Corporate Presentation

October 2026

BrightPath Biotherapeutics Co., Ltd.

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BrightPath Biotherapeutics

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BrightPath (Tokyo Stock Exchange Growth: 4594)

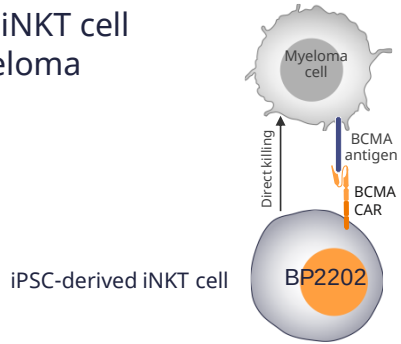
- A clinical-stage biopharmaceutical company focused on the development of novel cancer therapies to transform cancer treatment for refractory or progressive cancers that cannot be treated with conventional standard therapies
- Actively involved in developing cell therapies, currently in clinical trials, and immunomodulatory antibodies

Development Pipeline

Product	Mechanism / target	Indication	Discovery	Preclinical	Phase 1	Phase 2
Cell Therapy						
BP2201	iPSC-derived NKT cells	Head & neck cancer				
BP2202	iPSC-derived BCMA CAR-iNKT cells	Multiple myeloma				
BP2301	Autologous HER2 CAR-T cells	Sarcoma Gynecologic cancers				
Antibody						
BP1212	CD39×TIM-3 bispecific	Solid tumors				
BP1223	CD39×CD3 T cell engager	AML				

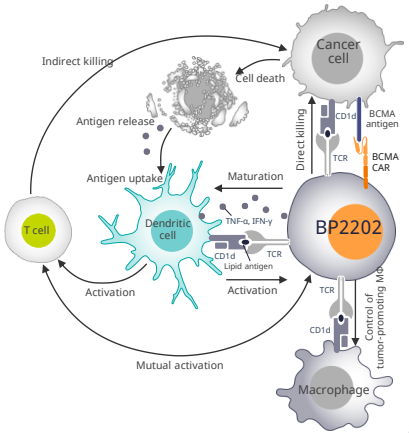
BP2202 (iPSC-derived BCMA CAR-iNKT Cell Therapy)

1. iPSC-derived BCMA CAR-iNKT cell therapy for multiple myeloma



2. Invariant Natural killer T (iNKT) cell effector

- BCMA CAR-iNKT cells kill myeloma cells directly and also drive endogenous T cells into an anti-tumor response
- May overcome the lack of durable responses seen with conventional allogeneic CAR-T



3. Allogeneic cell therapy

- No manufacturing wait; can be administered promptly
- Consistent quality, independent of the patient's T-cell status

4. Manufactured from a master iPS cell bank

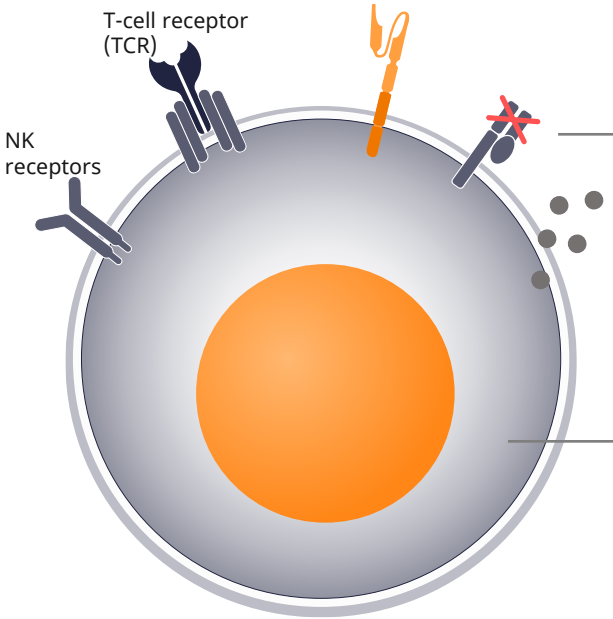
- Consistency: avoids the efficacy/safety variability of autologous or donor-derived allogeneic products — a predictable therapy that is easy for physicians to use
- Scalable format: cost of goods competitive with non-cell-based off-the-shelf products

5. Clinical-stage

- Open-label, dose-escalation, multicenter Phase 1 trial in relapsed/refractory multiple myeloma ongoing in the US

BP2202 Cell Product

iPSC-derived BCMA CAR-iNKT cells (BP2202)



Gene editing

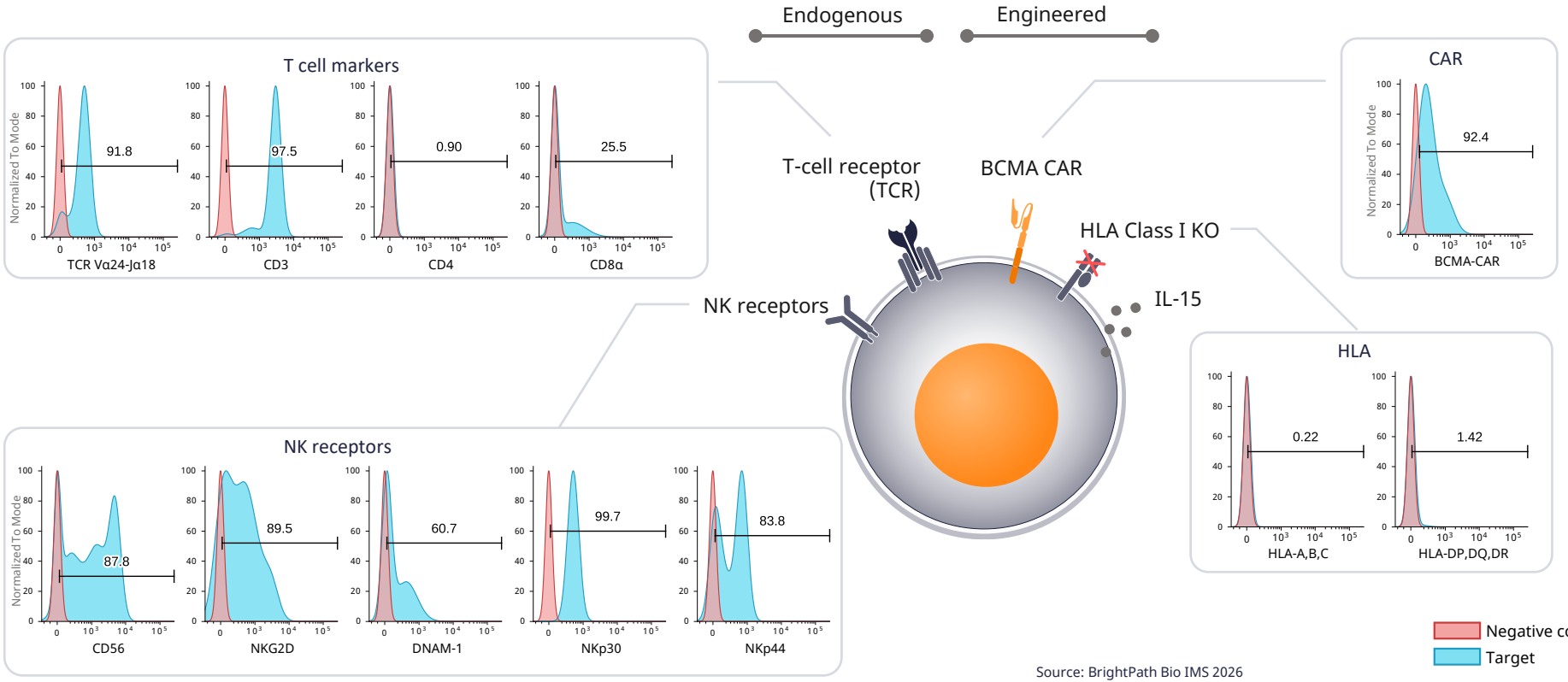
- **BCMA CAR transduction**
  - Chimeric antigen receptor (CAR) recognizing BCMA (B-cell maturation antigen), which is highly expressed on myeloma cells
  - Upon target recognition, transmits activation signals to the NKT cell, which kills myeloma cells by releasing cytotoxic molecules and cytokines
- **HLA class I knockout**
  - Removes HLA class I, the marker by which the patient's killer T cells recognize allogeneic cells as non-self, to avoid rejection
- **IL-15 transduction**
  - Autonomous survival signaling: greater persistence, less exhaustion

Effector cells

- **Invariant Natural killer T (iNKT) cells derived from iPS cells**
  - In addition to directly killing myeloma cells, they drive the patient's endogenous T cells into an anti-tumor response
  - Manufacturing from a master iPS cell bank enables continuous supply at consistent quality, an advantage for clinical scale-up and commercial production

BP2202 Cell Product (cont'd)

Phenotype and CAR expression



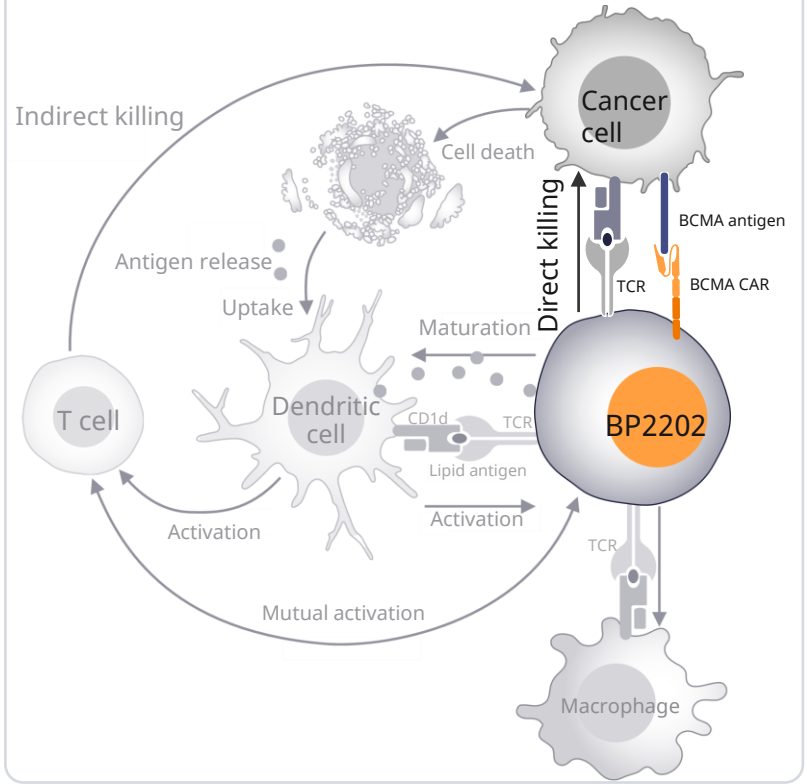
Source: BrightPath Bio IMS 2026

BP2202 Key Differentiators

Invariant Natural killer T (iNKT) cell effector

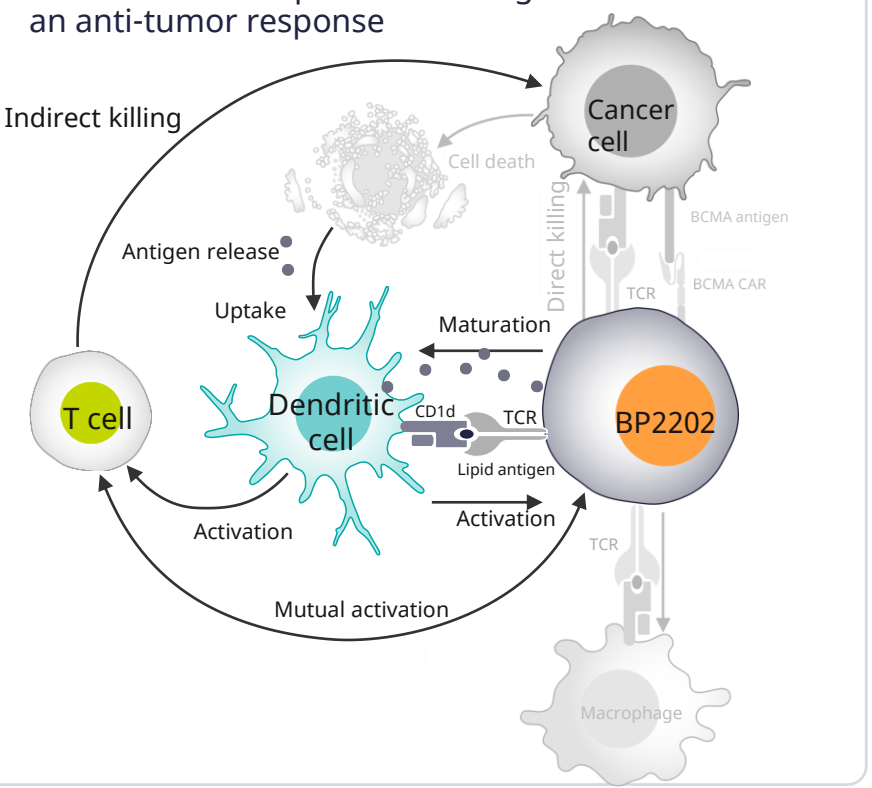
May overcome the lack of durable responses seen with conventional allogeneic CAR-T

- CAR -mediated direct killing of myeloma cells



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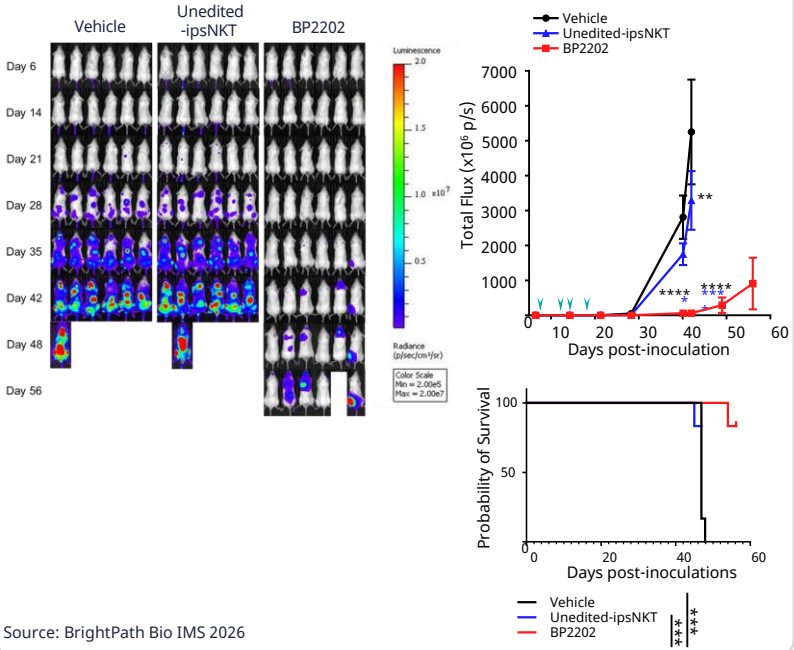
- Orchestrates the patient's endogenous T cells into an anti-tumor response



BP2202 Key Differentiators (cont'd)

Anti-tumor effects

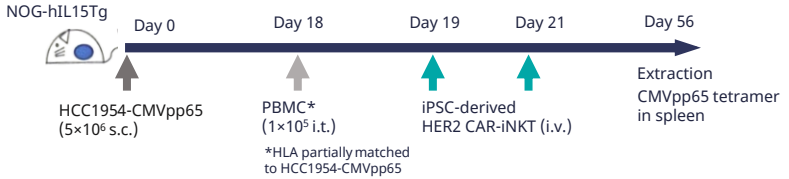
- CAR -mediated direct killing of myeloma cells
  - BP2202 suppressed tumor burden and prolonged survival in MM.1S-luc–bearing NOG-hIL-15 Tg mice compared with vehicle (HBSS) and non-transduced iPSC-derived iNKT cells.

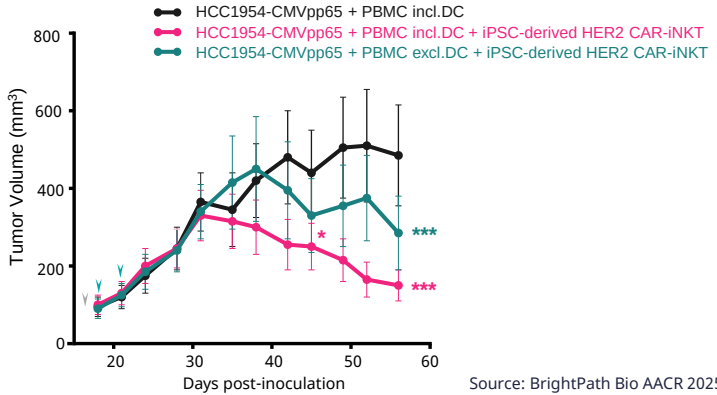


Source: BrightPath Bio IMS 2026

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- Orchestrates the patient's endogenous T cells into an anti-tumor response
  - Anti-tumor efficacy of HER2 CAR-iNKT cells and DC-dependent induction of tumor antigen-specific T cell responses



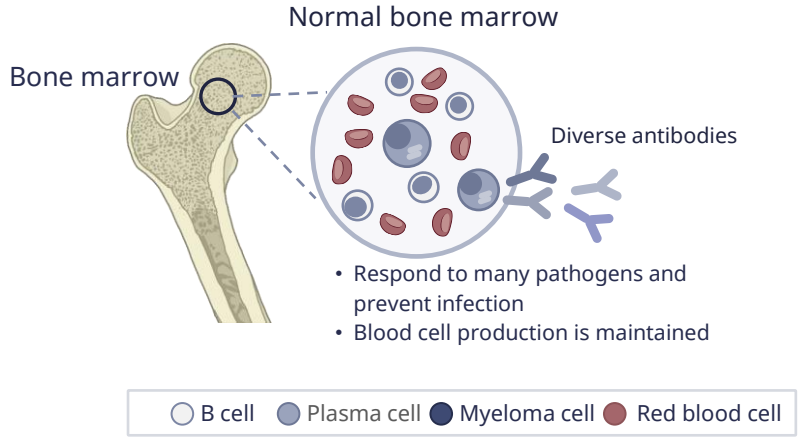


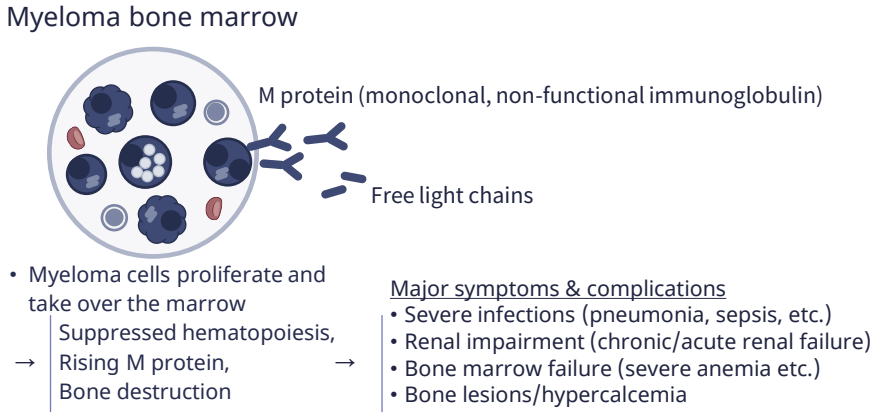
Source: BrightPath Bio AACR 2025

BP2202 Target Indication

BP2202 target indication: Multiple myeloma (MM)

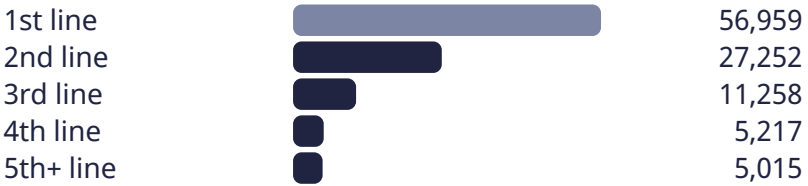
- A blood cancer arising from malignant plasma cells in the bone marrow; most patients experience repeated relapses





- 10–15% of blood cancers; median age at diagnosis 69 (US SEER)
- 5-year relative survival 64% (US)
- Estimated incidence and mortality
  - Global: 196,000 new cases/yr; 119,000 deaths/yr (2024)
  - US: 36,000 new cases/yr; 10,900 deaths/yr (2026)
  - Japan: 8,100 new cases/yr; 4,400 deaths/yr (2025)

- Estimated patients on treatment by line of therapy (US, 2020)



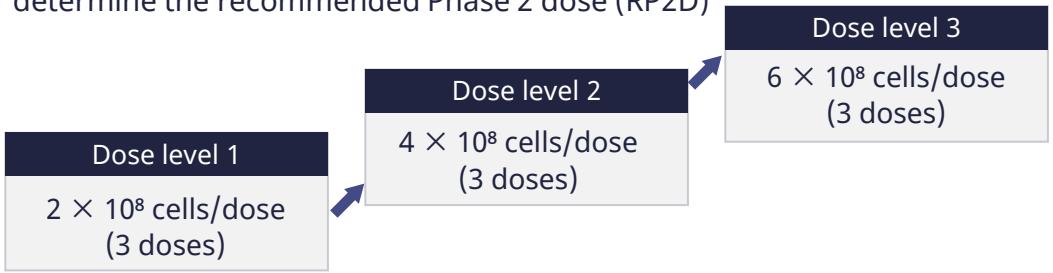
Sources: Sonneveld P, et al. *Nat Rev Clin Oncol* 2025; Cowan AJ, et al. *JAMA* 2022; SEER (NCI); GLOBOCAN 2024 (IARC); National Cancer Center Japan, 2025 cancer statistics projections; Nikolaou A, et al. *Value Health* 2022

BP2202 Clinical Trial Design

Open-label, multicenter Phase 1 study in relapsed/refractory multiple myeloma

- Study design
  - Part A Dose escalation (3+3 design)

    Start at dose level 1 and escalate if no dose-limiting toxicity (DLT) occurs, to determine the recommended Phase 2 dose (RP2D)



  - Part B Dose expansion

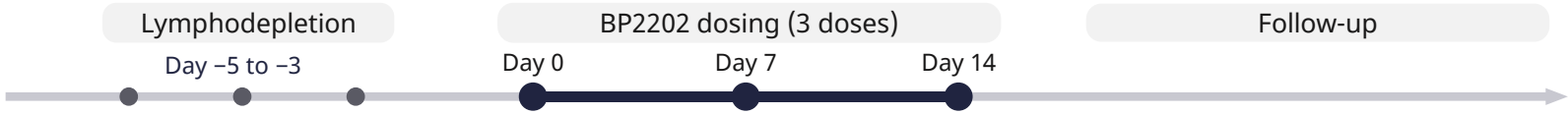
    Further evaluate safety and anti-tumor activity



    All Part B patients receive the RP2D

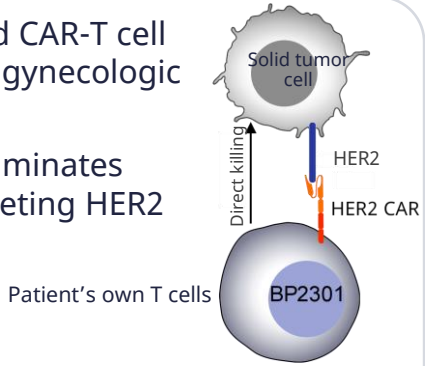
    Up to 12 patients (including Part A patients treated at the RP2D)

- Treatment schedule

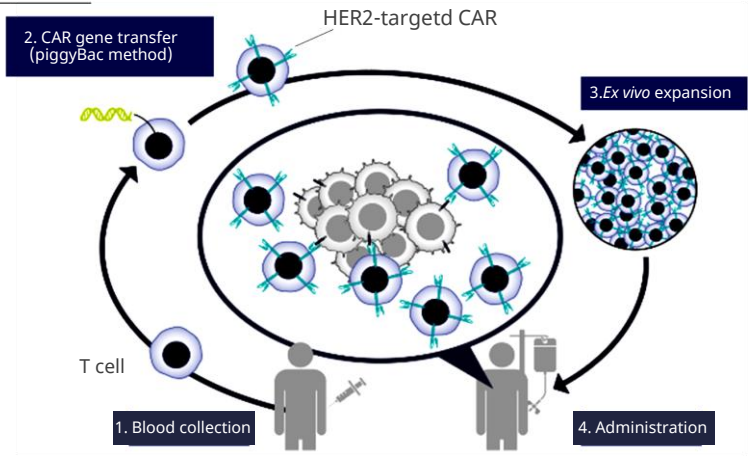


BP2301 (Autologous HER2 CAR-T Cell Therapy)

1. Autologous HER2-targeted CAR-T cell therapy for HER2-positive gynecologic cancers or sarcoma
   - Mechanism of action: eliminates solid tumor cells by targeting HER2

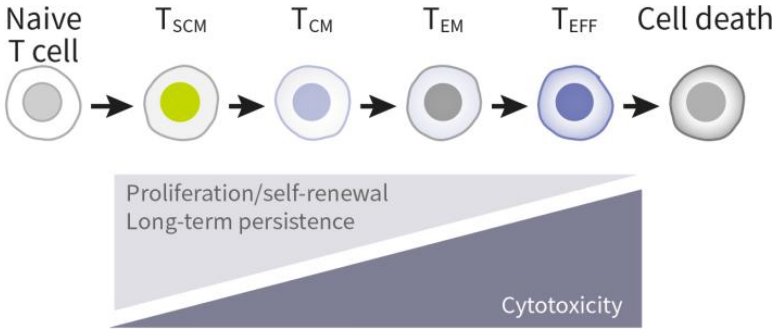


CAR-T treatment flow



2. Autologous CAR-T designed for solid tumors
   - Stem cell memory-like T cells (T_{SCM}) harnessed for CAR-T
     - Escape early exhaustion in immunosuppressive solid tumors and keep killing cancer cells
     - Manufacturing process enriches for less-differentiated T_{SCM} with high self-renewal capacity and persistence

T cell differentiation



3. Phase 1 investigator-initiated trial ongoing
   - Indications: HER2-positive sarcoma and gynecologic cancers
   - Study site: Shinshu University Hospital

BP2301 (Cont'd)

Prior HER2 CAR-T clinical programs

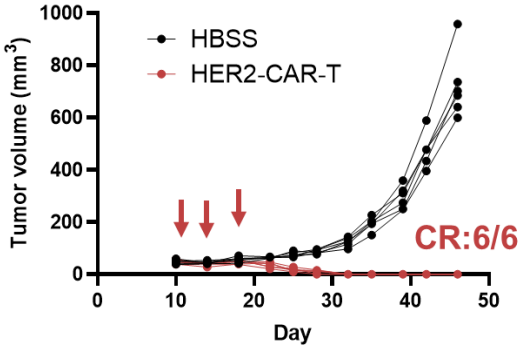
Trial	Baylor College of Medicine 2017*1	Baylor College of Medicine (HEROS2 trial) 2024*2	EXUMA Biotech 2025 (interim analysis)*3	BP2301 FY2026E
CAR gene transfer	Retroviral vector	Retroviral vector	Lentiviral vector	piggyBac (non-viral)
Indication	Glioblastoma	Sarcoma (relapsed/refractory, with metastases)	Solid tumors (gastric, esophageal, colorectal, breast)	Sarcoma Gynecologic cancers
Patients	17	13	12	
Adverse events	Not reported	G≥3 CRS: 2 pts	G≥3 AEs: 2 pts	
Clinical response	PR: 1 pt	CR: 2 pts	"Deep" PR: 1 pt 100% target lesion reduction	

*1 *JAMA* Oncol 2017;3(8):1094–1101 *2 *Nat Cancer* 2024;5:880–894 *3 ASCO GI 2025
CRS: cytokine release syndrome; PR: partial response; CR: complete response
Sources: publications and conference presentations

- Enabled by the non-viral CAR gene transfer method of Prof. Yozo Nakazawa et al. (Shinshu University) combined with a cell culture method co-developed with BrightPath
- Manufacturing process patents granted in Japan, the US and China

Non-clinical proof of concept

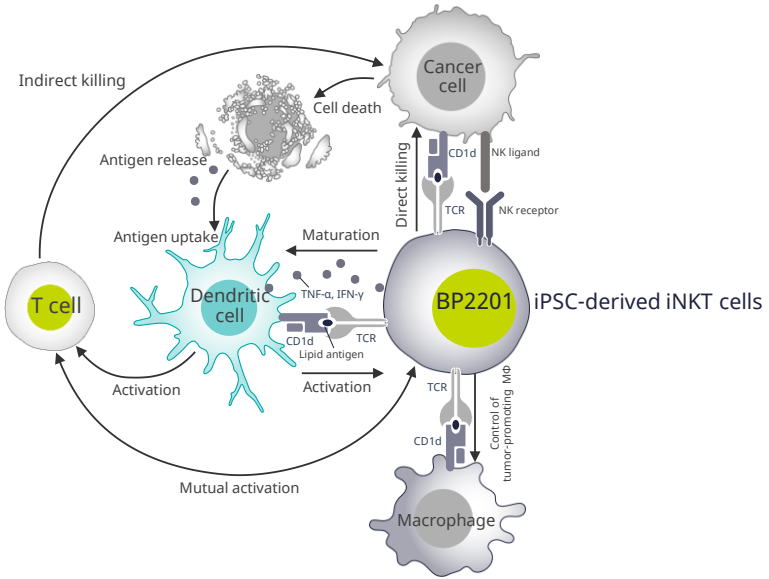
- In mice bearing human ovarian cancer SK-OV-3 xenografts, BP2301 induced complete tumor regression in all 6 mice, with no regrowth



Source: BrightPath Bio, AACR 2022

BP2201 (iPSC-derived, Non-gene-modified NKT Cells)

1. iPSC-derived iNKT cell therapy for head and neck cancer



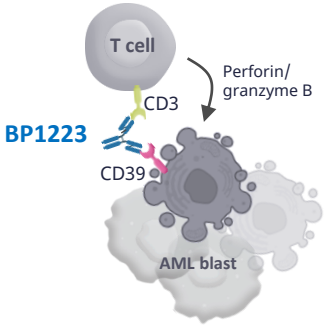
2. Phase 1 investigator-initiated trial completed (2024)
   - Indication: relapsed/refractory head and neck cancer
   - Study site: Chiba University Hospital

- World's first administration of iPSC-derived NKT cells to humans, confirming safety and early clinical activity
  - In relapsed/refractory head and neck cancer, 4 of 5 evaluable patients (80%) in the high-dose group achieved stable disease (SD)
  - Including a patient with a trend toward tumor shrinkage
  - Most frequent treatment-related AE (TRAE): Grade 1–2 fever (1 patient, low dose; 4 patients, high dose)
- In 2 patients with tumor growth suppression, cytotoxic T cells increased, suggesting an association with iPS-NKT cell-derived cytokines

Iinuma et al., Allogeneic iPSC-derived iNKT cells in recurrent head and neck cancer: a phase 1 trial. *Nature Communications* 16:11666 (2025)

BP1223 (CD39-targeted T Cell Engager)

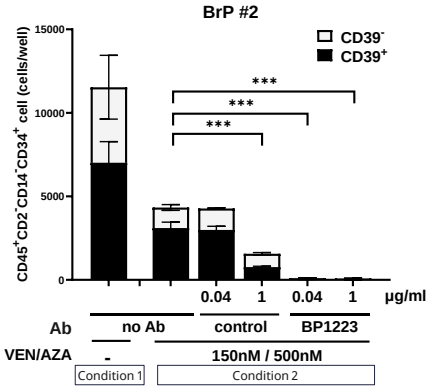
1. CD39-targeted T cell engager for acute myeloid leukemia (AML)
   - [MOA] Binds simultaneously to CD39 on AML blasts and CD3 on T cells, bridging the two; T cells activated via CD3 signaling eliminate the adjacent AML blasts



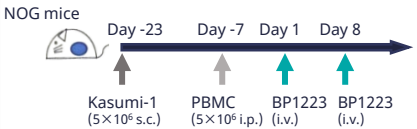
2. Target Validation
   - CD39 is highly expressed on AML blasts resistant to venetoclax, the standard of care for newly diagnosed and relapsed unfit AML — a key difference from targets of previous candidates (CD123, CD33, CD47, TIM-3, etc.)

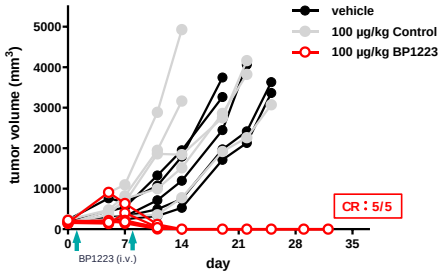
   ⇒ Complementary to venetoclax in combination

3. Anti-leukemia effect
   - BP1223 demonstrated enhanced reduction of patient-derived AML cells in combination with Ven+Aza



   - BP1223 monotherapy completely eliminated engrafted human AML tumors in all 5 mice





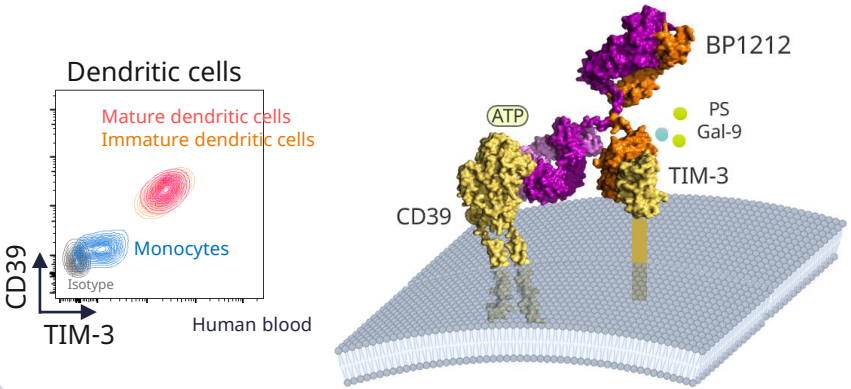
BrightPath Bio, ASH 2024

BP1212 (CD39 × TIM-3 Bispecific Inhibitory Antibody)

1. Dendritic cell-targeting CD39 × TIM-3 bispecific inhibitory antibody for solid tumors

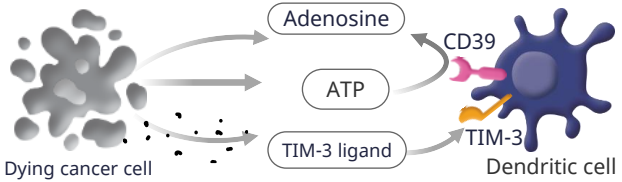
   [MOA]

   - CD39 and TIM-3 are highly expressed on dendritic cells, the “command center” of immunity, keeping them inactive and thereby playing a central role in creating an immunosuppressive tumor microenvironment
   - By blocking both immunosuppressive pathways, BP1212 promotes T cell stimulation by dendritic cells and induces robust anti-tumor immunity



2. BP1212 acts locally within tumors

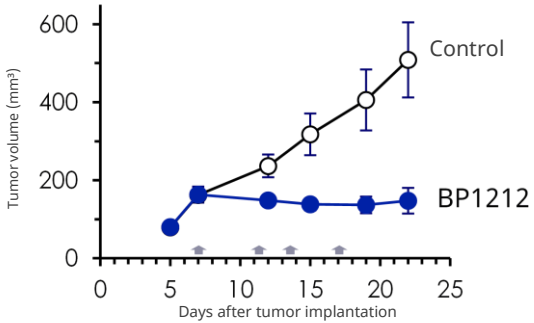
   - Acts within tumors rich in eATP (CD39 substrate) and PS (TIM-3 ligand), reactivating dendritic cells rendered dysfunctional by the immunosuppressive environment



3. Solid tumor mouse model study

   - Monotherapy markedly suppressed tumor growth

Syngeneic model in CD39/TIM-3 double knock-in mice



BrightPath Bio, IRCI 2025

Company Overview

Company Overview

Name	BrightPath Biotherapeutics Co., Ltd. (TSE Growth: 4594)	
Locations	Headquarters	2-2-4 Kojimachi, Chiyoda-ku, Tokyo
	Head Office / Kawasaki Research Laboratory	3-25-22 Tonomachi, Kawasaki-ku, Kawasaki, Kanagawa
	Cell Technology Laboratory	3-25-22 Tonomachi, Kawasaki-ku, Kawasaki, Kanagawa
Established	May 8, 2003	
Business	Development and commercialization of cancer immunotherapies	
Capital	JPY 2,574 million (as of March 31, 2026)	
Employees	23 (as of March 2026)	
Officers	Representative Director & CEO	Kenichi Nagai
	Director (part-time)	Akira Yamada
	Director (outside, independent)	Hirotaka Takeuchi
	Auditor (outside)	Tsutomu Kishino
	Auditor (outside, independent)	Taketoshi Abe
	Auditor (outside)	Yoshiyasu Yamaguchi

	Professor Emeritus, Kurume University
	Chair, ICU Board of Trustees; Prof. Emeritus, Hitotsubashi U.
	Partner, TMI Associates

Locations and History

Headquarters

Kojimachi Central Bldg. 7F,
2-2-4 Kojimachi, Chiyoda-ku, Tokyo



Head Office / Kawasaki Lab
Cell Technology Lab

Life Innovation Center, 3-25-22 Tonomachi,
Kawasaki-ku, Kawasaki, Kanagawa



May 2003
Founded in Kurume, Fukuoka

January 2006
Phase 1 trial of ITK-1 in CRPC initiated

August 2013
ITK-1 Phase 3 trial initiated

October 2015
Listed on TSE Mothers

US Phase 1 trial of GRN-1201 (melanoma) initiated

August 2016
Kawasaki Research Lab opened

April 2018
Joined RIKEN iPS-NKT project

June 2020
Investigator-initiated trial of BP2201 started

May 2022
Investigator-initiated trial of BP2301 started

November 2022
Exercised RIKEN option for exclusive worldwide rights to develop, manufacture and sell iPS-NKT

August 2026
US Phase 1 trial of BP2202 (BCMA CAR-NKT) initiated

BrightPath

Biotherapeutics