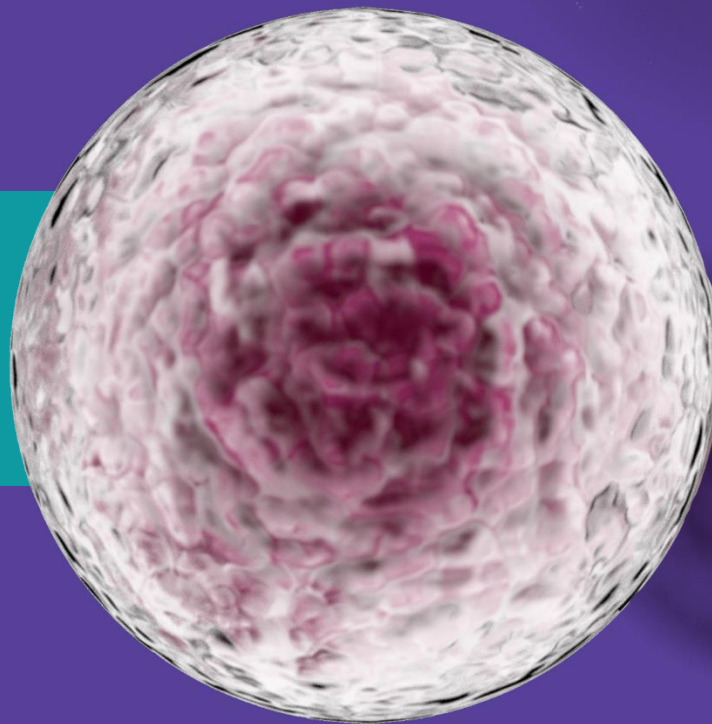




celularity



The Next Evolution in Cellular Medicine

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OUR VISION

Next Evolution in Off-the-shelf Allogeneic Cellular Therapies, at Greater Scale & Quality with Attractive Economics



To harness the placenta's unique biology and ready availability to develop therapeutic solutions

Lead the evolution in placental-derived therapeutics: advance the discovery of the placenta as a limitless, renewable source of neonatal cells, which are biologically preferred to cells from adult bone marrow or peripheral blood

Target large markets with high unmet need: broad therapeutic application including cancer, degenerative, and infectious diseases

Develop safe and effective therapies: leverage inherent advantages of placental-derived cells to produce uniform, scalable and optimized cellular therapies

Deliver off-the-shelf, cost effective therapies: cryopreserved allogeneic cellular therapies that clinicians can access on demand and off-the-shelf, enabling repeat dosing/multiple cycles as required in an outpatient setting

KEY INVESTMENT Highlights



1

Proprietary placenta-based platform developed over a 20-year history

2

Broad pipeline of novel, investigational product candidates across therapeutic areas and indications of high unmet need

3

Robust preclinical differentiation, encouraging clinical data and rapid path to approval

4

Purpose-built 150,000 sqft cell manufacturing facility with a highly scalable and optimized production process

5

Strong intellectual property portfolio with over 1,500 issued and pending patents worldwide

6

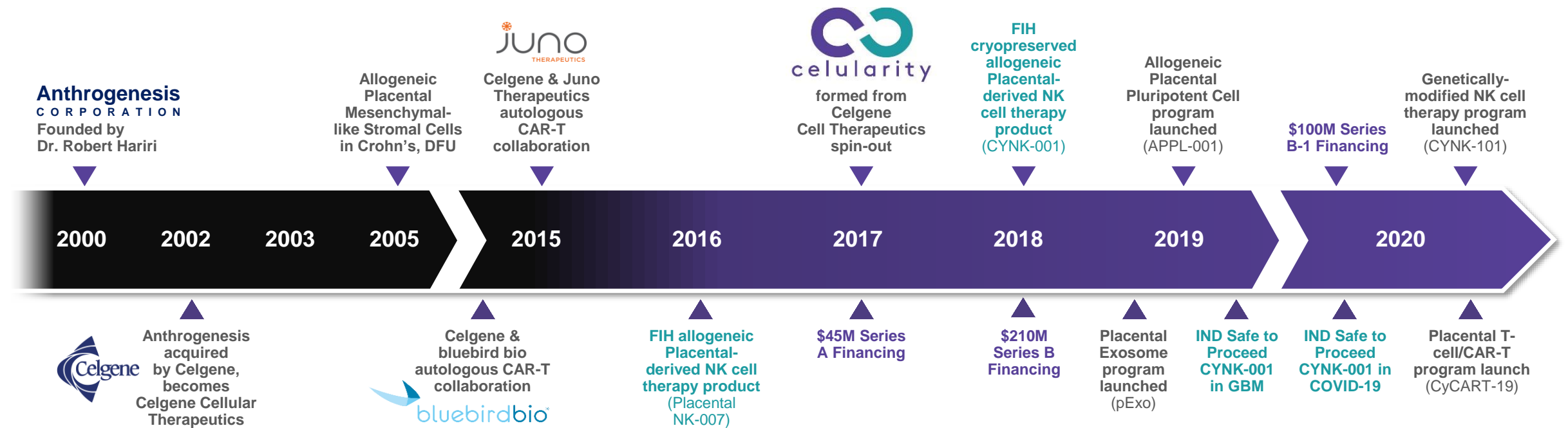
Experienced management team with deep expertise in cell therapy to advance the Company

Celularity

A Leader In Cellular Therapeutics

CELULARITY: COMPANY HISTORY

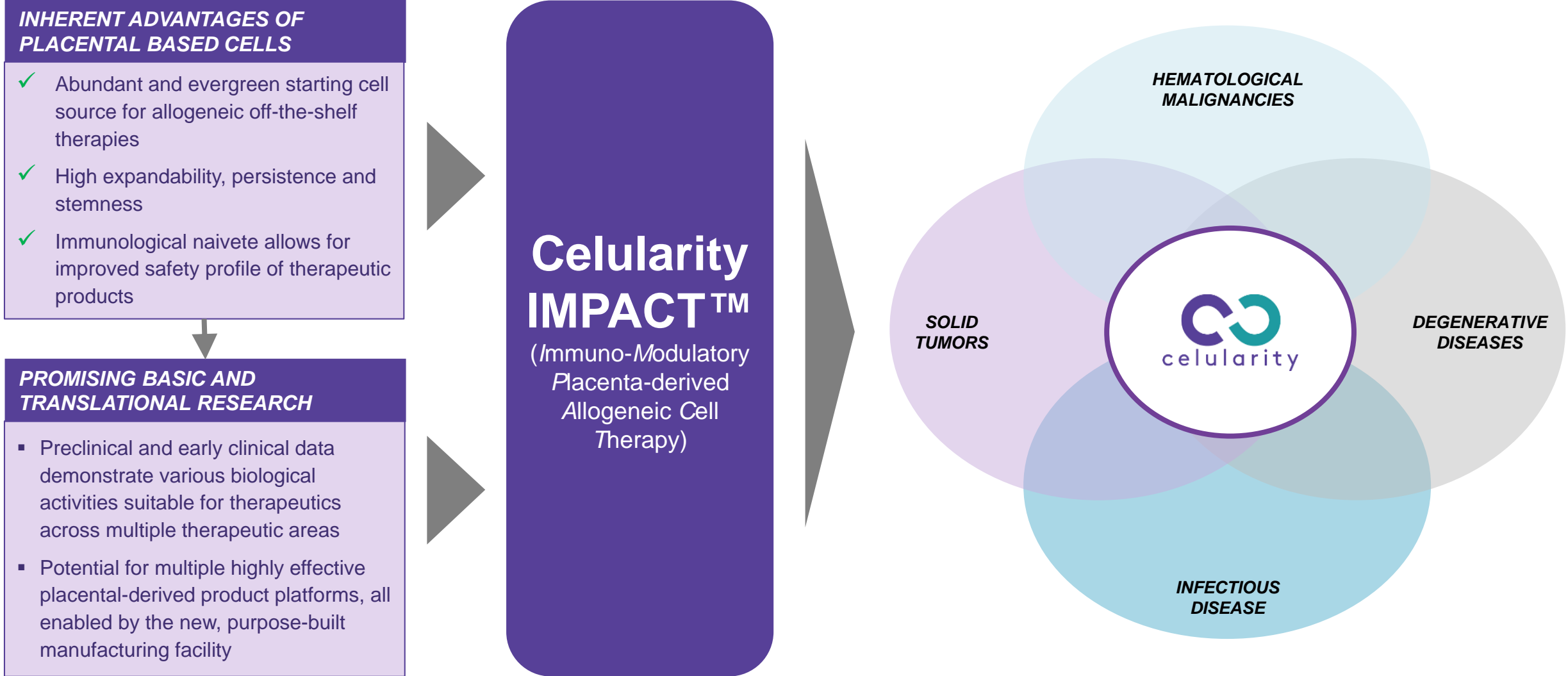
Celgene Spin-out (2017) Leveraging 20+ Years of Cellular Therapeutics Innovation



KEY: CORPORATE MILESTONE CLINICAL MILESTONE FINANCIAL MILESTONE

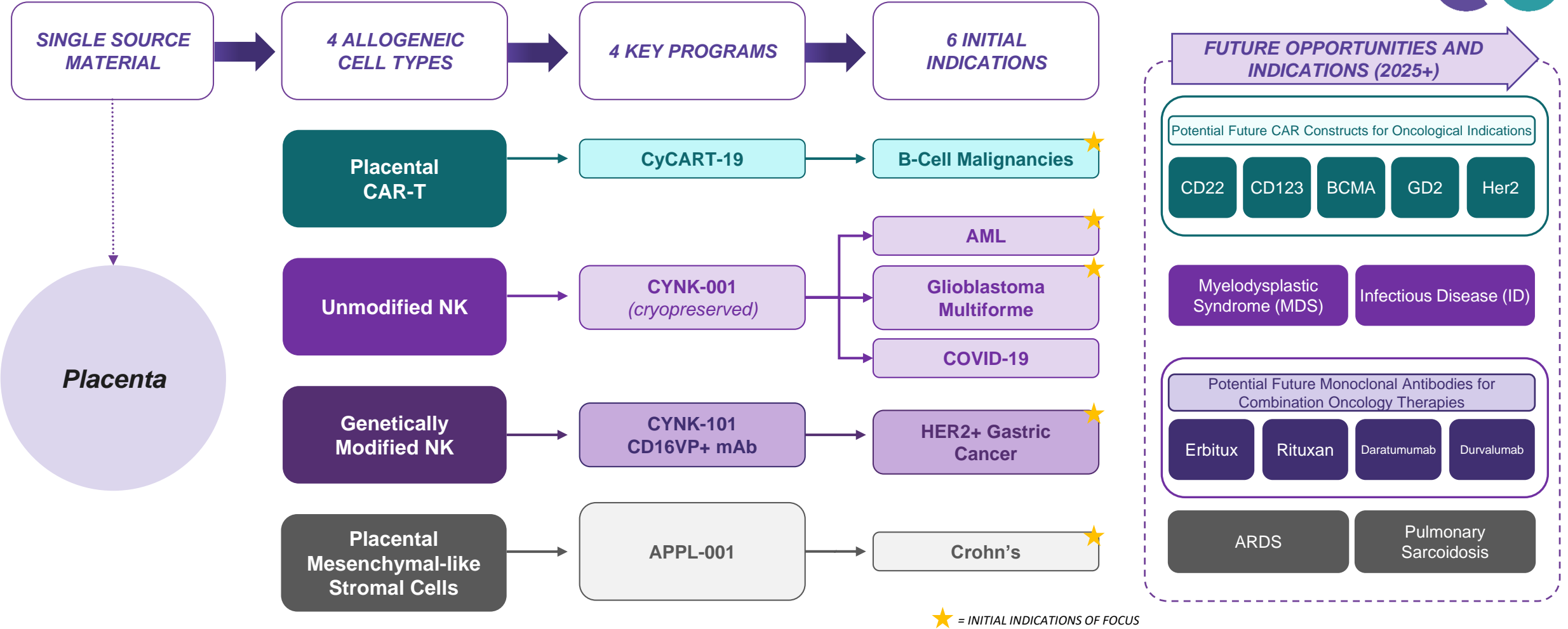
CELULARITY IMPACT™ PLATFORM

Capitalizing on the Benefits of Placenta Derived Cells to Target Multiple Diseases



CELULARITY'S SINGLE-SOURCE, PLACENTA-BASED PLATFORM DRIVING BROAD PIPELINE

Four Key Cell Types Driving Six Initial Indications and Potential for Further Expansion



MANUFACTURING >> Purpose-built, fully integrated manufacturing facility; rapidly scalable, end-to-end supply chain

Pipeline Overview

Broad Pipeline Across Oncology and Degenerative Diseases; Catalyst Rich 24 Months Ahead



	CELL TYPE	PROGRAM	INDICATION	2021	2022
HEMATOLOGIC MALIGNANCIES	CAR-T	CyCART-19	B-Cell Malignancies	IND Submission Phase I	Pivotal Phase II
	Unmodified Natural Killer Cell	CYNK-001 (cryopreserved)	Acute Myeloid Leukemia (AML)	Phase I Pivotal Phase II	
SOLID TUMORS	Genetically Modified Natural Killer Cell	CYNK-101 + mAb	HER2+ Gastric Cancer	IND Submission Phase I/IIa	Pivotal Phase II
	Unmodified Natural Killer Cell	CYNK-001 (cryopreserved)	Glioblastoma Multiforme (GBM)	Phase I/IIa Pivotal Phase II	
DEGENERATIVE DISEASES	Placental Mesenchymal-like Stromal Cell	APPL-001	Crohn's Disease	IND Submission	Phase I/IIa



3 Upcoming IND Submissions (2021E)
5 Programs in the clinic by end of 2021

MANUFACTURING OVERVIEW

Fully Integrated, Purpose Built Commercial Scale Manufacturing Site Including Translational Research & Biorepository



PURPOSE BUILT FACILITY FOR COMMERCIAL-SCALE CELLULAR THERAPEUTIC MANUFACTURING

- \$80M investment in cGMP/cGTP manufacturing
- Enables greater control, efficiency and optimization than is achievable by outsourcing to contract manufacturing organizations (CMOs) alone

STAFFED BY OVER 100 HIGHLY SPECIALIZED SCIENTISTS, ENGINEERS & TECHNICIANS.

- Optimized, product-specific CMC, QA/QC and manufacturing processes accelerate product development, production and commercialization
- Over 2 decades of experience with source material procurement

COMMERCIAL SCALE, GMP-READY

- 9 Grade C/ISO 7 suites
- 6 Grade D/ISO 8 labs
- Dedicated translational research labs

Celularity benefits from Celgene's 20 year+ investment in developing the technologies and capabilities required to manufacture cellular products at scale with consistent and reliable quality

EXPERIENCED MANAGEMENT TEAM

With Deep Expertise in Cell Therapy



Executive Leadership Team



Robert J. Hariri, MD, PhD
Founder & CEO



Xiaokui Zhang, PhD
Chief Scientific Officer



John Haines
Chief Operating Officer



David Beers
Chief Financial Officer



Andrew Pecora, MD, FACP, CPE
President of Medical Affairs



Gregory Berk, M.D.
Chief Medical Officer



Senior Medical Team



Solveig Ericson, MD, PhD
VP Medical Affairs



Sharmila Koppiseti, MD
VP Drug Safety Pharmacovigilance



Krzysztof Grzegorzewk, MD
VP Medical Affairs



Chi Li, PhD, MBA, RAC
SVP Regulatory Affairs



Selected Approvals by Medical Team



Celularity Pipeline Overview



CyCART-19

B-Cell Malignancies

CyCART-19 OVERVIEW

Celularity Approach and Advantages



CAR-T approach

Background

Allogeneic approaches have important advantages

- Off-the-shelf for on-demand use
- Eliminates lengthy wait time for patient
- Scalable manufacturing and simplified logistics instead of “one batch, one patient”

The Placenta Advantage

Among allogeneic, placenta may be key differentiator

- Rationale for greater stemness, expandability, persistence
- Abundant renewable starting cell source for allogeneic therapies
- Potential for improved safety profile due to immunological naivety

Celularity Approach

Strong pre-clinical evidence of anti-tumor activity

CyCART-19 in R/R B-Cell NHL IND submission : Q2 2021

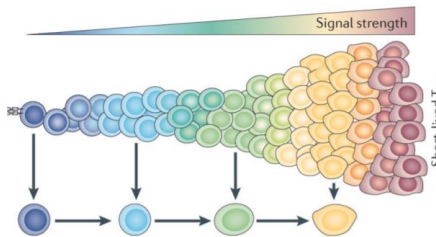
Phase 1 (safety and dose finding) start Q3 2021

Advantages

		CAR-T THERAPIES		
		AUTOLOGOUS	OTHER ALLOGENEIC	CELULARITY CyCART-19
MANUFACTURING COMPLEXITY	Cell Therapy Technology Scorecard			
	Source Procurement Non-invasive Collection / Reliable Procurement	✗	✗	✓
	Lower COGs Standardized, Scalable Manufacturing	✗	✓	✓+
	Starting Material Consistent Quality and Phenotype	✗	✗	✓
	Ability to Readily Expand While Maintaining a Less Differentiated Phenotype	✗	✗	✓
	“Off-the-Shelf” Treatment	✗	✓	✓+
	Ability to Re-dose Patients (if Necessary)	✗	✓	✓+

CELULARITY CyCART-19

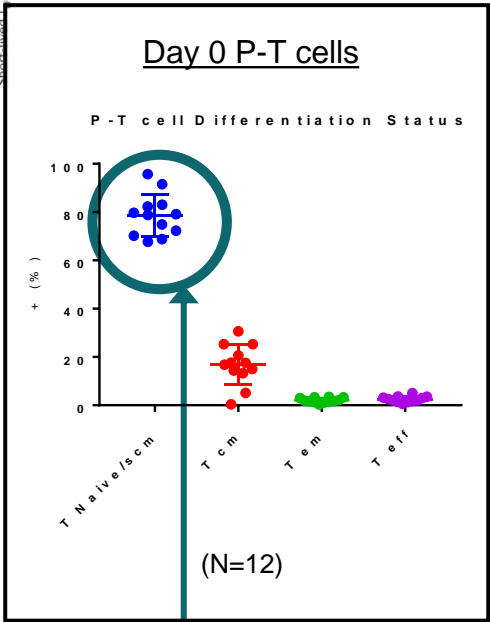
Demonstrated T Stem Cell Memory Characteristics



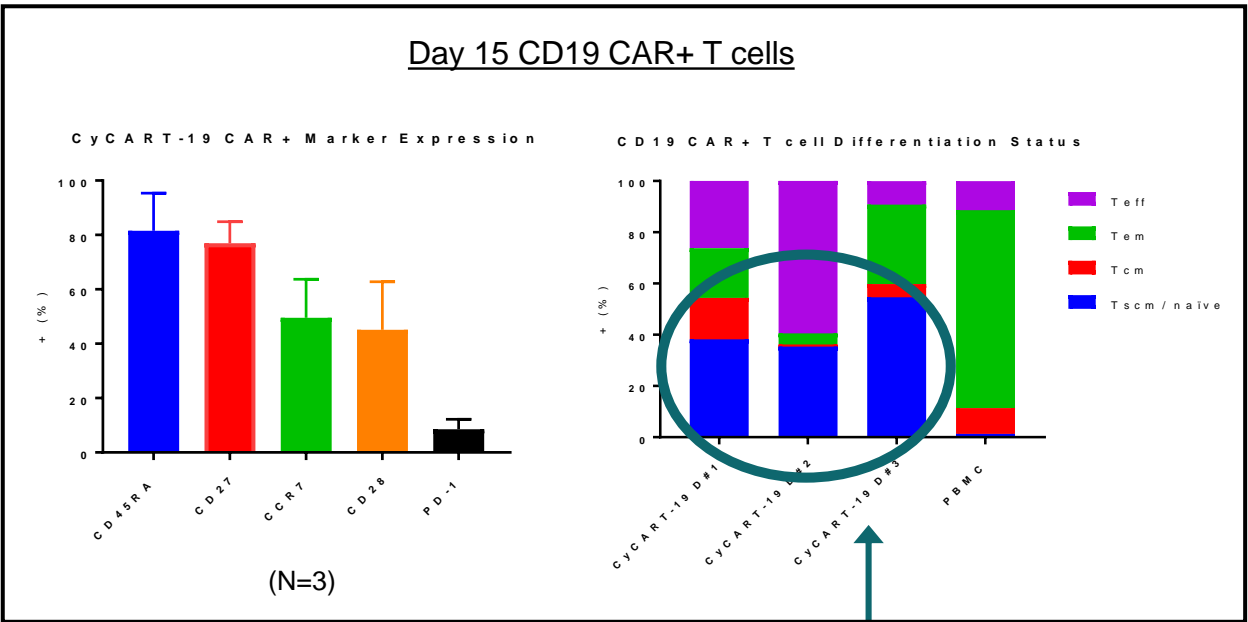
	Marker	Naïve	Stem Cell Memory	Central Memory	Effector Memory
Phenotype	CD45RA	+	+	-	+/-
	CD27	+++	+++	++	+/-
	CCR7	+++	+++	++	-
	CD28	++	+++	+++	+/-
Function	Telomere	+++	+++	++	+
	Self-renewal	+	+++	++	+
	IFN-γ	-	+	++	+++
	IL-2	-	++	+++	+/-
	Cytotoxicity	-	+/-	+	+++

Adopted from Gattinoni *et al.* Nature Reviews Cancer 2012

Stem Cell Memory = Greater Proliferative Potential, Increased Persistence in vivo



CyCART-19 starting material consists mostly of T stem cell memory (Tscm) cells



Established Robust Process to Ensure High Product Quality

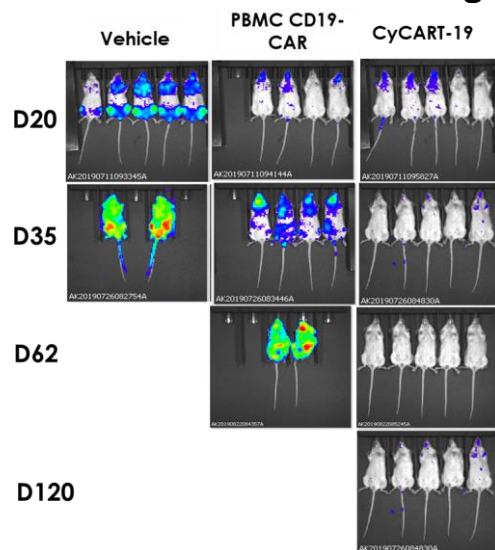
High proportion Tscm cells remain in CyCART-19 post expansion

CyCART-19 DEMONSTRATES SUPERIOR ANTI-LYMPHOMA ACTIVITIES & SURVIVAL

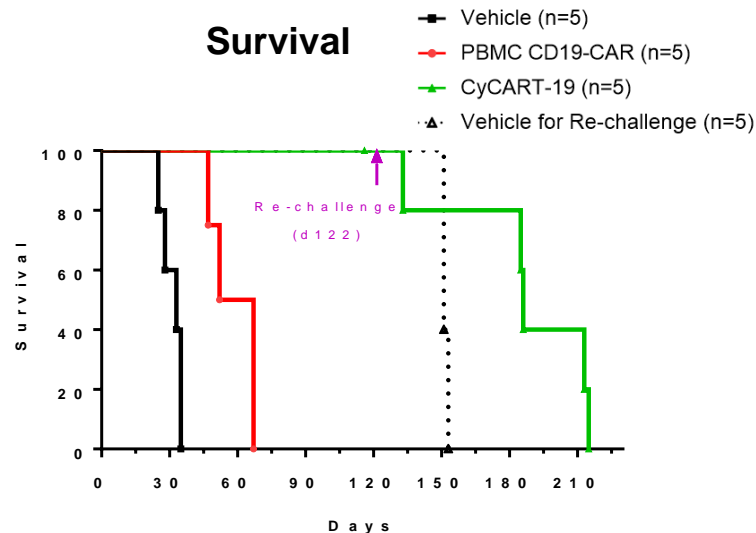
Greater Efficacy & Persistence, Prolonged Immune Attack upon Tumor Recharging



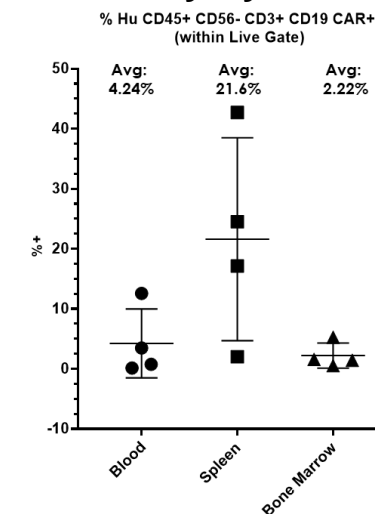
Bioluminescence Imaging



Survival



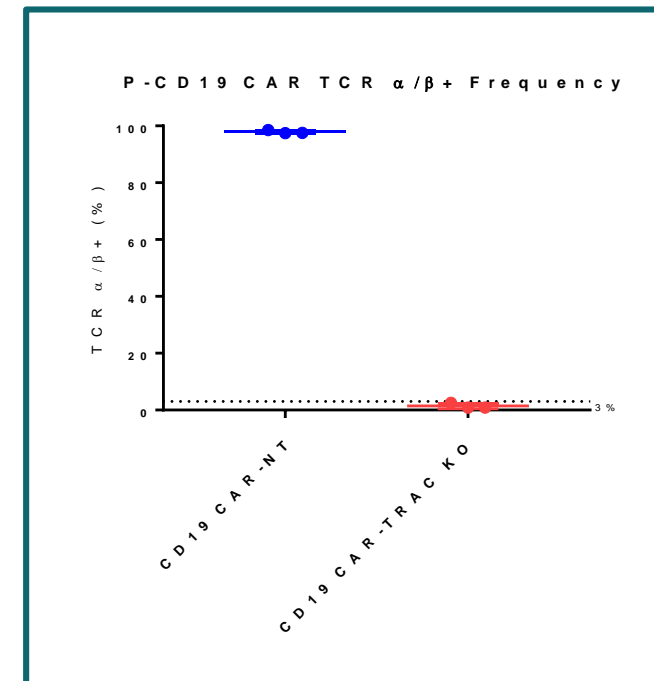
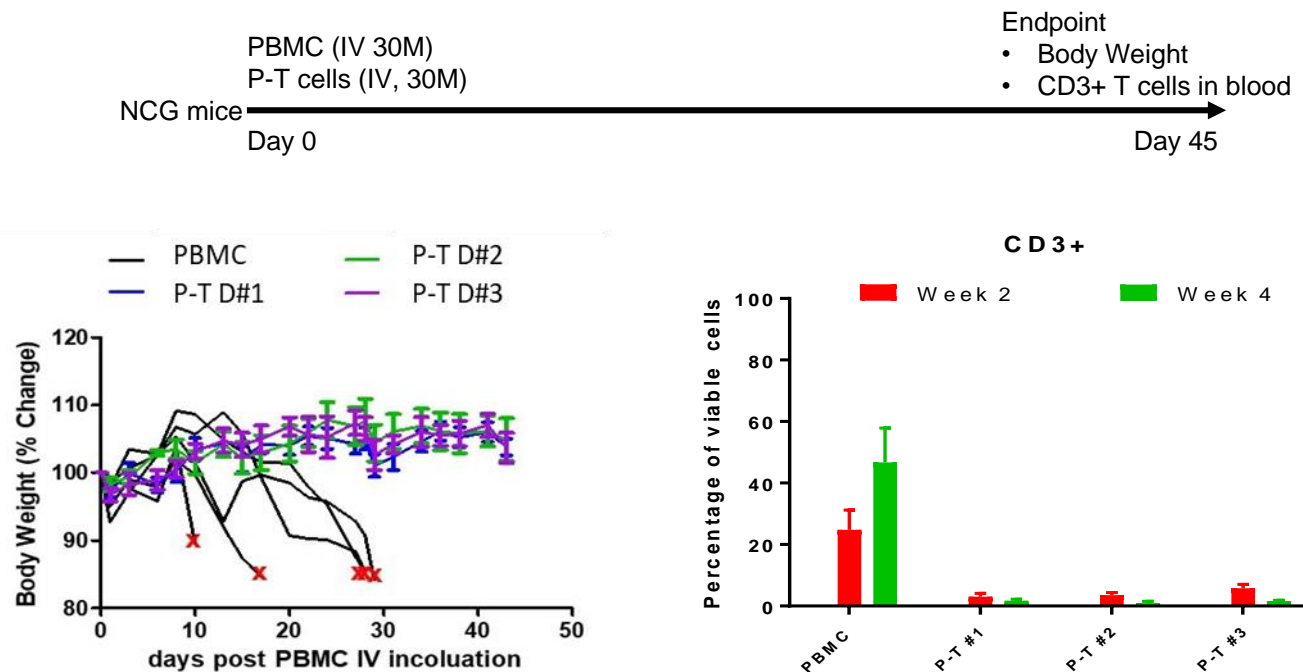
End of Study CyCART Analysis



- CyCART-19 demonstrates **significantly reduced tumor burden and survival benefit** compared to adult blood-derived CD19 CAR-T cells
- CyCART-19 **eliminated tumor** and resulted in **100% survival** out to **120 days**
- CyCART-19 “**memory**” characteristics demonstrated via:
 - **Extended survival out to 215 days** upon tumor re-challenge on Day 122
 - **Differentiated persistence** at end of study to elicit **prolonged antitumor** activities

CyCART-19 CELLS DO NOT INDUCE XENOGENEIC GvHD IN VIVO

Efficient TCR Knockout as Risk Mitigation



- Placental T (P-T) cells do not induce xenogeneic GvHD in vivo
 - Evidenced by **100% survival**, **no weight loss**, no increase in detection of any human CD3+ T cells in P-T treated mice
 - PBMC-treated mice exhibited significant weight loss, death of all mice, and increase of detection of human CD3+ T cells at Day 28
- Celularity includes CRISPR-mediated TRAC KO in its process as a risk mitigation strategy to prevent GvHD
 - **97-99% TRAC KO efficiency** achieved in CyCART-19 cells



KEY TAKEAWAYS

- Celularity has established a robust process to obtain placental T naive/scm population as source materials to produce off-the-shelf, highly scalable CyCART-19 cells
- CyCART-19 demonstrates stem cell memory characteristics as evidenced by superior in vivo persistence and durable antitumor activity in preclinical models
- CyCART-19 cells outperform adult blood-derived CART cells by significantly greater persistence and longer survival in preclinical studies
- Early data suggesting no signs of GvHD
- Note: If Phase 1 successful, Celularity plans to pursue a pivotal Phase 2 basket trial across major B-cell malignancies (subject to FDA discussions)

CLINICAL PLAN

- 2Q21: IND Submission Expected
- 3Q21: Phase I Study Start
- 1Q22: Phase II Study Start

CYNK-001

AML



NK CELL OVERVIEW

Celularity Approach and Advantages



NK approach

Background

NK cells are natural immune cells that eradicate both cancer and virus-infected cells

The Placenta Advantage

Placental-derived NK cells exhibit distinct characteristics:

- Different maturation and activation state
- Immature phenotype
- CYNK cells possess longer telomere length in comparison to peripheral blood (PB) NK cells, which suggests high in-vivo proliferation and persistence

Celularity Approach

Phase 1 study in R/R AML showed early signs of clinical benefit (2 out of 8 efficacy evaluable pts)

CYNK-001 moving into randomized Phase 2 (Q1 2021)

- High-risk patients (MRD+ disease)
- Leukemia free survival at 12 months is primary end-point
- Potential registrational study

CYNK-101 moving into Phase 1/2a (Q3 2021)

Advantages

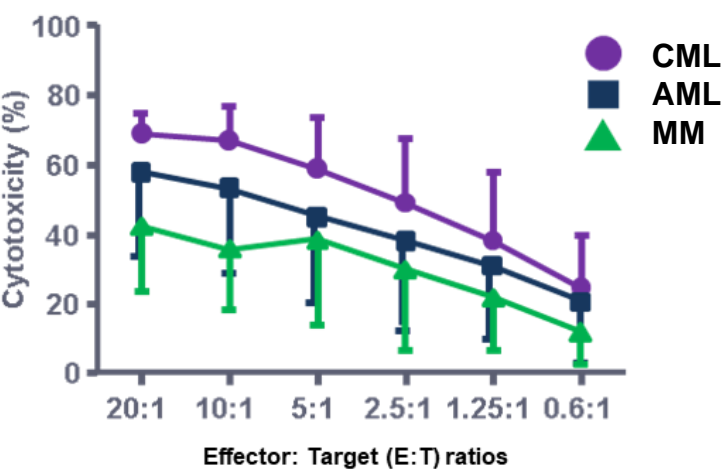
		NK CELL THERAPIES	
		ADULT DONOR DERIVED	CELULARITY CYNK-001 & CYNK-101
MANUFACTURING COMPLEXITY	Cell Therapy Technology Scorecard		
	Source Procurement Non-invasive Collection / Reliable Procurement	✓	✓
	Lower COGs Standardized, Scalable Manufacturing	✓	✓ +
	Starting Material Consistent Quality and Phenotype	✓	✓
	Ability to Readily Expand While Maintaining a Less Differentiated Phenotype	✗	✓
	"Off-the-Shelf" Treatment	✓	✓ +
	Ability to Re-dose Patients (if Necessary)	✗	✓ +

AML: PRE-CLINICAL DATA

Shows Evidence of Significant Leukemia Killing

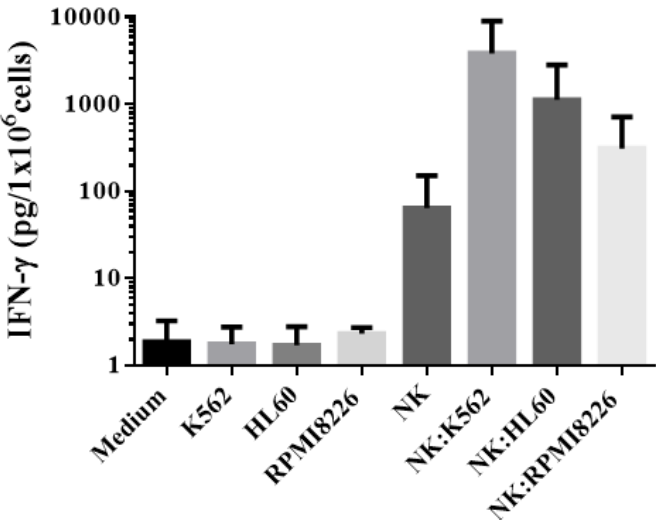


CML, AML, MM IN VITRO KILLING



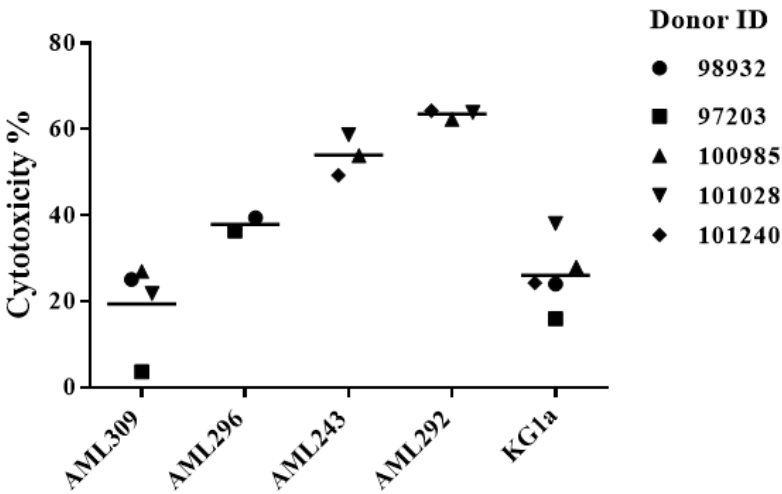
CYNK-001 demonstrates robust killing (cytolytic) against CML, AML, MM cell lines and primary AML samples

IFN-G PRODUCTION



CYNK-001 activation releases high concentration of IFN-g, favoring Th1 responses

PRIMARY AML KILLING



CYNK-001 exerted up to 60% specific lysis against primary AML samples at an Effector: Target (E:T) ratio of 3:1

FIRST ALLOGENEIC PLACENTAL HSCS-DERIVED NK CELL IMMUNOTHERAPY

Biological Characteristics Suggest Greater Therapeutic Potential



Proprietary process generates highly scalable, allogeneic, off-the-shelf NK cells from placental hematopoietic stem cells

- CYNK-001 investigated in two Phase I studies for refractory and relapsed Acute Myeloid Leukemia (r/r AML) and Multiple Myeloma (MM) post autologous stem cell transplant
 - CYNK-001 was well tolerated in 25 participants treated

Placental derived cells potentially differentiated from PB NK cells

- High expression of NCRs and low expression of CD16 and KIR, indicating immature phenotype characteristics
- Longer telomere length, suggesting potential high in vivo proliferation and persistence

High in vitro cytolytic activity against a broad range of tumor cell lines, in vivo maturation and anti-tumor activities, high cytolytic cytokines (e.g. IFN γ , Granzyme) production

- Low to no expression of PD-1, TIGIT, LAG-3, TIM-3

CYNK-001-AML-001 FIRST-IN-HUMAN STUDY

Phase I Study in Relapsed / Refractory Acute Myeloid Leukemia Showed Early Signs of Clinical Benefit



DESIGN

- Dose escalation study
- Conditioning with cyclophosphamide and fludarabine
 - Fludarabine 25 mg/m² x 5 days start day -6
 - Cyclophosphamide 60 mg/kg x 2 days on day -5 and -4 (omit Day -4 if within 4 months of prior transplant)
- CYNK-001 administered IV followed by up to 6 rhIL-2 injections
 - rhIL-2 at 6 million units subcutaneously beginning Day 0, every other day for 6 total doses

PHASE I RESULTS

- CYNK-001 well tolerated in a heavily pre-treated AML patient population
 - 11 r/r AML patients enrolled, 10 treated with single dose of CYNK-001, no DLTs¹, no GvHD, no detectable HLA allo-antibody
 - 8 of 10 patients were efficacy evaluable; the other 2 patients were not due to inadequate bone marrow (BM) for evaluation
 - 2 of 8 efficacy evaluable patients (both treated at 10M cells/kg) had evidence of clinical benefit
 - CRp² at Day 21
 - MLFS³ at Day 14

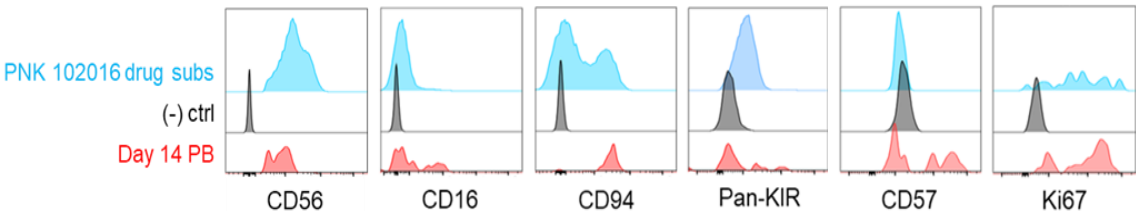
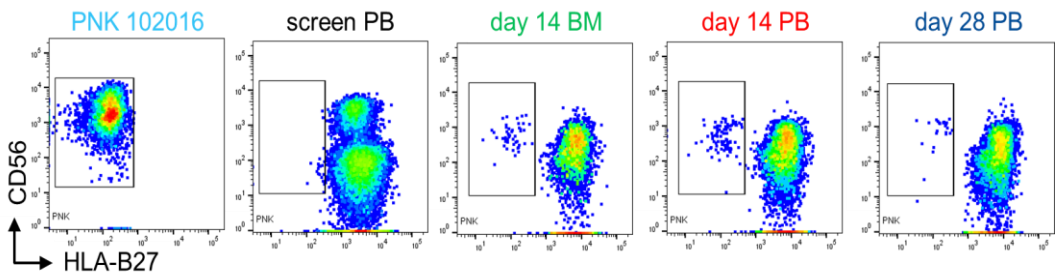
CYNK-001

Persisted, Matured and Proliferated in AML



CYNK-001 demonstrated **persistence up to 28 days**
(mean=11days)

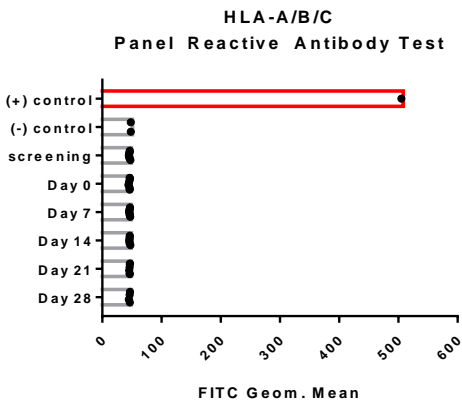
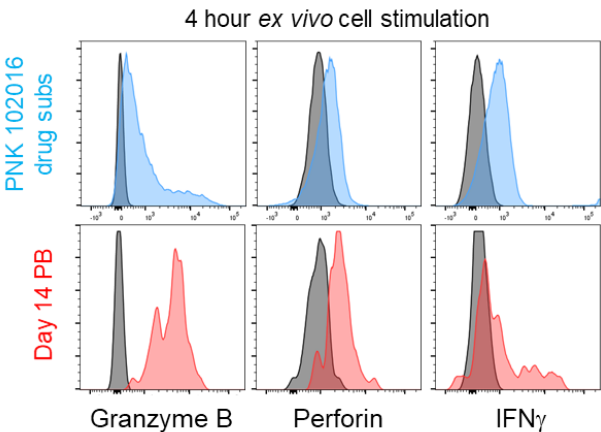
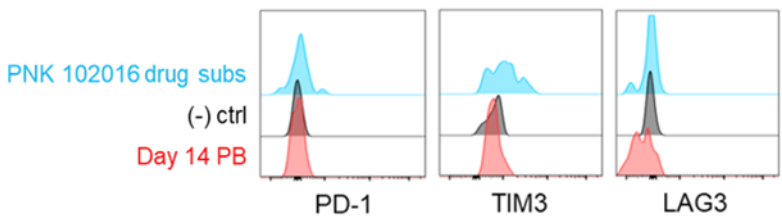
Persistent CYNK-001 cells **matured and proliferated**



No detectable exhaustion on CYNK-001 cells

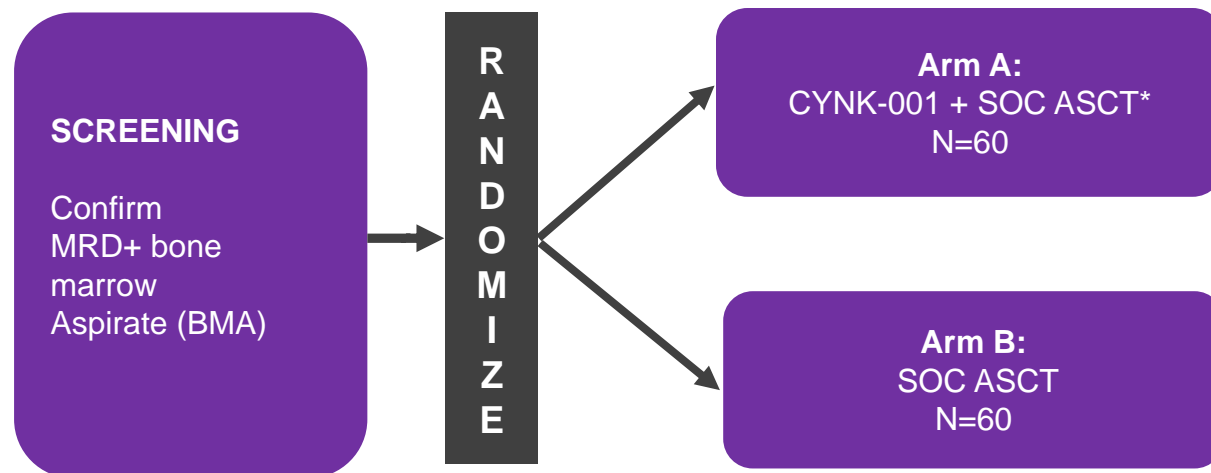
CYNK-001 **demonstrated effector function** post infusion

Absence of allo-HLA antibodies in all subjects



PIVOTAL RANDOMIZED PHASE 2 TRIAL

Evaluating CYNK-001 in MRD+ AML



RESPONSE ASSESSMENTS

- **Clinical response (BMA):**
 - Day 28 and 3, 6, 9, and 12 months post ASCT
- **MRD assessment:**
 - Arm A: Post-CYNK-001/pre ASCT
 - Arm A and B: Day 28 and 3, 6, 9, and 12 months post ASCT

PIVOTAL RANDOMIZED PHASE 2 STUDY (EFFICACY)

- **CYNK-001 + SOC ASCT vs SOC ASCT**
- Two-arm randomized (1:1) SOC ASCT with or without CYNK-001
- CYNK-001 dose from Phase 1 CYNK-001-AML-001 study
- N=120
- **Primary Endpoints:** Leukemia free survival at 12 months
- **Secondary Endpoints:** OS at 12 months, MRD conversion rate

KEY ELIGIBILITY CRITERIA:

- Subjects with AML in morphologic CR with MRD+ disease
- Transplant eligible with an identified donor



KEY TAKEAWAYS

- NK cells are natural immune cells that eradicate both cancer and virus-infected cells
 - Key mediators of antibody-dependent cellular cytotoxicity
- Placental derived NK cells exhibit distinct characteristics:
 - Different maturation and activation state
 - Immature phenotype
- CYNK cells possess longer telomere length in comparison to PB NK cells, which suggests **high in-vivo proliferation and persistence**

CLINICAL PLAN

- Current: Phase I Enrollment
- 1Q21: End of Phase I Meeting with FDA
- 1Q21: Phase II Study Start

CYNK-101

HER2+ Advanced Esophageal /
Gastric Adenocarcinoma

GENETICALLY MODIFIED NK PROGRAM

Improving Anti-Tumor Killing Power of CYNK Program



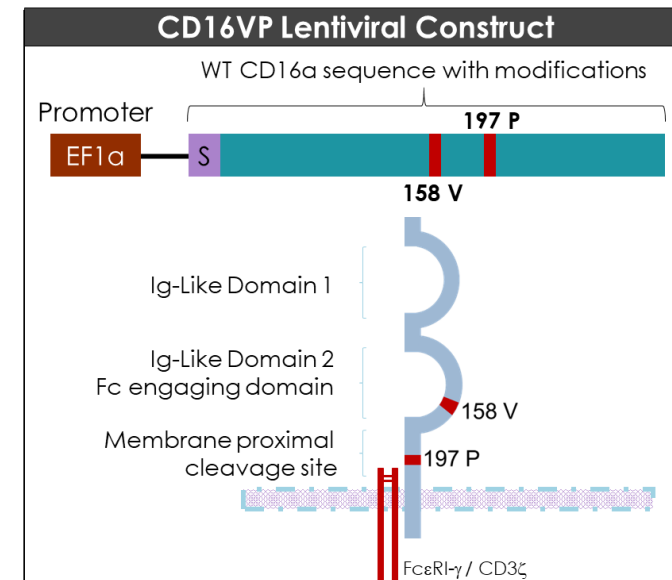
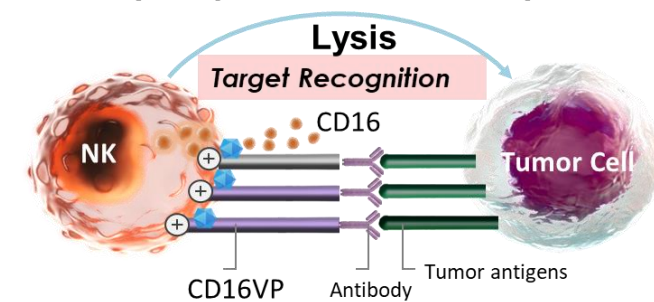
RATIONALE

- Engineering CYNK cells with high affinity and cleavage resistant (CD16VP) expected to improve affinity for IgG1 therapeutic antibodies, resist activation induced cleavage and improve overall ADCC potential
 - CD16 polymorphism impacts IgG affinity and thus ADCC
 - CD16 158 V/V – highest affinity for IgG1 and IgG3 and directly correlate with clinical responses
 - ~10% of population are homozygous for high affinity CD16 158V/V
 - Activation by cytokines or tumor cells leads to CD16 cleavage
 - CD16 cleavage by ADAM-17 – blocked by S→P mutation at position 197

OPPORTUNITIES

- Enable combination therapy with ADCC mediating therapeutic mAb therapies
- Augment CYNK clinical program with added “punching power” of Genetic Modification

Antibody-Dependent Cellular Cytotoxicity



CYNK-101 DEMONSTRATES EFFECTIVE ANTITUMOR ACTIVITY

Against Gastric Cancer Cell Lines in Conjunction with Anti-HER2 Monoclonal Antibody

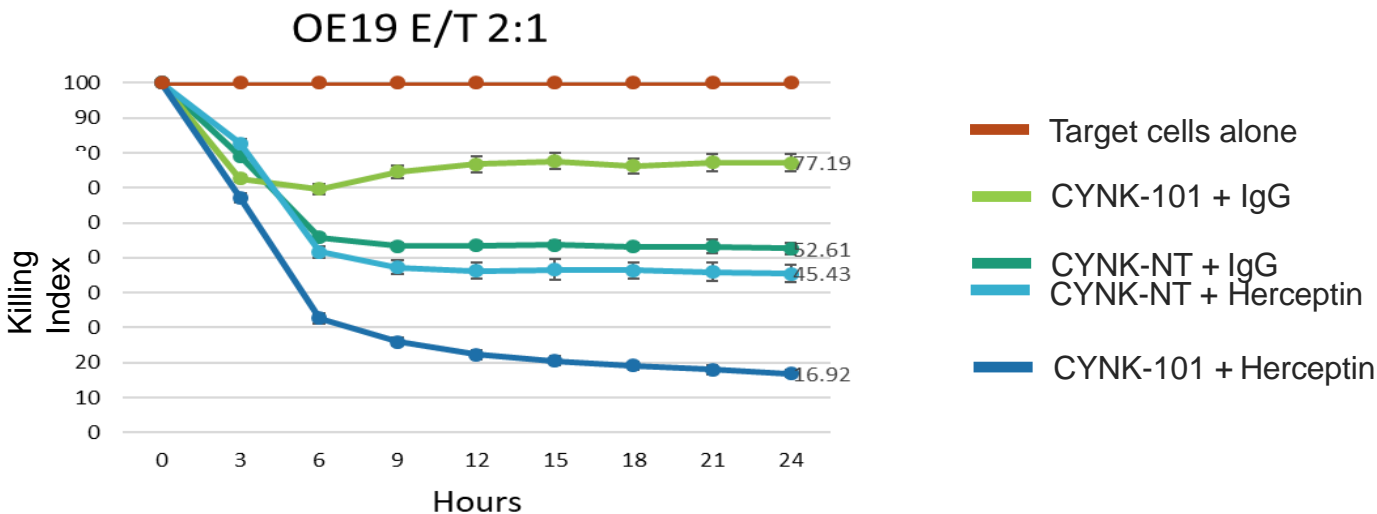
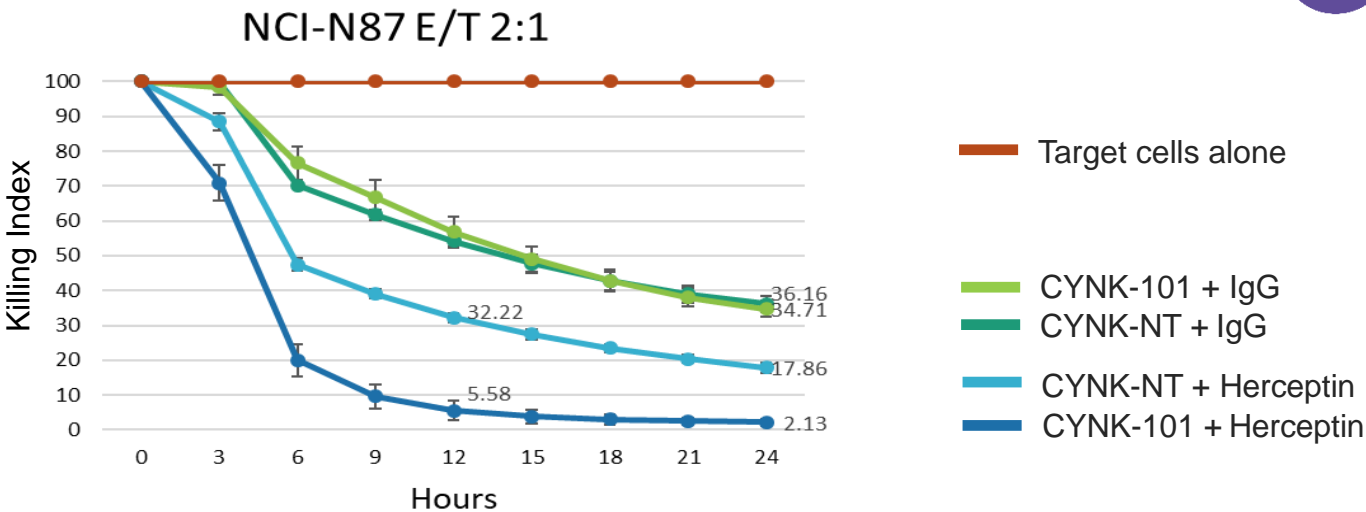


RESULTS

- Significant ADCC activity of CYNK-101 in combination with Herceptin against both gastric cancer cell lines was shown at E:T ratio of 2:1 over 24h in comparison with that of CYNK Non-Transduced (NT) or IgG control

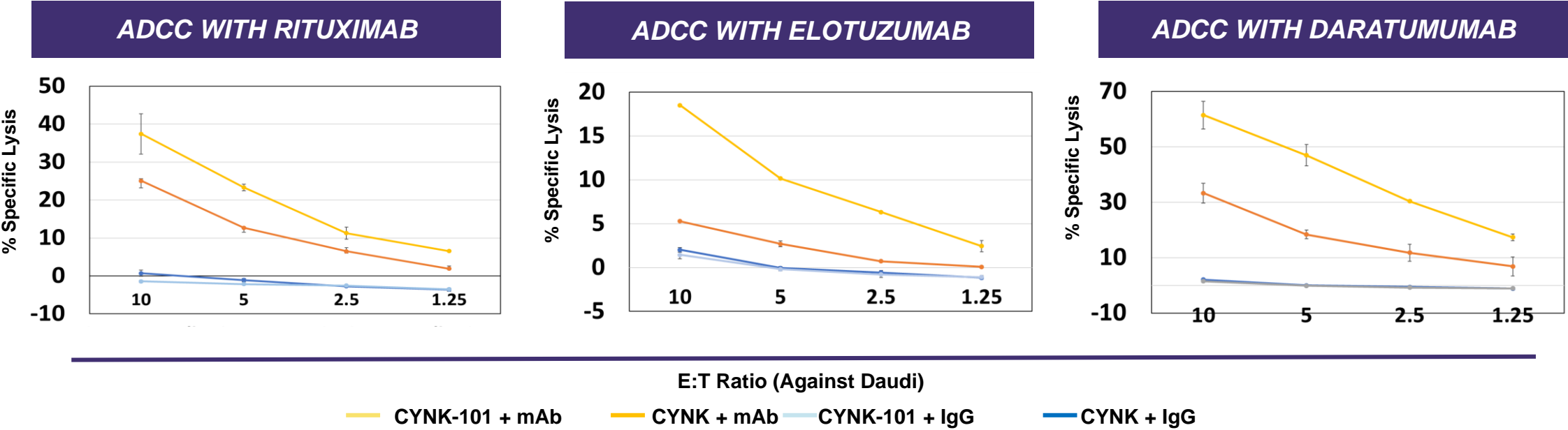
CONCLUSION

- Demonstrated ADCC activity of CYNK-101 in combination with Herceptin against HER2+ gastric cancer cells
 - HER2+ Gastric demonstrated to be an immunologically susceptible tumor type with evidence of strong NK cell infiltration



CYNK-101 PROVIDES A PLATFORM

For a Variety of mAb Combination Therapies



- Improved ADCC response observed from CYNK-101 compared to unmodified CYNK cells against lymphoma cell lines in combination with: Rituximab, Daratumumab and Elotuzumab antibodies
- IND-enabling studies on-going to evaluate CYNK-101 + mAbs in subcutaneous and orthotopic tumor models

CYNK-101 IN HER2+ GASTRIC CANCER

Summary



KEY TAKEAWAYS

- CYNK-101 adds “punching power” to the CYNK-001 platform via genetic modification
- When combined with Herceptin demonstrates ADCC activity against HER2+ Gastric Cancer cells
 - Joint impact of modified NK cells + mAb shows improved immunologic response with added NK cell killing

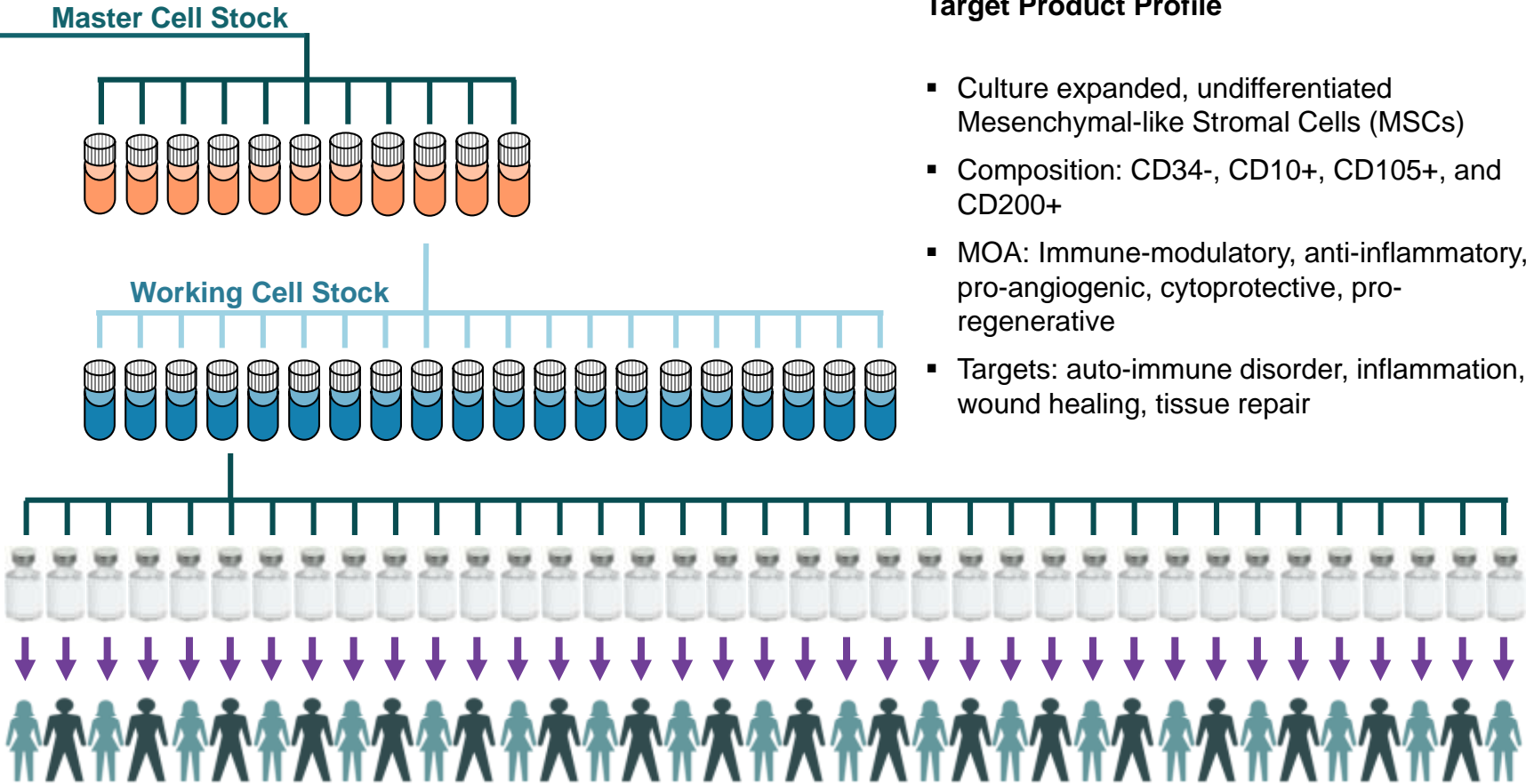
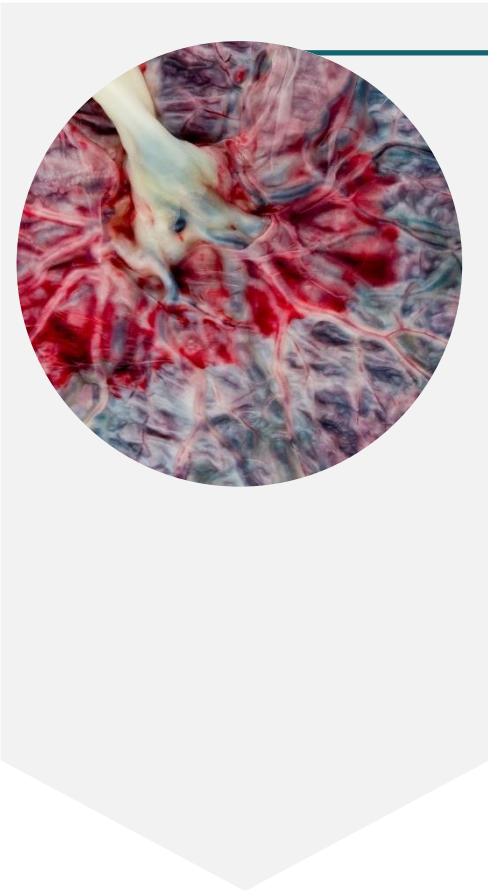
CLINICAL PLAN

- 2Q21: IND Submission
- 3Q21: Phase I/IIa Trial Start
- 2Q22: Pivotal Phase II Study Start

Degenerative Diseases

ALLOGENEIC PLACENTAL PLURIPOTENT CELLS: SCALABLE & OFF-THE-SHELF

Clinical Stage



One Placenta > 100,000 Doses

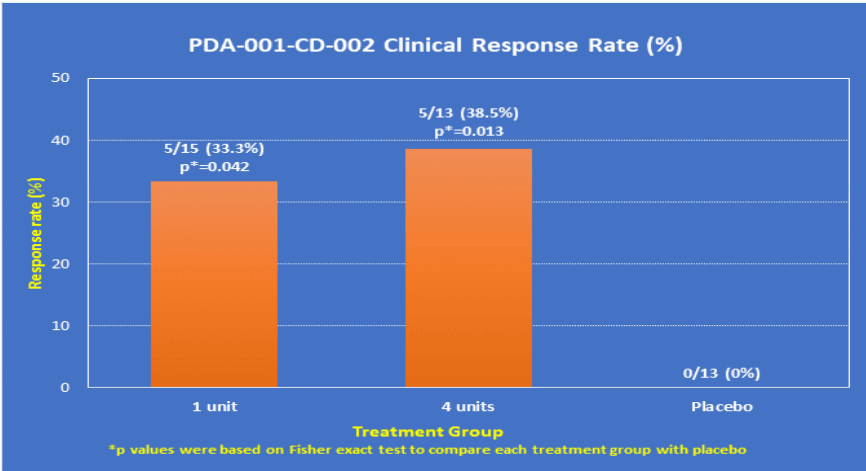
NEWLY DEVELOPED APPL PROGRAM

Leveraging Legacy Placental Mesenchymal-like Stromal Cell Studies to Expand to Degenerative Diseases



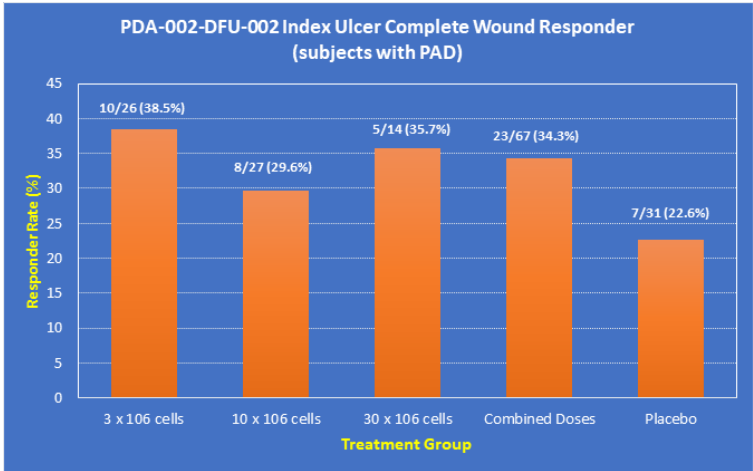
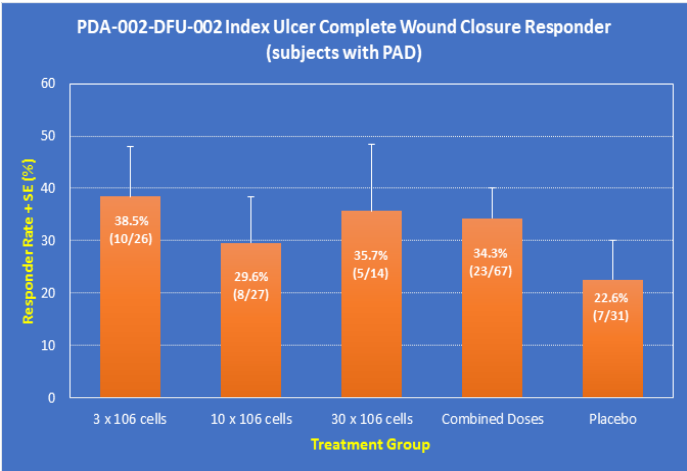
IV Formulation

- 50+ patients dosed in **multiple Crohn's Disease studies**
- Clinical response rates were significantly higher in IV Formulation treatment groups compared with the placebo group
 - Response rates were 43% points in the treatment group vs 0% in the placebo group on Day 365
 - Well-tolerated, no SAEs at therapeutic dose



IM Formulation

- 140+ patients dosed in **Diabetic Foot Ulcer (DFU) and Diabetic Peripheral Neuropathy Ph II studies**
- IM Formulation has systemic microvascular/neovascularization effects
 - Enhanced healing of diabetic foot ulcers compared to placebo
 - Improvement of retinopathy
 - Well-tolerated, no SAEs at therapeutic dose



NEWLY DEVELOPED APPL PROGRAM

Leveraging PDA Cells and Develop New APPL Candidate

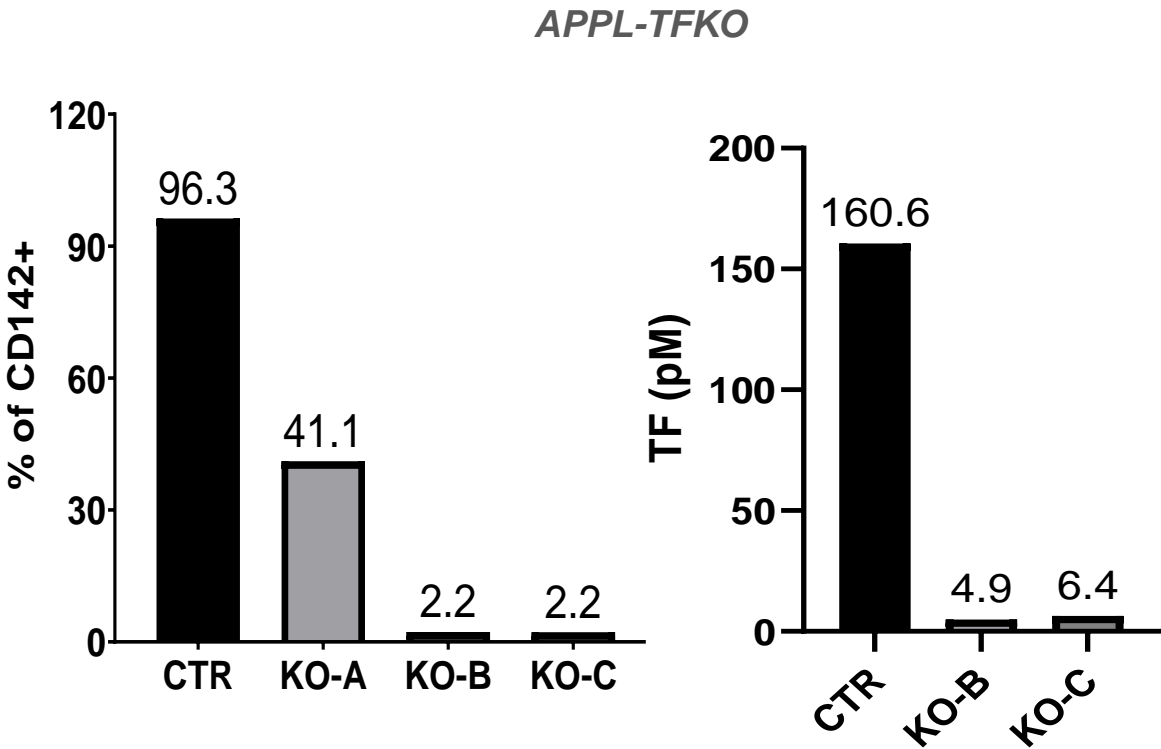


Genetically Modified APPL with Greater Safety Profile

- Tissue factor (TF) Knockout (KO) in APPL using CRISPR/Cas9 to reduce potential safety risk associated with TF
- Identified two of four CRISPR guide RNAs showing >95% high KO efficiency
- Demonstrated sustained TF KO throughout culture expansion
- APPL-TFKO cells significantly reduced TF activity
- TF KO showed no effect on cell proliferation and viability

Novel Media and Culture Method Established to Develop APPL with Greater Potency

- Demonstrate immune modulation and regenerative functionality
- New IP opportunities in process and product composition



APPL IN CROHN'S DISEASE

Summary



KEY TAKEAWAYS

- Culture-expanded, undifferentiated mesenchymal-like stromal cells
 - Genetically modified with tissue factor (TF) knockout (KO)
- Mechanism of Action:
 - Immune-modulatory, anti-inflammatory, pro-angiogenic, cytoprotective and pro-regenerative

CLINICAL PLAN

- 2H21: IND Submission
- 1H22: Phase I/IIa Trial

Transaction Summary



TERMS OF TRANSACTION

Overview



Transaction Summary

- Celularity Inc. (“Celularity”) intends to combine with GX Acquisition Corp. (“GX”, NASDAQ: GXGX) pursuant to a merger agreement and plan of reorganization (the “Merger Agreement”)
 - Celularity is a clinical-stage biopharmaceutical company that is leveraging the unique biology and availability of the placenta to deliver off-the-shelf allogeneic cellular therapies at unparalleled scale and quality with competitive economics
 - GX is a special purpose acquisition company whose sponsor, GX Sponsor LLC, is managed by the principals of Trimaran Capital Partners
- The transaction values Celularity’s equity at \$1.25bn
- The transaction will be supported by a PIPE placement of ~\$80 million¹. The implied post-transaction equity value at \$10 / share and assuming all warrants remain outstanding at close, no redemptions from GX public stockholders and PIPE proceeds of ~\$80mm will be ~\$1.7bn.
- Transaction expected to close in Q2 2021

Robust, Long-Term Investor Base

- Pursuant to the Merger Agreement, all existing Celularity stockholders will roll their equity into the newly-formed public company
- Strong investor group to support the transaction via participation in the PIPE, including affiliates of Starr Insurance Companies, Dragasac Limited, Sorrento Therapeutics and other unaffiliated institutional investors

Use of Proceeds

- As of 12/31/20 and pro forma for the business combination, the company is expected to have ~\$375mm in cash assuming a PIPE placement of ~\$80 million and no GX stockholder redemptions
 - Proceeds will be used fund Celularity’s operations into 2024, including R&D efforts and the clinical development and commercialization of the placental CAR-T (CyCART-19), unmodified NK (CYNK-001), genetically modified NK (CYNK-101) and allogeneic placental pluripotent cell (APPL) programs
 - Proceeds will also be used to pay Celularity’s transaction expenses and GX’s expenses

Management & Board

- Company to be led by Celularity’s existing senior management team
- Company’s directors to include two GX designees and one mutually agreed upon independent director, with remaining directors designated by Celularity

TERMS OF TRANSACTION

Pro Forma Valuation and Ownership



Illustrative Pro Forma Valuation

\$mm, except per share; mm shares

Price per Share (illustrative)	\$	10.00
<i>Pro Forma Fully Diluted Shares Outstanding</i>		167
Pro Forma Equity Value	\$	1,668
Estimated standalone Celularity cash		(53)
Cash to Balance Sheet from Business Combination		(322)
Estimated pro forma debt at close		-
Pro Forma Enterprise Value	\$	1,293

Sources of Funds

\$mm

Cash Held in Trust (12/31/20E)	\$	292
PIPE Proceeds		80
Celularity Shareholder Equity Rollover		1,250
Total Sources of Funds	\$	1,622

Pro Forma Ownership

	Shares (mm)	% Ownership
Current Celularity shareholders, optionholders and warrant holders	123	74%
Public GX Shareholders	29	17%
GX Sponsor	7	4%
PIPE Investors	8	5%
Total	167	100%

Uses of Funds

\$mm

Equity Issued to Celularity shareholders, optionholders and warrant holders	\$	1,250
Cash to Balance Sheet		322
Estimated Transaction Fees & Expenses		50
Total Uses of Funds	\$	1,622

USE OF PROCEEDS

Transaction Overview



- Approximately \$375 million¹ of cash as of 12/31/20, pro forma for the business combination, projected on the combined company balance sheet to pursue Celularity's research and development programs
 - Expected to provide cash runway into 2024, based on management's current clinical development assumptions
- Projected proceeds will be primarily used to fund Celularity's research and development programs, including:
 - Approximately \$20 – \$30 million to fund Phase 1 and Phase 2 pivotal trials for its CyCART-19 program in relapsed refractory B-cell NHL
 - Approximately \$30 – \$40 million to fund Phase 1 and Phase 2 pivotal trials for its CYNK-001 program in MRD+ AML
 - Approximately \$40 – \$60 million to fund Phase 1 and Phase 2 for its CYNK-001 program in Glioblastoma Multiforme
 - Approximately \$80 – \$100 million to fund Phase 1 and Phase 2 pivotal trials for its CYNK-101 program in Gastroesophageal Junction / Gastric HER2+ Adenocarcinoma
 - Approximately \$20 – \$30 million to fund Phase 1/2a pivotal trial for its APPL-001 program in Crohn's Disease

NEAR-TERM MILESTONES

To Achieve the Next Advance in Placenta-based Cell Therapy



Achievements to Date

- June 2019:** Submitted IND for CYNK-001 in AML
- December 2019:** Completion of Phase 1/2 (manufacturing) at Florham Park
- January 2020:** Received FDA Safe to proceed on IND for CYNK-001 in GBM
- March 2020:** Completed \$100mm Series B-1 financing
- April 2020:** Received FDA Safe to proceed on IND for CYNK-001 in COVID-19
- Expanded collaboration with Lung Biotechnology for CYNK-001 to include COVID-19 and ARDS indications
- September 2020:** Completion of Facility at Florham Park

Key Near-Term Development Milestones

CyCART-19

- 1H21: IND Submission Expected
- 3Q21: Phase I Study Start

CYNK-001

- 1H21: End of Phase I Meeting with FDA (AML)
- 1H21: Phase II Study Start (AML)
- 2H21: Pivotal Phase II Trial (GBM)

CYNK-101

- 1H21: IND Submission
- 3Q21: Phase I/IIa Trial Start

APPL-001

- 2H21: IND Submission

Appendix

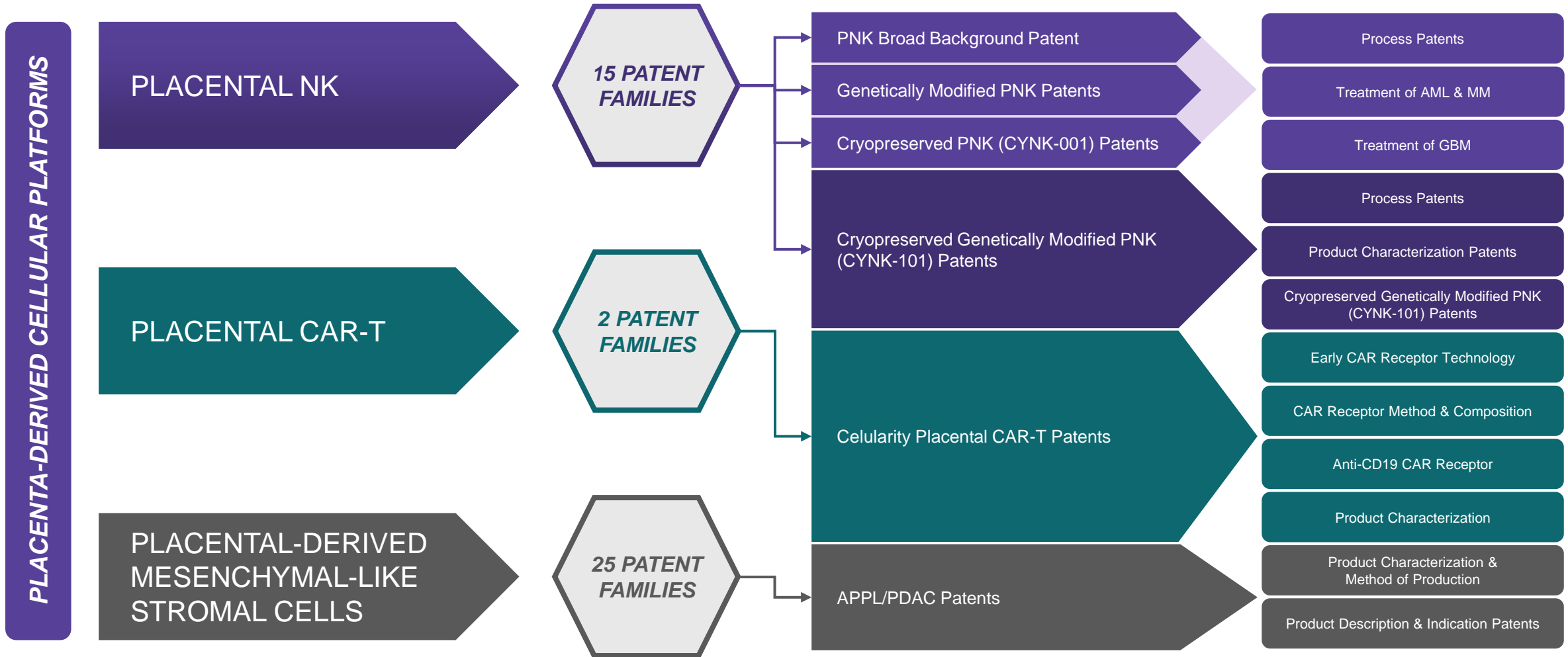
Clinical Programs

Additional Detail



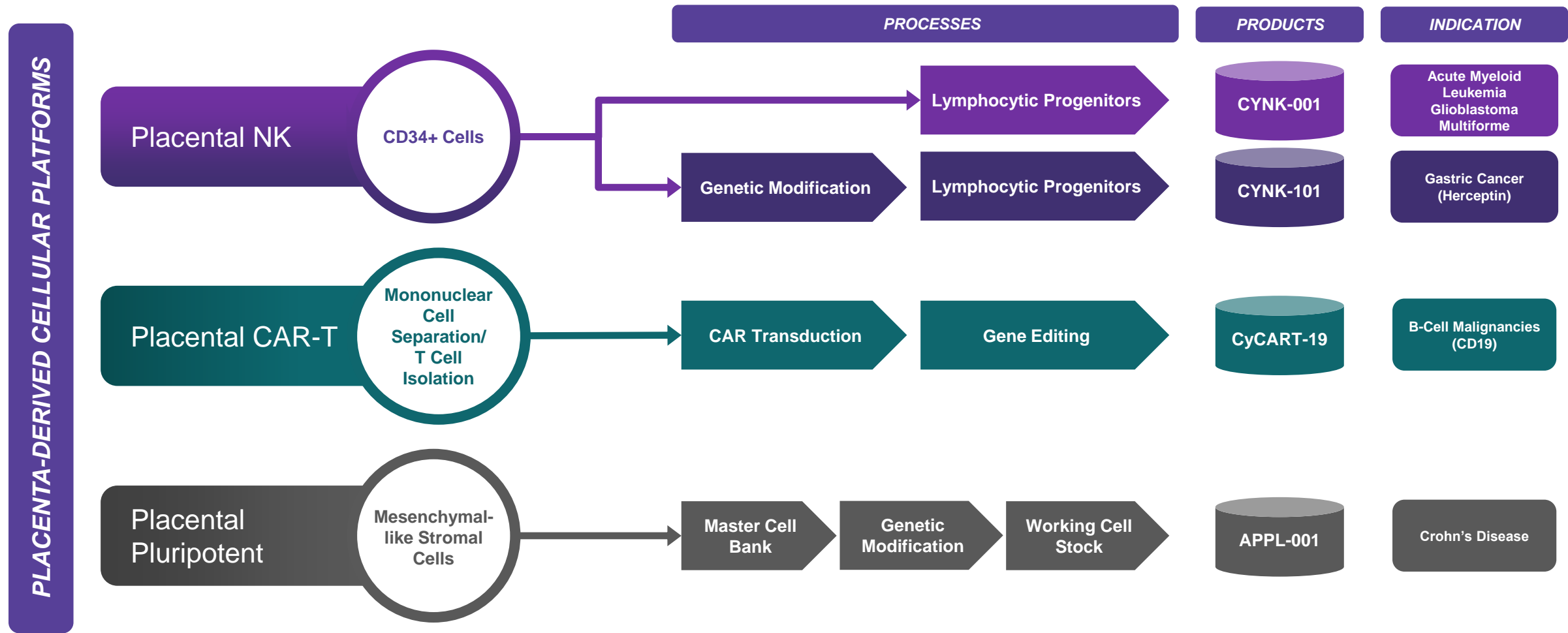
CELULARITY IMPACT™ PLATFORM

Broad IP Protection Across All Lead Programs



CELULARITY IMPACT™ PLATFORM

The Placenta as a Renewable Allogeneic Source, with Purpose-Built Commercial Scale Manufacturing



CELULARITY PLACENTAL CAR-T (CyCART)

Solving the Downside of Autologous CAR-T Therapies



AUTOLOGOUS CAR-T THERAPY		ALLOGENEIC PLACENTAL CAR-T
Status Quo	Downside	Celularity's Scalable Solution
<ul style="list-style-type: none"> ➤ All CAR T-cell therapies on the market and most (~75%) of clinical assets are autologous 	<ul style="list-style-type: none"> ▪ Complex, high COGS manufacturing and <i>one-batch, one-patient</i> supply chain 	<ul style="list-style-type: none"> ✓ No apheresis capacity constraints ✓ High volume manufacturing ✓ On-demand, off-the-shelf cryopreserved packaged product
<ul style="list-style-type: none"> ➤ Peripheral blood-derived T-cell is the immune cell 'vehicle' used to express a CAR 	<ul style="list-style-type: none"> ▪ Multiple rounds of lymphocyte-depleting therapies cause inconsistent apheresis cell recovery in relapsed or refractory patients 	<ul style="list-style-type: none"> ✓ Placentas provide a profuse, renewable source of healthy, ready to use lymphocytes ✓ Placental T-Cells containing abundance of stem cell memory conferring greater expansion and persistence potential
<ul style="list-style-type: none"> ➤ "Patient as their own donor" automatically makes the patient part of the supply chain 	<ul style="list-style-type: none"> ▪ Therapeutic outcomes affected by <i>collection-manufacturing-release-administration</i> timeframe "Long vein-to-vein time" 	<div>UNIQUE ADVANTAGES OF PLACENTAL-DERIVED CELLS</div> <ul style="list-style-type: none"> ✓ Dynamic & flexible supply chain ✓ Patient-responsive, not patient-dependent ✓ Simplified logistics, ability to pre-position cryopreserved product at treatment sites

CELULARITY PLACENTAL CAR-T (CyCART)

Providing Upside to Adult-donor Allogeneic CAR-T Therapies



ALLOGENEIC CAR-T THERAPY		ALLOGENEIC PLACENTAL CAR-T
Status Quo	Downside	Celularity's Scalable Solution
<ul style="list-style-type: none"> ➤ Requires selection, screening & testing T cells from healthy adult donors e.g. donor bone marrow 	<ul style="list-style-type: none"> ▪ Complex logistics, multistep manufacturing process to source, limited scalability, improved speed vs. autologous but still measured in days 	<ul style="list-style-type: none"> ✓ No apheresis capacity constraints ✓ High volume manufacturing ✓ On-demand, off-the-shelf cryopreserved packaged product
<ul style="list-style-type: none"> ➤ High cost of treatment inherent of engineered T cell therapy 	<ul style="list-style-type: none"> ▪ Requires separate engineering for each new therapeutic candidate 	<ul style="list-style-type: none"> ✓ Placentas provide an abundant, renewable source of healthy, ready to use lymphocytes ✓ Placental T-Cells containing abundance of stem cell memory conferring greater expansion and persistence potential
<ul style="list-style-type: none"> ➤ Adult donor ≠ universal donor 	<ul style="list-style-type: none"> ▪ Potential safety complications observed from graft versus host disease (GvHD), as well as CRS and cerebral edema 	<div>UNIQUE ADVANTAGES OF PLACENTAL-DERIVED CELLS</div> <ul style="list-style-type: none"> ✓ Dynamic & flexible supply chain ✓ Patient-responsive, not patient-dependent ✓ Simplified logistics, ability to pre-position cryopreserved product at treatment sites

CELULARITY PLACENTAL NK CELLS

Providing Upside to both Adult-donor NK Cells



ADULT DONOR NK CELL THERAPY

Peripheral Blood NK

iPSC NK

ALLOGENEIC PLACENTAL NK

Celularity's Scalable Solution

- Apheresis of peripheral blood from healthy donor / patient
- Requires voluntary donor

- De-differentiated adult fibroblasts
- Additional processing required

- ✓ No apheresis capacity constraints
- ✓ High volume manufacturing
- ✓ On-demand, off-the-shelf cryopreserved packaged product

- Cytokine activation without expansion or direct expansion on feeder cell platform

- Two-stage differentiation:
 - First from iPSC's to iCD34 cells, and then to NK cells
- Expression of multiple de-differentiation genes higher risk of insertional mutagenesis

- ✓ Feeder cell-free, cytokine cocktail-based NK cell expansion and differentiation
- ✓ No prior exposure to physiological or environmental factors; no exhaustion

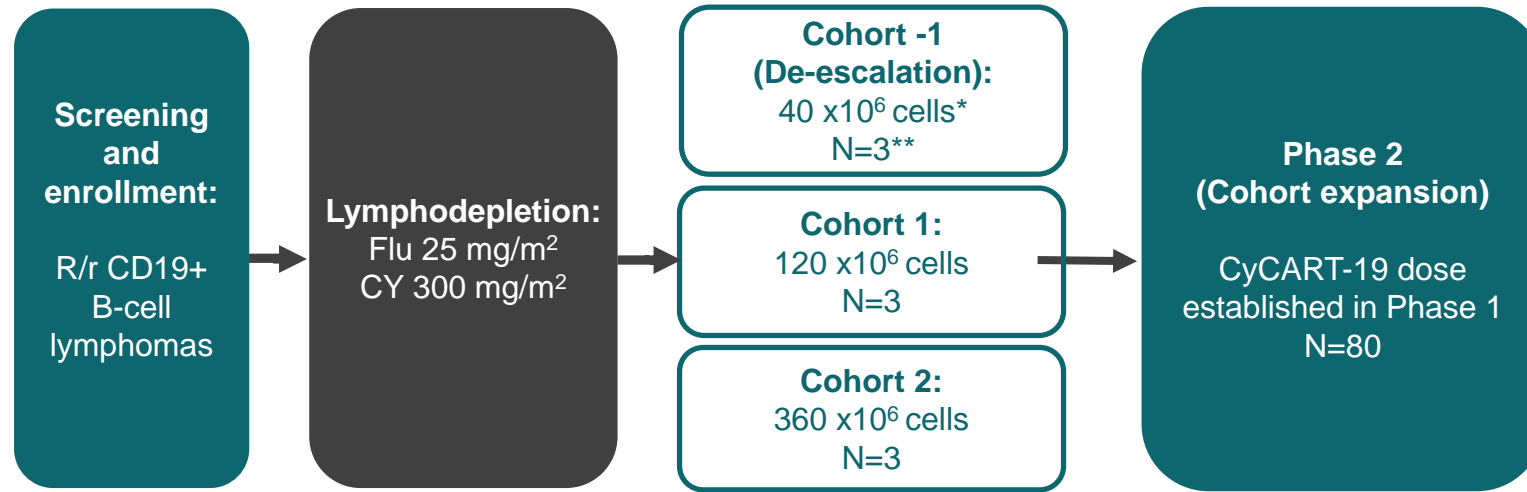
- Heterogeneous NK cells with high expression of both NK cell activating receptors and inhibitory receptors (KIRs)
- Potential for fratricide exists with CD38 mAb

- NK cells with high expression of both NK cell activation and inhibitory receptors (KIRs)
- Potential for fratricide necessitated knock out of CD38 when combined with CD38 mAb

- ✓ Heterogeneous NK cells with high expression of natural cytotoxicity receptors (NCRs) with low expression of inhibitory receptors (KIRs)
- ✓ No fratricide observed in combination with CD38 targeted approaches

CyCART-19 IN R/R B-CELL NHL

Phase I/II Study Design



*Cells= Transduced, viable CyCART-19 cells. **3+3 Design; N up to 6 per Cohort

Projected Timeline/Key Assumptions:

- Q2 2021: IND Submission
- Q3 2021: Phase I Study Start
 - 6-month: Dose Finding
- Q1 2022: Phase II Start
 - 9-month Accrual
 - 6-month Follow-up
 - 6 months: Preparation for Filing

PHASE 1 STUDY (SAFETY AND DOSE FINDING)

- Three dose cohorts (40, 120 and 360 x10⁶ transduced, viable CAR-T cells)
- 3+3 design
- N=up to 18
- **Primary Endpoints:** Determine safety and maximum tolerated dose
- **Secondary Endpoints:** ORR (CR+PR), DOR, PFS, OS
- *Exploratory Endpoints: Persistence of CyCART-19*

PHASE 2 (EFFICACY)

- CyCART-19 dose from Phase 1 Cohort study
- N=80
- **Primary Endpoints: Determine ORR at(CR+PR)**
- **Secondary Endpoints:** Safety, Time to response, DOR, PFS, OS
- *Exploratory Endpoints: Persistence of CyCART-19*

NK CELL THERAPY FOR CANCER IMMUNOTHERAPY

Preclinical & Clinical Data Supporting Role of NK cells in the Treatment of Cancer

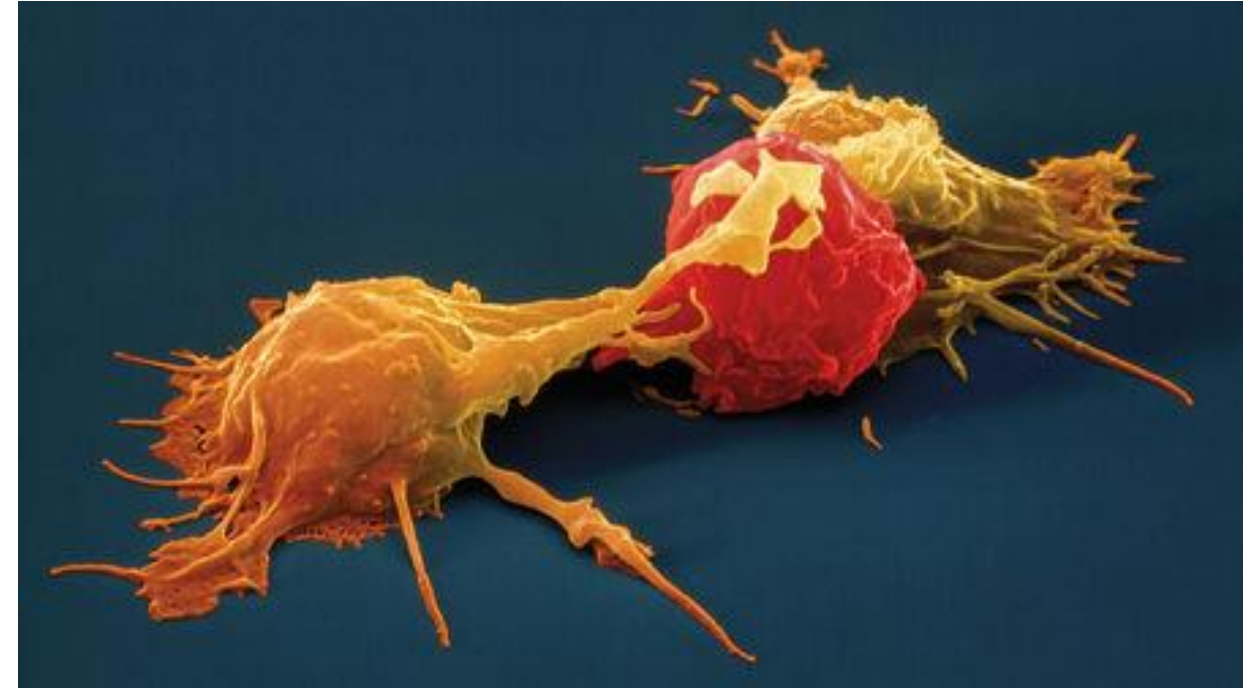


NK CELLS ARE A MAJOR COMPONENT OF THE INNATE IMMUNE SYSTEM

- Natural immune cells that eradicate both cancer and virus-infected cells
 - Directly via cytolytic granule mediated lysis
 - Indirectly via secretion of immunoregulatory cytokines (e.g. IFN-g)

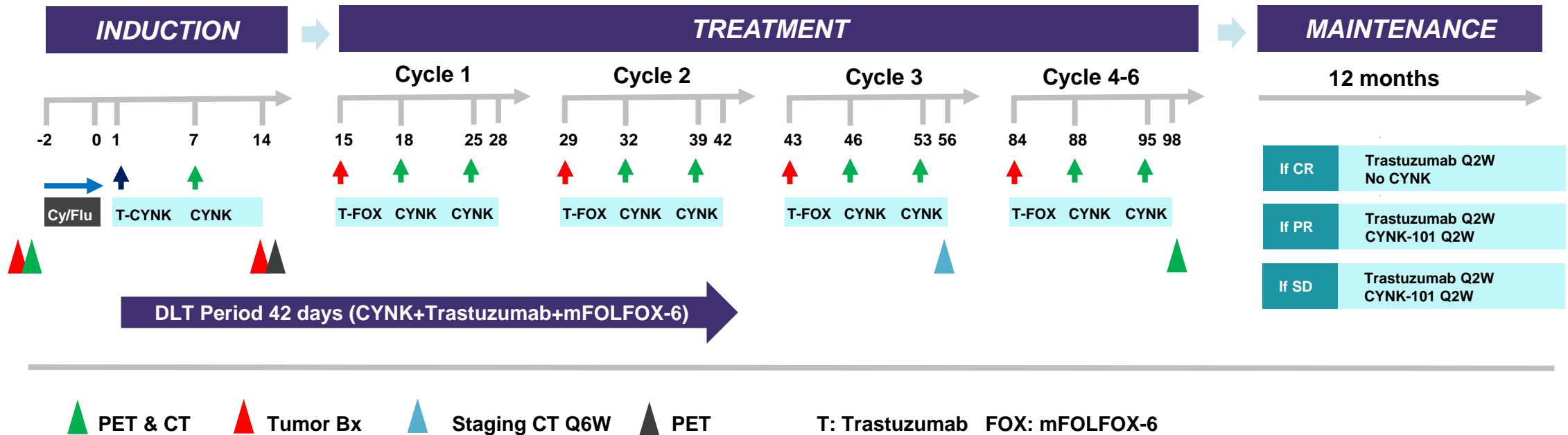
NK CELL ACTIVITY IS THERAPEUTICALLY RELEVANT

- Kills cancer cells (e.g., leukemic blasts) without prior sensitization, in a non-MHC restricted or tumor antigen-restricted manner
- Key mediators of ADCC (e.g. Rituximab, Cetuximab)
- Defective NK cell number & function has been linked to increased cancer risk and tumor development
- NK cell activity inversely correlated to relapse (anti-metastatic)
- NK cells infiltration predicts immune checkpoint blockade responsiveness



CYNK-101 PHASE 1/2A TRIAL

Treatment Schedule



Biopsies (2) - at the screening, after completing induction (before starting chemotherapy),

PET Scans (3) - at screening, after induction phase and before starting maintenance,

Planned interim efficacy analyses:

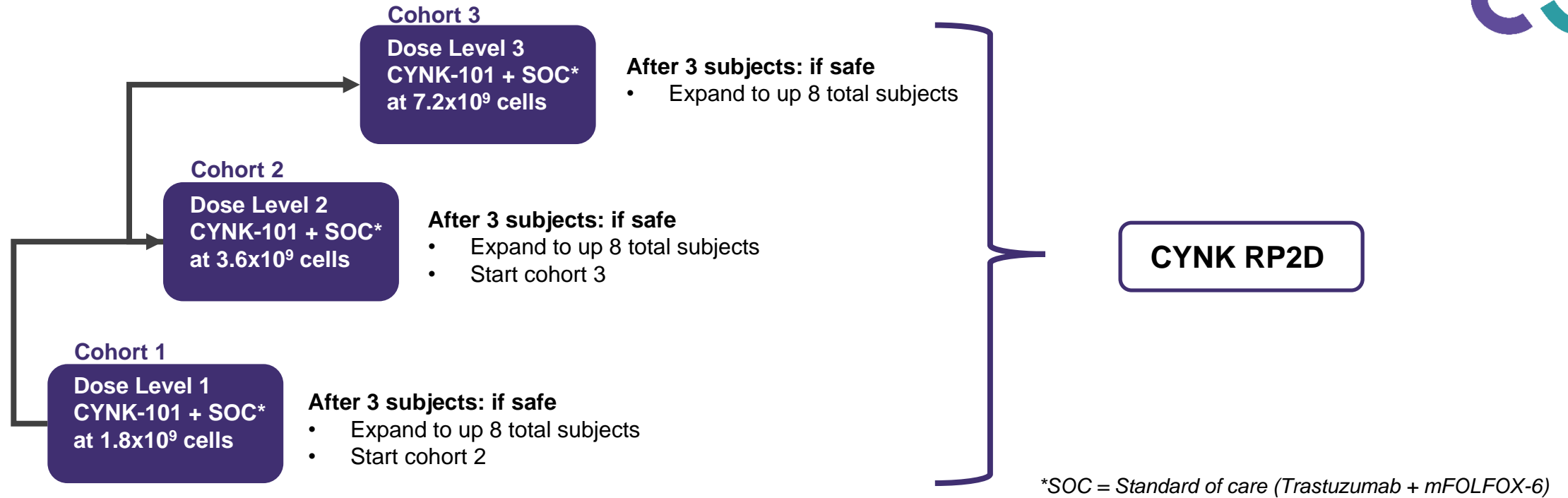
- After induction
- After 3 cycles
- After 6 cycles

Planned primary efficacy analysis – 6 months from starting treatment



CYNK-101 PHASE 1/2A TRIAL

Design



PHASE 1/2A (SAFETY AND FEASIBILITY)

- **CYNK-101 + SOC (Trastuzumab + mFOLFOX-6)**
- Dose escalation (3+3) with DLT period of 42 days followed by expansion up to 8 subjects per dose
- N=24
- HER2+ GEJ/Gastric adenocarcinoma
- Trastuzumab naïve
- General objective: to establish MTD and recommended phase 2 dose (RP2D)

ENDPOINTS/ASSUMPTIONS:

- **Primary Endpoint:**
 - Phase 1 portion: safety (MTD), CR rate
 - Phase 2a portion (expansion): PFS (6m), CR Rate
- **Secondary Endpoints:** ORR, DOR, PFS, mOS, safety, pharmacodynamics/translational
- **Assumptions for primary analysis at 6 months:**
 - PFS at 6 month - 75% or better
 - CR Rate – 25% or better

CYNK-101 PHASE 1/2A TRIAL

Timeline



	Phase 1/2a
Label Indication	Previously untreated HER2+ metastatic GEJ/Gastric Adenocarcinoma
Target Patient Population	Previously untreated subjects with metastatic or advanced unresectable gastroesophageal junction (GEJ) or gastric adenocarcinoma over-expressing HER-2
Patient Enrollment	<ul style="list-style-type: none"> 3 cohorts, 24 subjects total – for phase 1/2a
Primary Endpoint	<ul style="list-style-type: none"> mPFS (6m), CR Rate
Secondary Endpoints	<ul style="list-style-type: none"> Overall Response Rate as measured by RECIST 1.1, Duration of Response (DoR), mPFS, mOS and safety
Trial Duration	<ul style="list-style-type: none"> 10 months accrual , 6 months follow-up for efficacy
Logistics	<ul style="list-style-type: none"> North America, 10 sites
Data Availability	<ul style="list-style-type: none"> 2022 Q2 (1st interim data) 2022 Q4 (Final)

APPL CROHN'S DISEASE (CD)

Pivotal Study Design



Study Design: Randomized, double-blind, Placebo-controlled study in adults with 5 doses of 1/4th unit APPL (~ 37 million cells) over 8 weeks vs. Humira treatment.

Study Population: Moderate-to-Severe CD (CDAI score: 220-450) who are refractory to Corticosteroids

Primary objective: To assess the clinical efficacy by measuring response/remission rates during the induction phase as well as to explore durability of response during the maintenance phase in subjects with moderate to severe CD. Subjects shall be re-treated if a flare is developed during the 1-year.

Secondary Objective: The secondary objectives of this study are to assess clinical improvement by endoscopic measurements and quality of life assessments.

Primary Endpoint: To assess clinical efficacy, the modified Crohn's Disease Activity Index (CDAI) scoring system will be used to measure the following:

- Clinical Remission: Reduction of CDAI score to less than 150 points 4-6 weeks
- Clinical Remission: Reduction of CDAI score to less than 150 points 1-year

Secondary Endpoints:

- Clinical Response Rate: Reduction in CDAI score by 100 points to the baseline at 1-year
- Evaluation of mucosal healing as measured by Simple Endoscopic Score for Crohn's Disease (SES-CD) at week 4-6 and 1-year
- Patient-reported outcome of quality of life as measured by Inflammatory Bowel Disease Questionnaire (IBDQ)

Sample Size: 162 subjects in each arm (80% power and 10% drop out) involving APPL versus Humira with NI margin of 12%

Timeline Estimate:

- IND: 1H 2022



celularity