Corporate Presentation

December 2025

BrightPath Biotherapeutics

Disclaimer

The material that follows is a presentation of general background information about BrightPath Biotherapeutics Co., Ltd. (“BrightPath”) as of the date of this presentation. This material prepared solely for information purpose and is not to be construed as a solicitation or an offer to buy or sell any securities and should not be treated as giving investment advice to recipient.

This material and any material distributed in connection with this presentation may contains forward-looking statements, beliefs or opinions regarding BrightPath’s future business, future position and result of operations, including estimates, forecasts, targets and plans for BrightPath. Without limitation, forward-looking statements often include words such as “target”, “plans”, “believes”, “hopes”, “continues”, “expects”, “aims”, “intends”, “ensures”, “will”, “may”, “should”, “would”, “could”, “anticipate”, “estimates”, “projects” or similar expressions or the negative thereof. Such forward-looking statements involve known and unknown risks, uncertainties and other factors which may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by such forward-looking statements. Such forward-looking statements are based on numerous assumptions regarding our present and future business strategies and the environment in which BrightPath will operate in the future. The important factors that could cause our actual results, performance or achievements to differ materially from those in the forward- looking statements include, among others, risks associated with product discovery and development, uncertainties related to the outcome of clinical trials, slower than expected rates of patient recruitment, unforeseen safety issues resulting from the administration of our products in patients, uncertainties related to product manufacturing, the lack of market acceptance of our products, our inability to manage growth, the competitive environment in relation to our business area and markets, our inability to attract and retain suitably qualified personnel, the unenforceability or lack of protection of our patents and proprietary rights, our relationships with affiliated entities, changes and developments in technology which may render our products obsolete, and other factors. These factors include, without limitation, those discussed in our public reports filed with the Tokyo Stock Exchange and the Financial Services Agency of Japan. Although BrightPath believes that the expectations and assumptions reflected in the forward-looking statements are reasonably based on information currently available to BrightPath's management, certain forward-looking statements are based upon assumptions of future events which may not prove to be accurate.

BrightPath does not undertake to update any of the forward-looking statements contained in this presentation or any other forrard-looking statements it may make, except as required by law or stack exchange rule.

No representation or warranty, either express or implied, by BrightPath is made as to the accuracy, fairness, or completeness of the information presented herein and no reliance should be placed on the accuracy, fairness, or completeness of such information. Various factors may cause actual results to differ materially from those described in this material.

The presentation contains information on drugs currently under development. Nothing contained herein should be considered any solicitation, promotion, advertisement, or medical advice.

The information contained in this material may be changed without prior notice. In addition, BrightPath may alter, modify or otherwise change in any manner the contents of this presentation, in its own discretion without the obligation to notify anu person of such revision or changes. Neither BrightPath nor its sources or informants assume any responsibility or liability for any loss or damage that may be incurred by those who will rely on the information in this document.

This presentation and its contents are proprietary confidential information and may not be reproduced, published or otherwise disseminated in whole or in part without BrightPath’s prior written consent. These materials are not intended for distribution to, or use by, any person or entity in any jurisdiction or country where such distribution or use would be contrary to local law or regulation.

In the event of any inconsistency between the statement in the English-language presentation and the statement in the Japanese-language presentation, the statements in the Japanese-language version should prevail.

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

Developed product	Mechanism/target	Cancer type	Discovery	Preclinical	PI	PII
Cell Therapy						
BP2201	iPSC-derived NKT cells	Head & Neck SCC				
BP2202	iPSC-derived BCMA CAR NKT	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 Vaccine						
BP1209	Personalized neoantigen	Solid Tumor				

Cell Therapy Pipeline

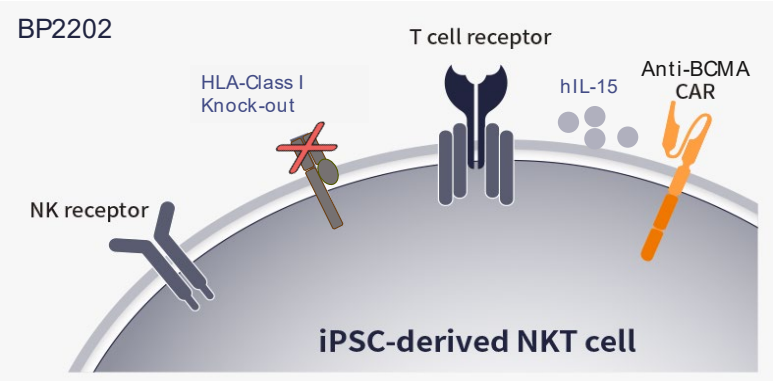
BP2202 (iPSC-derived BCMA CAR-iNKT)

- iPS cell-derived BCMA CAR-NKT cells for the treatment of multiple myeloma

- Allogeneic CAR-*Natural Killer **T*** (NKT) cells orchestrate endogenous T cells toward anti-tumor activity, addressing the limitation in clinical response durability observed with conventional allogeneic CAR-T therapies

- Master iPS-Cell Bank: constant, clinically-scalable, commercially viable cell source

- Currently in an IND-enabling stage
  - Orphan Drug Designated in multiple myeloma by FDA



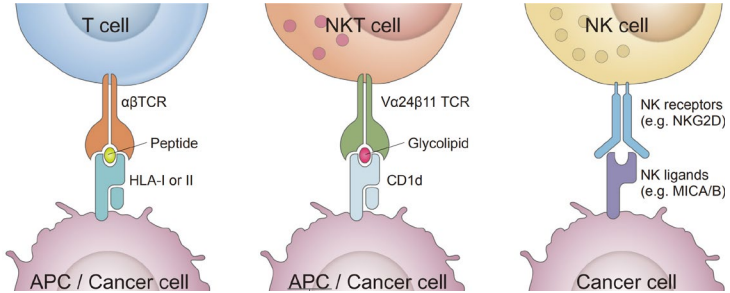
The CAR and hIL-15 coding genes were inserted, and the β2M gene was knocked out, using non-viral CRISPR/Cas12-based gene editing in a single iPS cell clone.

BP2202 (cont'd)

- Natural Killer T cell has a unique TCR that enables them to interact with and orchestrate surrounding antigen-presenting cells and myeloid cells upon activation

No GvHD, No need of TCR knock-out

- Unlike conventional αβ T cells, their invariant TCR recognizes a monomorphic, MHC class I-like molecule independent of HLA type, which reduces the risk of GvHD and eliminates the need to knock out the endogenous TCR



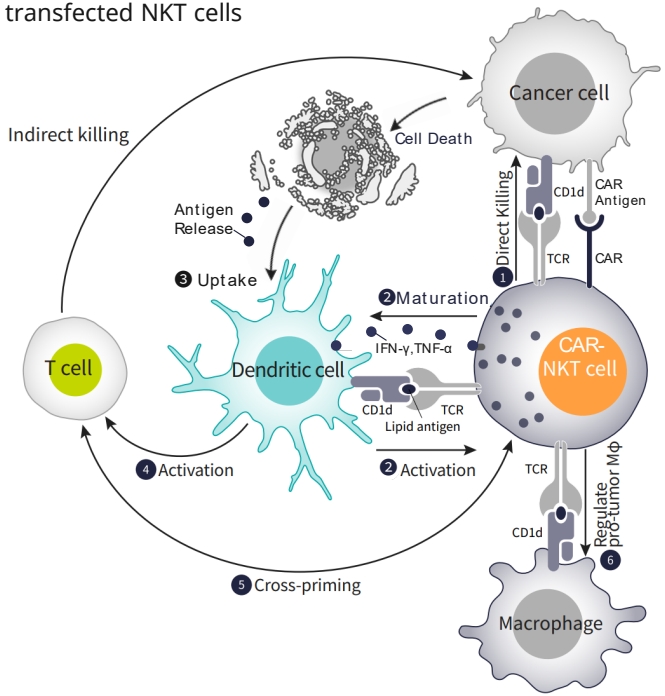
*Multiple myeloma cells are CD1d-positive.

Application of iPS cell technology

- The use of NKT cells as effector cells in CAR-T therapy has been limited by their extremely low frequency in peripheral blood, making it difficult to manufacture clinical-scale doses from a single healthy donor.
- iPSC technology not only overcomes this hurdle but also provides a consistent and renewable source of NKT cells.

Orchestration of immune cells

- This unique TCR is key to the NKT cell's ability to orchestrate the surrounding immune environment
- TCR-mediated orchestration can be partially reproduced even in CAR-transfected NKT cells

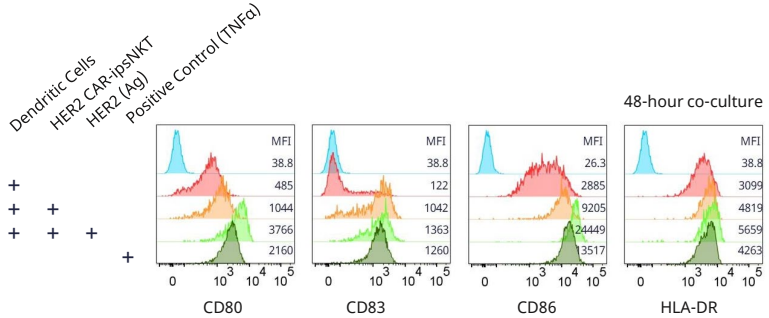


BP2202 (cont'd)

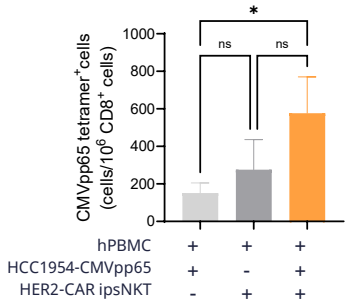
- *In vitro* and *in vivo* data supporting the immune-orchestrating capacity of NKT cells

Proof of concept data using HER2 CAR-ipsNKT cell

Promotion of Dendritic Cell maturation

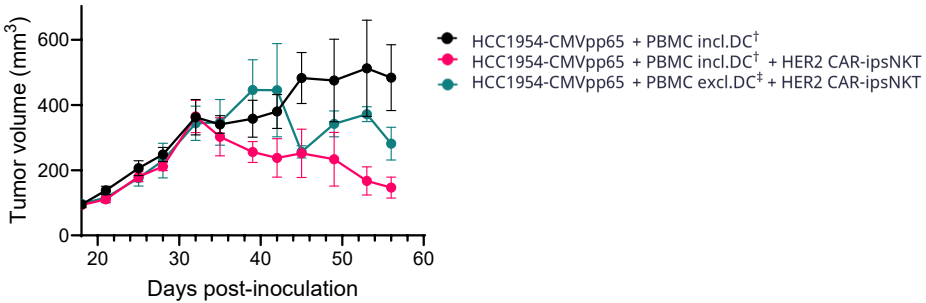


Induction of antigen-specific T cells



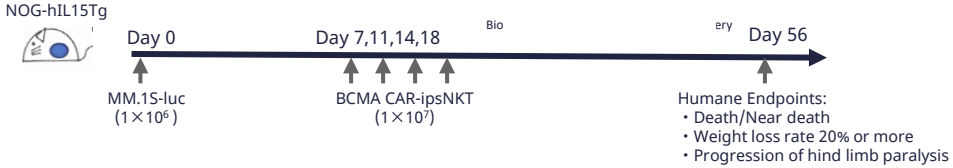
7-day co-culture of HLA-matched PBMC HCC1954-CMVpp65 cells (tumor) and HER2 CAR-ipsNKT cells

CAR-ipsNKT cells induce antigen-specific T cells *in vivo*



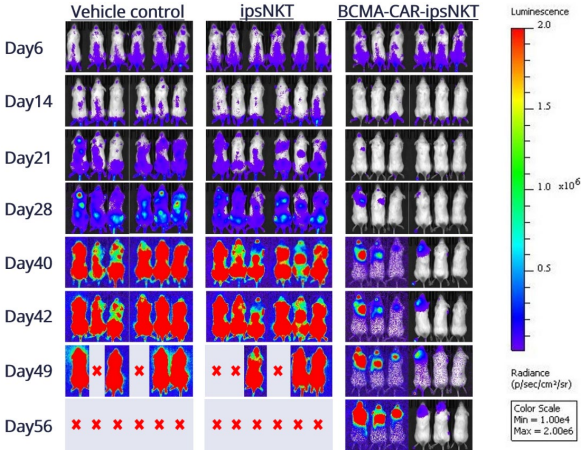
BP2202 (cont'd)

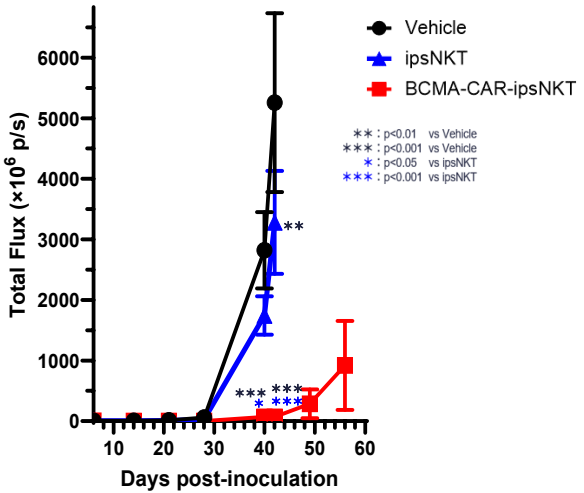
- BP2202 demonstrated a potent anti-tumor activities *in vivo*



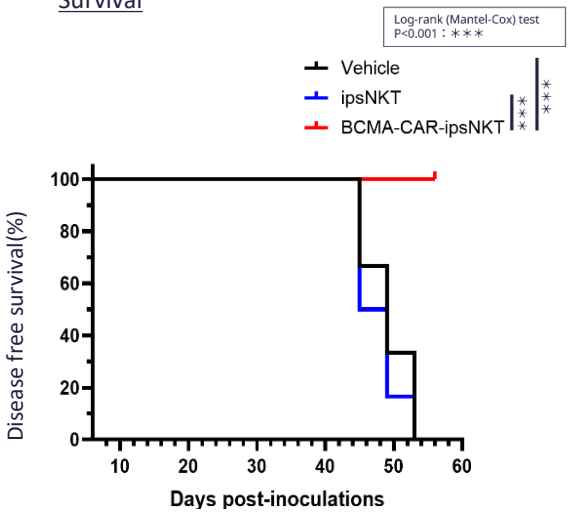
Tumor burden

MM1.S-luc xenograft mouse model





Survival



BP2201 (unmodified iPS cell-derived iNKT cells)

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

  - First-in-human Phase I investigator-initiated trial of non-gene-edited, non-CAR-transfected iPSC-derived NKT cells was conducted in r/r HNSCC, demonstrating early clinical activity with an 80% disease control rate, including tumor shrinkage

  - 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.

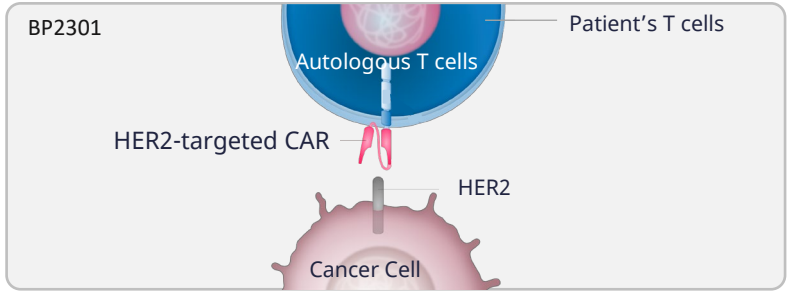
    - Low-dose (n=3) : 1 SD, 2 PD DCR 33.3%
      High-dose (n=6): 4 SD, 1 PD, 1 NE DCR 80% (4 of 5 evaluable patients)

    - The most frequently observed trAEs were Grade 1 or 2 fever (1 patient at low-dose, 4 patients at high-dose)

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

BP2301 (HER2 CAR-T Cell therapy)

- A novel autologous HER2-targeted CAR-T cell therapy
  - Phase I clinical trial is currently being conducted in Japan
  - Indications: HER2-positive sarcoma and gynecological cancers (e.g., ovarian and cervical cancers)

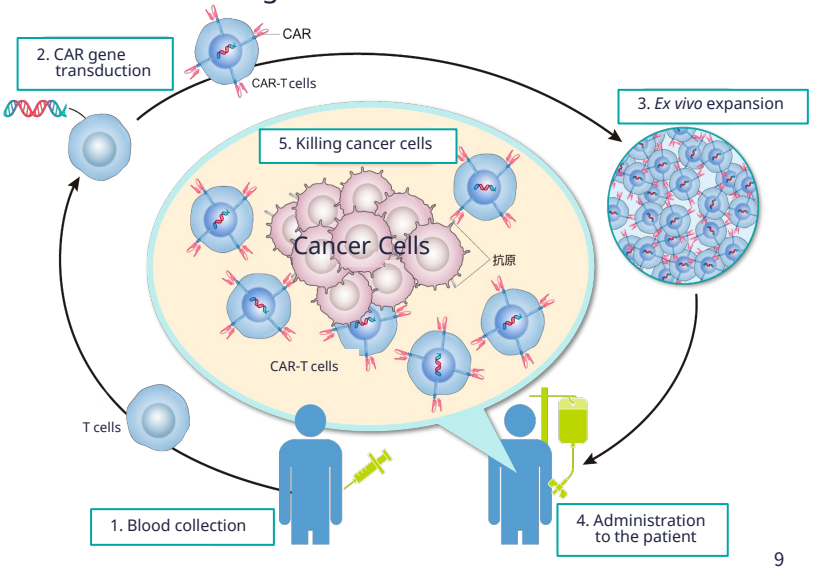


- Phase I study
  - 3 + 3 Dose-escalation

8.3×10^5 cells/kg → 2.7×10^6 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

Autologous CAR-T treatment flow

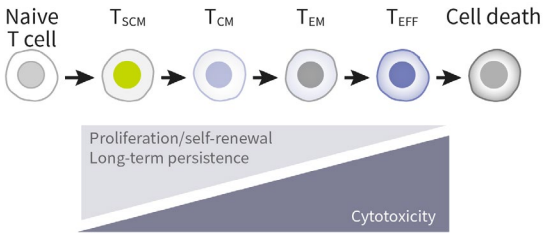


BrightPath Biotherapeutics

BP2301 (cont'd)

- 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

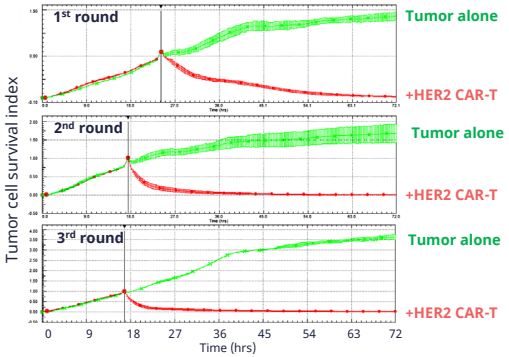
• T cell differentiation and phenotypes



- PiggyBac-mediated, TSCM-rich BP2301 demonstrated potent and sustained killing activity

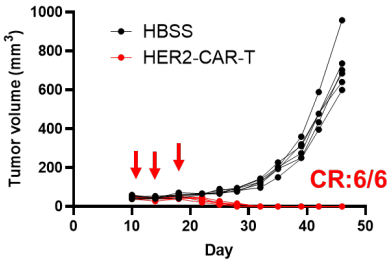
• BP2301 showed persistent cytotoxicity against HER2+ sarcoma in a serial killing assay Data

U2-OS (Osteosarcoma), E:T =1:1



• BP2301 eradicated inoculated tumor in an ovarian cancer xenograft model

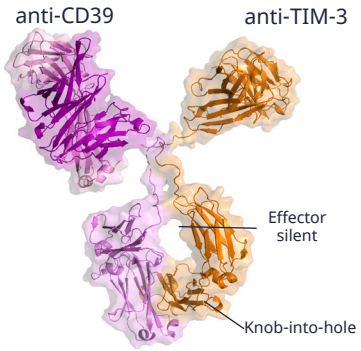
SK-OV-3 (Ovarian)



Antibody Pipeline

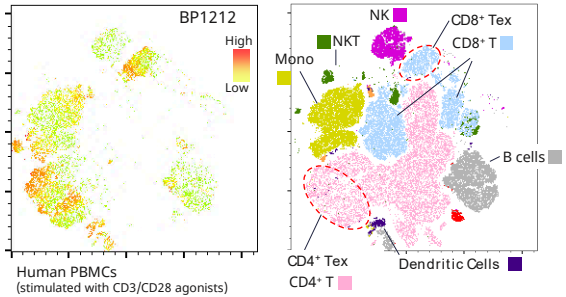
BP1212 (CD39 x TIM-3 bispecific antibody)

- First-in-class Inflammasome promoting antibody that works on dendritic cells
  - BP1212 preferentially binds to CD39 and TIM-3 double positive cells and strongly inhibits the ligand-binding of TIM-3 and the enzymatic activity of CD39.
  - BP1212 inhibits the degradation of extracellular ATP, an inflammasome inducer, and releases TIM-3-mediated inflammasome inhibition by preventing ligand binding to TIM-3 on dendritic cells.
  - Both TIM-3 and CD39 are expressed on exhausted T cells and dendritic cells, modulating the tumor microenvironment unfavorable to T cells. BP1212 promotes a favorable tumor microenvironment for anti-tumor immunity.
  - BP1212 is exclusively active within the tumor microenvironment, thereby restricting unnecessary immune activation.



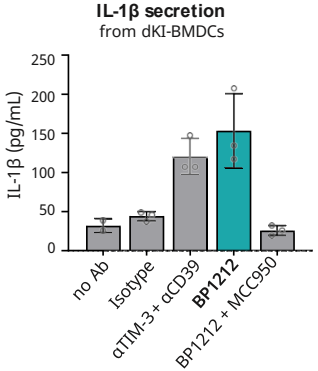
Selectively binds to CD39 & TIM3 co-expressing DC and Tex

- TIM-3 and CD39 are co-expressed in dendritic cells (DCs) and exhausted T cells (Tex) that are key targets for promoting anti-tumor immunity

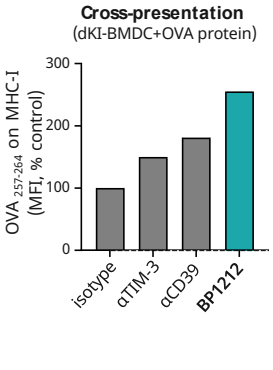


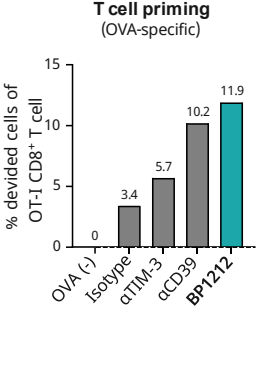
Inflammasome activation in DCs, enhancing antigen cross-presentation and T cell priming

- BP1212 induces IL-1β secretion from huTIM-3/huCD39 double knock-in (dKI-) mice BMDCs via inflammasome activation.



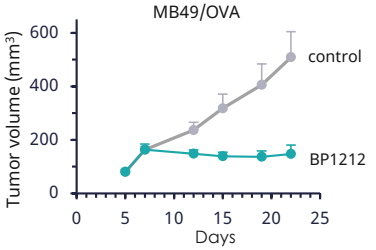
- BP1212 increases cross-presentation of OVA antigen ($OVA_{257-264}$) and enhances $OVA_{257-264}$ specific T cell (OT-I) proliferation in vitro.





Tumor growth inhibition in syngeneic model

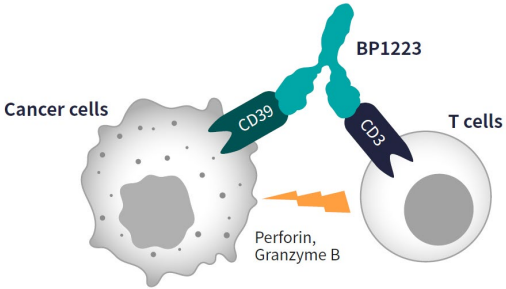
- BP1212 monotherapy significantly blocked tumor growth and lead to rejection of tumor



BP1223 (CD39-targeted T cell engager)

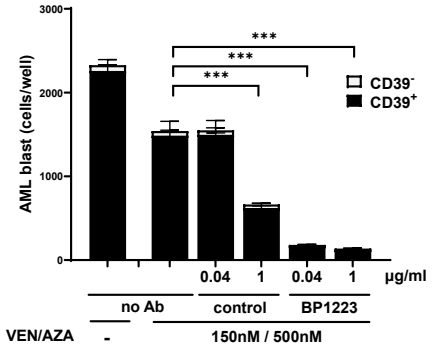
■ Novel T cell engager targeting CD39

- CD39 is expressed in acute myeloid leukemia (AML) blasts, which correlates with increased resistance to a standard-of-care treatment Venetoclax
- CD39-targeted T cell engager BP1223 could serve as a combination agent with Venetoclax to overcome the resistance



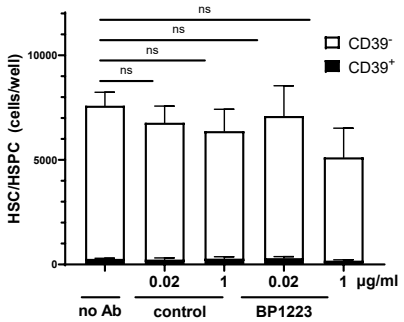
Cytotoxicity to AML blasts

- BP1223 demonstrated enhanced reduction of patient-derived AML cells in combination with Ven+Aza



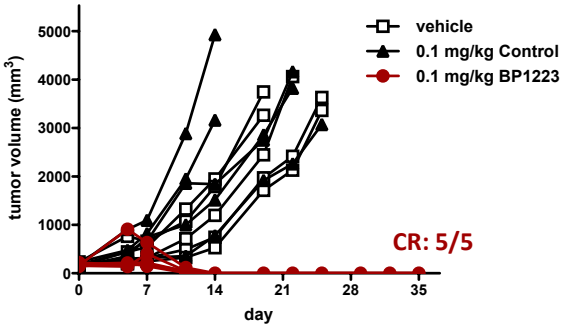
Cytotoxicity to Normal BMMC

- BP1223 spares BMMCs from healthy donors



AML xenograft model

- BP1223 demonstrated complete response (CR) of 5/5 AML xenograft mice in monotherapy

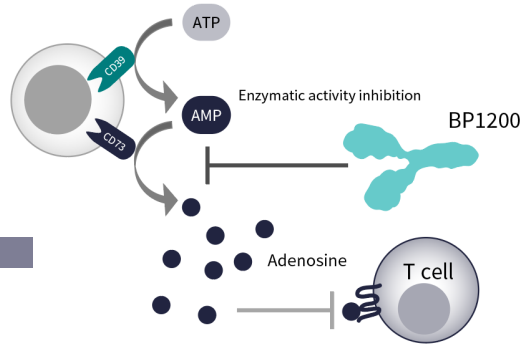


BrightPath Bio ASH2024

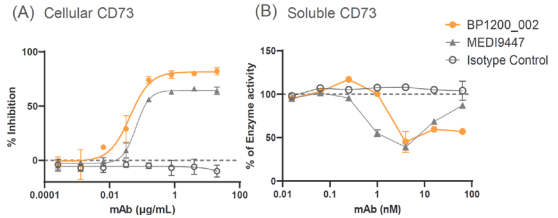
BP1200 (anti-CD73 Antibody)

- 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

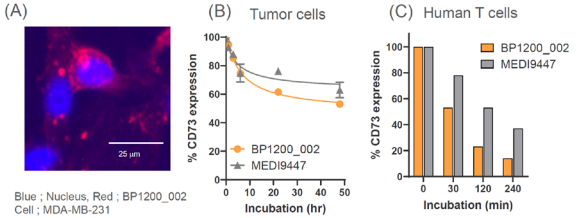
Enzyme inhibition



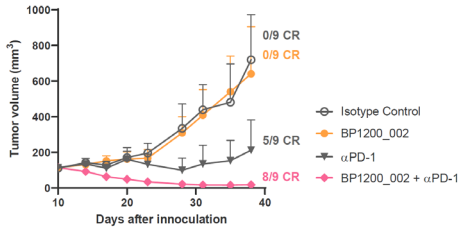
CD73 enzyme activity inhibition



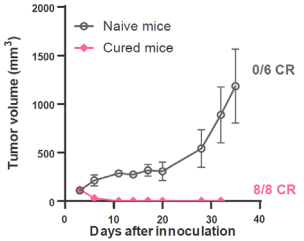
BP1200 Internalization



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

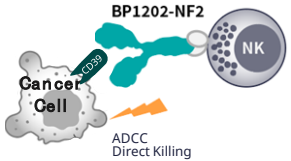
Table 2.Toxicokinetics of single or multiple intravenous dose of BP1200 in female cynomolgus monkeys

Dose	Route	Day	C_{max}	$AUC_{0-\infty}$	CL	V_{ss}	$t_{1/2}$	$MRT_{0-\infty}$
mg/kg			μg/mL	μg · hr/mL	mL/hr/kg	mL/kg	hr	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

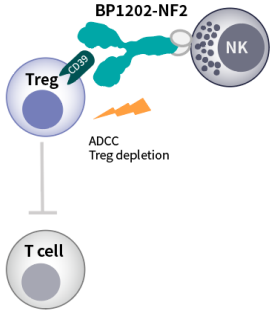
BP1202 (anti-CD39 Antibody)

■ Novel CD39-targeting strategy that emphasizes the depletion of these cells rather than the enzyme inhibition

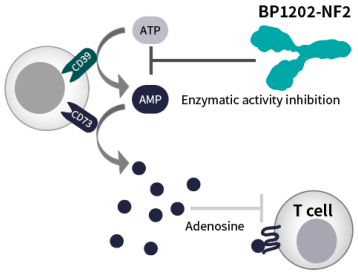
MOA (1)
Direct killing



MOA (2)
Treg depletion



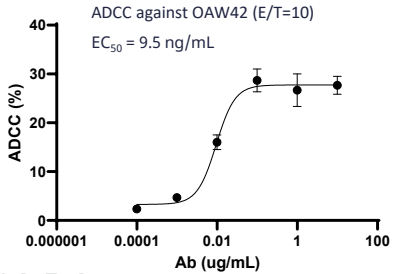
MOA (3)
Enzyme Inhibition



- 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

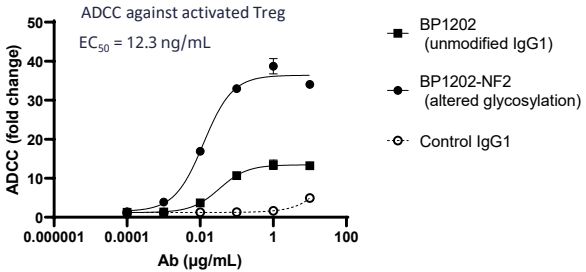
Direct Tumor Killing

- 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

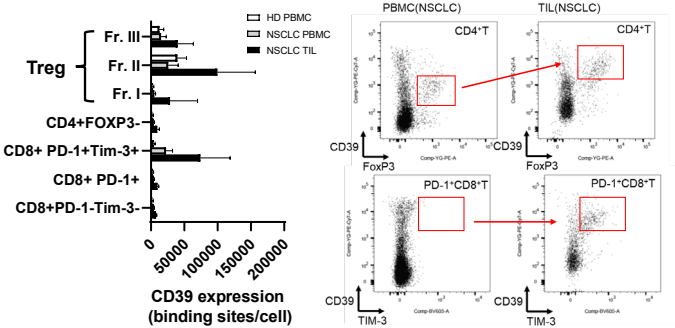


Treg depletion

- BP1202-NF2 demonstrated high ADCC activity against Treg



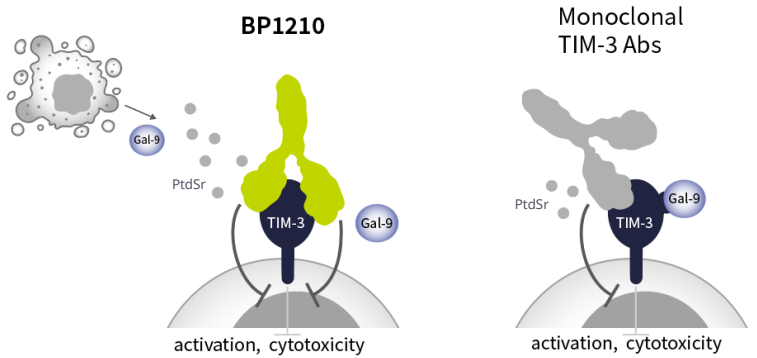
- CD39 expression was elevated on tumor-infiltrating Tregs and exhausted CD8⁺T cells in NSCLC patients



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

Anti–TIM-3 antibody that blocks galectin-9 binding

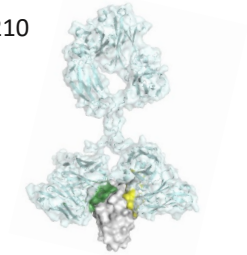
- TIM-3 and galectin-9 (Gal-9) are overexpressed in multiple poor-prognosis cancers and contribute to tumor-associated immune suppression.
- Gal-9 binding to TIM-3 on dendritic cells inhibits their maturation and downstream T-cell immunity. However, conventional anti-TIM-3 antibodies fail to effectively block Gal-9 binding.
- BrightPath overcomes this limitation with a biparatopic antibody that blocks both TIM-3 ligand-binding epitopes.



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 $x10^{-7}$

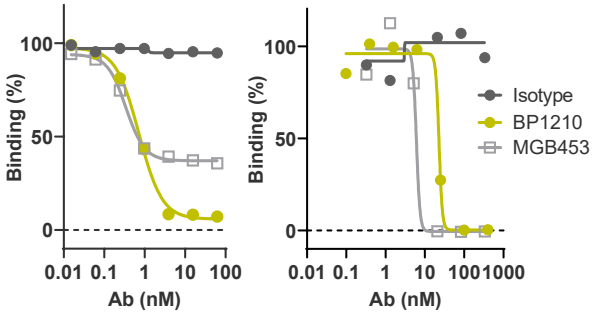
BP1210



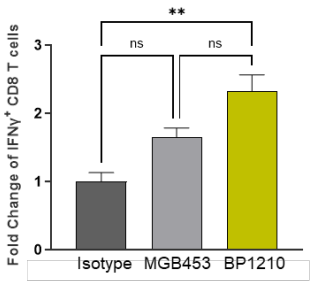
hTIM-3 IgV domain

Inhibition of the ligand-binding

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

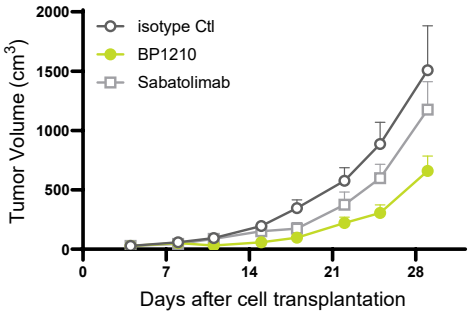


IFNγ-producing T cells



Robust Anti-tumor effect

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



BrightPath Biotherapeutics

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	23 (as of December 2025)
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
Akira Yamada	Director (part-time)	久留米大学 (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