Clinical Stage Cell Therapy for AL Amyloidosis and Other Serious Diseases

September 2025

IMMiX BIOPHARMA

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Pioneering Cell Therapy in AL Amyloidosis and Other Serious Diseases

Sterically-optimized, proprietary CAR-T construct from Immix N-GENIUS platform

- Immix N-GENIUS platform produced NXC-201
- NXC-201 is our lead, sterically-optimized CAR-T with "digital filter" that reduces non-specific activation (enhancing tolerability)
- NXC-201 CAR-T construct provides barrier to entry

Dedicated team for NXC-201 in AL Amyloidosis and other serious diseases

- Ex-NCI/NIH scientists designed cell therapy for benign tolerability, being developed by Immix (licensed from Israel)
- Senior regulatory team with multiple BLAs at Pfizer/BMS
- Scientific advisors from Stanford, Memorial Sloan Kettering, Columbia, Tufts, UCLA
- Experienced management and Board of Directors

Sizable AL Amyloidosis market

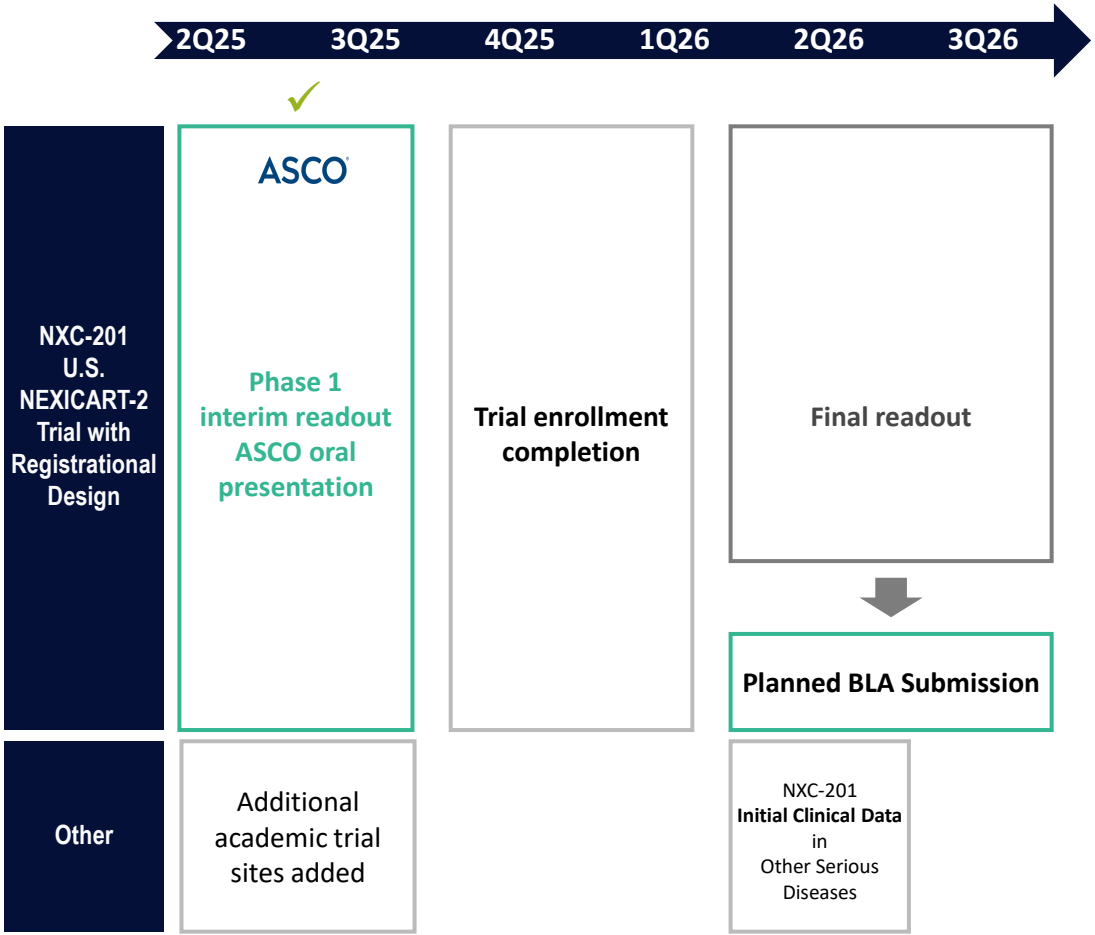
- Relapsed/refractory AL Amyloidosis target market: 34,600 U.S. patient prevalence (multi billion $ value)
- Established billing code establishes pricing floor for BCMA CAR-T at $425,000 per dose
- No drugs currently FDA approved in relapsed/refractory AL Amyloidosis

NXC-201: The only CAR-T in development for AL amyloidosis

- We believe NXC-201 high complete response rates to-date significantly improve treatment options for relapsed/refractory AL Amyloidosis patients (compared to real-world 0-10% complete response rates in r/r AL)
- ASCO oral presentation of interim results for NEXICART-2 Phase1/2 clinical trial with registrational design

Source:. E Lebel et al. Efficacy and Safety of Anti-BCMA Chimeric Antigen Receptor T-Cell (CART) for the Treatment of Relapsed and Refractory AL Amyloidosis. Presentation. ASH 2024. M. Assayag, et al. Asherie N, et al. Development and manufacture of novel locally produced anti-BCMA CAR T cells for the treatment of relapsed/refractory multiple myeloma: results from a phase I clinical trial. Haematologica. 2023 Jul 1;108(7):1827-1839. doi: 10.3324/haematol.2022.281628. PMID: 36200421; PMCID: PMC10316256. Quock TP, et al. Epidemiology of AL amyloidosis: a real-world study using US claims data. Blood Adv. 2018 May 22;2(10):1046-1053. doi: 10.1182/bloodadvances.2018016402. PMID: 29748430. Bazarbachi AH et al. Timing and outcomes of second-line therapy in the era of daratumumab-based frontline therapy in AL amyloidosis. Am J Hematol. 2024 Nov;99(11):2225-2228. doi: 10.1002/ajh.27450. Epub 2024 Aug 3. PMID: 39096115. Zanwar S, et al. Treatment patterns for AL amyloidosis after frontline daratumumab, bortezomib, cyclophosphamide, and dexamethasone treatment failures. Leukemia 2024.

Significant Near-Term Milestones



Prior

- ✓ Secured rights to NXC-201, N-GENIUS platform
- ✓ FDA **Orphan Drug Designation (ODD)** and **Regenerative Medicine Advanced Therapy (RMAT) Designation** Granted
- ✓ **Mentioned in New England Journal of Medicine (NEJM) AL Amyloidosis Review**
- ✓ Reported ex-U.S. NEXICART-1 AL Amyloidosis data at **ASGCT 2023**, **ASH 2023**, **ASGCT 2024, ASH 2024**, **JCO published 2024**
- ✓ NEXICART-2 U.S. AL Amyloidosis clinical trial first 6 patients dosed; first patient at Memorial Sloan Kettering Cancer Center (met guidance)
- ✓ Reported first 4 patients U.S. NEXICART-2 AL Amyloidosis clinical data 4Q 2024 (met guidance)
- ✓ Reported first 10 patients U.S. NEXICART-2 AL Amyloidosis clinical data Q2 2025 at ASCO 2025

ASCO

N-GENIUS Platform: Sterically-Optimized CAR-T construct “Digital Filter” reduces non-specific activation, leading to better tolerability

ALL BCMA CAR-TS ARE NOT CREATED EQUAL

N-GENIUS PLATFORM

NXC-201 sterically-optimized CAR-T’s “Digital Filter”…. **…reduces non-specific activation**

1 Proprietary Optimized CD3 – “CD3ζγ”

✓ Delivers “Digital” Intracellular Signaling

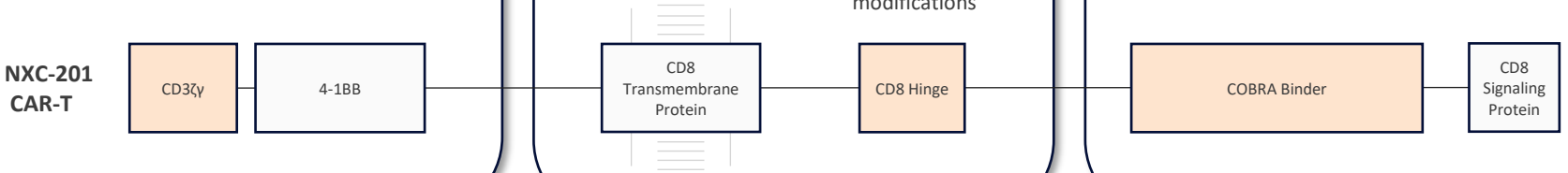
2 Proprietary Optimized CD8 Hinge Flexibility

✓ Reduces cytokine release

3 Proprietary Optimized COBRA Binder

✓ Enhances Plasma Cell Binding
✓ Ensures High Expression

Sterically-optimized key construct modifications



Source: M. Assayag, et al. Academic BCMA-CART cells (HBI0101), a promising approach for the treatment of LC Amyloidosis. 27th Annual Meeting of The American Society of Gene and Cell Therapy (ASGCT). Late Breaking Oral Presentation. Baltimore, MD. May, 2024. Feucht, M. Sadelain, et al. Calibration of CAR activation potential directs alternative T cell fates and therapeutic potency. Nature Medicine. 2019 Jan;25(1):82-88. doi: 10.1038/s41591-018-0290-5. Epub 2018 Dec 17. PMID: 30559421 PMCID: PMC6532069. O. Harush C. J. Cohen, et al. Preclinical evaluation and structural optimization of anti-BCMA CAR to target multiple myeloma. Haematologica. 2022 Oct 1;107(10):2395-2407. doi: 10.3324/haematol.2021.280169. PMID: 35354252 PMCID: PMC9521250. Adapted from PEGS 2021. Zanwar S, et al. Eyal Lebel et al., Efficacy and Safety of Anti–B-Cell Maturation Antigen Chimeric Antigen Receptor T-Cell for the Treatment of Relapsed and Refractory AL Amyloidosis. JCO. JCO-24-02252. DOI:10.1200/JCO-24-02252.

NXC-201 Referenced in June 2024 New England Journal of Medicine Publication

The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

Dan L. Longo, M.D., *Editor*

Systemic Light Chain Amyloidosis

Vaishali Sanchorawala, M.D.

TREATMENT OF RELAPSE AND PROGRESSION AFTER INITIAL THERAPY

No consensus has been established on the criteria for commencing second-line therapy in patients with progressive disease after initial therapy.[73,74] Patients with relapsed disease can be treated by repeating first-line therapy if the response lasted for more than a year, although such patients have a shorter time to relapse without a reduction in overall survival than patients who are treated with a different therapy for relapsed disease.

The potential options available for the treatment of relapsed systemic AL amyloidosis include proteasome inhibitors,[75,76] anti–CD-38 monoclonal antibodies,[77,78] immunomodulatory agents,[79] venetoclax for patients with t(11;14),[80] bendamustine,[81] high-dose melphalan with autologous SCT,[82,83] bispecific antibodies,[84,85] and even chimeric antigen receptor T-cell therapy.[86] Although it is not possible to be prescriptive regarding the sequencing of therapies, the two guiding considerations are the depth and duration of the initial response and the choice of a class of agents not previously used. The limitations imposed by a patient's reduced level of fitness or frailty and end-organ damage must also be considered. Enrollment in clinical trials is encouraged.

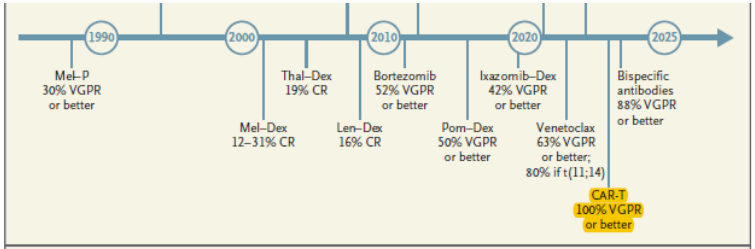


Figure 3. Therapeutic Landscape of AL Amyloidosis.

The therapeutic landscape of AL amyloidosis has seen substantial expansion in the past three decades. The majority of treatment regimens are adapted from myeloma therapies, with a focus on targeting the underlying plasma cell clone. Hematologic response has improved significantly with more effective and contemporary treatments, contributing to an overall increase in survival and a reduction in the rate of early death. CAR-T denotes chimeric antigen receptor T-cell therapy, CR complete hematologic response, CTD cyclophosphamide–thalidomide–dexamethasone, CyBorD cyclophosphamide–bortezomib–dexamethasone, HDM–SCT high-dose melphalan and stem-cell transplantation, Ixazomib–Dex ixazomib–dexamethasone, Len–Dex lenalidomide–dexamethasone, Mel–Dex melphalan–dexamethasone, Mel–P melphalan–prednisone, Pom–Dex pomalidomide–dexamethasone, Thal–Dex thalidomide–dexamethasone, and VGPR very good partial hematologic response.

tory AL amyloidosis: a multinational retrospective case series. Blood 2024;143:734-7.

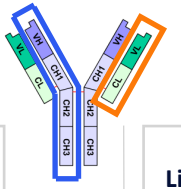
86. Kfir-Erenfeld S, Asherie N, Grisariu S, et al. Feasibility of a novel academic BCMA-CART (HBI0101) for the treatment of relapsed and refractory AL amyloidosis. Clin Cancer Res 2022;28:5156-66.

87. Nuvolone M, Nevone A, Merlini G. Targeting amyloid fibrils by passive immunotherapy in systemic amyloidosis.

Footer Sanchorawala et al. Systemic Light Chain Amyloidosis. New England Journal of Medicine. June 2024.

AL Amyloidosis: 34,600 Relapsed/Refractory U.S. Patients with No FDA Approved Drugs

NXC-201 TARGETS AL AMYLOIDOSIS PLASMA CELLS THAT EXPRESS BCMA ON CELL SURFACE

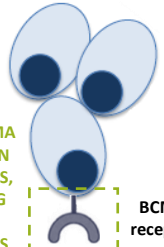


Light chains produced by dysfunctional plasma cells in bone marrow

Light chains misfold and aggregate

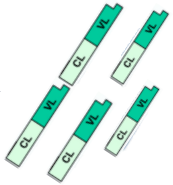
Light chains deposit in organs, damaging organs

Bone marrow plasma cells

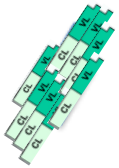


NXC-201 TARGETS BCMA RECEPTOR ON PLASMA CELLS, ELIMINATING SOURCE OF LIGHT CHAINS

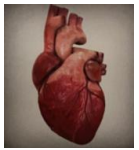
BCMA receptor



Light chains

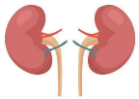


Sheet of misfolded light chains (amyloid)



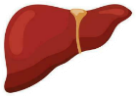
Heart

- Heart failure
- Dangerous, irregular rhythms
- Difficulty breathing
- Low blood pressure



Kidney

- Kidney failure
- Elevated protein in the urine
- Swelling of the limbs



Liver

- Enlarged liver
- Abnormal enzyme levels

Pre-existing heart failure caused by AL Amyloidosis

(immediately prior to heart transplant)

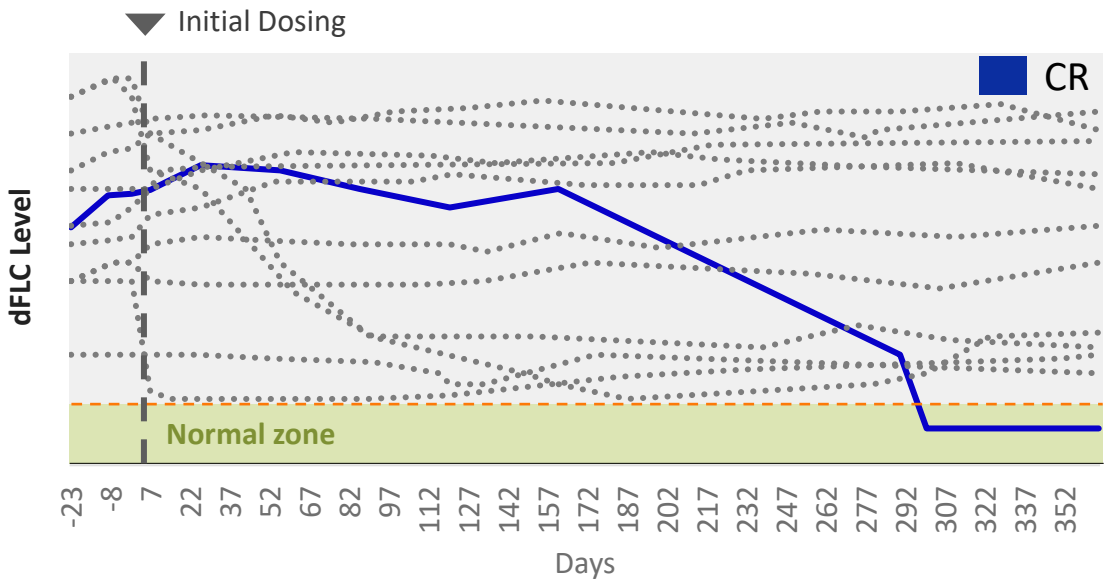
✓ Goal with NXC-201 is to treat R/R patients early enough to prevent reaching heart failure stage shown here

Bone marrow | **Circulation** | **Target tissues**

Source: Merlini, G., et al. Nat Rev Dis Primers. Oct 2018, Front. Cardiovasc. Med., Dec 2022, Hemato 2022, 3(1), 47-62; https://doi.org/10.3390/hemato3010005. Quock TP, Yan T, Chang E, Guthrie S, Broder MS. Epidemiology of AL amyloidosis: a real-world study using US claims data. Blood Adv. 2018 May 22;2(10):1046-1053. doi: 10.1182/bloodadvances.2018016402. PMID: 29748430; PMCID: PMC5965052.
Lu R, Richards TA. AL Amyloidosis: Unfolding a Complex Disease. J Adv Pract Oncol. 2019;10(8):813-825.

Standards of Care Produce 0-10% Complete Response Rate

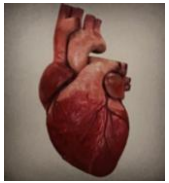
12 Patient Series Relapsed/Refractory AL Amyloidosis Receiving Second Line Therapy



This Is Heart Failure Caused by AL Amyloidosis

AL AMYLOIDOSIS CAUSES PHYSICALLY IRREVERSIBLE DISTORTION OF HEART STRUCTURE, SHAPE AND SIZE

Increased Mortality



Remission

There are no drugs approved in relapsed/refractory AL amyloidosis. Current investigators' choice agents produce an unsatisfactory reduction in AL amyloidosis disease markers (dFLC) with a low (0-10%) complete response (CR) rate

Note: Patient values are illustrative. R/R AL investigator's choice therapies included: Dara-VCd, Dara-Vd, Dara-VRd, Dara-Porn-Dex, Dara-Cd, Dara-Porn-Dex, Bendamustine-Dex

Source: Bazarbachi AH et al. Timing and outcomes of second-line therapy in the era of daratumumab-based frontline therapy in AL amyloidosis. Am J Hematol. 2024 Nov;99(11):2225-2228. doi: 10.1002/ajh.27450. Epub 2024 Aug 3. PMID: 39096115. Zanwar S, et al. Treatment patterns for AL amyloidosis after frontline daratumumab, bortezomib, cyclophosphamide, and dexamethasone treatment failures. Leukemia 2024.

NXC-201 Addresses Sizable U.S. Relapsed/Refractory AL Amyloidosis Patient Population

Prevalence: Relapsed/Refractory ("R/R")

Incidence: Newly Diagnosed / Front Line

Population

34,600

patients eligible for treatment with NXC-201 in the U.S.

Subtract 4% Cardiac stage 3b (not eligible for NXC-201)

Beginning prevalence 33,300 + 2,800 = ~36,100 U.S. total R/R patients

… Of which, 2,800 become R/R

Existing therapies

- ~35% of patients on Darzalex combos reach a CR — ~80% Darzalex combo eligible
- 12 - 21 months median duration on therapy
- 8% of all patients in long-term remission with ASCT (20%*40% = 8%) — ~20% ASCT eligible

~4,300 newly diagnosed

Blue Ocean Opportunity

- **0-10% complete response rate** for existing therapies in R/R AL
- No FDA Approved Drugs in Relapsed / Refractory AL Amyloidosis

Therapies

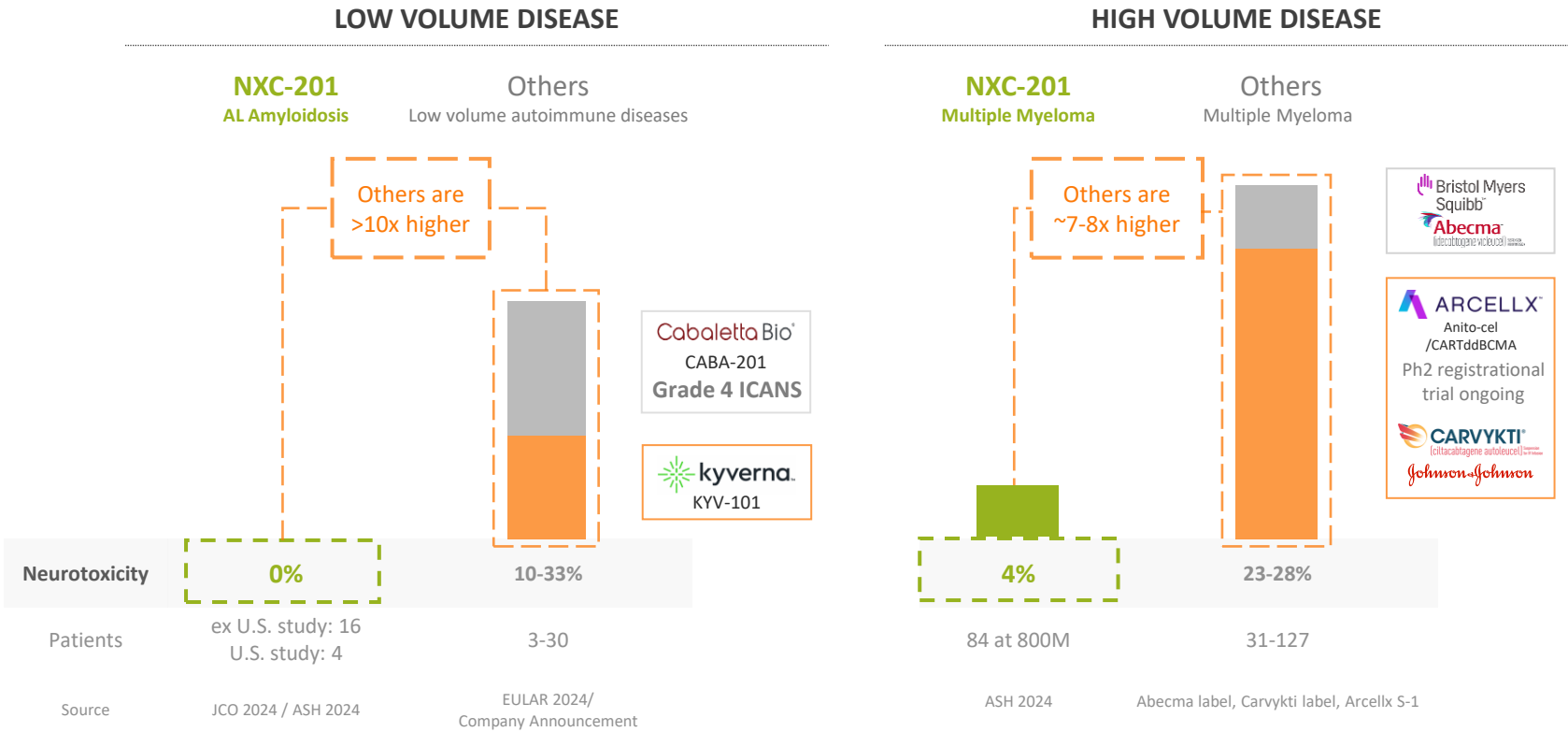
Front-line only	Approved
	DARZALEX (daratumumab) injection for intravenous infusion 100 mg/5 mL, 400 mg/20 mL

(Darzalex combined with cyclophosphamide, bortezomib, and/or dexamethasone)

Source: Incidence and prevalence: Quock T et al, Epidemiology of AL amyloidosis: a real-world study using US claims data. Blood 2018. Incidence growth rate: Laires P et al, Prevalence, Incidence, and Characterization of LIGHT Chain Amyloidosis in the USA: A Real-World Analysis Utilizing Electronic Health Records (EHR). Blood 2023. Daratumumab: Bellofiore C, et al. A real-life study of daratumumab combinations in newly diagnosed patients with light chain (AL) amyloidosis. Hematol Oncol. 2024. Chakraborty R et al, Reduced early mortality with Daratumumab-based frontline therapy in AL amyloidosis: A retrospective cohort study. AJH 2024. Bazarbachi AH et al. Timing and outcomes of second-line therapy in the era of daratumumab-based frontline therapy in AL amyloidosis. Am J Hematol. 2024 Nov;99(11):2225-2228. doi: 10.1002/ajh.27450. Epub 2024 Aug 3. PMID: 39096115. ASCT: Bomsztyk J et al, Recent guidelines for high-dose chemotherapy and autologous stem cell transplant for systemic AL amyloidosis: a practitioner's perspective. Expert Review of Hematology 2022. Gustine J et al, Predictors of hematologic response and survival with stem cell transplantation in AL amyloidosis: A 25-year longitudinal study. AJH 2022. Mayo staging: Zanwar S, et al. Treatment patterns for AL amyloidosis after frontline daratumumab, bortezomib, cyclophosphamide, and dexamethasone treatment failures. Leukemia 2024.

NXC-201 Advantage: Overcoming Neurotoxicity

ALL BCMA CAR-TS ARE NOT CREATED EQUAL



Source: Carvykti and Abecma FDA labels, Arcellx S-1. Assayag, et al. Academic BCMA-CART cells (HBI0101), a promising approach for the treatment of LC Amyloidosis. 27th Annual Meeting of The American Society of Gene and Cell Therapy (ASGCT). Late Breaking Oral Presentation. Baltimore, MD. May, 2024 Assayag, N., et al. European Society for Blood and Marrow Transplantation 49th Annual Meeting. Lebel E, et al. Efficacy and Safety of a Locally Produced Novel Anti-BCMA Chimeric Antigen Receptor T-Cell (CART) (HBI0101) for the Treatment of Relapsed and Refractory Multiple Myeloma, International Myeloma Society 20th Annual Meeting. 2023. Differences exist between trial designs and subject characteristics, and caution should be exercised when comparing data across studies. Figures reflect cross-trial comparison and not results from a head-to head study. Kyverna corporate presentation June 14, 2024. Accessed through https://www.sec.gov/ix?doc=/Archives/edgar/data/0001994702/000095017024073312/kytx-20240614.htm. Low volume diseases refers to ANCA vasculitis, autoimmune encephalitis, anti-synthetase syndrome, CIDP, DAGLA encephalitis, IgG4 related disease, Lambert-Eaton myasthenic syndrome, lupus nephritis, myasthenia gravis, multiple sclerosis, rheumatoid arthritis, systemic sclerosis, Stiff Person syndrome Cabaletta 2Q 2024 earnings press release: https://www.cabalettabio.com/investors/news-events/press-releases/detail/114/cabaletta-bio-reports-second-quarter-2024-financial-results. High volume disease NXC-201 CRS data from ASH 2024 Abstract which included 84 MM patients. NXC-201 data from NEXICART-1 clinical study.

NXC-201 Tolerability Drives AL Amyloidosis Leadership

ALL BCMA CAR-TS ARE NOT CREATED EQUAL

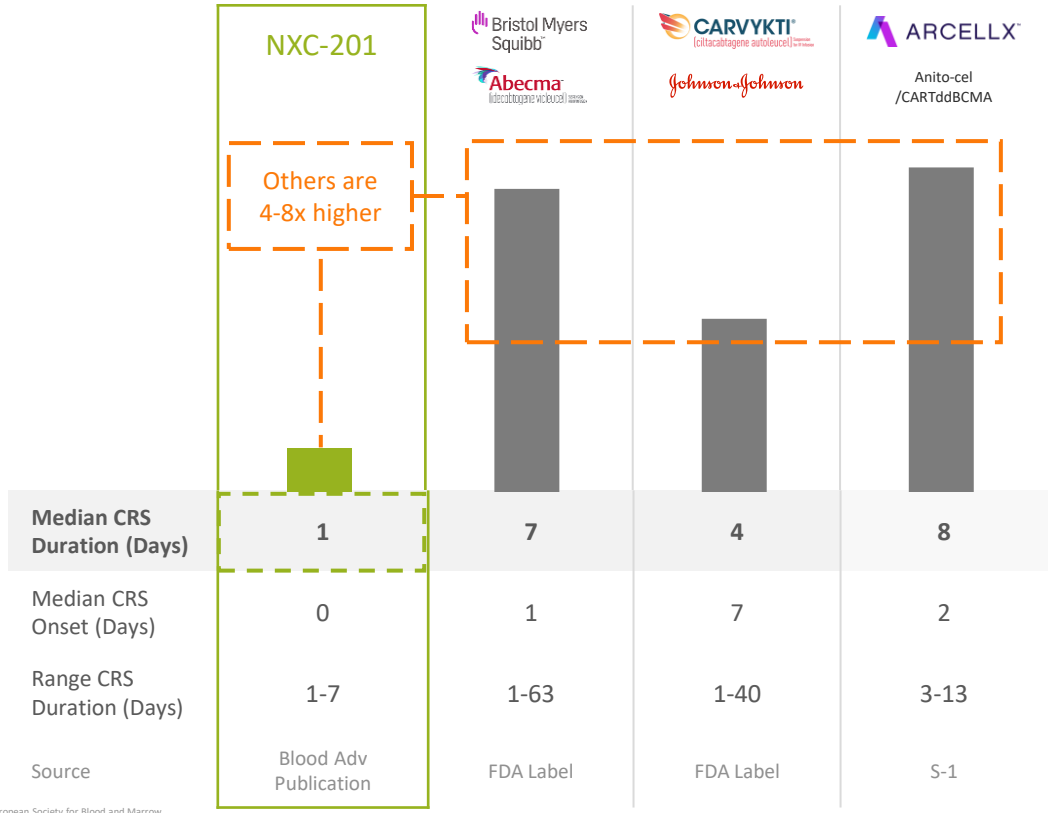
NXC-201's short CRS duration makes it **uniquely suitable to treat ALA patients** (in whom the #1 source of mortality is heart failure)

Cardiovascular stress is the key determinant for ability to treat relapsed/refractory ALA patients

- Long CRS duration causes extended cardiovascular stress
- Other CARTs have 4-8x longer CRS duration

"The biggest challenge … has been applicability of these therapies in amyloidosis **when the patients are particularly frail and have organ dysfunction** … where the key lies in the safety rather the efficacy in a low-volume disease setting is going to be key … "

– *Dr. Susan Bal, MD*
Assistant Professor, Hematology
University of Alabama at Birmingham



Data in Multiple Myeloma

Source: M. Assayag, et al. Point-of-care CART manufacture and delivery for the treatment of multiple myeloma and AL amyloidosis: the experience of Hadassah Medical Center. European Society for Blood and Marrow Transplantation 49th Annual Meeting. Poster Presentation. April 2023. Nov 2023 KOL discussion https://lifescievents.com/event/immixbio/;NXC-201 (formerly HBI0101) American Society of Hematology Presentation, Abecma FDA approval label, Carvykti FDA approval label, Arcelx S-1. NXC-201 data from NEXICART-1 clinical study.

Pipeline: Only CAR-T in AL Amyloidosis; Expanding To Other Serious Diseases

Lead Program: NXC-201, a next-generation BCMA-targeting CAR-T for AL Amyloidosis and Other Serious Diseases

Indication	Therapy	Pre-clinical	Phase 1	Phase 2	Upcoming Milestones
Relapsed/Refractory AL Amyloidosis	NXC-201	US FDA Regenerative Medicine Advanced Therapy (RMAT) and Orphan Drug Designation (ODD); EU EC ODD			✓ **2Q 2025:** Report interim clinical data readout for NEXICART-2 trial in relapsed/refractory AL Amyloidosis **4Q 2025 / 1Q 2026:** Planned NEXICART-2 enrollment completion **2Q/3Q 2026:** Report final topline clinical data readout for NEXICART-2 trial in AL Amyloidosis
Undisclosed select Other Serious Diseases	NXC-201	IND enabled			**2Q 2026:** Report NXC-201 interim clinical data in unaddressed immune-mediated diseases

Other Emerging Pipeline

Preclinical Candidates	Not yet announced				

World-Class Team

Management

Ilya Rachman, MD, PhD
Chief Executive Officer

Gabriel Morris, BA
Chief Financial Officer

Denise Bruns
Senior Regulatory Advisor

David Marks, MBBS, PhD
Chief Medical Officer

Gerhard Bauer
Head of Cell Therapy Manufacturing

Board of Directors

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Former Prometheus Biosciences Board Member

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GI Research Foundation

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Founder & Chairman, Rayliant Global Advisors

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Vaishali Sanchorawala, MD
Director, Amyloidosis Center

Marko Radic, PhD
Autoimmune CAR-T Pioneer

Suzanne Lentzsch, MD, PHD
Director, Multiple Myeloma and Amyloidosis

U.S. Multi-Center NEXICART-2 Trial Designed Based on ex-US NEXICART-1 Trial Experience

40 patient NEXICART-2 U.S. multi-center trial with registrational design

Relapsed/Refractory AL Amyloidosis NXC-201 trial	Excludes pre-existing severe cardiac patient enrollment?	Excludes patients with prior BCMA-targeted therapy exposure?	Excludes patients with concomitant Multiple Myeloma?
NEXICART-1: ongoing Israel trial	X No	X No	X No
NEXICART-2: ongoing US trial	✓ Yes	✓ Yes	✓ Yes

NEXICART-2: 40 patient, single-arm, multi-site, US trial → submit data to FDA

Source: Feasibility of a Novel Academic Anti-BCMA Chimeric Antigen Receptor T-Cell (CART) (HBI0101) for the Treatment of Relapsed and Refractory AL Amyloidosis, 65th ASH Annual Meeting and Exposition, San Diego, CA. October 2023. R. Chakraborty, et al. Safety and efficacy of teclistamab in systemic immunoglobulin light chain amyloidosis. Blood Cancer Journal. October 2023. N. Forgeard, et al. Teclistamab in relapsed or refractory AL amyloidosis, a multinational retrospective case series. Blood. February 2024. One NXC-201 relapsed/refractory AL amyloidosis patient died of COVID-19

NEXICART-2: Multi-Center U.S. CAR-T NXC-201 Clinical Trial with Registrational Design

IMMIX BIOPHARMA

NEXICART-2 U.S. Relapsed/Refractory AL Amyloidosis Trial (NCT06097832)

U.S. TRIAL WITH REGISTRATIONAL DESIGN ONGOING; PLANNED ENROLLMENT COMPLETION 4Q 2025 / 1Q 2026

Study design

- **Open-label, single-arm, multi-site phase 1/2 study**
- **n=40 patients**

Key criteria

Inclusion	• **AL Amyloidosis patients exposed to at least 1 line of therapy including a CD38 monoclonal antibody**
Exclusion	• **Prior anti-BCMA directed therapy** • **Cardiac: Mayo stage 3b, NYHA stage III/IV** • **Concomitant Multiple Myeloma**

Outcome measures

Phase 1

- **Safety**
- **Efficacy: Complete hematologic response (CR) based on validated criteria**

Phase 2

- **Efficacy: CR based on validated criteria in AL amyloidosis**
- **Safety**

Note: Complete Response according to consensus recommendations in AL amyloidosis (Palladini, et al. 2012. "Consensus guidelines for the conduct and reporting of clinical trials in systemic light-chain amyloidosis." Leukemia 26(11): 2317-2325.)

NEXICART-2 (U.S.): Patient enrollment focused on patients with preserved heart function at enrollment

Preserved heart function

	NX2-001	NX2-002	NX2-003	NX2-004	NX2-005	NX2-006	NX2-007	NX2-008	NX2-009	NX2-010	Median (range)
Age	56	67	82	64	62	72	77	66	63	80	67 (56-82)
Gender	Female	Female	Male	Female	Female	Male	Male	Male	Male	Male	-
Prior lines of therapy	4*	6**	2	4	4*	3	12*	4*	4*	3*	4 (2-12)
dFLC (mg/L)	65	24	-	86	42	26	47	121	84	-	56 (24-121)
M-spike (g/dL) ¥	-	-	0.79	-	-	-	-	-	-	0.65	-
Organ involvement	Heart	Heart/GI/ nerve	Kidney	Heart/GI	Kidney	Heart	Nerve	Heart	Heart	Kidney/ Heart	-
NYHA stage	I	II	I	I	I	I	I	II	I	II	-
NT-ProBNP (ng/L)	146	560	1,297	218	805	989	143	909	289	290	425 (143-1,297)
hs-Troponin-I (ng/L)	7	6	42	7	9	31	14†	47	6	52	9 (6-52)
Mayo Stage At Diagnosis	II	II	II	IIIa	I	IIIa	I	II	IIIb	IIIa	
At Enrollment	I	II	-	I	-	IIIa	-	IIIa	I	II	-
Creatinine (mg/dL)	0.7	1.1	2.2	1.8	2.7	0.8	1.3	0.8	0.9	0.9	1.0 (0.7-2.7)
Albuminuria (mg/24 hrs)	143	0	3,032	10	10,274	0	135	360	13	2,153	143 (0-10,274)

* Prior autologous stem cell transplantation (ASCT)
** Two prior ASCT
¥ M-spike value if used as measurable disease

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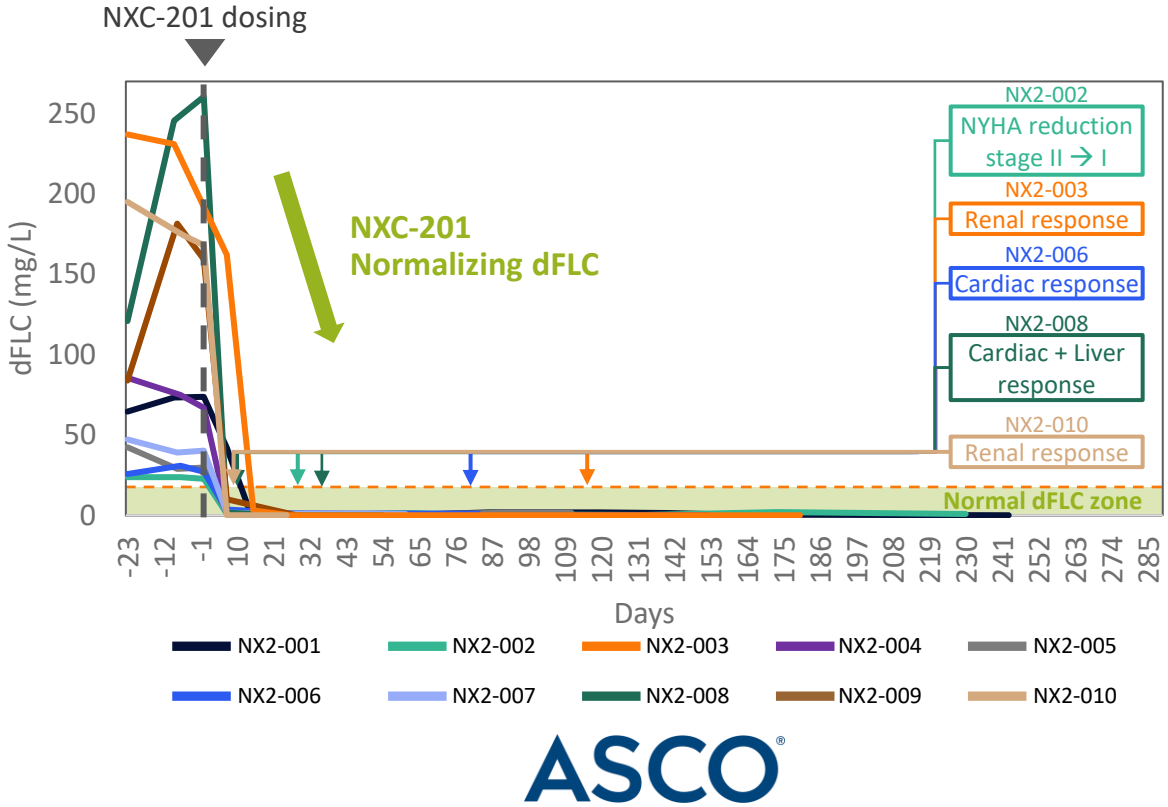
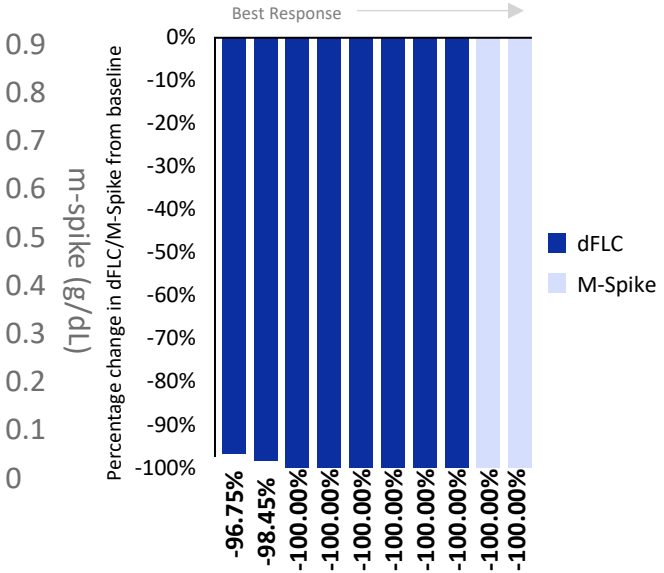
NEXICART-2 (U.S. 2025): Rapid Normalization of Diseased Light Chains (FDA Endpoint) within First ~Month; Consistent with Ex-US Dataset





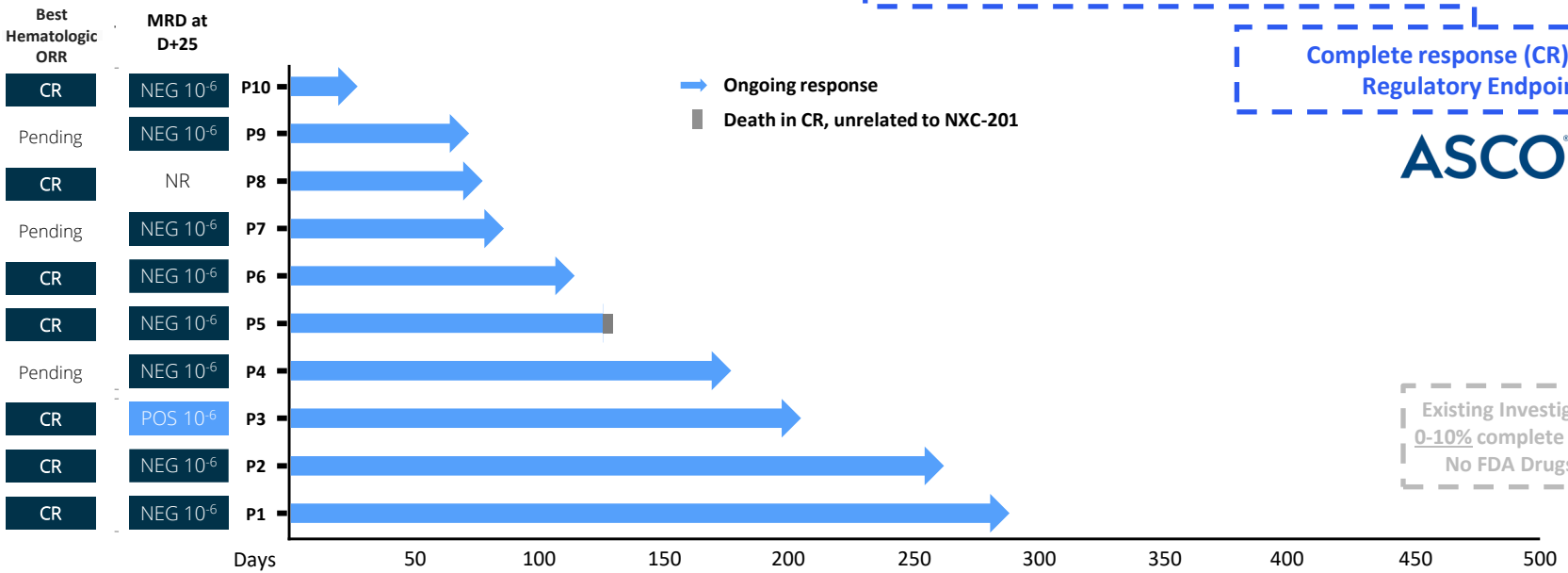
Subject #	NX2-006	NX2-007	NX2-001	NX2-002	NX2-004	NX2-005	NX2-008	NX2-009	NX2-003	NX2-010
Time to response (days)	7	7	14	7	7	7	7	7	15	7
Disease Marker status as of data cutoff	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Normal
Follow-up (days)	114	86	289	261	177	127	79	71	205	29

ASCO®

Note: Data cut-off as of April 11, 2025. Both graphs show trend of relapsed/refractory AL Amyloidosis pathologic disease markers after NXC-201 dosing. dFLC: difference in free light chain (disease marker). Renal response based on AL Amyloidosis consensus criteria for renal response (Palladini G et al 2014 doi: 10.1182/blood-2014-04-570010). 2 out of 2 cardiac organ responses evaluable – NX2-006, NX2-008 responded. 2 out of 3 renal responses evaluable – NX2-003, NX2-010. 1 out of 1 liver response evaluable – NX2-008. Most recent available dFLC reading for patient NX2-001 as of day 243. For patient NX2-002, as of day 230. AL Amyloidosis disease markers on line graph: All patient data is dFLC (left-hand side vertical axis), except for patients NX2-003 and NX2-010, which are m-spike (right-hand side vertical axis)

NEXICART-2 (U.S.) Clinical Activity: 70% Complete Responses (CR) in 7/10 Patients; Remaining Three MRD- negative 10^{-6}, predicting future CR

	Subject #	NX2-001	NX2-002	NX2-003	NX2-004	NX2-005	NX2-006	NX2-007	NX2-008	NX2-009	NX2-010
	NXC-201 Dose (million CAR+T cells)	150	150	150	450	450	450	450	450	450	450
AL Amyloidosis Disease Markers	Status as of data cutoff	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Normal
	Time to normalization (days)	14	7	15	7	7	7	7	7	7	7
	Hematologic response	**CR**	**CR**	**CR**	Pending (already MRD(-)10^{-6})	**CR**	**CR**	Pending (already MRD(-)10^{-6})	**CR**	Pending (already MRD(-)10^{-6})	**CR**
	Downstream Organ Response- Renal 2/3, Cardiac 2/2, Liver 1/1: ("-" indicates not eligible for organ response evaluation)	-	-	✓ Renal	-	-	✓ Cardiac	-	✓ Cardiac ✓ Liver	-	✓ Renal



Complete response (CR) is FDA Regulatory Endpoint

ASCO®

Existing Investigator's choice
0-10% complete response rate
No FDA Drugs approved

Note: Data cut-off as of April 11, 2025. Complete Response according to consensus recommendations in AL amyloidosis (Palladini, et al. 2012. "Consensus guidelines for the conduct and reporting of clinical trials in systemic light-chain amyloidosis." Leukemia 26(11): 2317-2325.)
Source: Zanwar S, et al. Treatment patterns for AL amyloidosis after frontline daratumumab, bortezomib, cyclophosphamide, and dexamethasone treatment failures. Leukemia 2024.

NEXICART-2 (U.S.) Safety: Consistent or Improved Compared to Ex-US Dataset

- No ICANS neurotoxicity of any kind
- Grade 2 CRS in one patient, Grade 1 CRS in seven patients, median 1-day duration

Subject		NX2-001	NX2-002	NX2-003	NX2-004	NX2-005	NX2-006	NX2-007	NX2-008	NX2-009	NX2-010	Median (Range)
Dose	CART Cell Dose (x10^6)	150	150	150	450	450	450	450	450	450	450	-
	Neurotoxicity	None	None	None	None	None	None	None	None	None	None	-
	CRS	None	None	Grade 2	Grade 1	Grade 1	Grade 1	Grade 1	Grade 1	Grade 1	Grade 1	1 (1-2)
	CRS Onset (days)	None	None	3	3	1	1	1	1	1	3	1 (1-3)
	CRS Duration (days)	None	None	1	1	1	1	1	4	1	2	1 (1-4)
Other	Neutropenia	Grade 3	Grade 3	Grade 3	Grade 4	Grade 4	Grade 2	Grade 4	Grade 4	Grade 4	Grade 2	4 (2-4)
	Febrile Neutropenia	None	None	None	None	None	None	None	Grade 3	None	None	-
	Anemia	Grade 1	Grade 2	Grade 3	Grade 1	Grade 3	Grade 1	Grade 1	Grade 2	Grade 1	Grade 1	1 (1-3)
	Thrombocytopenia	Grade 1	Grade 1	Grade 1	Grade 1	Grade 3	Grade 2	None	Grade 4	Grade 3	Grade 1	1 (1-4)
	Acute kidney injury	None	None	None	None	Grade 4 acute on chronic kidney Injury (pre-existing stage 4 chronic kidney disease at enrollment)	None	None	None	None	None	-
	LFT Abnormalities	Grade 2	None	None	None	None	None	None	Grade 1	None	None	-
	≥ Grade 3 Infections	None	Grade 3	Grade 3	None	Grade 5*	None	None	None	None	None	-
	Fatigue	None	Grade 2	Grade 2	Grade 2	None	Grade 1	None	None	None	None	2 (1-2)
	Cardiac Event	None	None	None	Grade 2**	None	None	None	None	None	Grade 2**	-

*Acute on chronic kidney injury in patient with stage 4 CKD at enrollment

**Two patients with pre-existing atrial fibrillation experienced transient arrythmias responsive to beta-blockers

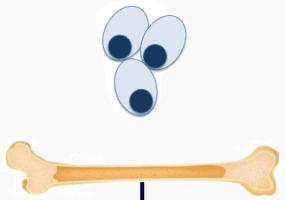
Note: Data cut-off as of April 11, 2025. CRS and ICANS reported according to ASTCT Consensus Grading (Lee et al. 2019)

NEXICART-2 (U.S.): Drivers of Complete Response (CR) in relapsed/refractory AL Amyloidosis

Drives

Drives

MRD Negativity



Normalization of Light Chains

Faster (days or weeks)

Immunofixation

Slower (weeks or months)

Complete Response (CR)

As of April 11 data cutoff, all 10 patients are:
either classified as a **CR**, **or** are MRD negative (10^{-6}) (predicting future CR)

Single-arm potentially pivotal NEXICART-2 trial designed considering NEXICART-1 and precedents in AL

		2021 daratumumab (DARZALEX) FDA Approval	NXC-201 NEXICART-2
Patient Characteristics	Line of Therapy	Newly Diagnosed	Relapsed/Refractory
	Standard of Care (SoC) at time of trial	3-drug combination: Cyclophosphamide, bortezomib, dexamethasone	✓ **None (no FDA approvals)**
	Randomization vs. Standard of Care?	x **Randomization vs. SoC**	✓ **No SoC to randomize against**
	Lines of therapy prior to receiving study drug	x **None**	✓ **At least 1 line of therapy including a CD38 monoclonal antibody**
Study Design	Statistical Power	Based on the assumption that the percentage of patients with a hematologic complete response would be 15 percentage points higher in the daratumumab group than in the control group; approximately **360 patients were required** to provide 85% power to detect this difference (two-sided alpha level of 0.05).	Based on NEXICART-1 complete response (CR) rates, with a sample size of **40 patients**, there is a >99% probability that the lower limit of 95% CI for the NXC-201 CR rate is statistically significantly higher compared to historical controls based on the Clopper-Pearson exact method.
	Primary Endpoint	✓ **Hematologic complete response rate for both studies**	

Single-arm, open-label FDA approval precedents include: Abecma/BMS (single arm study 100 patients in efficacy results population, FDA approved 2021); Carvykti/J&J (single arm study 97 patients in efficacy results population, FDA approved 2022); Elrexfio/Pfizer (single arm study 97 patients in efficacy results population, FDA approved 2023)

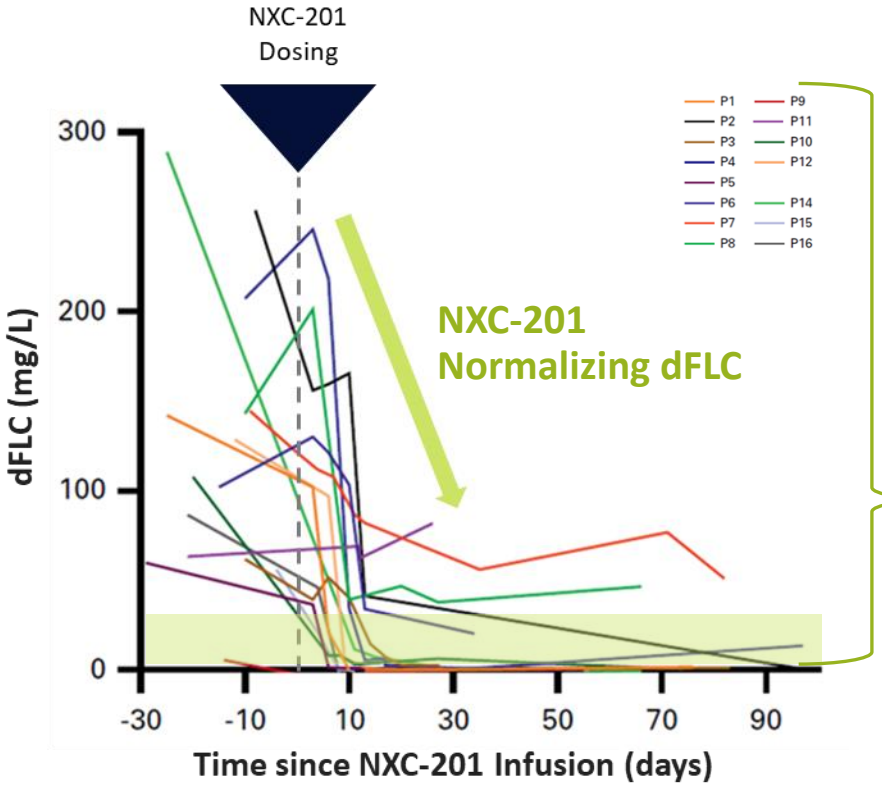
Note: Source for daratumumab information is ANDROMEDA (NCT03201965). NXC-201 information on this slide is illustrative only and represents current plan.

NEXICART-1: Single-Center Ex-US CAR-T NXC-201 Clinical Trial

IMMIX BIOPHARMA

NEXICART-1 (Israel): Normalization of Diseased Free Light Chains 30 Days after Dosing

NXC-201 RAPIDLY ELIMINATES DISEASED AL AMYLOIDOSIS PLASMA CELLS WITHIN ~30 DAYS



(Each line represents 1 patient clinical data readout after NXC-201)

The NEW ENGLAND JOURNAL of MEDICINE

"An early and deep hematologic response has been found to lead to significantly prolonged survival"

– *Vaishali Sanchorawala, M.D.*
Professor, Hematology and Oncology
Director, Amyloidosis Center at Boston University School of Medicine
Director, Stem Cell Transplantation at Boston Medical center

doi: 10.1056/NEJMra2304088

NXC-201
75% complete response rate
(NEXICART-1)

Existing Investigator's choice
0-10% complete response rate
No FDA Drugs approved

Note: Data cut-off as of December 9, 2024. E Lebel et al. Efficacy and Safety of Anti-BCMA Chimeric Antigen Receptor T-Cell (CART) for the Treatment of Relapsed and Refractory AL Amyloidosis. Presentation. ASH 2024.
Source: Zanwar S, et al. Treatment patterns for AL amyloidosis after frontline daratumumab, bortezomib, cyclophosphamide, and dexamethasone treatment failures. Leukemia 2024.

NEXICART-1 (Israel): 6 patients had pre-existing heart failure; 10 patients had preserved heart function at enrollment

PRE-EXISTING HEART FAILURE CAUSES PHYSICALLY IRREVERSIBLE DISTORTION OF HEART STRUCTURE, SHAPE AND SIZE

Preserved heart function Pre-existing heart failure

Patient #	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	Median (range)
Age	64	58	82	63	64	72	55	68	78	59	64	64	63	67	70	58	64 (55-82)
Gender	Male	Female	Male	Male	Male	Female	Female	Male	Male	Male	Female	Male	Female	Male	Male	Male	11/16 M 5/16 F
Prior lines of therapy	8	6	6	10	3	4	4	7	4	3	8	4	4	3	6	3	4 (3-10)
dFLC (mg/L)	143	177	50	550	51	103	196	408	41	108	64	129	83	275	49	86	106 (41-550)
BMPCs (%)	3	15	1	15	0.1	1	1	10	15	1	1	1	0.5	1.5	0.5	0.3	1 (0.1-15)
FISH cytogenetics	t(11:14)	t(14:16) 1Q+	14Q-	t(11:14)	t(11:14)	t(11:14) 1Q+	14Q-	17p-	Normal	17p-	t(4:14) 1Q+	t(11:14)	t(11:14)	Normal	t(11:14)	Normal	7/16 (44%) t(11:14)
Organ involvement	Cardiac, Renal, PNS	Cardiac, Renal, Liver	Renal, GI	Cardiac, Liver, Lung, Soft tissue, PNS	Cardiac, Soft tissue, PNS	Cardiac, Renal, Liver	Cardiac, Soft tissue	Cardiac, Renal, Soft tissue	Renal	Cardiac, Renal, PNS	Cardiac, Renal, GI, Liver, Soft tissue, PNS	Cardiac, Renal	Cardiac, Renal, Soft tissue, GI	Liver	Cardiac, PNS, GI	Cardiac, Renal, GI, Liver	Heart: 13/16 (81%) Kidney: 11/16 (69%) Liver: 6/16 (38%)
NYHA stage	3	4	1	3	2	4	4	2	1	2	2	1	2	3	2	2	1-2: 10/16 (38%) 3: 3/16 (19%) 4: 3/16 (19%)
ProBNP (ng/L)	7,500	2,008	119	2,773	731	28,000	6,600	220	930	669	211	3,158	281	191	107	964	831 (107-28,000)
Trop T (ng/L)	60	40	8	78	18.3	110	30	12	9	8	20	--	--	--	--	--	--
Creatinine (mmol\L)	80	72	110	100	82	108	83	69	220	227	79	--	--	--	--	--	--
Albuminuria (g/24h)	0.3	0.3	2.4	0.1	0.1	1.0	0	0	0.3	1.4	0	--	--	--	--	--	--
ALKP (u/L)	45	218	84	140	84	186	166	106	160	59	160	--	--	--	--	--	--
MAYO stage	3a	3a	1	3a	2	3b	2	1	1	2	2	3a	1	1	1	2	--
ECOG PS	0	2	0	0	1	2	4	0	1	1	1	1	1	2	0	1	1 (0-4)
Concomitant MM	Yes	no	no	No	yes	no	No	no	no	no	no	no	no	no	no	no	2/16
Compassionate use	No	no	no	yes	no	no	yes	no	no	no	no	no	no	no	no	no	2/16

Note: Data cut-off as of December 9, 2024. E Lebel et al. Efficacy and Safety of Anti-BCMA Chimeric Antigen Receptor T-Cell (CART) for the Treatment of Relapsed and Refractory AL Amyloidosis. Presentation. ASH 2024. Eyal Lebel et al., Efficacy and Safety of Anti–B-Cell Maturation Antigen Chimeric Antigen Receptor T-Cell for the Treatment of Relapsed and Refractory AL Amyloidosis. JCO 0, JCO-24-02252. DOI:10.1200/JCO-24-02252.

NEXICART-1 (Israel) NXC-201 Produces Durable Complete Responses in Patients with Preserved Heart Function

Duration of response (ASH 2024)

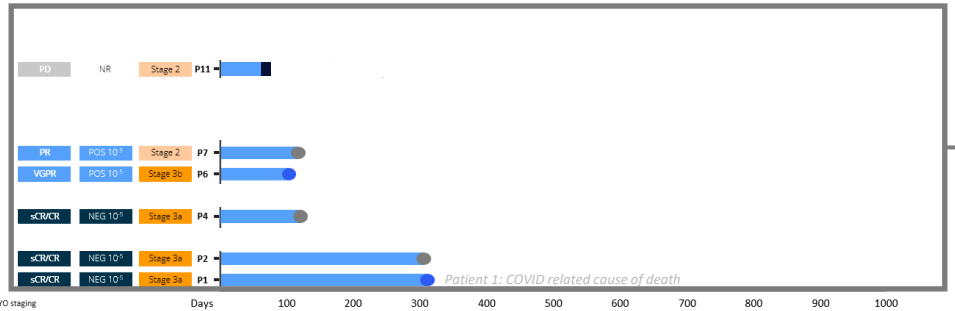
Preserved heart function

- Ongoing response
- Cardiac death in CR/VGPR
- Cardiac death while in PD
- Discontinued

Target For U.S. AL Amyloidosis Clinical Trial Patient Enrollment:

- 90% complete response rate
- Extended response duration

Pre-existing heart failure



Would have been excluded from U.S. clinical trial

- 50% complete response rate
- Limited response duration due to pre-existing heart failure

sCR: strict complete response, CR: complete response

Note: E Lebel et al. Efficacy and Safety of Anti-BCMA Chimeric Antigen Receptor T-Cell (CART) for the Treatment of Relapsed and Refractory AL Amyloidosis. Presentation. ASH 2024. Exclusion criteria: Mayo Stage 3b, NYHA 3/4, prior BCMA exposure. Patient 9 death due to depression. Eyal Lebel et al., Efficacy and Safety of Anti–B-Cell Maturation Antigen Chimeric Antigen Receptor T-Cell for the Treatment of Relapsed and Refractory AL Amyloidosis. JCO 0, JCO-24-02252. DOI:10.1200/JCO-24-02252.

NEXICART-1 (Israel): 75% Complete Response Rate (is the FDA Regulatory Endpoint)

Preserved heart function Pre-existing heart failure

Patient #	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
CART cells infused ($x10^6$)	150	450	800	450	800	800	800	800	800	800	800	800	800	800	800	800
Best hematologic response	CR	CR	CR	CR	CR	VGPR	PR	VGPR	CR	CR	PD	CR	CR	CR	CR	CR
Follow-up (months)	10.3	10.2	31.5, ongoing	4.0	24.0, ongoing	3.3	3.8	5.5	6.0	8.7, ongoing	1.3	6.2, ongoing	4.2, ongoing	2.2, ongoing	2.0, ongoing	1.5, ongoing

Complete response (CR) is FDA Regulatory Endpoint

- **75% (12/16) Complete Response (CR) rate** (9 out of 16 were MRD- 10^{-5})
- Best responder: 31.5 months complete response ongoing as of December 9, 2024 cut-off
- Historical complete response rates for investigator's choice is ~0-10%

Note: Data cut-off as of December 9, 2024. E Lebel et al. Efficacy and Safety of Anti-BCMA Chimeric Antigen Receptor T-Cell (CART) for the Treatment of Relapsed and Refractory AL Amyloidosis. Presentation. ASH 2024. Follow-up duration, estimated internally based on ASH 2024 published swimmer plot. Zanwar S et al.. Treatment patterns for AL amyloidosis after frontline daratumumab, bortezomib, cyclophosphamide, and dexamethasone treatment failures. Leukemia. 2024 Jun;38(6):1423-1426. doi: 10.1038/s41375-024-02243-5. Epub 2024 Apr 9. PMID: 38594348; PMCID: PMC11147750.

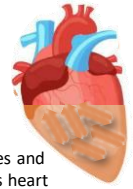
NXC-201: Potential to Expand to Other Serious Diseases

IMMIX BIOPHARMA

CAR-T NXC-201 Targets Plasma Cells (antibody factories of the body)

ANTIBODY FACTORY PLASMA CELLS PRODUCE ANTIBODIES THAT DRIVE IMMUNE-MEDIATED DISEASES

AL Amyloidosis



Infiltrates and damages heart

AL amyloid antibody deposits

Light chain antibody fragments

Neurology

- Myasthenia Gravis
- NMO Spectrum Disorder

Hematology

- Thrombotic thrombocytopenic purpura
- Immune Thrombocytopenia

Rheumatology

- Systemic Lupus Erythematosus
- Rheumatoid Arthritis

Vascular

- ANCA vasculitis

Disease-causing antibodies

ANTIBODY FACTORY PLASMA CELL

(NXC-201 therapeutic target)

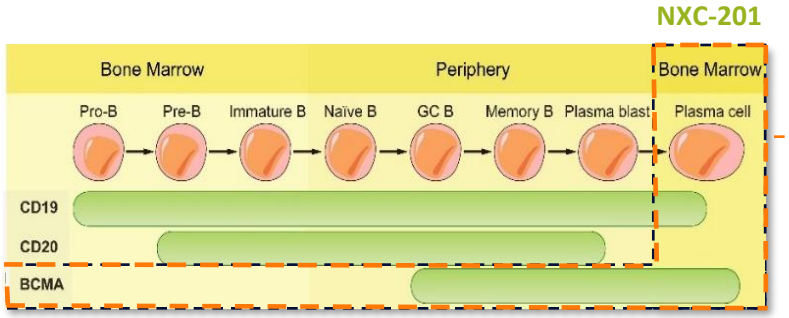
Note: select indications noted above are for illustrative purposes only.

*Illustrative list of immune-mediated diseases where B cells may play a role in initiating or maintaining disease, and where NXC-201 may provide a potential treatment

Source: MedicTests. Lee, J. et al. Antigen-specific B cell depletion for precision therapy of mucosal pemphigus vulgaris. J. Clin. Invest. 2020. Mackensen, A. et al. Anti-CD19 CAR T cell therapy for refractory systemic lupus erythematosus. Nat. Med. 2022. Qin C, et al. Anti-BCMA CAR T-cell therapy CT103A in relapsed or refractory AQP4-IgG seropositive neuromyelitis optica spectrum disorders: phase 1 trial interim results. Signal Transduct Target Ther. 2023. Granit V, et al. Safety and clinical activity of autologous RNA chimeric antigen receptor T-cell therapy in myasthenia gravis (MG-001): a prospective, multicentre, open-label, non randozised phase 1/2a study. Lancet Neurol. 2023. McGlothlin J, et al. Bortezomib and daratumumab in refractory autoimmune hemolytic anemia. Am J Hematol. 2023. Yu TS, et al. Abnormalities of bone marrow B cells and plasma cells in primary immune thrombocytopenia. Blood Adv. 2021. Zhang Z, Xu Q, Huang L. B cell depletion therapies in autoimmune diseases: Monoclonal antibodies or chimeric antigen receptor-based therapy? Front Immunol. 2023. Pioli PD. Plasma Cells, the Next Generation: Beyond Antibody Secretion. Front Immunol. 2019

NXC-201 BCMA CAR-T targeting is uniquely suited to address Other Serious Diseases

NXC-201 BCMA CAR-T TARGETS OTHER DISEASE CAUSING LONG-LIVED PLASMA CELLS



~80% of all antibodies are produced by plasma cells…

BCMA is expressed on long lived plasma cells

NXC-201 BCMA CART targets long lived plasma cells (LLPC), targeting the source of disease-causing antibodies

- CD19 therapies target earlier lineage B-Cells, allowing LLPCs to persist

Immix Biopharma unaddressed other serious disease indication selection criteria

High unmet medical need	✓
Limited therapies in development	✓
Biological basis for plasma cell-mediated therapy	✓

Appendix 1: Technology

September 2025

N-GENIUS Platform: Sterically-Optimized CAR-T construct “Digital Filter” reduces non-specific activation, leading to better tolerability

ALL BCMA CAR-TS ARE NOT CREATED EQUAL

N-GENIUS PLATFORM

NXC-201 sterically-optimized CAR-T’s “Digital Filter”…. **…reduces non-specific activation**

1 Proprietary Optimized CD3 – “CD3ζγ”

✓ Delivers “Digital” Intracellular Signaling

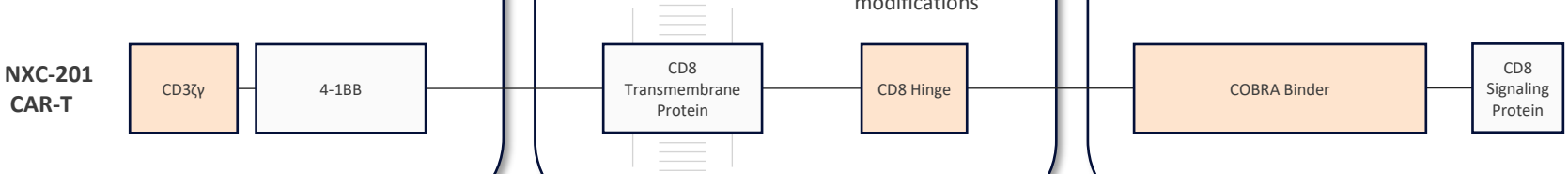
2 Proprietary Optimized CD8 Hinge Flexibility

✓ Reduces cytokine release

3 Proprietary Optimized COBRA Binder

✓ Enhances Plasma Cell Binding
✓ Ensures High Expression

Sterically-optimized key construct modifications



Source: M. Assayag, et al. Academic BCMA-CART cells (HBI0101), a promising approach for the treatment of LC Amyloidosis. 27th Annual Meeting of The American Society of Gene and Cell Therapy (ASGCT). Late Breaking Oral Presentation. Baltimore, MD. May, 2024. Feucht, M. Sadelain, et al. Calibration of CAR activation potential directs alternative T cell fates and therapeutic potency. Nature Medicine. 2019 Jan;25(1):82-88. doi: 10.1038/s41591-018-0290-5. Epub 2018 Dec 17. PMID: 30559421 PMCID: PMC6532069. O. Harush C. J. Cohen, et al. Preclinical evaluation and structural optimization of anti-BCMA CAR to target multiple myeloma. Haematologica. 2022 Oct 1;107(10):2395-2407. doi: 10.3324/haematol.2021.280169. PMID: 35354252 PMCID: PMC9521250. Adapted from PEGS 2021. Zanwar S, et al. Eyal Lebel et al., Efficacy and Safety of Anti–B-Cell Maturation Antigen Chimeric Antigen Receptor T-Cell for the Treatment of Relapsed and Refractory AL Amyloidosis. JCO. JCO-24-02252. DOI:10.1200/JCO-24-02252.

NXC-201 sterically-optimized CAR-T's "Digital Filter" ...reduces non-specific activation

CD3ζ

1

Proprietary Sterically-Optimized CD3ζ + CD8 Delivers "Digital" Intracellular Signaling, Eliminates Neurotoxicity, Reduces CRS Duration

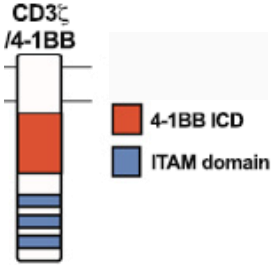
CARs rely on activation of CAR-T cells through CD3ζ derived immunoreceptor tyrosine-based activation motifs (ITAMs), typically 3 ITAM motifs per CAR

NXC-201 adds a positively charged amino acid (lysine) next to a tyrosine phosphorylation site, therefore:

- ✓ Impeding phosphorylation of ITAM1 (by affecting protein folding dynamics which block the tyrosine site), thus reducing intracellular reactivity
- ✓ Adding an additional site for ubiquitination, allowing the CAR to be marked for degradation more rapidly that a traditional CAR

The combined effect of these modifications is to drive a "digital" signaling of extracellular activity, that is on when antigen is present and off when not

Modification of ITAMs is a common theme in third-generation CAR design, with publications in Nature Medicine and by Memorial Sloan Kettering on the topic



nature Signal Transduction and Targeted Therapy

"In activated T cells, the CD3ζ chain gets ubiquitinated by CBLB at its multiple lysine residues and induces degradation of surface TCRs"

doi: 10.1038/s41392-021-00823-w

nature medicine

Memorial Sloan Kettering Cancer Center

"We hypothesized that the redundancy of CD28 and CD3ζ signaling in a chimeric antigen receptor (CAR) design incorporating all three CD3ζ immunoreceptor tyrosine-based activation motifs (ITAMs)11,13 may foster counterproductive T cell differentiation and exhaustion. Therefore, we calibrated ITAM activity by mutating tyrosine residues to impede their phosphorylation and downstream signaling"

doi: 10.1038/s41591-018-0290-5

Source: Feucht J, et al. Nat Med. 2019; Shah K, et al. Signal Transduct Target Ther. 2021

NXC-201 sterically-optimized CAR-T's "Digital Filter" ...reduces non-specific activation

2

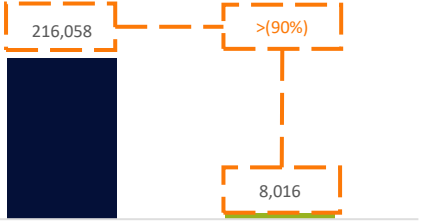
Proprietary Sterically-Optimized CD3ζ + CD8 Delivers "Digital" Intracellular Signaling, Eliminates Neurotoxicity, Reduces CRS Duration

CD8 Hinge

PreClinical

NXC-201: Optimized (Decreased) CD8 Hinge Flexibility Resulted In:

- ✓ >90% reduction in cytokine release in preclinical studies
- ✓ improved human efficacy
- ✓ zero neurotoxicity

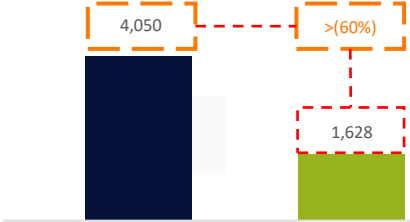


IFNγ (pg/mL): K562-BCMA co-culture

Interferon-c (IFN-c) release after 24 h of co-culture of CAR T cells with BCMA+ (K562-BCMA, RPMI-8226, U266-B1, H929) targets.

Literature: Optimized (Decreased) CD8 Hinge Flexibility Resulted In

- ✓ >60% reduction in cytokine release in preclinical studies
- ✓ improved human efficacy
- ✓ zero neurotoxicity



IFNγ (pg/mL): K562-CD19 co-culture

CAR+ T cells were stimulated with K562 cells expressing human CD19. Supernatant was harvested after 24 hours of incubation, and the indicated cytokines were measured by cytokine bead array. Results are representative of two-to-four independent experiments.

Clinical

		Abecma in MM	NXC-201 in MM	Kymriah	CD19-BBz(86)
Efficacy	ORR (%)	72	92	52	73
	CR (%)	28	64	40	55
CRS	CRS, any grade (%)	85	95	58	28
	CRS, Grd3+ (%)	9	19	22	0
	Duration, CRS (days)	7	1 at 800M	7	n/a
Neurotoxicity	Neurotoxicity, any grades (%)	28	4% (all Grade 1-2)	21	0

Sterically-optimized (Decreased) CD8 Hinge Flexibility Results in Zero Neurotoxicity, Improved Human Efficacy, and reduction in CRS duration

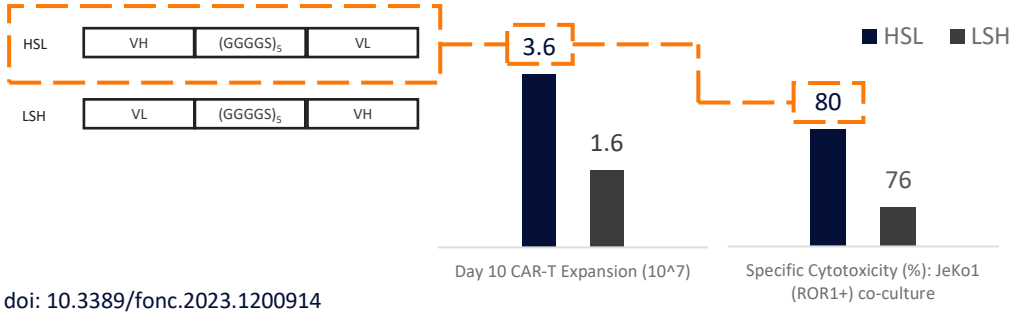
MM: multiple myeloma

Source: E Lebel et al. Efficacy of HBI0101, an Anti-BCMA Chimeric Antigen Receptor T-Cell (CART) for Relapsed/Refractory Multiple Myeloma. Abstract. ASH 2024. S Kfir-Erenfeld et al. Clinical evaluation and determinants of response to HBI0101 (BCMA CART) therapy in relapsed/refractory multiple myeloma. Blood Adv. 2024 Aug 13;8(15):4077-4088. doi: 10.1182/bloodadvances.2024012967. Ying Z, et al. Nat Med. 2019; Schuster SJ, et al. N Engl J Med. 2019; Assayag, M., et al EBMT 2023; Abecma FDA label; Harush O, et al. Haematologica. 2022; Friedman KM, et al. Hum Gene Ther. 2018. Kymriah: Preclinical is an average of CD8+ and CD4+ T-cells, source: Milone MC, et. Al. Mol Ther. 2009 Aug;17(8):1453-64. doi: 10.1038/mt.2009.83. Epub 2009 Apr 21. Erratum in: Mol Ther. 2015 Jul;23(7):1278. PMID: 19384291; PMCID: PMC2805264. *1 Day CRS occurred in high dose MM cohort as of EBMT 2023. NXC-201 in multiple myeloma data from ASH 2023 95% ORR in patients without prior anti-BCMA therapy exposure

3 Sterically-Optimized COBRA Binder Ensures High Expression And Binding Affinity

COBRA Binder

COBRA Binder Leads with Heavy Chain



doi: 10.3389/fonc.2023.1200914

Proven Linker of Heavy and Light Chain Employed

Biomarker Research

"Glycine (Gly) and serine (Ser) residues provide the flexibility necessary for antigen-binding sites to change conformation and maintain good stability in aqueous solutions… prevent[ing] formation of secondary structures and reduc[ing] likelihood of the linker interfering with the folding and function of the scFv"

September 19, 2022
doi: 10.1186/s40364-022-00417-w

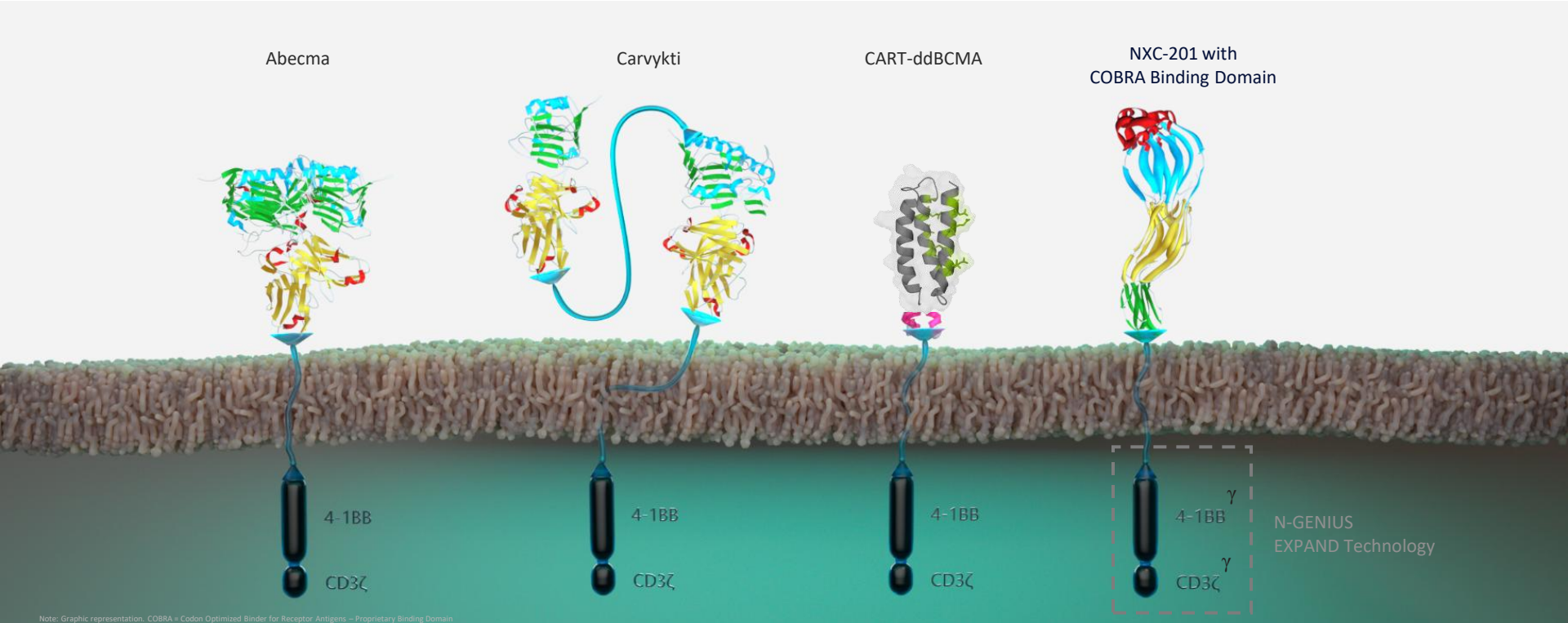
NXC-201
COBRA Binder: Heavy Chain – Proven Linker – Light Chain Configuration, enabling:

- ✓ Rapid, Sustained CAR-T Expansion
- ✓ Improved Cytotoxicity in the presence of antigen

Source: Moreno-Cortes E,. et al Front Oncol. 2023; Mazinani M, et al. Biomark Res. 2022.

Proprietary EXPAND Technology and COBRA Binding Domain Are Differentiated Innovations

N-GENIUS TECHNOLOGY PLATFORM: STERICALLY-OPTIMIZED BCMA CAR-T NXC-201



Note: Graphic representation. COBRA = Codon Optimized Binder for Receptor Antigens – Proprietary Binding Domain

N-GENIUS Sterically-Optimized CARTs Drive Immix's Immune-Mediated Disease Leadership

N-GENIUS Platform

Through partnership Hadassah Medical Organization, Jerusalem and Bar-Ilan University, Immix is a pioneer in sterically-optimized CAR-T construct development, driving our next-generation industry-leading CAR-Ts in select immune-mediated diseases.

N-GENIUS developed sterically-optimized CAR-Ts allow for a differentiated safety and efficacy profile through modification of key regions of the CAR-T construct, driving a "digital" intracellular signal and overcoming limitations of current clinical stage and commercial CAR-Ts.

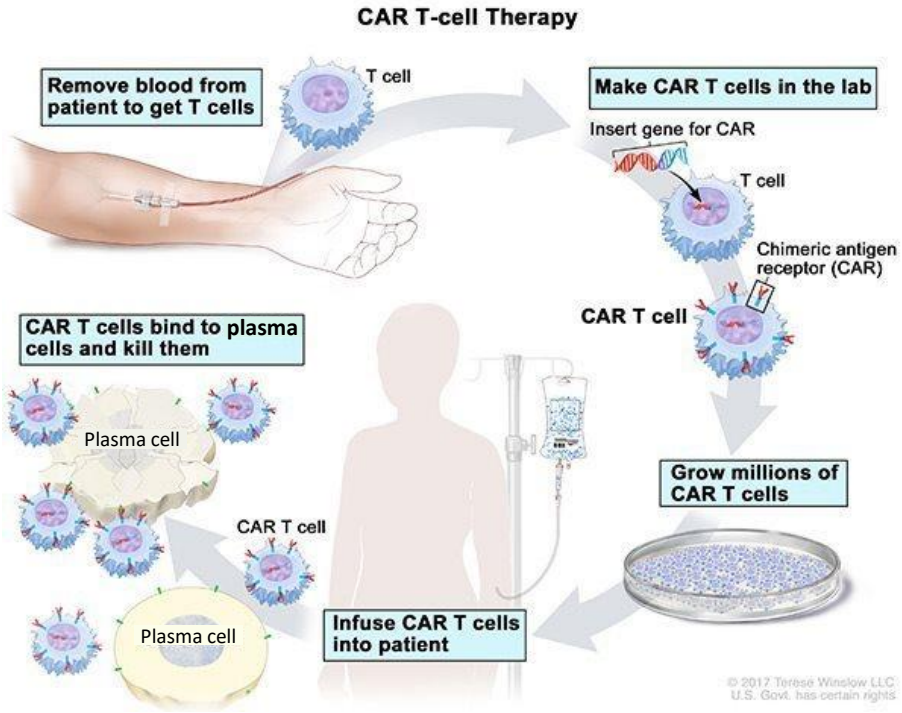
Portfolio of U.S. patents, pending applications, and exclusive patent licenses, which cover our core technology platforms and product

Hadassah University Hospital

Bar-Ilan University
אוניברסיטת בר־אילן

What is autologous chimeric antigen receptor (CAR)-T cell therapy?

NXC-201 IS A NEXT-GENERATION BCMA TARGETED AUTOLOGOUS CAR-T CELL THERAPY



Patient Specific

Personalized treatment using patient's own T cells

Genetic Modification

Genetically engineered CARs (chimeric antigen receptors) on T cell surface

Targeted Therapy

Target cells that express antigens recognized by CARs

Source: Adapted from Texas Oncology. CAR-T Therapy. https://www.texasoncology.com/services-and-treatments/treatments/car-t-therapy

N-GENIUS Platform Process

1

Problem Identification

CAR-T Toxicity Main Block to Widespread Use: Neurotoxicity

2

Research Group Formation

Reduce CAR-T Toxicity while Improving Efficacy

3

Platform Foundation

N-GENIUS Platform with EXPAND Technology aims to achieve research group goal

4

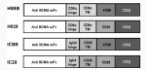
Candidate Creation

Entire CAR-T construct optimized with N-GENIUS Platform and EXPAND Technology In Selected Indications

5

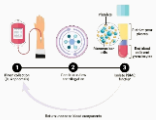
Final Candidate Selection

4 promising candidates selected among a larger number of options



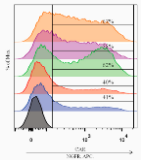
6

Human Peripheral blood mononuclear cells (PBMC) were transduced with the CAR candidates



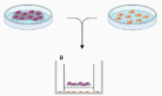
7

CAR-T Transgene expression was assessed by flow cytometry



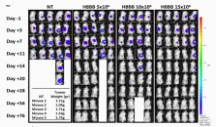
8

Co-culture of CAR-T cells and target cells with target antigens and in vitro analysis of cytokine release



9

Evaluation CAR-T activity in vivo (tumor growth, cytokine release, safety evaluation)



10

NXC-201

Source:: Harush O, et al. Haematologica. 2022;

Appendix 2: AL Amyloidosis Clinical

Advantages of CAR-T NXC-201 in Relapsed/Refractory AL Amyloidosis

		NXC-201	Antibody-drug conjugates	Bispecifics
1	One-time treatment	✓	✗	✗
2	High Complete Response Rates	✓	✗	✗
3	Low rates of severe infection	✓	✓	✗
4	No ICANS/Neurotoxicity	✓	✓	✗

NXC-201 uniquely suited for Relapsed/Refractory AL Amyloidosis

Note: High complete response rates defined as >50%. Low rates of severe infection refers to <30%

Source: Feasibility of a Novel Academic Anti-BCMA Chimeric Antigen Receptor T-Cell (CART) (HBI0101) for the Treatment of Relapsed and Refractory AL Amyloidosis, 65th ASH Annual Meeting and Exposition, San Diego, CA. October 2023. R. Chakraborty, et al. Safety and efficacy of teclistamab in systemic immunoglobulin light chain amyloidosis. Blood Cancer Journal. October 2023. N. Forgeard, et al. Teclistamab in relapsed or refractory AL amyloidosis, a multinational retrospective case series. Blood. February 2024. Chakraborty R, Bhutani D, Maurer MS, Mohan M, Lentzsch S, D'Souza A. Safety and efficacy of teclistamab in systemic immunoglobulin light chain amyloidosis. Blood Cancer J. 2023 Nov 27;13(1):172. doi: 10.1038/s41408-023-00950-3. PMID: 38012151; PMCID: PMC10682473. One NXC-201 relapsed/refractory AL amyloidosis patient died of COVID-19. Kastritis, et al. Efficacy And Safety Of Belantamab Mafodotin Monotherapy In Patients With Relapsed Or Refractory Light-chain Amyloidosis: A Phase 2 Study By The European Myeloma Network. Abstract. EHA 2024.

In AL Amyloidosis, NXC-201 Overcomes Limitations of Other Modalities in Performance and Tolerability

Challenges of bispecifics/ T-cell engagers	NXC-201 overcomes these challenges
• No clinical trials with clinical data available in relapsed/refractory AL amyloidosis	✓ **75% CR in relapsed/refractory AL amyloidosis**
• Early data from select centers indicates bispecific responses and tolerability are inferior to CAR-T (NXC-201) in relapsed/refractory AL amyloidosis	✓ **0 deaths from drug-related infection in relapsed/refractory AL amyloidosis**
• Retrospective study with 17 R/R multiple myeloma + AL Amyloidosis patients: ✗ 41% CR ✗ 35% severe infections including death ✗ Grade 3 ICANS neurotoxicity reported	✓ **0% neurotoxicity (0/16) in relapsed/refractory AL amyloidosis patients**
• Repeat/ongoing dosing with need for healthcare provider to administer	• **One-time dosing with durable responses**

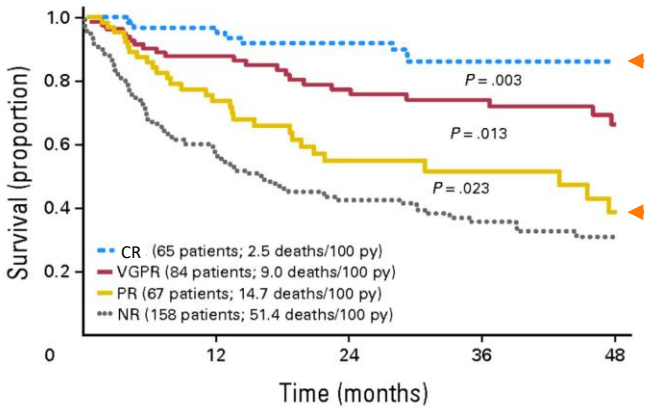
Advantages of NXC-201 CAR-T in AL Amyloidosis

Source: Feasibility of a Novel Academic Anti-BCMA Chimeric Antigen Receptor T-Cell (CART) (HBI0101) for the Treatment of Relapsed and Refractory AL Amyloidosis, 65th ASH Annual Meeting and Exposition, San Diego, CA. October 2023. R. Chakraborty, et al. Safety and efficacy of teclistamab in systemic immunoglobulin light chain amyloidosis. Blood Cancer Journal. October 2023. N. Forgeard, et al. Teclistamab in relapsed or refractory AL amyloidosis, a multinational retrospective case series. Blood. February 2024. One NXC-201 relapsed/refractory AL amyloidosis patient died of COVID-19

Complete Hematologic Response is correlated with longer survival

COMPLETE HEMATOLOGIC RESPONSE WAS PRIMARY ENDPOINT IN 2021 DARATUMUMAB APPROVAL STUDY AND ONGOING NEXICART-2 TRIAL

Complete Hematologic Response (CR) associated with improved survival in AL



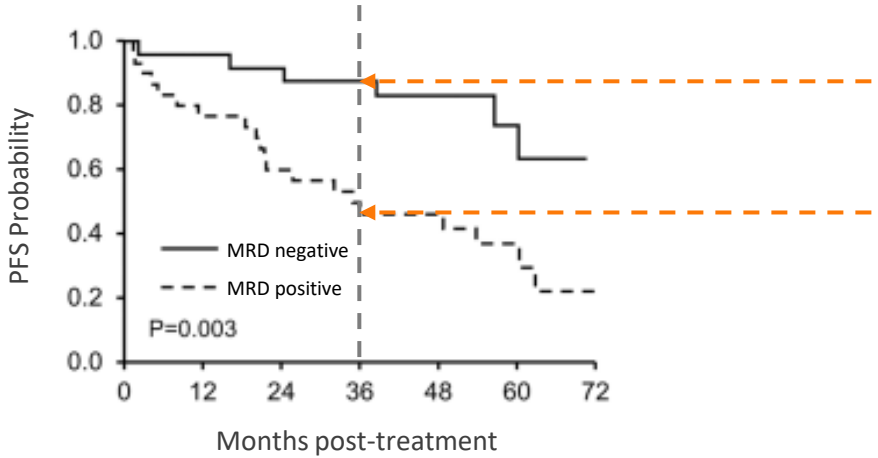
2x survival at 48 months for CR vs PR

- Complete Hematologic response patients have 85% survival at 48 months
- Partial hematologic response patients have 40% survival at 48 months

Source: Adapted from Palladini G, Dispenzieri A, Gertz MA, Kumar S, Wechalekar A, Hawkins PN, Schönland S, Hegenbart U, Comenzo R, Kastritis E, Dimopoulos MA, Jaccard A, Klersy C, Merlini G. New criteria for response to treatment in immunoglobulin light chain amyloidosis based on free light chain measurement and cardiac biomarkers: impact on survival outcomes. J Clin Oncol. 2012 Dec 20;30(36):4541-9. doi: 10.1200/JCO.2011.37.7614. Epub 2012 Oct 22. PMID: 23091105.

MRD- is Correlated with Improved PFS in AL Amyloidosis

MRD negativity is associated with improved Progression Free Survival in AL



2x PFS at 36 months for MRD- vs MRD+
(patients with CR or VGPR)

- MRD negative patients have 88% 36-month PFS
- MRD positive patients have 46% 36-month PFS

Note: Adapted from Muchtar E, Dispenzieri A, Jevremovic D, Dingli D, Buadi FK, Lacy MQ, Gonsalves W, Warsame R, Kourelis TV, Hayman SR, Kapoor P, Leung N, Russell S, Lust JA, Lin Y, Go RS, Zeldenrust S, Kyle RA, Rajkumar SV, Kumar SK, Gertz MA. Survival impact of achieving minimal residual negativity by multi-parametric flow cytometry in AL amyloidosis. Amyloid. 2020 Mar;27(1):13-16. doi: 10.1080/13506129.2019.1666709. Epub 2019 Sep 23. PMID: 31544536; PMCID: PMC7372715.

Principal Investigator for NEXICART-2: Heather Landau, MD

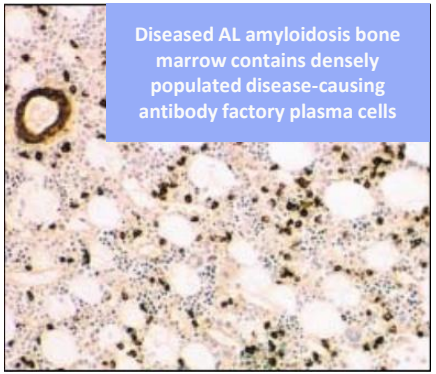
- **Director of the Amyloidosis Program and a Bone Marrow Transplant Specialist & Cellular Therapist at Memorial Sloan-Kettering Cancer Center in New York.**
- Authored more than 100 peer-reviewed publications.
- Dr. Landau received her medical degree from SUNY Upstate Medical University, completed her Internal Medicine residency at University of Colorado and her Hematology & Oncology fellowship at Memorial Sloan Kettering Cancer Center.

NXC-201 CAR-T cells penetrate tissues and repeat kill disease-causing cells in AL Amyloidosis

AL Amyloidosis pathologic light chains emerge from deep in the bone marrow where they are produced by pathologic antibody factory plasma cells

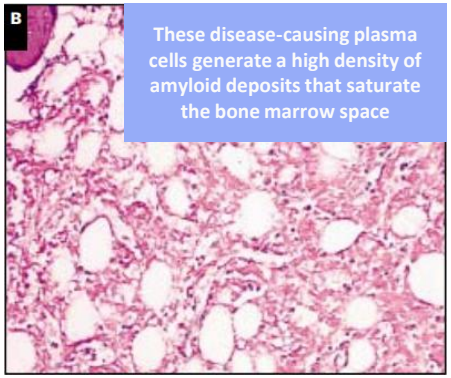
Traditional antibody-based therapies primarily address passively accessible target cells

NXC-201 is the only AL amyloidosis therapy in clinical development that actively penetrates deep into the bone marrow



Diseased AL amyloidosis bone marrow contains densely populated disease-causing antibody factory plasma cells

Immunoperoxidase with hematoxylin counterstain, ×100



These disease-causing plasma cells generate a high density of amyloid deposits that saturate the bone marrow space

Periodic acid–Schiff, ×100

Antibody-based therapeutics have difficulty penetrating deep into organs where disease causing plasma cells reside

Inserm

"Plasma cells (PC) were detected in the [organs] of patients … up to 6 months after rituximab treatment, and the PC population displayed a long-lived program"

doi:10.1172/JCI65689

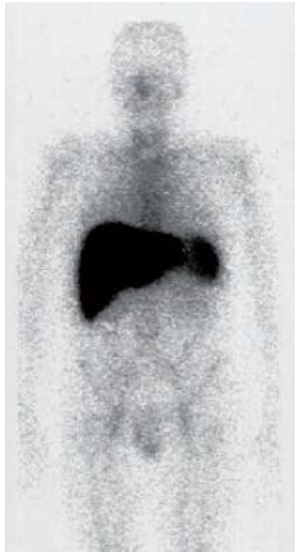
Source: N Swan et al. Bone Marrow Core Biopsy Specimens in AL (Primary) Amyloidosis. Hematopathology. Am J Clin Pathol 2003. DOI: 10.1309/PFUGHBX0TY20E08U.. Mahévas M, et al. B cell depletion in immune thrombocytopenia reveals splenic long-lived plasma cells. J Clin Invest. 2013 Jan;123(1):432-42. doi: 10.1172/JCI65689. Epub 2012 Dec 17. PMID: 23241960; PMCID: PMC3533302.

Amyloid deposits in AL Amyloidosis are cleared naturally after treatment

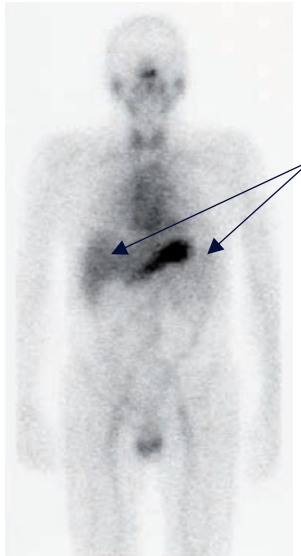
BEFORE TREATMENT

2 YEARS AFTER TREATMENT

Pre-treatment imaging shows dense amyloid deposits in liver and spleen in AL patient



High-dose melphalan and peripheral blood stem-cell transplantation



Post-treatment imaging shows clearance of amyloid in organs throughout the body

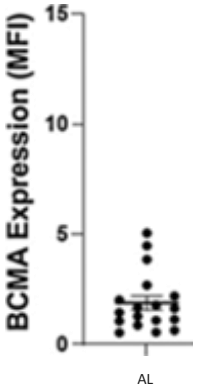
Source: Adapted from van Gameren I Ingrid, et al. Novel treatments for systemic amyloidosis. Int. J. Clin. Rheumatol. (2009)

NXC-201 Designed for High Activity Against Disease-causing AL Amyloidosis Plasma Cells

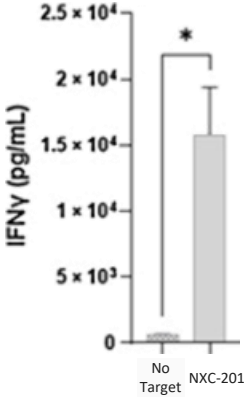
ALL BCMA CAR-TS ARE NOT CREATED EQUAL

a) Uneven BCMA expression and b) frail patient condition has historically prevented conventional, approved CAR-T use in AL Amyloidosis
NXC-201 was designed and tested for human AL Amyloidosis treatment, with preclinical testing revealing that:

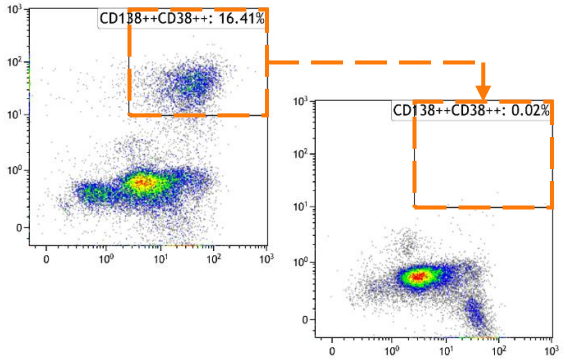
1 BCMA is expressed in 18 AL Amyloidosis patients at a low-medium level…



2 …NXC-201 CAR-Ts are activated in presence of the AL Amyloidosis target cells…



…completely eliminating AL Amyloidosis aberrant plasma cells from patient bone marrow.



NXC-201's uniquely favorable tolerability profile (no neurotoxicity + "Single-day CRS") combined with proven preclinical and clinical efficacy make NXC-201 uniquely suited to treat AL Amyloidosis.

Note: NXC-201 (formerly NBI0101). Infy TNFa for two patients, AL1, AL2. Far right top right quartile selected diseased AL Amyloidosis plasma cell elimination. Far right graph after 33 days co-incubated with NXC-201.
Source: Kfir-Erenfeld S, et al. Feasibility of a Novel Academic BCMA-CART (HBI0101) for the Treatment of Relapsed and Refractory AL Amyloidosis. Clin Cancer Res. 2022; Raje N, et al. Anti-BCMA CAR T-Cell Therapy bb2121 in Relapsed or Refractory Multiple Myeloma. N Engl J Med. 2019.;

NXC-201 May Be a Curative Treatment for AL Amyloidosis

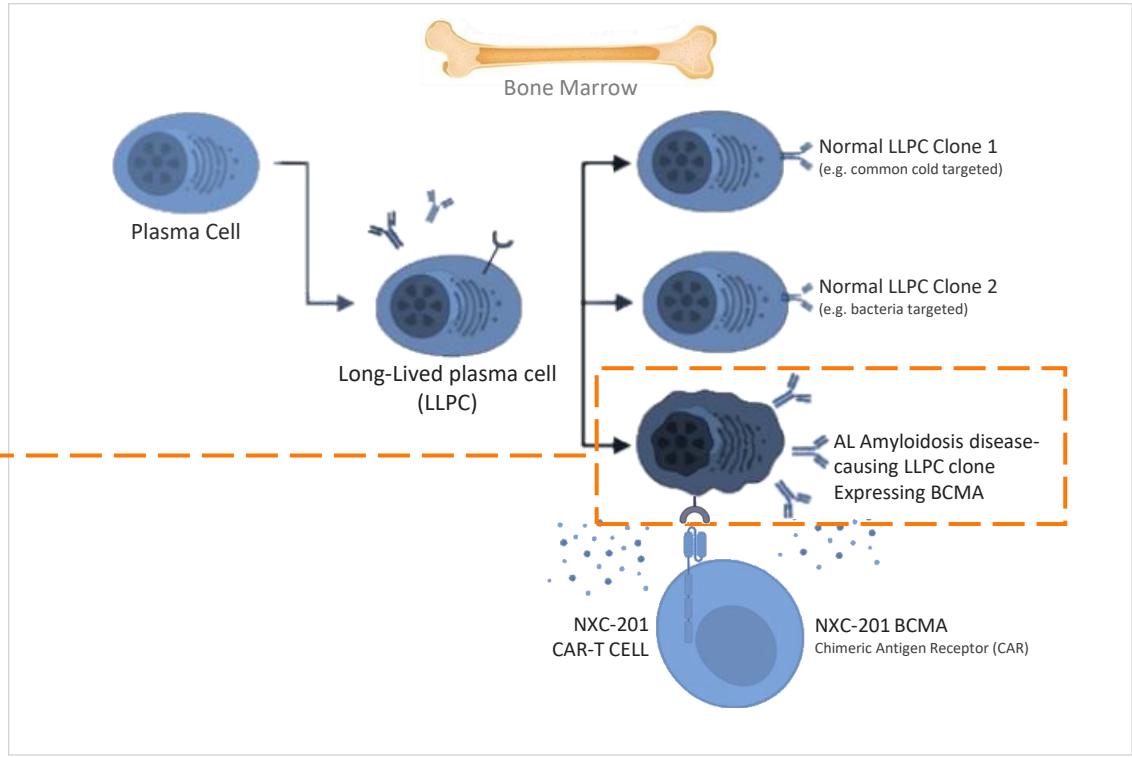
ALL BCMA CAR-TS ARE NOT CREATED EQUAL

AL amyloidosis is caused by dysfunctional plasma cells that can live for years, continuously producing toxic, misfolded amyloid light chain antibody fragments

AL Amyloidosis disease-causing plasma cells:

- Are a minority of total plasma cells
- Are 1 of thousands of antibody-specific plasma cells, each of which produces 1 antibody, specific to 1 target (e.g. common flu, cold, a type of bacteria…)
- Are eliminated by NXC-201 treatment

Then healthy plasma cells regenerate in the months after NXC-201 treatment, potentially preventing disease relapse



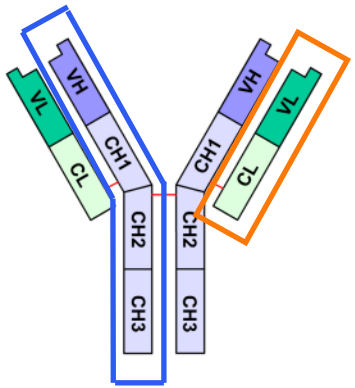
Source: Alberts B, Johnson A, Lewis J, et al. Molecular Biology of the Cell. 4th edition. New York: Garland Science; 2002. The Generation of Antibody Diversity. Available from: https://www.ncbi.nlm.nih.gov/books/NBK26860/; Wang Y, Li C, Xia J, Li P, Cao J, Pan B, Tan X, Li H, Qi K, Wang X, Shi M, Jing G, Yan Z, Cheng H, Zhu F, Sun H, Sang W, Li D, Zhang X, Li Z, Zheng J, Liang A, Zhou J, Xu K. Humoral immune reconstitution after anti-BCMA CAR T-cell therapy in relapsed/refractory multiple myeloma. Blood Adv. 2021 Dec 14;5(23):5290-5299. doi: 10.1182/bloodadvances.2021004603. PMID: 34587230; PMCID: PMC9153033.

Pathologic Light Chains in AL Amyloidosis are an Antibody Building Block That Are Overproduced by Plasma Cells

A LIGHT CHAIN IS A PORTION OF AN ANTIBODY

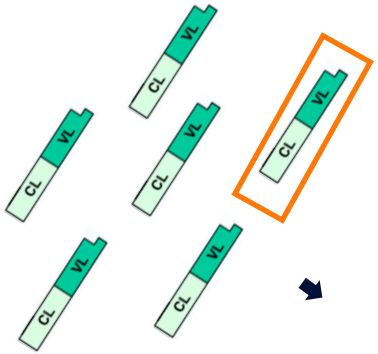
Light chain portion of antibody

Heavy chain portion of antibody



Normal antibody produced by plasma cell

IN AL, PLASMA CELLS PRODUCE TOO MANY LIGHT CHAINS



Diseased light chains in AL Amyloidosis: excessive production by plasma cells

AL amyloid antibody deposits

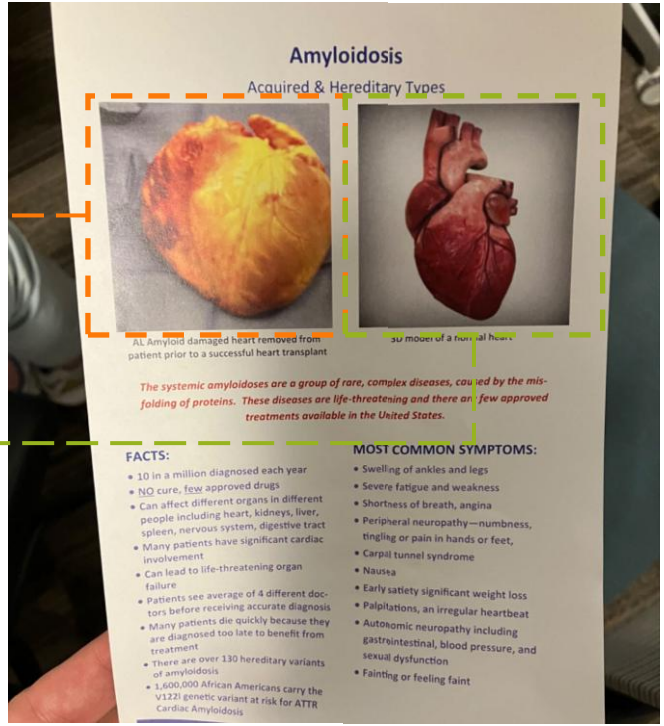
Infiltrates and damages heart

This Is Pre-Existing Heart Failure in AL Amyloidosis

PRE-EXISTING HEART FAILURE CAUSES PHYSICALLY IRREVERSIBLE DISTORTION OF HEART STRUCTURE, SHAPE AND SIZE

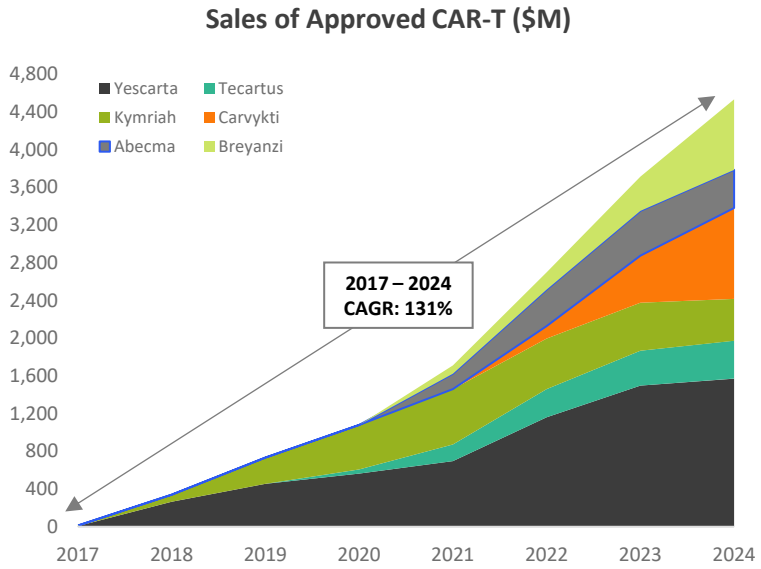
Pre-existing heart failure

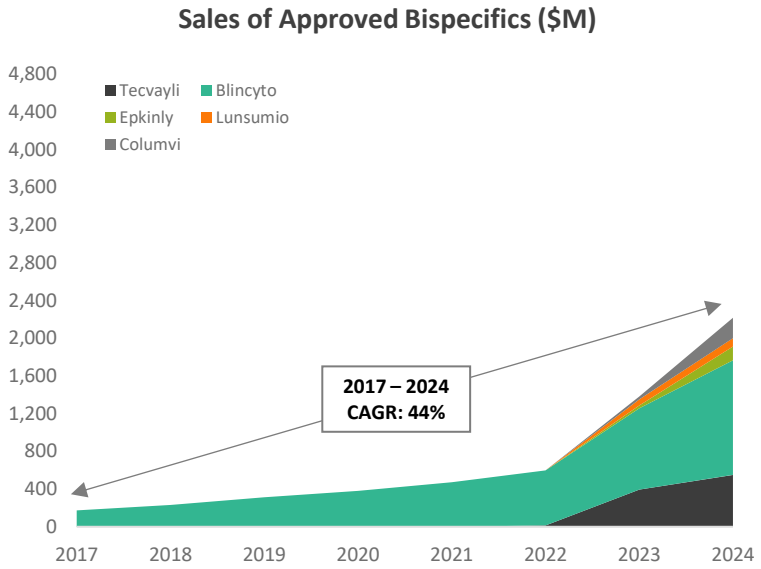
Preserved heart function



Appendix 3: Market

Robust Global Sales of CAR-T Continue





Source: Company reports

Clinical Stage Cell Therapy for AL Amyloidosis and Other Serious Diseases

September 2025

IMMiX BIOPHARMA