distinct feature of priming endogenous anti-tumor T cells.
  - Low-dose (n=3) : 1 SD, 2 PD DCR 33.3%
    High-dose (n=6): 4 SD, 1 PD, 1 NE DCR 80% (4 of 5 evaluable patients)
  - The most frequently observed trAEs were Grade 1 or 2 fever (1 patient at low-dose, 4 patients at high-dose)

Source: Professor Shinichiro Motohashi MD, Ph.D, of Chiba University, at CD1-MR1 2024 Conference

BP2202 (cont'd)

Landscape of iPS-derived immune cell development and out-licensing status

Target (IND filing year)

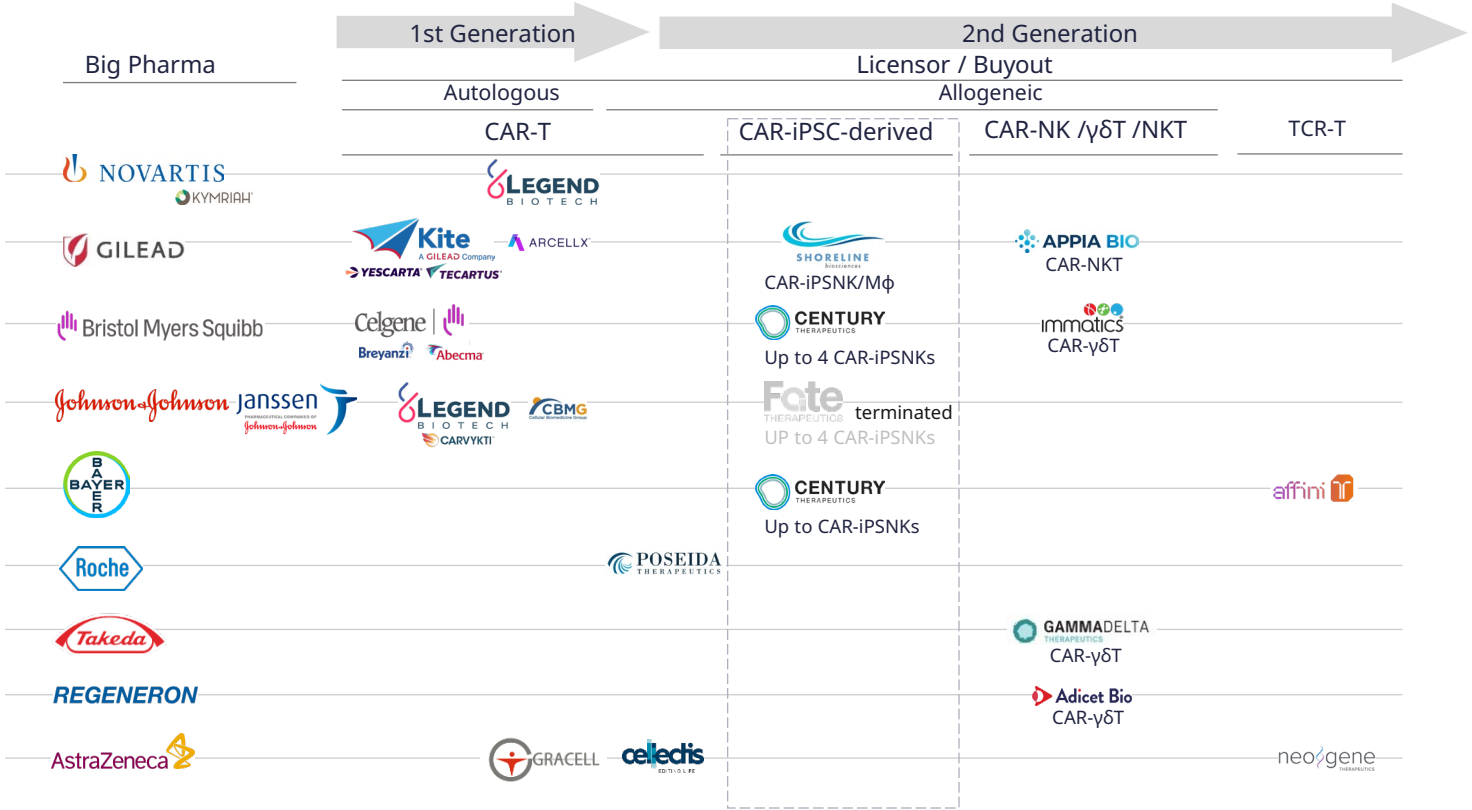
	Company	unmodified	Blood Cancer CAR: Lymphoma	Blood Cancer CAR: Leukemia	Blood Cancer CAR: Multiple Myeloma	Solid Tumor CAR	Collaborations (Platform type licensee)
iPS-T	Fate Therapeutics	2018				HER2 (2023)	Ono 2018 (2 Targets)
iPS-NK	Fate Therapeutics		CD19 (2023)				
iPS-NK	Shoreline Biosciences		Gilead CD19/CD20(−) CD5 (−)	Gilead Not disclosed (−)	Gilead TACI/BCMA (−)		Gilead 2021(Multiple Target); BeiGene 2021
iPS-NK	Century Therapeutics		Bayer CD19 (2022)	Bristol Myers Squibb Not disclosed (2024)	Bristol Myers Squibb Not disclosed (2024)		Bayer 2019 (3 Target); Bristol Myers Squibb 2021 (4 Target)
iPS-NK	Cytovia Therapeutics				CD38 (2025)	EGFR (2025) GPC3 (2024)	
iPS-NKT	BrightPath Biotherapeutics	2020 (RIKEN)	→	→	→		

Source: Company

-: IND schedule undisclosed at the research stage

BP2202 (cont'd)

Platform licensors and big pharma (licensees)

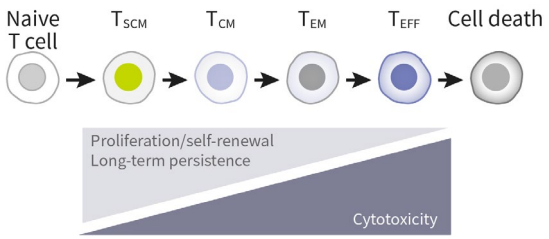


BP2301 (HER2 CAR-T)

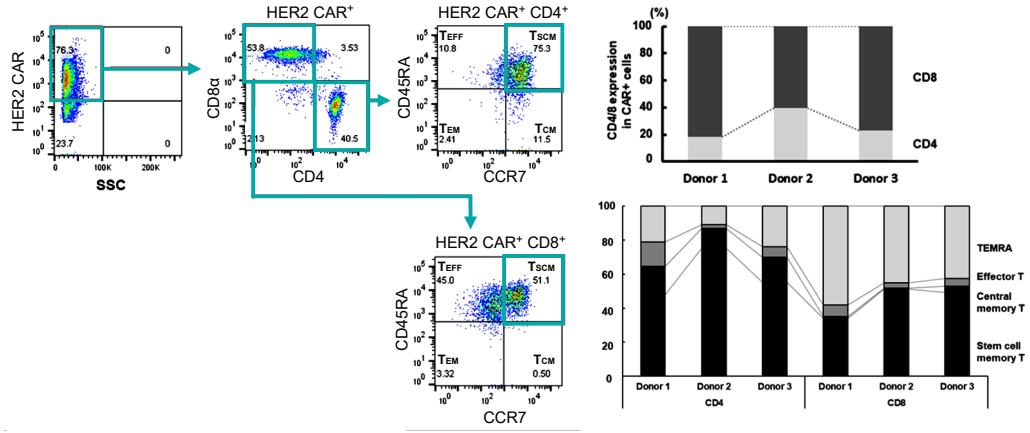
Autologous, non-virally CAR transduced, HER2-targeting CAR-T cells

- Stem cell memory-like T (T_{SCM}) phenotype-rich CAR-T cells, mediated by the non-viral piggyBac transposon system for CAR transduction
- T_{SCM} effector exhibiting continuous proliferation capacity and self-renewal ability, and long-lived in vivo
- Able to overcome T cell exhaustion in an immunosuppressive solid tumor microenvironment, leading to durable clinical responses

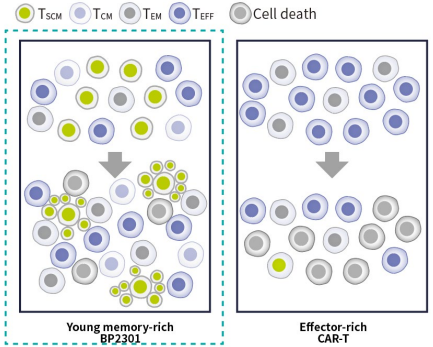
• T cell differentiation and phenotypes



• BP2301 exhibited memory-like phenotype



• Persistence of memory-rich CAR-T cells

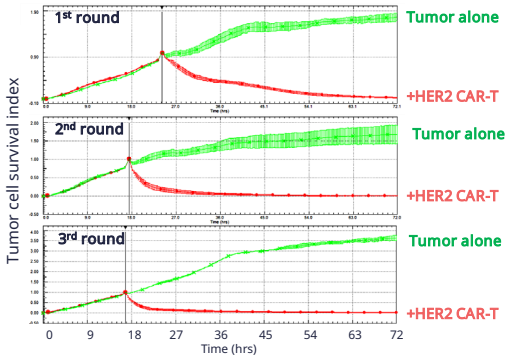


BP2301 (cont'd)

■ PiggyBac-mediated, T_{SCM}-rich BP2301 demonstrated potent and sustained killing activity

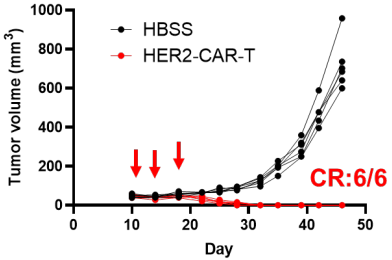
- BP2301 showed persistent cytotoxicity against HER2+ sarcoma in a serial killing assay Data

Target Cells: U2-OS (Osteosarcoma)
E:T =1:1



- BP2301 eradicated inoculated tumor in an ovarian cancer xenograft model

Target Cells: SK-OV-3 (Ovarian)



■ Phase 1 clinical trial ongoing

- 3 + 3 Dose-escalation Design (n=12)

8.3 x 10^5 cells/kg → 2.7 x 10^6 cells/kg

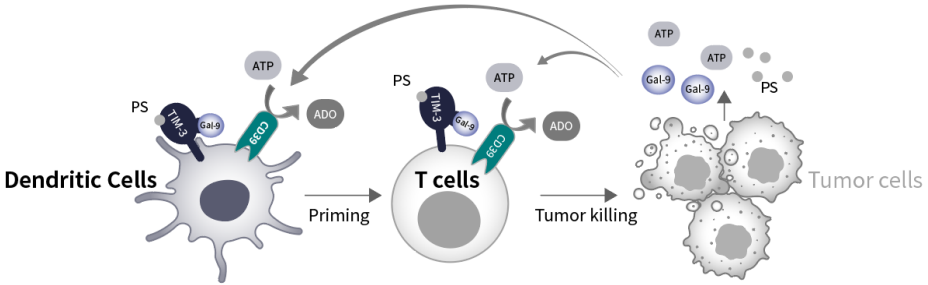
- Primary objective: Safety and tolerability
- Secondary objective: Expansion and persistence of BP2301, efficacy
- Lymphodepletion: 3-day regimen
  FLU 25 mg/m^2 + Cy 250 mg/m^2

Antibody Pipelines

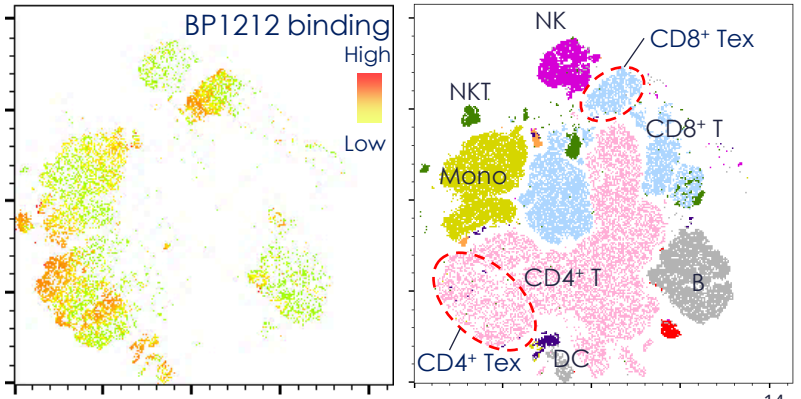
BP1212 (CD39 x TIM-3 bispecific antibody)

First-in-Class Dual Booster of Dendritic Cells and Cytotoxic T Cells for Innate and Adaptive Immune Responses in Cancer Treatment.

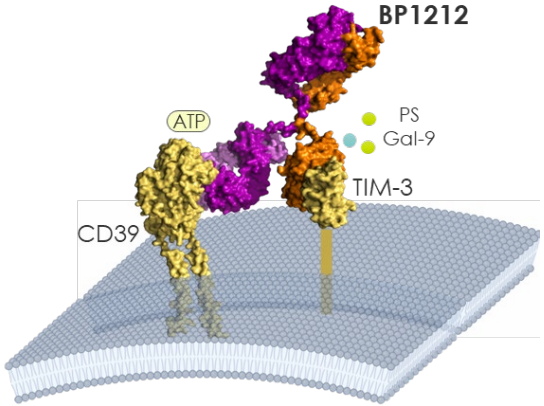
- CD39 is an intervening molecule that disrupts tumor-derived DAMP (exATP). TIM-3 had been recognized as a suppressor of T cells. BP1212 is the first strategy to enhance the innate and adaptive anti-tumor immunity mediated by dendritic cells and T cells.
- We redefined CD39 and TIM-3 as a new immune checkpoint that is distinct from the conventional understanding of the innate and adaptive immune systems.
- TIM-3 and CD39 are co-induced on exhausted T cells as well as on dendritic cells, modulating the tumor microenvironment unfavorable to T cells and suppressing activation signals.
- The expression of TIM-3 and CD39 on dendritic cells suppresses anti-tumor immunity by hindering the activation of innate immunity and the induction of adaptive immunity.



Targeting and enhancing anti-tumor dendritic cells and T cells

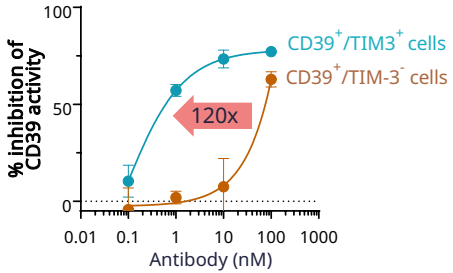


BP1212 (cont'd)



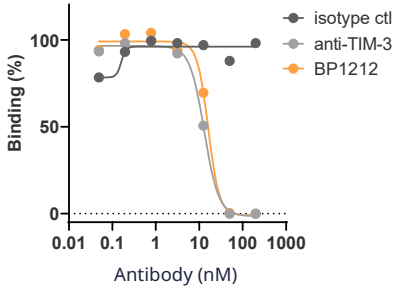
CD39 Inhibition

- Inhibition of CD39 enhanced on TIM3⁺ CD39⁺ cells



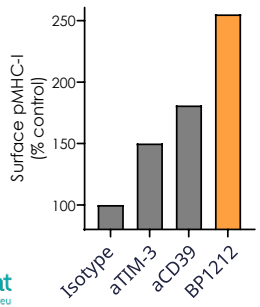
TIM-3 blockade

- Inhibition of PtdSr binding



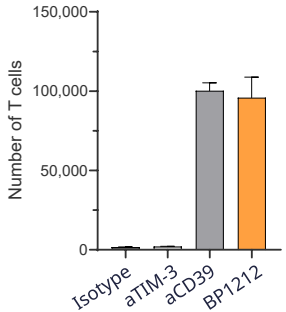
Dendritic cells

- Enhanced antigen presentation by DCs

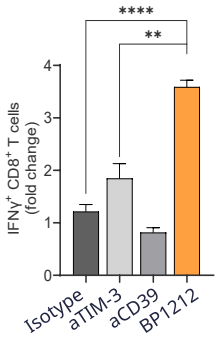


Cytotoxic T cells

- Proliferation of T cells in the tumor microenvironment

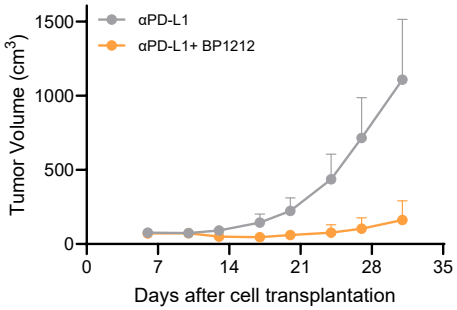


- BP1212 Enhances the expansion of IFNg-producing T cells



Robust anti-tumor potential

- BP1212 strongly suppresses the proliferation of syngeneic tumor cells in combination with PD-L1



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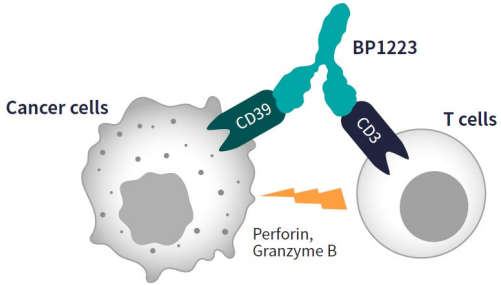
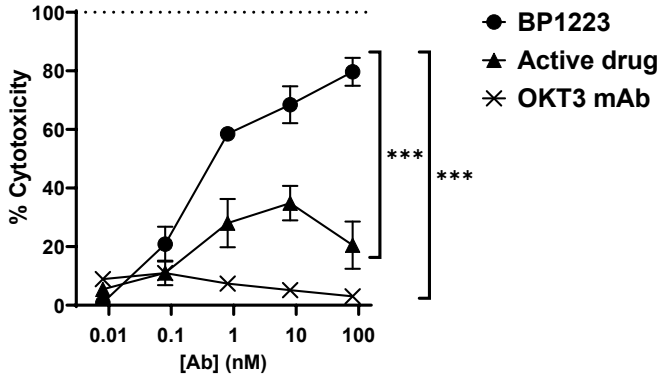
BP1223 (anti-CD39 T cell engager)

Novel T cell engager targeting CD39

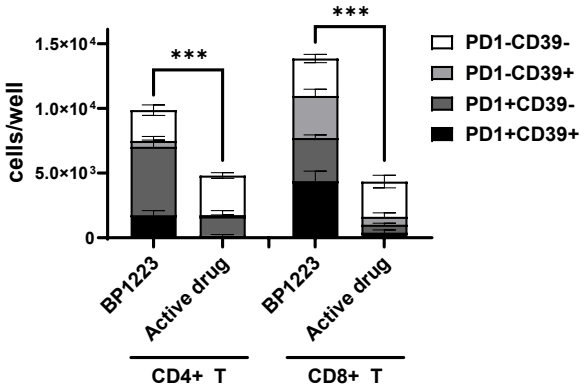
- CD39 is an ectoenzyme and expressed in hematopoietic tumors such as acute myeloid leukemia (AML) and multiple myeloma (MM)
- BP1223 is a bispecific antibody consisting of anti-CD39 scFV and anti-CD3 Fab, exerting anti-tumor effects by attracting and activating T cells to CD39-expressing tumors
- Minimal effects on $CD39^+$ normal cells (i.e., endothelial cell HUVEC or $CD34^+$ bone marrow cells) was observed
- BP1223 demonstrated complete elimination of AML blasts in *in vitro* study using patient samples and complete tumor regression in AML xenograft model



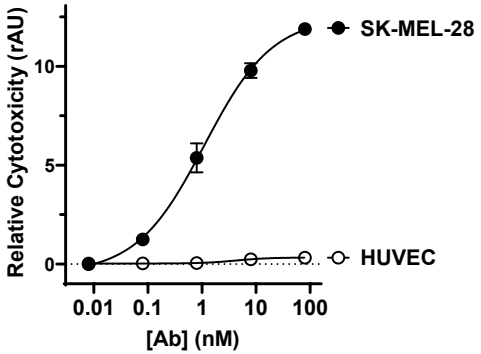
Cytotoxic activity on CD39-expressed cancer cell line (SK-ME-28)



T cell activation in cancer cell and PBMC co-culture systems



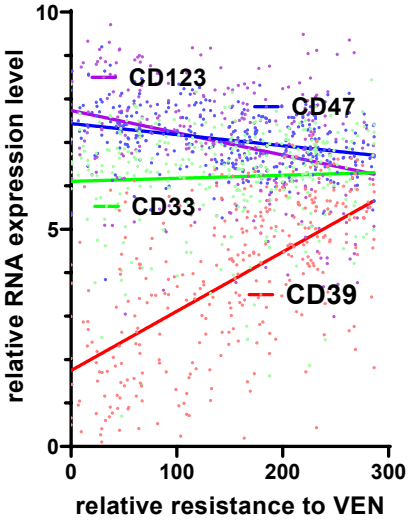
Cancer cell-selective cytotoxic activity



BP1223 (cont'd)

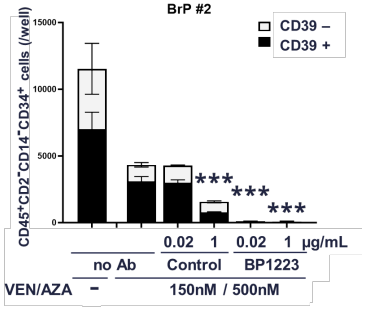
CD39 expression and resistance to Venetoclax

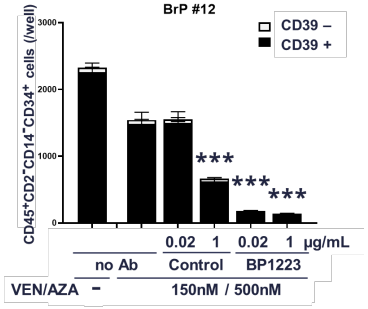
- A strong correlation has been observed between CD39 expression and resistance to standard therapies in AML patient samples



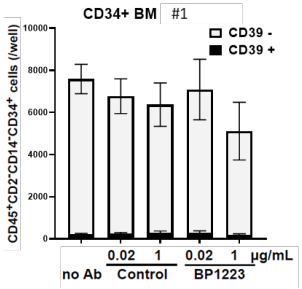
Patient data from a large cohort in the Beat AML 2.0 dataset

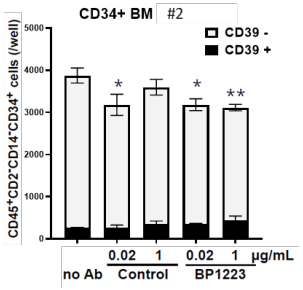
Depletion of AML blast by combination of BP1223 and Ven/Aza in AML patient-derived cells



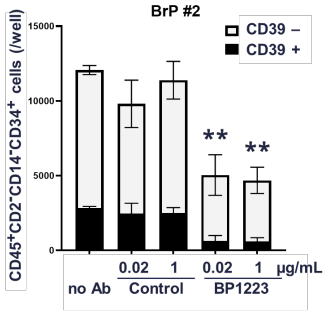


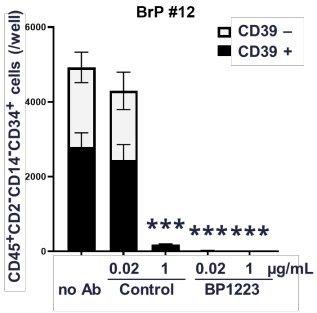
BP1223 has minor effects on CD34+ normal bone marrow cells



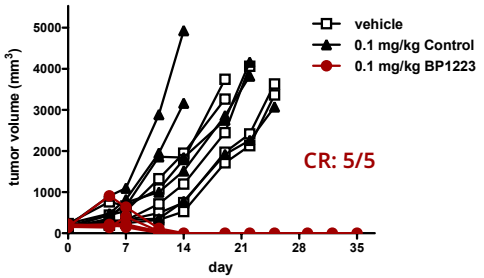


Anti-tumor effects of BP1223 on primary AML cells by autologus bone marrow immune cells





BP1223 shows complete tumor regression in AML xenograft model

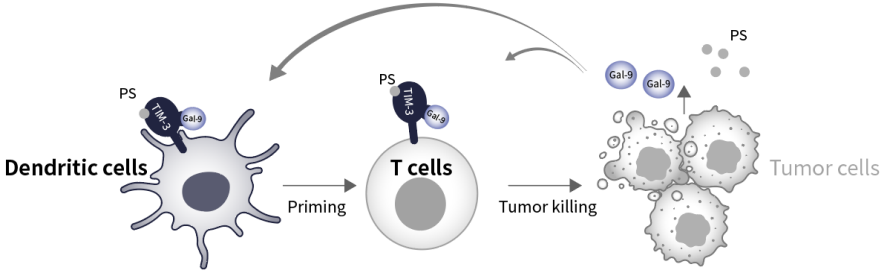


BrightPath Biotherapeutics

BP1210 (TIM-3 x TIM-3 biparatopic antibody)

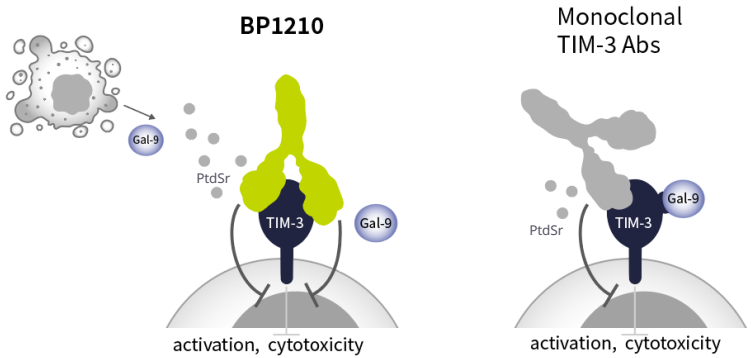
A Novel TIM-3 Targeting Strategy: Blocking the Binding of All Ligands

- Increasing expression of Tim-3 and Galectin-9, one of the four known ligands of Tim-3, are reported to relevant to poor prognosis cancers such as pancreatic carcinoma, glioma, cervical carcinoma, lymphoma and leukemia.
- We hypothesize that Gal-9 plays a pivotal role in facilitating immune suppression within tumors by binding to Tim-3 on the surface of dendritic cells. This binding inhibits dendritic cell maturation, thereby impeding the mediation of T cell immunity.
- Conventional anti-Tim-3 antibodies are not able to inhibit Gal-9 binding effectively, which impede Tim-3 antagonist to exert full potential of T cell anti-tumor activities.
- The limitation is derived from two distinct epitope bins of Tim-3, one of Gal-9 and the other of other three ligands such as PtdSr.
- BrightPath overcome the hurdle by bringing a biparatopic antibody that inhibits both epitope bin bindings.



TIM-3 restrains anti-tumor immunity by regulating inflamma-some activation in DC (Dixonet al. 2021)

TIM-3 expression can serve as a marker for terminally differentiated effector and exhausted cells (Wolf, et al. 2020)



BrightPath Biotherapeutics

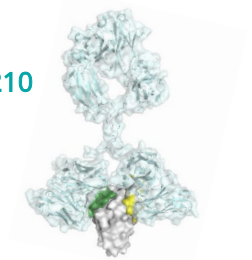
BP1210 (cont'd)

A novel humanized, IgG1-Fc silent biparatopic antibody

- BP1210 binds to two distinct TIM-3 epitopes: one is the same domain as sabatolimab (Novartis) and all other Abs advanced in clinical development, and the other is the one that enables full interference with Gal-9 binding and that those monoclonal Abs do not reach

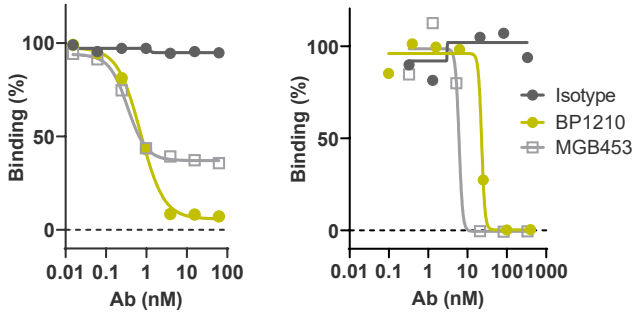
Binging Affinity Enhancement

- Biparatopic antibody BP1210's affinity is enhanced to KD(M) of $x10^{-10}$ in a combination of Clone A of $x10^{-9}$ and Clone B of $x10^{-7}$

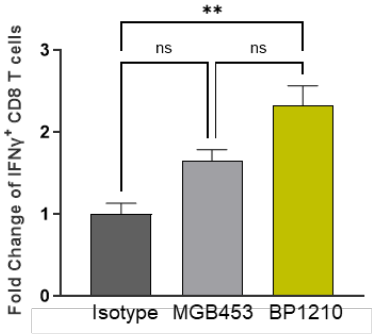


Inhibition of the ligand-binding

- Inhibition of Gal-9 binding
- Inhibition of PtdSr binding

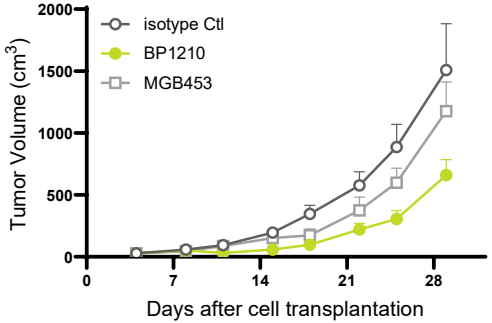


Enhanced IFNγ-producing T cells



Robust Anti-tumor effect

- Head-to-head monotherapy comparison (MC-38 mouse model)



BrightPath Biotherapeutics

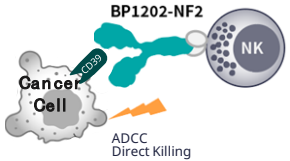
BP1202 (anti-CD39 Antibody)

A Novel Strategy on Targeting CD39

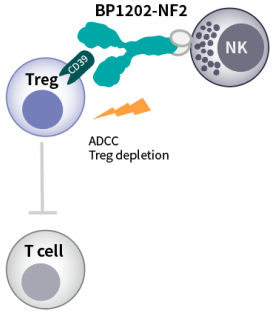
Rediscovery of CD39 as a target for depleting cancer cells and Tregs, while avoiding adenosine generation in the tumor microenvironment

- The conventional strategy to inhibit adenosine generation hasn't yielded promising results thus far
- We revisited CD39 expression by cancer cells themselves and Tregs within TME and proposed the CD39 targeting strategy that emphasizes the depletion of these cells rather than the inhibition of its enzyme activities

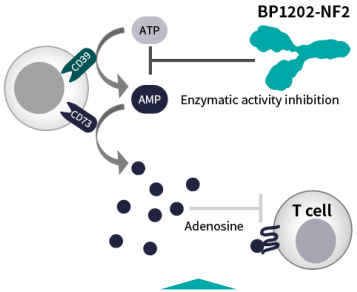
MOA (1)
Direct killing



MOA (2)
Treg depletion



MOA (3)
Enzyme Inhibition



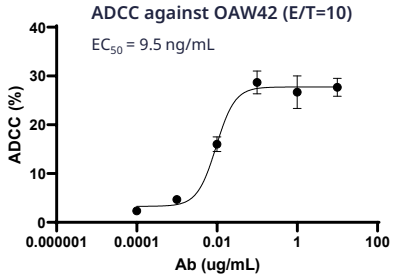
Conventional anti-CD39 antibodies rely solely on prevention of enzymatic activity

- BP1202-NF2, a glycoengineered anti-CD39 antibody, depletes CD39 expressing cancer cells and promotes immune response by CD39high Treg depletion and CD39 enzymatic activity blockade
- CD39 catalyzes the production of immunosuppressive and CD39 expression is elevated on tumor-infiltrating Tregs, whereas it is expressed broadly but moderately or slightly expressed by other tumor-associated immune cells
- BP1202-NF2 selectively depletes CD39high T cells and blockades CD39 enzymatic activity of CD39int/low immune cells in tumor

BP1202 (cont'd)

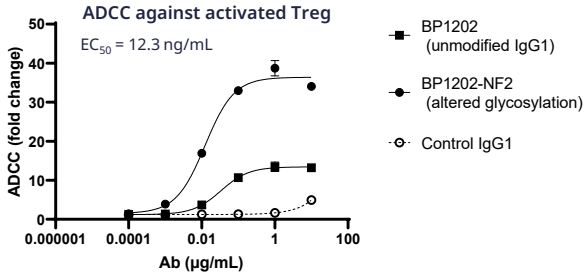
Direct Killing

- BP1202-NF2, of which glycosylation is optimized by CD39 density, affinity to CD39, and affinity against FcγRIIIa, showed potent killing of CD39+ cancer cell line in ADCC assay

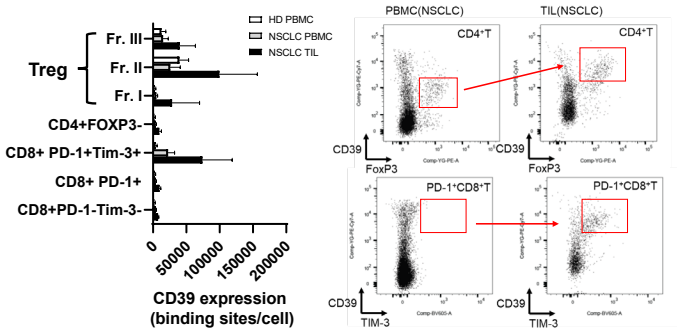


Treg depletion

- BP1202-NF2 demonstrated high ADCC activity against Treg



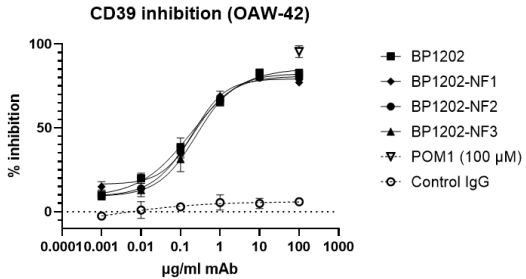
- CD39 expression was elevated on tumor-infiltrating Tregs and exhausted CD8$^+$T cells in NSCLC patients



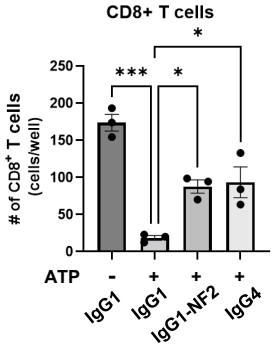
Ecto-enzyme inhibition

- BP1202 (unmodified IgG1) and BP1202-NF2 (altered glycosylation) show high affinity for recombinant human CD39 (KD(M) x10^{-10}) and Tregs(x10^{-9})
- BP1202-NF2 selectively depleted CD39hi population of CD4$^+$ T cells and CD8$^+$ T cells in ex vivo cultured human PBMCs

- Maximum blockade of membrane-associated CD39 is 80% at 10μg/mL, which is compar-able to IPH5201's 70% (industry high)



- BP1202-NF2 released the adenosine-inducing immunosuppression of CD8$^+$ T cells



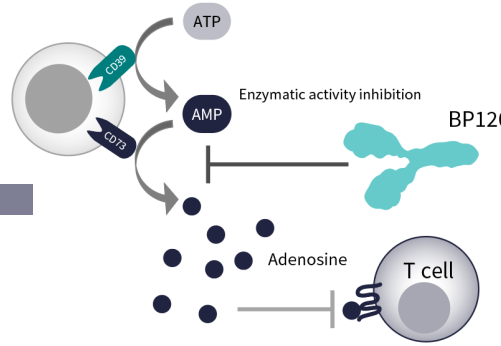
*ATP is catalyzed the conversion to adenosine by CD39 ecto-enzyme

BP1200 (anti-CD73 Antibody)

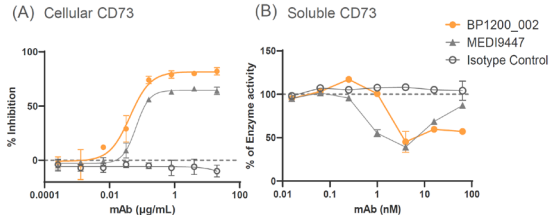
■ Novel anti-CD73 antibody taking standard strategy of adenosine generation blockade with a best-in-class profile

- Attenuates the activity of CD73 as a non-competitive inhibitor without hook effect
- Enhances the proliferation, cytotoxicity, and cytokine production of T cells under the TME condition
- The combination with immune checkpoint antibodies significantly suppressed tumor growth and lead long term immunotherapeutic efficacy
- Good PK/TK profiles without remarkable organ toxicity in mice and monkeys

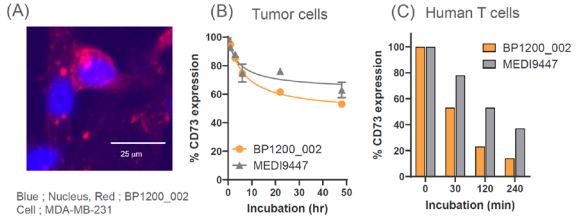
Enzyme inhibition



CD73 enzyme activity inhibition

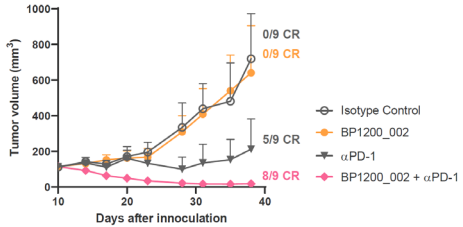


BP1200 Internalization

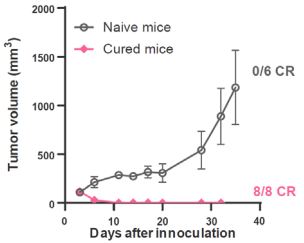


Blue ; Nucleus, Red ; BP1200_002
Cell ; MDA-MB-231

Combination therapy of BP1200 with ICB



Tumor-Rechallenge model



Cured mice treated with BP1200 and aPD1 combo were rechallenged with the MC-38 expressing hCD73

Pharmacokinetics and Toxicokinetics

Table 1. Pharmacokinetics of single intraperitoneal dose of BP1200 in female C57BL/6 mice

Dose mg/kg	C_{max} μg/mL	$AUC_{0-\infty}$ hr×mg/mL	Cl mL/hr/kg	V_{ss} mL/kg	$t_{1/2}$ hr	$MRT_{0-\infty}$ hr
10	91±15	24±2	0.41±0.03	119±12	201±27	290±39

Table 2.Toxicokinetics of single or multiple intravenous dose of BP1200 in female cynomolgus monkeys

Dose mg/kg	Route	Day	C_{max} μg/mL	$AUC_{0-\infty}$ μg · hr/mL	CL mL/hr/kg	V_{ss} mL/kg	$t_{1/2}$ hr	$MRT_{0-\infty}$ hr
5	iv, q1w	1	149	6900	0.7	52.8	51.2	73.9
5	iv, q1w	22	122	4600	2.6	173.3	42.9	61.9
25	iv, q1w	1	598	22200	1.1	68.4	41.9	60.4
25	iv, q1w	22	808	35700	0.7	57.6	57.7	83.2

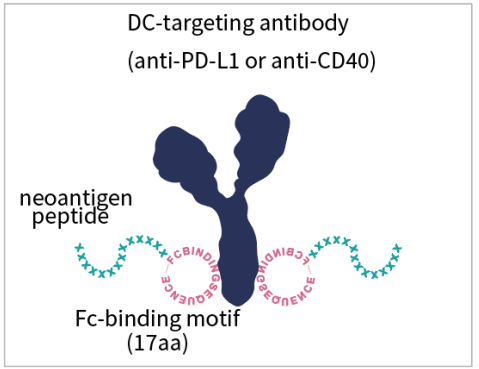
BrightPath Biotherapeutics

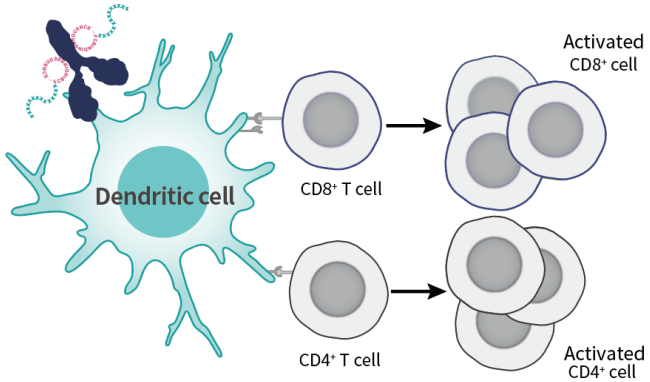
Cancer Vaccine Pipeline

BP1209 (Fully Personalized Neoantigen Vaccine)

A new platform of personalized neoantigen cancer vaccines directed by checkpoint inhibitor antibodies

- The BP1209 vaccine is delivered as a molecular complex of patient-specific neoantigen peptides and immune-checkpoint inhibitor antibody such as anti-PD-L1 and anti-CD40 antibodies.
- The neoantigen peptides consists of three modules: HLA-class I and -class II neoantigen epitopes, and an IgG-binding motif. The peptides non-covalently bind Fc domain of IgG, and self-assemble the antibody-vaccine complex without any chemical reaction which enables individual synthesis and manufacturing fully personalized neoantigen vaccine

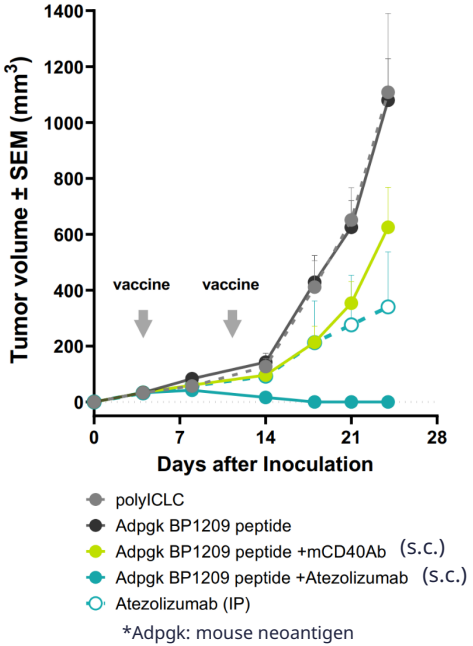




- BrightPath has developed in-house bioinformatic algorithms to identify highly immunogenic neoantigens from cancer patients and analyzed clinical samples from over 100 patients
- The new vaccine platform of BP1209 in combination with BrightPath's algorism to identify high quality neoantigens provides an ideal option to improve neoantigen vaccine therapy

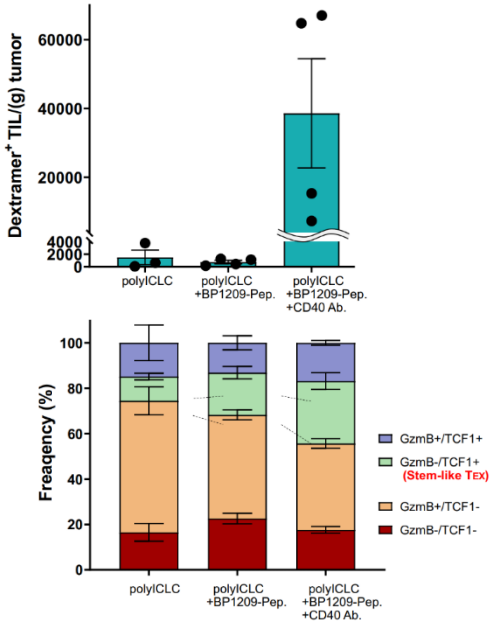
BP1209 (Fully Personalized Neoantigen Vaccine) (cont'd)

- BP1209 exerted robust anti-tumor effect in therapeutic setting
  - Atezolizumab conjugated BP1209 vaccine maintained complete tumor regression in all the mice until study end (n=9).



*Adpgk: mouse neoantigen

- BP1209 strongly enhances stem-like Tex infiltration into tumor
  - Mice treated with the BP1209 vaccine marked increase in neoantigen specific TIL
  - The BP1209 vaccine increased TCF1+/ Granzyme B- stem-like Tex



Company Profile

Company Profile

BrightPath Biotherapeutics Co., Ltd. (Tokyo Stock Exchange Growth: 4594)

Business	Development of novel cancer immunotherapy
Foundation	May 2003
Listing	November 2015
Employees	25 (as of September 2024)
Location	Headquarters: 2-2-4 Kojimachi, Chiyoda-ku, Tokyo Research Laboratories: 3-25-22 Tonomachi, Kawasaki-ku, Kawasaki, Kanagawa

Board Member

Kenichi Nagai	CEO	PPMX, MERRILL A BANK OF AMERICA COMPANY
Norihiro Nakamura	CSO	Genentech
Yoichi Takeshita	CFO	GlaxoSmithKline, amazon
Akira Yamada	Director (part-time)	久留米大学 KURUME UNIVERSITY (Present)
Hirotaka Takeuchi	Director (outside, independent)	HARVARD BUSINESS SCHOOL
Tsutomu Kishino	Auditor (outside)	DBJ Development Bank of Japan
Taketoshi Abe	Auditor (outside, independent)	Daiichi-Sankyo
Yoshiyasu Yamaguchi	Auditor (outside)	TMI Associates

BrightPath

Biotherapeutics