

Clinical Stage CAR-T for AL Amyloidosis and Select Immune-Mediated Diseases

November 2024



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Immix Biopharma Highlights



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

- NXC-201 could transform the treatment of Relapsed/Refractory AL amyloidosis (US patients: ~33,000 prevalence, ~4,300 annual incidence, \$3bn market)
- Ex-US NXC-201 study: 69% (9/13) Complete response rate in relapsed/refractory AL Amyloidosis in the largest CAR-T clinical study to-date
- NEXICART-2 US potentially pivotal 40 patient clinical trial initiated mid-2024

Clinical profile ideal for select immune-mediated diseases

- Established clinical profile across large 76 patient dataset dosed with NXC-201: well-suited to treat select immune-mediated diseases
- Short duration of cytokine release syndrome
- Lack of neurotoxicity

Sterically-optimized, proprietary CAR-T construct

- N-GENIUS platform produced NXC-201, our lead, sterically-optimized CAR-T
- NXC-201 CART construct provides barrier to entry: 3 key CAR modifications drive unique clinical profile - CD3 ζ γ , CD8 hinge, COBRA binder
- NXC-201 engineered specifically to solve for CAR-T tolerability (cytokine release syndrome, neurotoxicity)

Significant Near-Term Milestones

Upcoming Milestones	Anticipated Timing
Initial NEXICART-2 clinical data presentation in AL Amyloidosis	4Q 2024
Report interim clinical data readout for NEXICART-2 trial in AL Amyloidosis	2Q/3Q 2025
Report NXC-201 interim clinical data in 2 unaddressed immune-mediated diseases	4Q 2025
Report final topline clinical data readout for NEXICART-2 trial in AL Amyloidosis	2Q/3Q 2026

Completed	
NASDAQ IPO 2021	✓
Formed Cell Therapy R&D taskforce in 2022	✓
Secured global commercial rights for NXC-201 from Hadassah/Bar-Ilan in 2022	✓
Reported NEXICART-1 AL Amyloidosis interim clinical data at: ASGCT 2023	✓
ASH 2023	✓
ASGCT 2024	✓
Dosed first US patient in NEXICART-2 AL Amyloidosis clinical trial	✓ Met mid-2024 guidance



Pipeline: Only CAR-T in AL Amyloidosis; Tailor-Made for select Immune-Mediated Diseases



Lead Program: NXC-201, a next-generation BCMA-targeting CAR-T for AL Amyloidosis and select immune-mediated diseases

Indication	Therapy	Pre-clinical	Phase 1	Phase 2	Upcoming Milestones
Relapsed/Refractory AL Amyloidosis	NXC-201	US FDA and EU EC Orphan Drug Designation (ODD)			<p>4Q 2024: Initial NEXICART-2 clinical data presentation in AL Amyloidosis</p> <p>2Q/3Q 2025: Report interim clinical data readout for NEXICART-2 trial in AL Amyloidosis</p> <p>2Q/3Q 2026: Report final topline clinical data readout for NEXICART-2 trial in AL Amyloidosis</p>
Undisclosed select Immune-Mediated Diseases	NXC-201	IND enabled			<p>4Q 2025: Report NXC-201 interim clinical data in 2 unaddressed immune-mediated diseases</p>

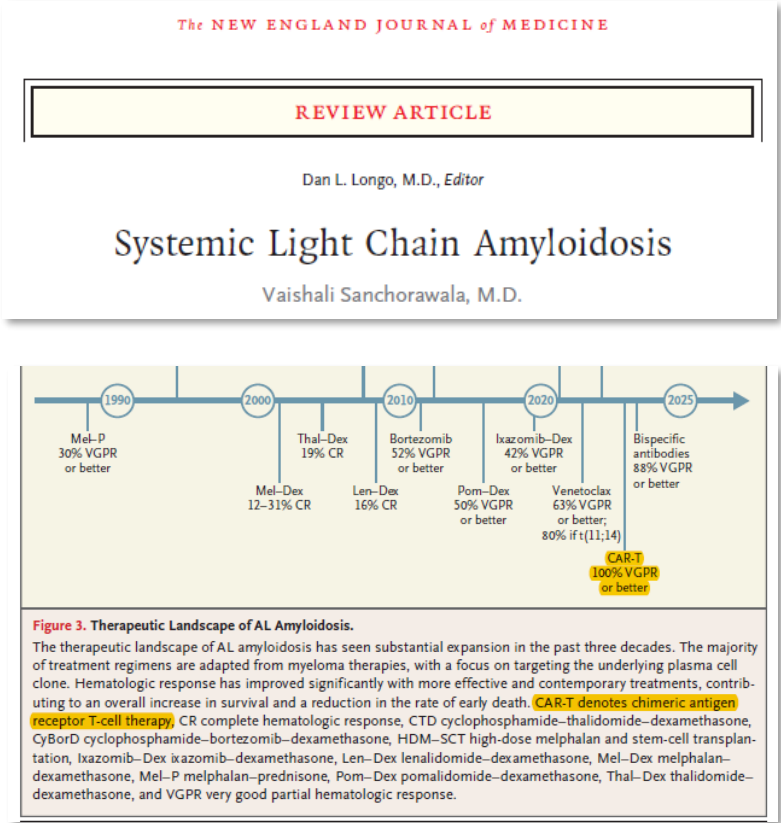
Other Emerging Pipeline

Preclinical Candidates	Not yet announced				
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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.



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 ECMA-CART (HB10101) 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.

World-Class Team



Leadership



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Gabriel Morris, BA
Chief Financial Officer



Graham Ross, MBChB, FFPM
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Director, Amyloidosis Program



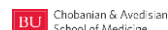
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Autoimmune CAR-T Pioneer



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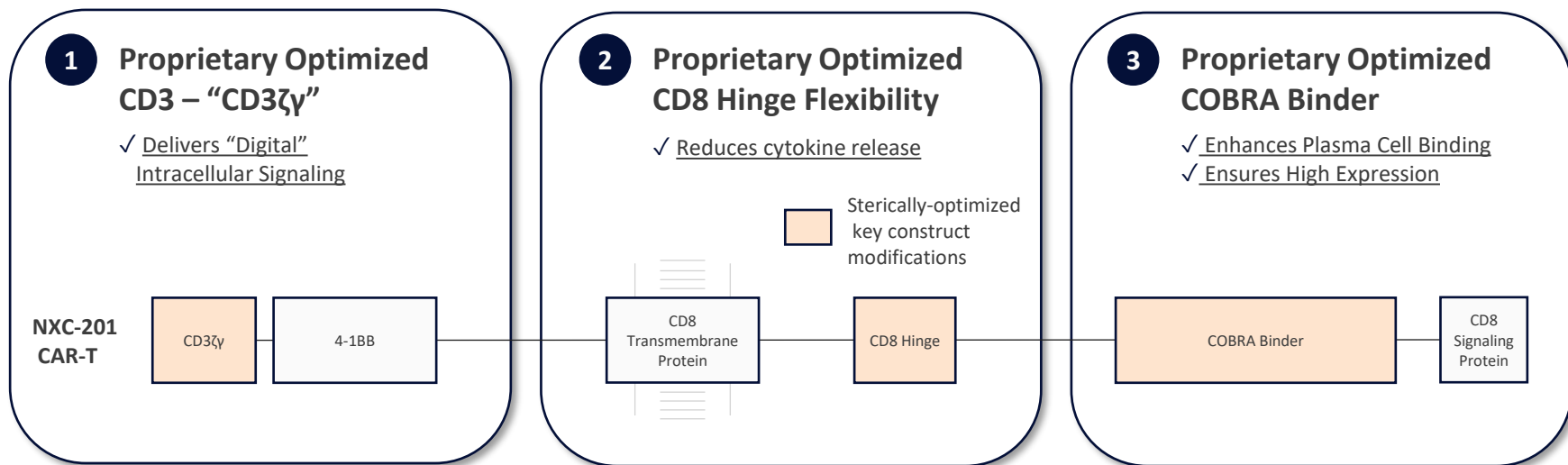


N-GENIUS Platform: Sterically-Optimized CAR-T Construct Drives Differentiated NXC-201 Clinical Profile

ALL BCMA CAR-TS ARE NOT CREATED EQUAL

N-GENIUS PLATFORM

Our CAR-T is differentiated based on 3 Key Construct Modifications



Immix’s proprietary N-GENIUS Platform can overcome the inherent limitations of conventional CAR-T methodologies that lack the sterically-optimized CD3ζγ (“Digital” Signaling), CD8 hinge flexibility, and COBRA binder, which drive NXC-201’s greatly reduced neurotoxicity, minimized CRS duration, and enhanced efficacy in heavily pretreated patients

“Single amino acid substitutions at key sites can affect CAR-T function over 200-fold range”

Source: M. Assayag, et al. Academic BCMA-CAR-T cells (HB0101), 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. Sadoun, 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.

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

CD3ζ

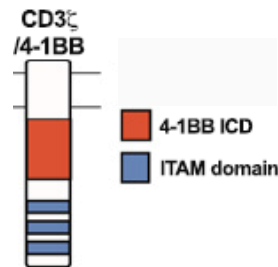
CARs rely on activation of CAR-T cells through CD3ζ derived immunoreceptor tyrosine-based activation motifs (ITAMs), typically 3 ITAM motifs per CAR

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 than 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

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

216,058

>(90%)

8,016

IFNγ (pg/mL): K562-BCMA co-culture

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

Abecma in MM

NXC-201 in MM

Literature: Optimized (Decreased) CD8 Hinge Flexibility Resulted In

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

4,050

>(60%)

1,628

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.

Kymriah

CD19-BBz(86)

Efficacy	ORR (%)	72	95
	CR (%)	28	56
CRS	CRS, any grade (%)	85	96
	CRS, Grd3+ (%)	9	14
	Duration, CRS (days)	7	1 at 800M
Neurotoxicity	Neurotoxicity, Grd1-5 (%)	28	0

Kymriah	52	73
	40	55
CD19-BBz(86)	58	28
	22	0
7	7	n/a
	21	0

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

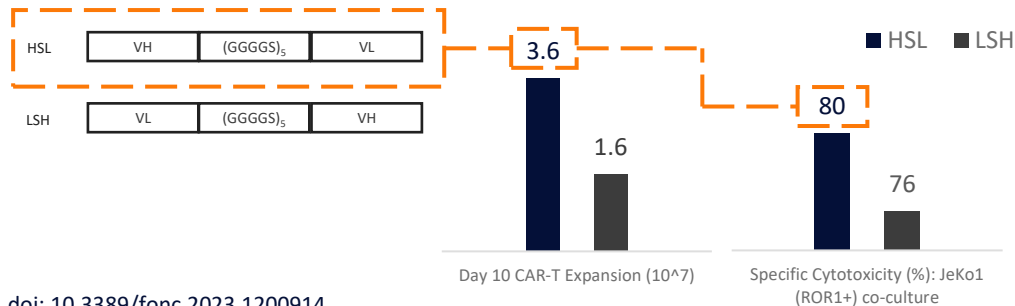
MM: multiple myeloma

Source: E. Lebel, et al. Safety And Efficacy of a Locally Produced Novel Anti-BCMA Chimeric Antigen Receptor T-Cell (CAR-T) (HB101) for the Treatment of Relapsed and Refractory Multiple Myeloma. 65th ASH Annual Meeting and Exposition, San Diego, CA, December 2023. 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: Predelineal 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

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

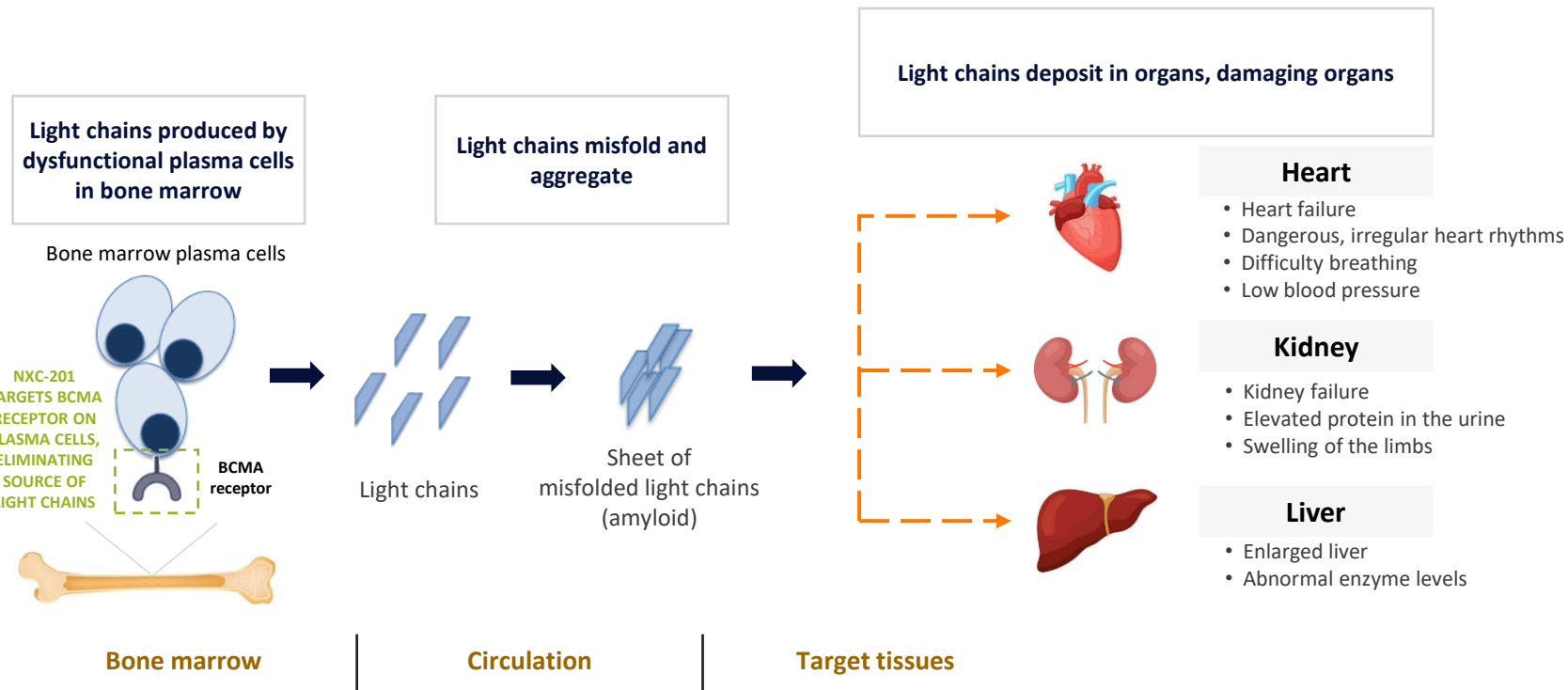
doi: 10.1186/s40364-022-00417-w

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

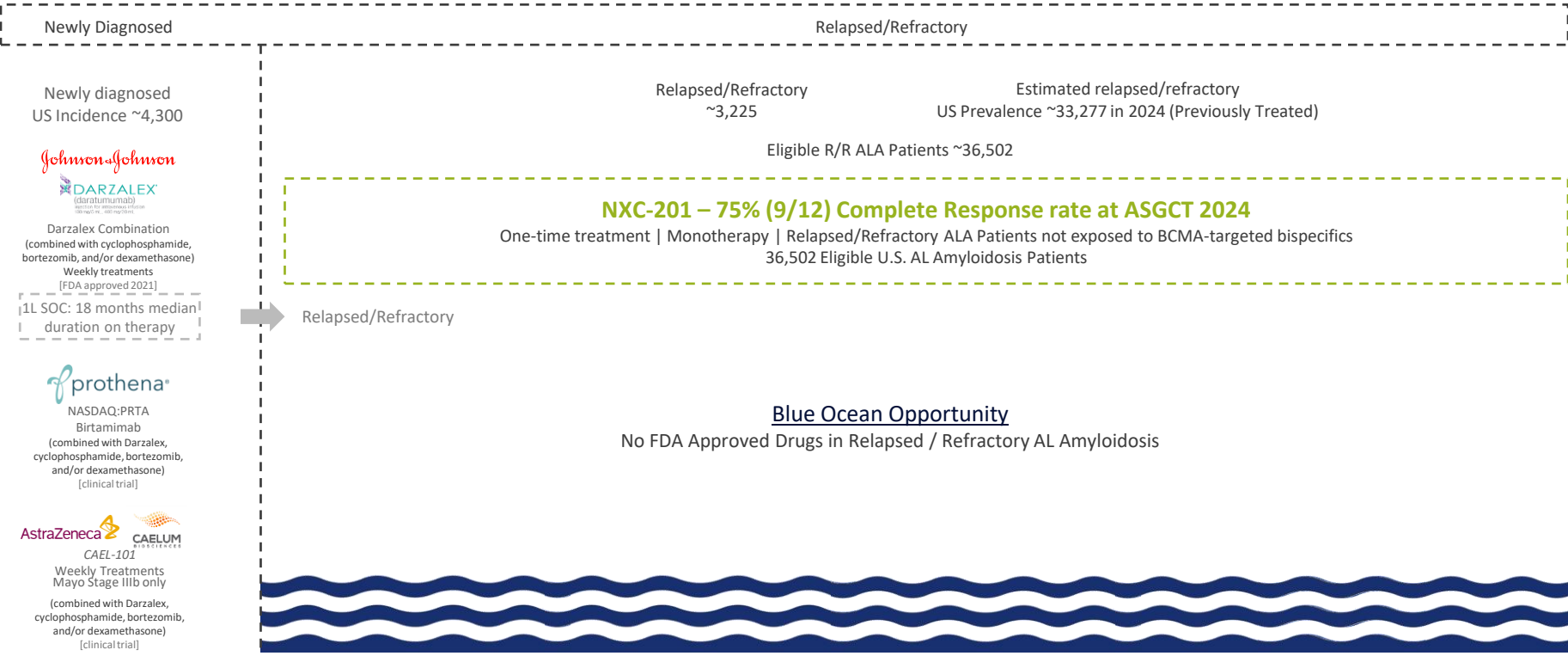
- ✓ Rapid, Sustained
CAR-T Expansion
- ✓ Improved Cytotoxicity in
the presence
of antigen

AL Amyloidosis: ~33,000 relapsed/refractory U.S. Patients With No FDA approved drugs

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



NXC-201 Addresses Sizable U.S. Relapsed/Refractory AL Amyloidosis Patient Population (1/2)



Note: Public information development plans as of 2023. Dara-CyBorD: Daratumumab, Bortezomib + cyclophosphamide + dexamethasone. BMD: bortezomib, melphalan, and dexamethasone. Source: Dima D, et al. Diagnostic and Treatment Strategies for AL Amyloidosis in an Era of Therapeutic Innovation. JCO Oncol Pract. 2023; Jimenez-Zepeda VH, et al. Understanding real-world treatment patterns and clinical outcomes in AL amyloidosis patients diagnosed in Canada: A population-based cohort study. EJHaem. 2022; Bou Zerdan M, et al. Systemic AL amyloidosis: current approach and future direction. Oncotarget. 2023; Kumar S, et al. Revised prognostic staging system for light chain amyloidosis incorporating cardiac biomarkers and serum free light chain measurements. J Clin Oncol. 2012; Browning S, et al. Hematologic relapse in AL amyloidosis after high-dose melphalan and stem cell transplantation. Blood. 2017; Palladini G, et al. Presentation and outcome with second-line treatment in AL amyloidosis previously sensitive to nontransplant therapies. Blood. 2018; 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;3(10):1046-1053. doi: 10.1182/bloodadvances.2018016402. PMID: 29748430; PMCID: PMC5965052. Sharon A, et al. Marked progress in AL amyloidosis survival: a 40-year longitudinal natural history study. Blood Cancer J. 2021;11(8):139; Lu R, Richards TA. AL Amyloidosis: Unfolding a Complex Disease. J Adv Pract Oncol. 2019;10(8):813-825. EQ Crusoe, E Kastiris, V Santhorawala, GMOBOTAS Group. Subcutaneous Daratumumab + Bortezomib, Cyclophosphamide, and Dexamethasone (Vcd) in Patients with Newly Diagnosed Light Chain (AL) Amyloidosis: Updated Results from the Phase 3 Andromeda Study. Hematology, Transfusion and Cell Therapy. Volume 43, Supplement 1, 2021. <https://doi.org/10.1016/j.htct.2021.10.364>.

NXC-201 Addresses Sizable U.S. Relapsed/Refractory AL Amyloidosis Patient Population (2/2)

Prevalence

Incidence

Standard of Care

1st Line

Newly diagnosed AL Amyloidosis
US Incidence ~4,300

75% Relapsed/Refractory

Estimated Relapsed/Refractory
US Prevalence ~33,277 in 2024

+

Relapsed/Refractory ~3,225

**Relapsed/
Refractory**
NXC-201

US Total Relapsed/Refractory patient
population ~36,502

✗ 8% long-term
remission from SCT

✗ 16% Mayo
Stage 3b

**27,741 US Relapsed/Refractory AL
Amyloidosis patients eligible for
treatment with NXC-201 in 2024**

1st Line

**Autologous stem cell transplant or
daratumumab + CyBorD (four drug combo)**
(± clinical development candidates Birtamimab / CAEL-101)

1L SOC: 18 months median duration on therapy
Darzalex + CyBorD (four drug combo)
becoming Relapsed/Refractory

**No FDA approved treatments for
Relapsed/Refractory AL Amyloidosis**

**Relapsed/
Refractory**
NXC-201

Note: 8% long-term remission estimated based on 20% eligible for SCT x 40% achieving CR (associated with superior long survival)

Source 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. Staron A, et al. Marked progress in AL amyloidosis survival: a 40-year longitudinal natural history study. *Blood Cancer J*. 2021;11(8):139; Lu R, Richards TA. AL Amyloidosis: Unfolding a Complex Disease. *J Adv Pract Oncol*. 2019;10(8):813-825. Santhorawala V, Sun F, Quillen K, Sloan JM, Berk JL, Seldin DC. Long-term outcome of patients with AL amyloidosis treated with high-dose melphalan and stem cell transplantation: 20-year experience. *Blood*. 2015 Nov 12;126(20):2345-7. doi: 10.1182/blood-2015-08-662726. Epub 2015 Oct 6. PMID: 26443620. Kumar S, Dispenzieri A, Lacy MQ, Hayman SR, Buadi FK, Colby C, Laumann K, Zeldenzust SR, Leung N, Dingli D, Greipp PR, Lust JA, Russell SJ, Kyle RA, Rajkumar SV, Gertz MA. Revised prognostic staging system for light chain amyloidosis incorporating cardiac biomarkers and serum free light chain measurements. *J Clin Oncol*. 2012 Mar 20;30(9):989-95. doi: 10.1200/JCO.2011.38.5724. Epub 2012 Feb 13. PMID: 22331953; PMCID: PMC3075680. EQ Cruone, E Kastritis, V Santhorawala, GIMBOTAS Group. Subcutaneous Daratumumab + Bortezomib, Cyclophosphamide, and Dexamethasone (Vcd) in Patients with Newly Diagnosed Light Chain (AL) Amyloidosis: Updated Results from the Phase 3 Andromeda Study. *Hematology, Transfusion and Cell Therapy*. Volume 43, Supplement 1, 2021. <https://doi.org/10.1016/j.htct.2021.10.364>.

NXC-201 May Be a Curative Treatment for AL Amyloidosis

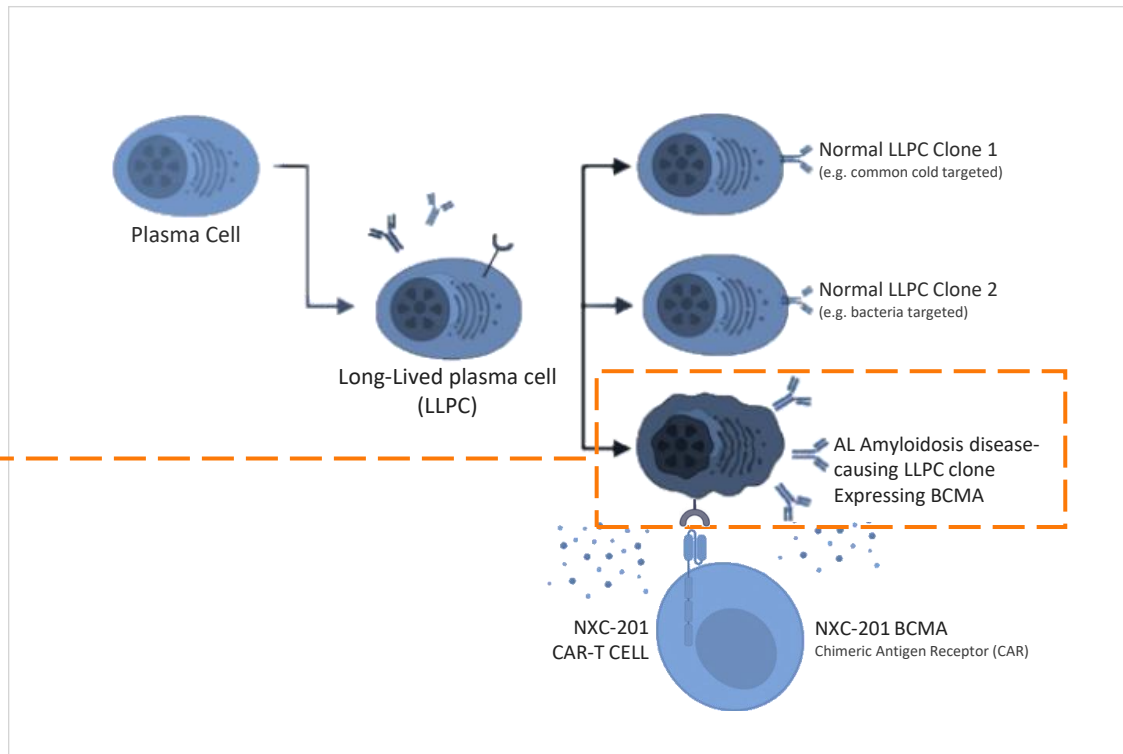
ALL BCMA CAR-TS ARE NOT CREATED EQUAL

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



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

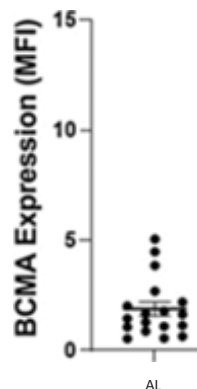
ALL BCMA CAR-TS ARE NOT CREATED EQUAL



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:

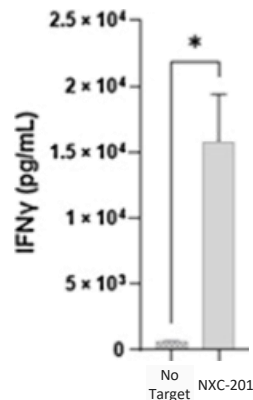
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BCMA is expressed in 18 AL Amyloidosis patients at a low-medium level...

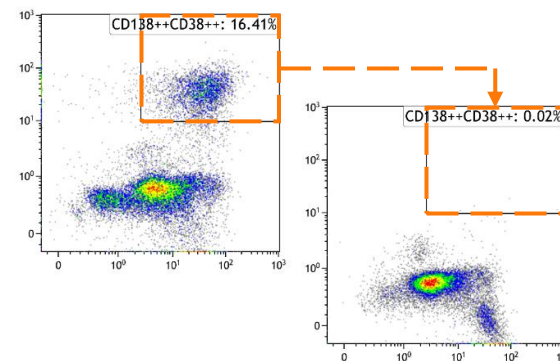


2

...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.

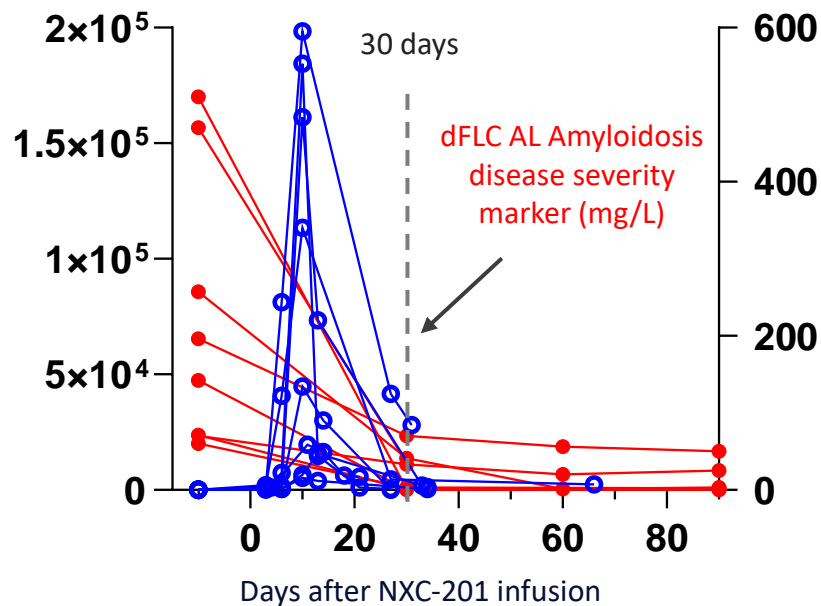
Note: NXC-201 (formerly NB10101). Infy TNFa for two patients, AL1, AL2. Far right top right quartile selected diseased AL Amyloidosis plasma cell elimination. Far right graph after 33 days co-incubated with NXC-201.

Source: Kfir-Erenfeld S, et al. Feasibility of a Novel Academic BCMA-CAR-T (HB10101) for the Treatment of Relapsed and Refractory AL Amyloidosis. Clin Cancer Res. 2022; Raju N, et al. Anti-BCMA CAR-T-Cell Therapy bb2121 in Relapsed or Refractory Multiple Myeloma. N Engl J Med. 2019;

Primary Efficacy Endpoint for NEXICART-2: Normalization of Diseased Free Light Chains 30 Days after Dosing

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

—○— NXC-201 /mL Blood —●— dFLC (mg/L)



NEXICART-1 Clinical Trial Data. Each line represents 1 patient clinical data readout after NXC-201
*dFLC (=involved free light chain - uninvolved free light chain), an AL amyloidosis disease severity marker



The NEW ENGLAND
JOURNAL of MEDICINE

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

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

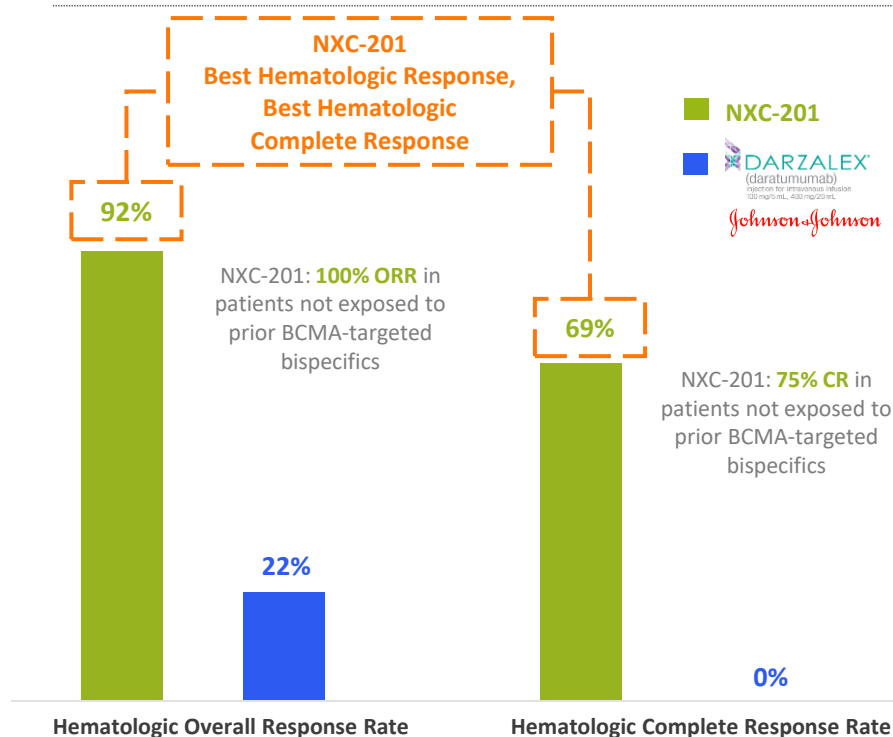
doi: 10.1056/NEJMra2304088

NXC-201: High Complete Response Rate in R/R AL amyloidosis (\$3bn market) at ASGCT 2024

APPROVAL OF DARZALEX CyBorD IN FRONT-LINE AL AMYLOIDOSIS BASED ON COMPLETE RESPONSE RATE



Relapsed/Refractory AL Amyloidosis



NXC-201 – NEXICART-1 Clinical Data

Only CAR-T in AL Amyloidosis

100% Overall Response Rate and 75% Complete Response Rate in Relapsed/Refractory AL amyloidosis for patients without prior BCMA-targeted bispecifics exposure

(median 4 lines of prior therapy prior to NXC-201 – all including Darzalex)

Zero Neurotoxicity of any grade in AL Amyloidosis

Ongoing potentially pivotal NEXICART-2 Trial Targeting Relapsed/ Refractory AL Amyloidosis Patients Most Likely to Benefit

NXC-201 clinical data indicate that R/R Amyloidosis patients with better pre-existing cardiac status and no prior BCMA-targeted bispecifics exposure are more likely to benefit from treatment

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

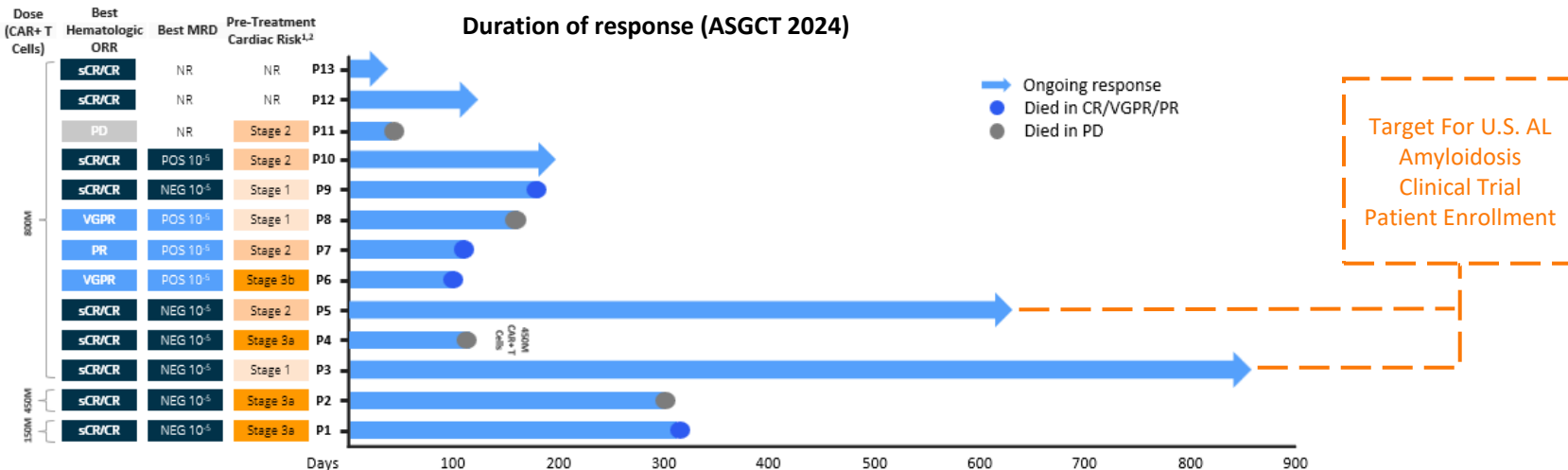
40 patient, single-arm, open-label US trial → submit data to FDA

NEXICART-1: Durable Responses in NXC-201 AL Amyloidosis Clinical Trial

ONGOING U.S. TRIAL TARGETING R/R AL AMYLOIDOSIS PATIENTS MOST LIKELY TO BENEFIT FROM DURABLE RESPONSES



- Complete hematologic response (CR) of 69% (9/13), and CR 75% (9/12) in patients without prior exposure to BCMA-targeted bispecific, a precedent approval endpoint based on the only commercial treatment for AL amyloidosis
- Ongoing NEXICART-2 US trial targeting patients reflective of durable responders



¹All but one (patient 9) had multi-organ involvement

² MAYO staging

sCR: strict complete response, CR: complete response

Darzalex FDA label. Assayag, et al. Academic BCMA-CART cells (HB0101), 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. E. Lebel, M.E. Gatt et al. Feasibility of a Novel Academic Anti-BCMA Chimeric Antigen Receptor T-Cell (CART) (HB0101) for the Treatment of Relapsed and Refractory AL Amyloidosis, 65th ASH Annual Meeting and Exposition, San Diego, CA, October 2023.

NEXICART-1: NXC-201 N-GENIUS Platform “Single-Day CRS” Drives AL Amyloidosis Leadership

ALL BCMA CAR-TS ARE NOT CREATED EQUAL

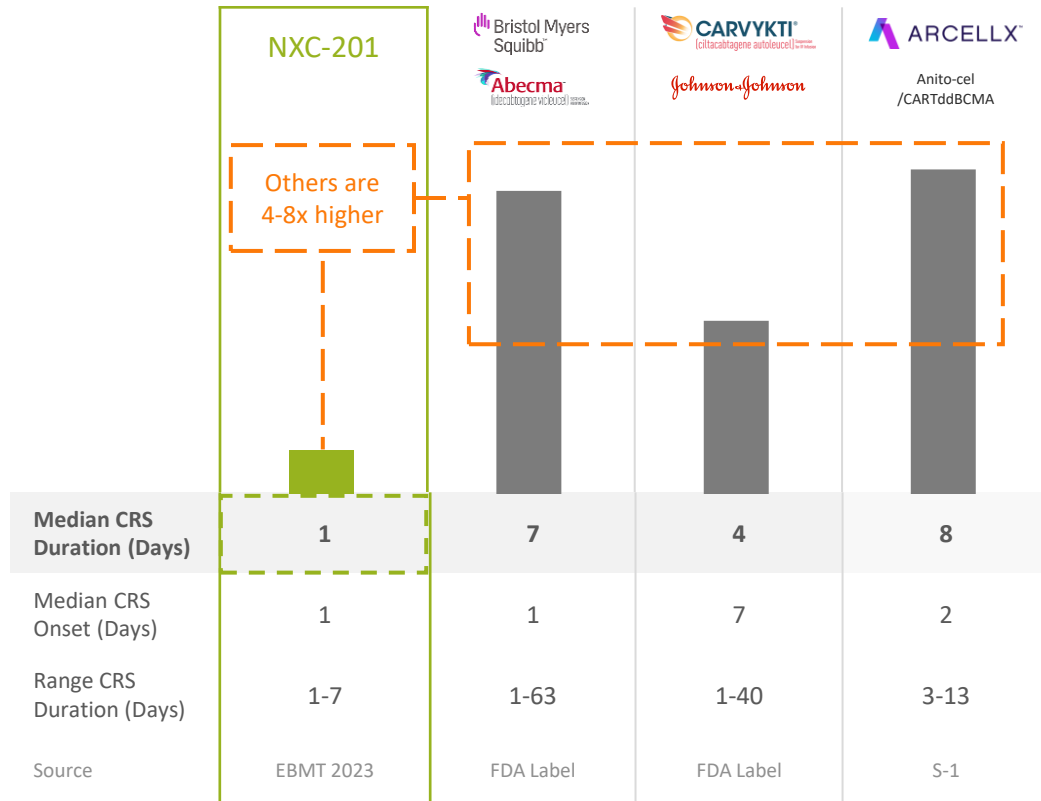
NXC-201’s short CRS duration makes it **uniquely suitable to treat ALA patients** (in whom the #1 source of mortality is heart failure)

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



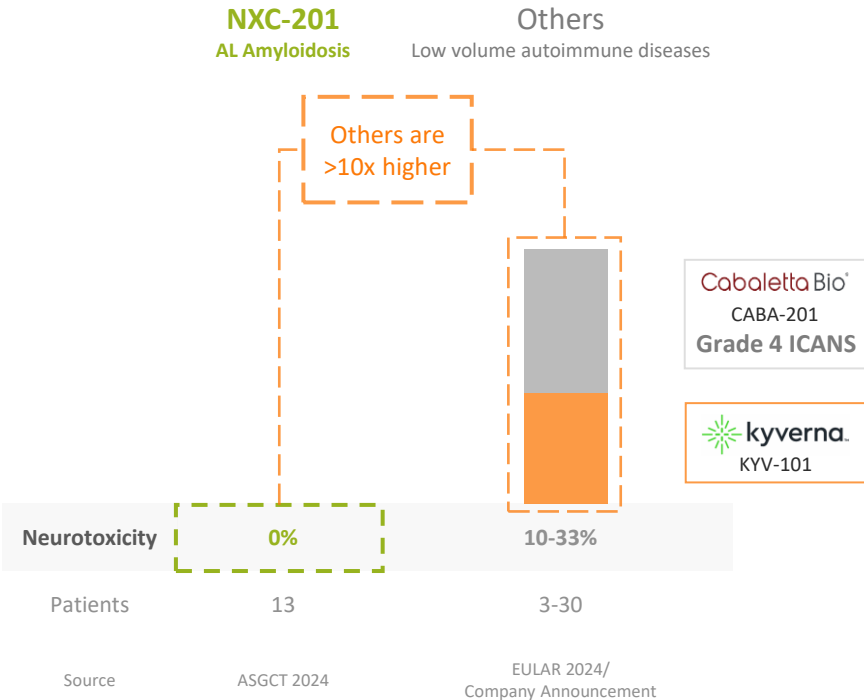
Data in Multiple Myeloma

NEXICART-1: Overcoming Neurotoxicity

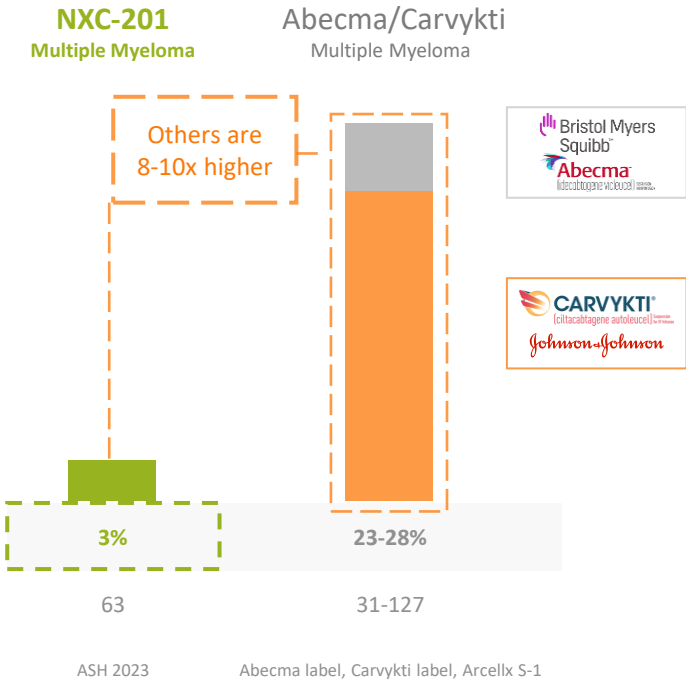
ALL BCMA CAR-TS ARE NOT CREATED EQUAL



LOW VOLUME DISEASE



HIGH VOLUME DISEASE



Source: Carvykti and Abecma FDA labels, Arcellx S-1. Assayag, et al. Academic BCMA-CAR-T cells (HB101), 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 (CAR-T) (HB101) 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/edgar/data/0001994702/0000995017024073312/kyv-20240614.htm>. Low volume diseases refers to ANCA vasculitis, autoimmune encephalitis, anti-synthetase syndrome, CIDP, DGLA 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>

NEXICART-2 US Relapsed/Refractory AL Amyloidosis Trial (NCT06097832)

NEXICART-2 NXC-201 US TRIAL INITIATED IN MID-2024



Study design

- Open-label, single-arm Phase 1b/2 study
- n=40 patients (majority of which expected to be enrolled in Phase 2 portion)

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 1b:
 - Safety
 - Efficacy: Hematologic response according to consensus recommendations in AL amyloidosis
- Phase 2:
 - Efficacy: Hematologic response according to consensus recommendations in AL amyloidosis
 - Safety

Status

- Lead site Memorial Sloan Kettering and other US sites started mid-2024



*Dosing informed by NEXICART-1 Israel trial in which Complete Responses in light chain Amyloidosis were observed at all dose levels: 150M, 450M

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

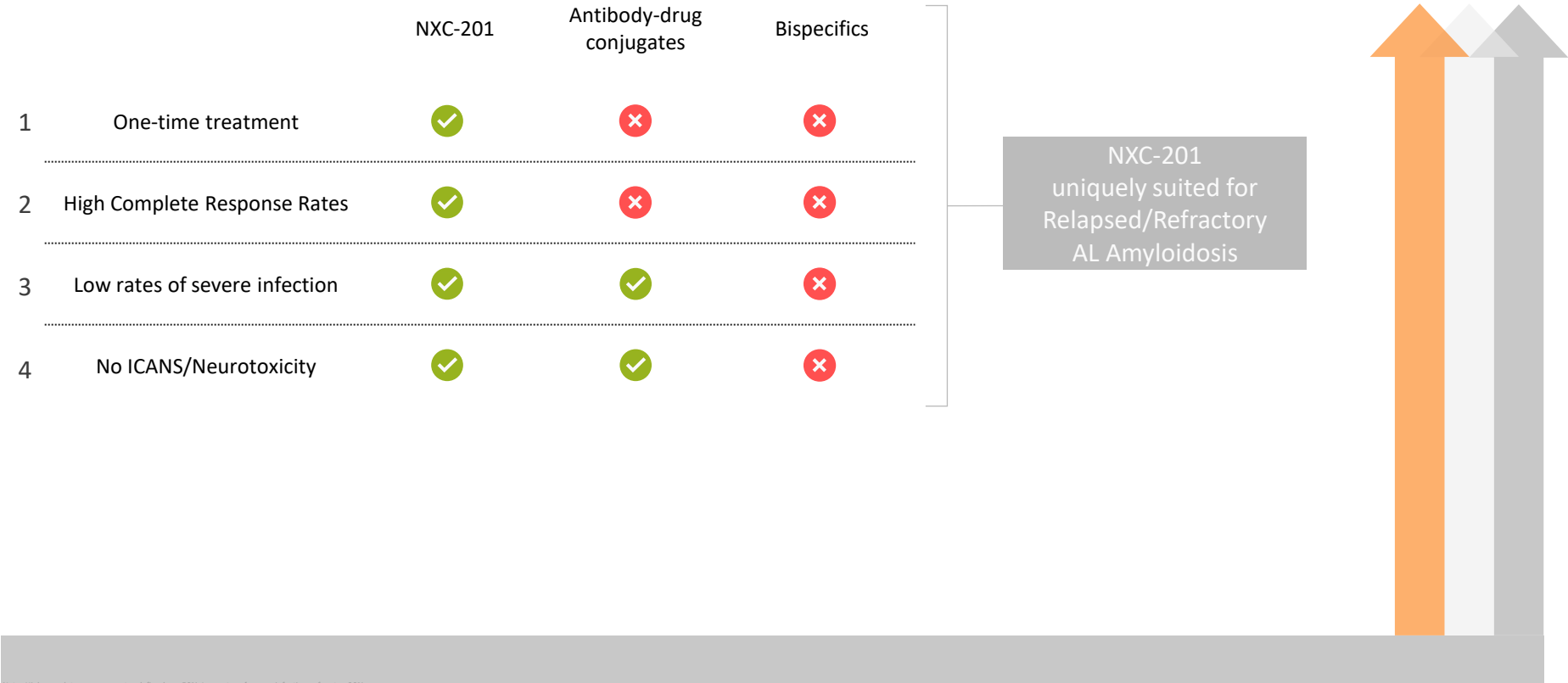
Could enrich ongoing NEXICART-2 US trial for patients more likely to benefit from therapy

Single-arm potentially pivotal NEXICART-2 trial designed considering NEXCIART-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?	✗ Randomization vs. SoC	✓ No SoC to randomize against
	Lines of therapy prior to receiving study drug	✗ 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)

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

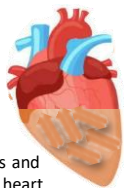


Note: High complete response rates defined as >50%. Low rates of severe infection refers to <30%
Source: Feasibility of a Novel Academic Anti-BiCMA Chimeric Antigen Receptor T-Cell (CART) (HBD101) for the Treatment of Relapsed and Refractory AL Amyloidosis, 65th ASH Annual Meeting and Exposition, San 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.

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

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)



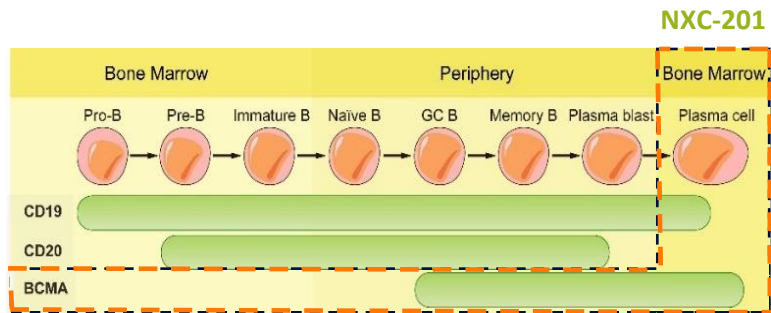
Note: select indications noted above are for illustrative purposes only.

*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 randomized phase 1b/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 Immune-Mediated Diseases

NXC-201 BCMA CAR-T TARGETS LONG-LIVED PLASMA CELLS, WHICH ARE OFF-TARGET FOR CD19 THERAPEUTICS



~80% of all auto-antibodies in immune-mediated disease are produced by long-lived 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 IMD indication selection criteria

High unmet medical need



Limited therapies in development

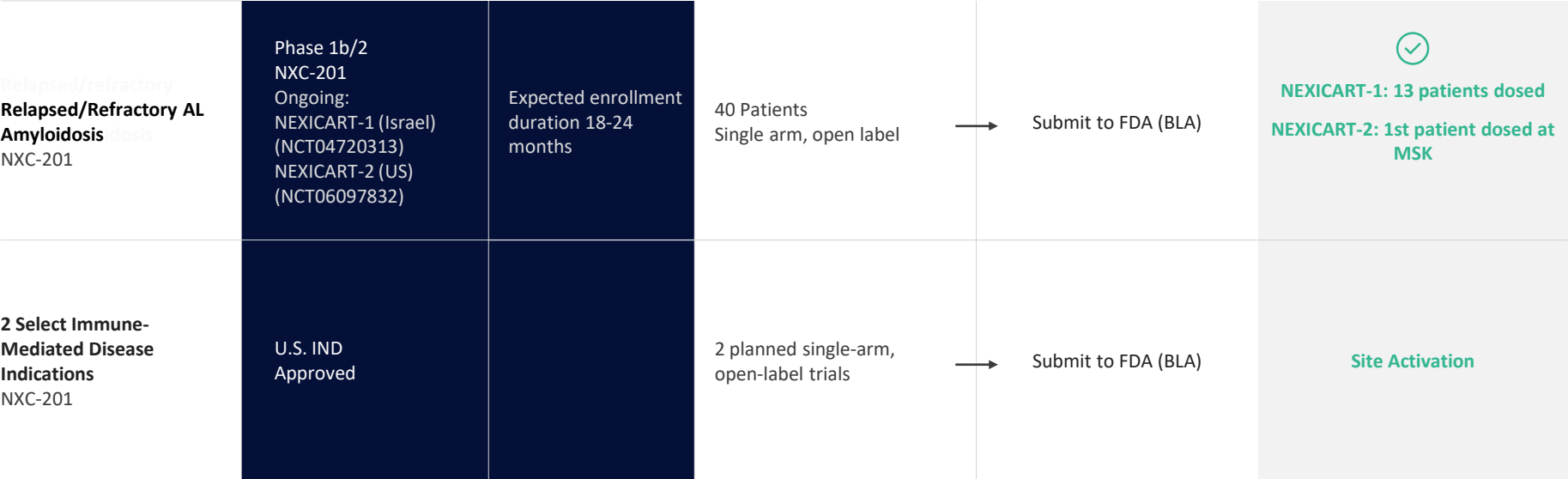


Biological basis for plasma cell-mediated therapy



NXC-201 Clinical Development Plan Through FDA BLA Submissions

RELAPSED/REFRACTORY AL AMYLOIDOSIS CLINICAL TRIAL TO ENROLL 40 PATIENTS PRIOR TO FDA BLA SUBMISSION



Note: 63 Relapsed/Refractory multiple myeloma patients have been dosed with NXC-201 in the ex-US NEXICART-1 clinical trial. Immix Biopharma is not actively pursuing US commercial approval in Relapsed/Refractory multiple myeloma at this time.

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)

Appendix

November 2024



Principal Investigator for NEXICART-2: Heather Landau, MD



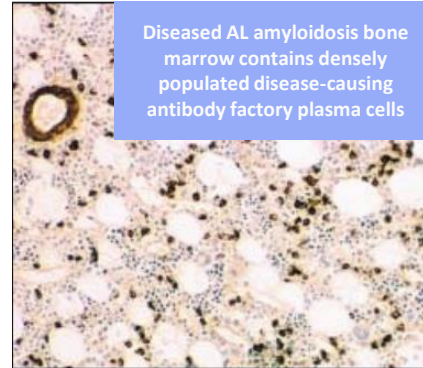
- **Director of the Amyloidosis Program and a Bone Marrow Transplant Specialist & Cellular Therapist at Memorial Sloan-Kettering Cancer Center in New York.**
- Authored more than 100 peer-reviewed publications.
- Dr. Landau received her medical degree from SUNY Upstate Medical University, completed her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



Immunoperoxidase with hematoxylin counterstain, ×100



Periodic acid-Schiff, ×100

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

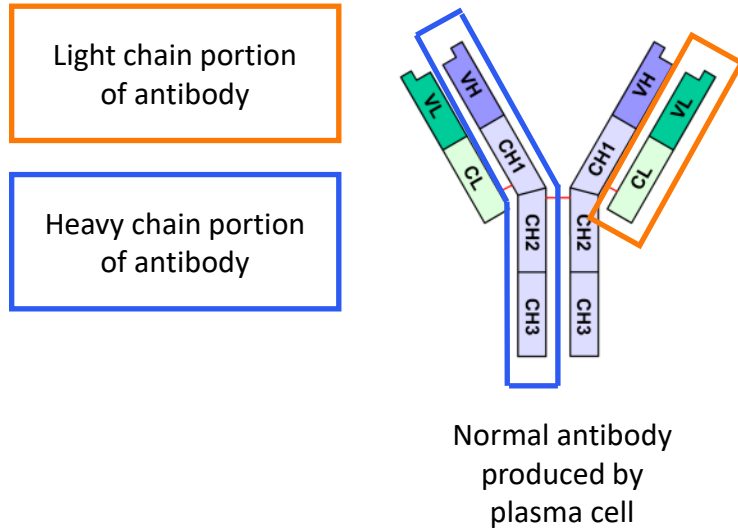
Inserm

“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”

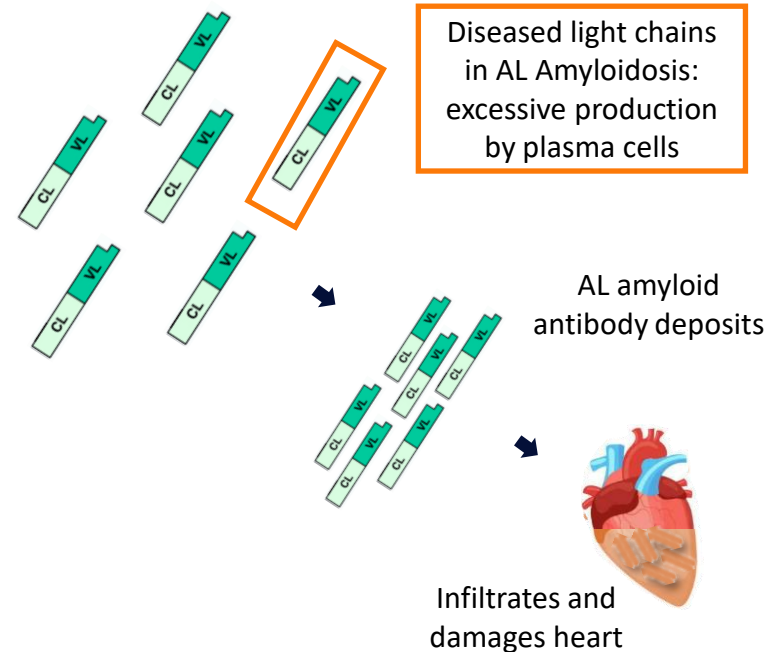
doi:10.1172/JCI65689

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



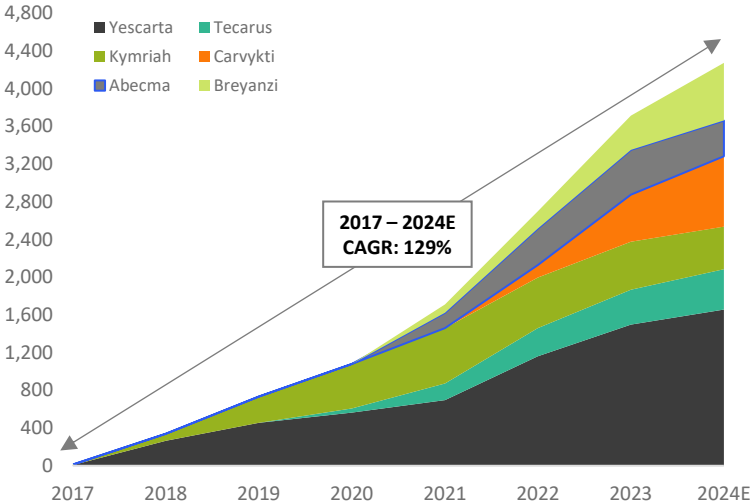
IN AL, PLASMA CELLS PRODUCE TOO MANY LIGHT CHAINS (AND NOT ENOUGH HEAVY CHAINS)



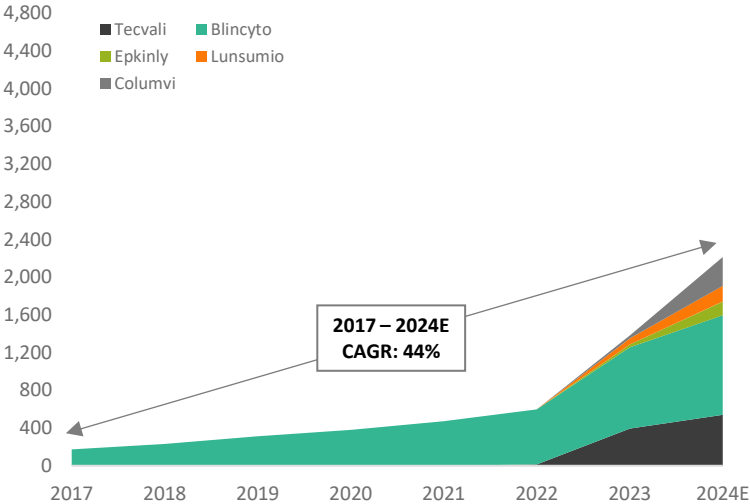
Robust Global Sales of CAR-T Continue



Sales of Approved CAR-T (\$M)



Sales of Approved Bispecifics (\$M)

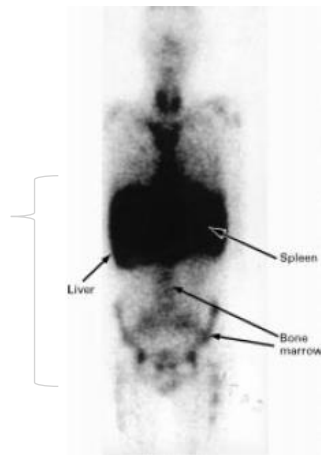


Note: Full year 2024 sales estimated based on annualized quarterly 2024 actual sales
Source: Company reports

Amyloid deposits in AL Amyloidosis are cleared naturally after treatment

BEFORE TREATMENT

Pre-treatment imaging shows dense amyloid deposits in liver, bone marrow, and other organs in AL patient

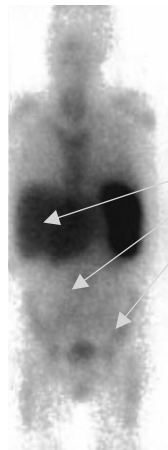


Cytotoxic treatments
(vincristine,
adriamycin, and
dexamethasone)



6-MONTHS AFTER TREATMENT

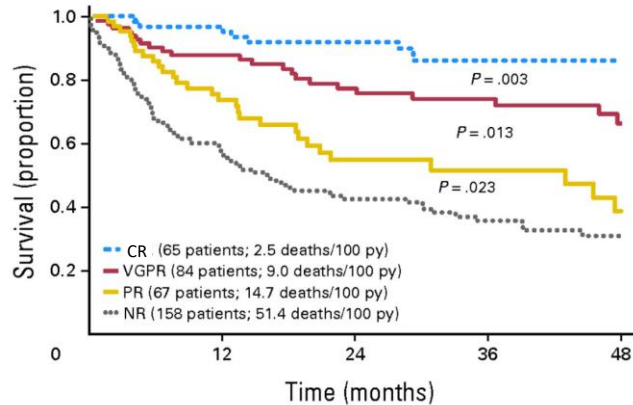
Post-treatment imaging shows clearance of amyloid in organs throughout the body



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

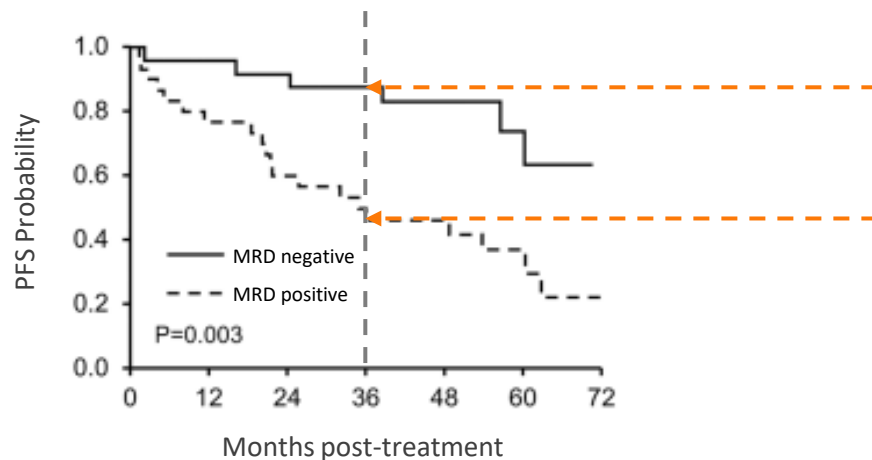


2x survival at 48 months for CR vs PR

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

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

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
<ul style="list-style-type: none">No clinical trials with clinical data available in relapsed/refractory AL amyloidosisEarly data from select centers indicates bispecific responses and tolerability are inferior to CAR-T (NXC-201) in relapsed/refractory AL amyloidosisRetrospective study with 17 R/R multiple myeloma + AL Amyloidosis patients:<ul style="list-style-type: none">✗ 41% CR✗ 35% severe infections including death✗ Grade 3 ICANS neurotoxicity reportedRepeat/ongoing dosing with need for healthcare provider to administer	<ul style="list-style-type: none">✓ 75% CR in relapsed/refractory AL amyloidosis in patients with no prior BCMA-targeted bispecifics exposure✓ 0 deaths from infection in relapsed/refractory AL amyloidosis✓ 0% neurotoxicity (0/13) in relapsed/refractory AL amyloidosis patients• One-time dosing with durable responses• Ongoing NEXCART-1 relapsed/refractory AL amyloidosis clinical trial with clinical data presented at ASGCT 2024

Advantages of NXC-201 CAR-T in AL Amyloidosis

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 US patents, pending applications, and exclusive patent licenses, which cover our core technology platforms and product



N-GENIUS Platform has produced sterically-optimized BCMA CAR-T NXC-201 with COBRA binder and EXPAND technology

ALL BCMA CAR-TS ARE NOT CREATED EQUAL



N-GENIUS PLATFORM

3 Key Elements



Purpose-Built Cell Therapy Evidence Capture Engine + Relational Database

Relating ImmixBio internal data to external to accelerate therapy design, manufacture, and preclinical



Proprietary EXPAND technology

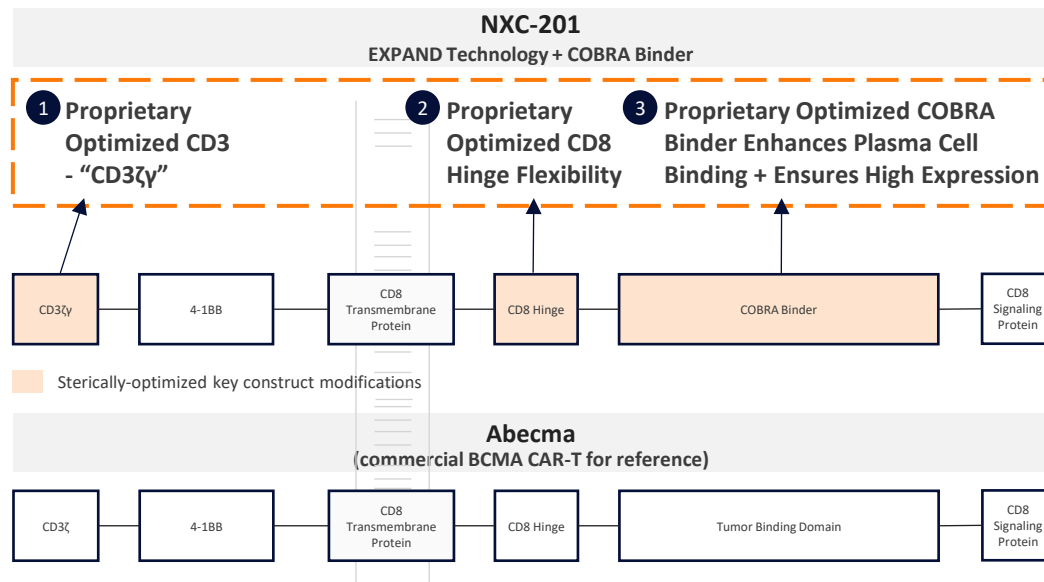
Applied to multiple cell therapy indications, already utilized to create NXC-201, to potentially increase efficacy and tolerability



Atomized, Novel Binding Scaffold Generation Engine

Allows us to make the correct binding for every molecule

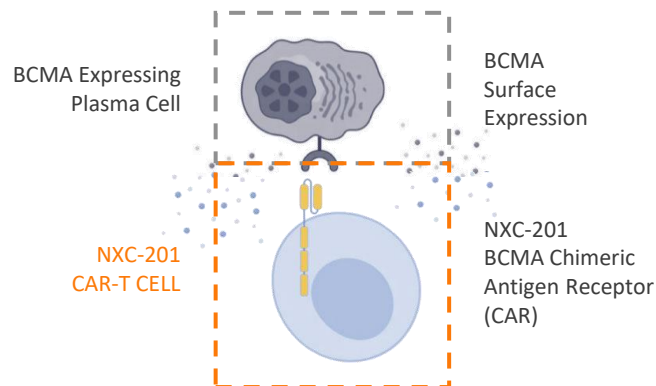
Produced NXC-201



NXC-201 MoA: Sterically-Optimized BCMA-targeted CAR-T

NXC-201: STERICALLY-OPTIMIZED BCMA CAR-T GENERATED BY THE N-GENIUS PLATFORM

NXC-201



NXC-201 is a next-generation chimeric antigen receptor (CAR) T-cell (CAR-T) produced by our N-GENIUS platform targeting B-cell maturation antigen (BCMA)

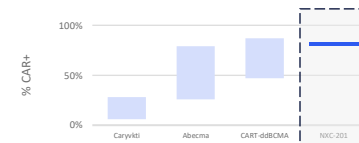
- High Overall Response Rates in AL Amyloidosis and Multiple Myeloma
- First Outpatient CAR-T: Short CRS duration (median 1 days) starting on day 1

NXC-201 — Key Characteristics



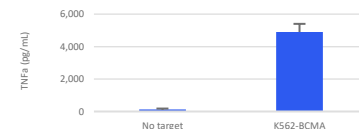
High Transduction Efficiency (Ensuring efficient manufacturing)

*Carvykti data presented at ASH 2019; Abecma data presented at ASH 2017. CART-ddBCMA source Arcellx. Analysis based on cross-trial comparisons of publicly available data reported in ASH 2017 and 2019 and not a head-to-head clinical trial



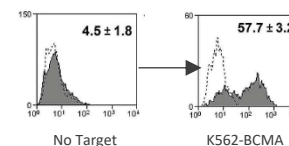
Low Tonic Signaling (Lower off-target toxicity may lead to lower toxicity)

NXC-201 was co-cultured with the indicated target T cells and TNFα (B) and IL-2 (C) concentrations secreted in the culture supernatant were determined by ELISA.



Anti-Exhaustion Capability (Increased Persistence may lead to efficacy over an extended period of time)

NXC-201 was co-cultured overnight then analyzed by flow cytometry for the expression of 4-1BB

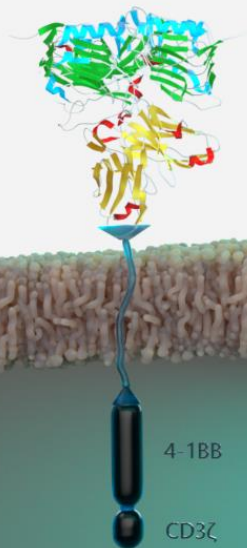


Proprietary EXPAND Technology and COBRA Binding Domain Are Differentiated Innovations

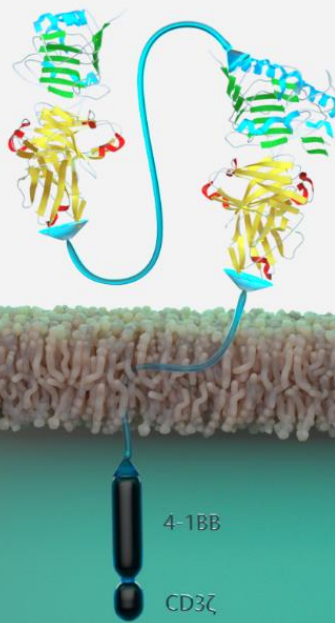
N-GENIUS TECHNOLOGY PLATFORM: STERICALLY-OPTIMIZED BCMA CAR-T NXC-201



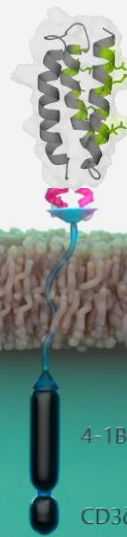
Abecma



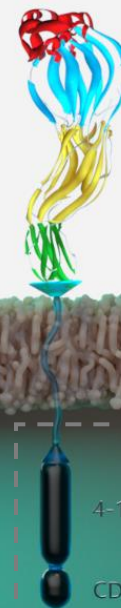
Carvykti



CART-ddBCMA



NXC-201 with
COBRA Binding Domain




N-GENIUS
EXPAND Technology

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

NXC-201 Best-in-Class AL Amyloidosis Clinical Results

Relapsed/Refractory Light chain (AL) Amyloidosis

			 	
	NXC-201 Monotherapy	Darzalex Combination (with cyclophosphamide, bortezomib, and/or dexamethasone)	CAEL-101	Birtamimab Combined with SOC CyBorD
Dosing frequency	1X	Weekly	Weekly	Weekly
Patient #s: n=	12	9	10 (renal), 24 (cardiac)	14 (cardiac), 15 (renal)
Hematologic Overall Response Rate	100%	22%	-	-
Hematologic Complete Response Rate	75%	0%	-	-
Hematologic CR + VGPR	92%	22%	-	-
Cardiac response – (NT-proBNP median reduction)	61%		39%	35%
Renal response (%)	67%		20%	60%
Median prior lines of therapy	4	1	2	2
% pretreated with CD38-targeted treatment (Darzalex/other)	100%	100%	?	0%

Birtamimab Source from JCO (Birtamimab development paused + restarted). CAEL-101 source: Edwards CV, et al. Phase 1a/b study of monoclonal antibody CAEL-101 (11-1F4) in patients with AL amyloidosis. Blood. 2021 Dec 23;138(25):2632-2641. doi: 10.1182/blood.2020009039. PMID: 34521113; PMCID: PMC8703360. Darzalex source from Blood. Point-of-care CART manufacture and delivery: Poster. Poster Presentation, ESBMT 2023. Poster ASGCT 2023. Figures reflect cross-trial comparison and not results from a head-to-head study. Differences exist between trial designs and subject characteristics, and caution should be exercised when comparing data across studies. Poster: IMS 2023. Poster: ASH 2023. Darzalex and Investigator's Choice : Theodorakakou, et al, Blood 2022. Astra Zeneca: Blood 2021 12 NXC-201 patients at ASGCT 2024 with no prior exposure to BCMA targeted bispecifics

Differentiated NXC-201 safety profile validated in historical Multiple Myeloma data



NXC-201 tolerability data in Relapsed/Refractory Multiple Myeloma

Cytokine release syndrome				
	Cohort 1 (N=6)	Cohort 2 (N=7)	Cohort 3 (N=50)	Overall (n=63)
Dose	150M	450M	800M	
CRS (n [%])				
Yes	5 (83%)	6 (86%)	48 (96%)	59 (94%)
No	1 (17%)	1 (14%)	2 (4%)	4 (6%)
CRS Start Day				
Median	6	0	0	
Min, Max	0, 21	0, 1	0, 4	
CRS Duration				
Median	3	2	1	
Min, Max	0, 5	1, 3	1, 7	
CRS Grade (n [%])				
No CRS	1 (17%)	1 (14%)	2 (4%)	4 (6%)
1	4 (67%)	2 (29%)	17 (34%)	23 (37%)
2	1 (17%)	4 (57%)	24 (48%)	29 (46%)
3	0 (0%)	0 (0%)	7 (14%)	7 (11%)
4	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Tocilizumab (n [%])				
Yes	2 (33%)	4 (57%)	40 (80%)	46 (73%)
No	4 (67%)	3 (43%)	10 (20%)	17 (27%)
Steroids (n [%])				
Yes	0 (0%)	0 (0%)	8 (16%)	8 (13%)
No	6 (100%)	7 (100%)	42 (84%)	55 (87%)
Vasopressors (n [%])				
Yes	0 (0%)	0 (0%)	7 (14%)	7 (11%)
No	6 (100%)	7 (100%)	43 (86%)	56 (89%)

ICANS neurotoxicity				
	Cohort 1 (N=6)	Cohort 2 (N=7)	Cohort 3 (N=50)	Overall (n=63)
Dose	150M	450M	800M	
ICANS (n [%])				
Yes	0 (0%)	0 (0%)	2 (4%)	2 (3%)
No	6 (100%)	7 (100%)	48 (96%)	61 (97%)
ICANS Grade (n [%])				
1-2	0 (0%)	0 (0%)	2 (4%)	2 (3%)
3-4	0 (0%)	0 (0%)	0 (0%)	0 (0%)

NXC-201 at 150M and 450M CAR+T cell dose
(US AL Amyloidosis trial dosing):

- 0% Grade 3+ CRS
- 0% ICANS of any grade

Relapsed/Refractory Multiple Myeloma: Key Inclusion Criteria

	NXC-201 (NEXICART-1 NCT04720313)	Abecma (pivotal NCT03361748)	Carvykti (pivotal NCT03548207)	CART-ddBCMA (pivotal NCT05396885)
Prior lines of therapy	≥3 different prior lines of therapy including proteasome inhibitor, immunomodulatory therapy and ≥1 antibody therapy, refractory/ responsive to the last line of therapy	≥3 different prior lines of therapy including a proteasome inhibitor, an immunomodulatory agent and an anti-CD38 antibody, refractory to the last treatment regimen	≥3 different prior lines of therapy including a proteasome inhibitor, an immunomodulatory agent and an anti-CD38 antibody, refractory to the last treatment regimen, refractory or non-responsive to their most recent line of therapy	≥3 different prior lines of therapy including a proteasome inhibitor, an immunomodulatory agent and an anti-CD38 antibody, refractory to the last treatment regimen
Toxicity recovery	Recovery to ≤Grade 2 or baseline of any non-hematologic toxicities due to prior treatments, excluding alopecia and Grade 3 neuropathy	Recovery to Grade 1 or baseline of any non-hematologic toxicities due to prior treatments	-	Resolution of adverse events (AEs) from any prior systemic anticancer therapy, radiotherapy, or surgery to Grade 1 or baseline
ECOG	0-2	0-1	0-1	0-1
Measurable disease	<ul style="list-style-type: none"> Serum M-protein greater or equal to 0.5 g/dL Urine M-protein greater or equal to 200 mg/24 h Serum free light chain (FLC) assay: involved FLC level greater or equal to 5 mg/dL (50 mg/L) provided serum FLC ratio is abnormal 	<ul style="list-style-type: none"> Serum M-protein greater or equal to 1.0 g/dL Urine M-protein greater or equal to 200 mg/24 h Serum free light chain (FLC) assay: involved FLC level greater or equal to 10 mg/dL (100 mg/L) provided serum FLC ratio is abnormal 	<ul style="list-style-type: none"> Serum monoclonal paraprotein (M-protein) level more than or equal to (≥) 1.0 gram per deciliter(g/dL) Urine M-protein level ≥200 milligram per 24 hours (mg/24hr) Light chain multiple myeloma without measurable disease in the serum or the urine: Serum immunoglobulin free light chain 10 mg/dL and abnormal serum immunoglobulin kappa lambda free light chain ratio 	<ul style="list-style-type: none"> Serum M-protein ≥1.0 g/dL Urine M-protein ≥200 mg/24 hours Involved serum free light chain ≥10 mg/dL with abnormal κ/λ ratio (i.e., >4:1 or <1:2)

Relapsed/Refractory Multiple Myeloma: Key Exclusion Criteria



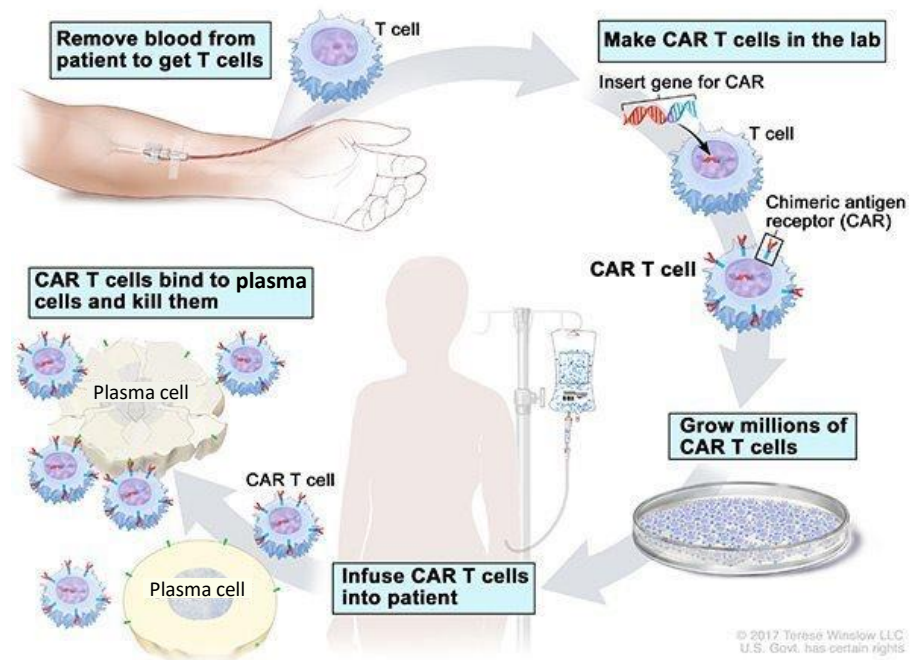
	NXC-201 (NEXICART-1 NCT04720313)	Abecma (pivotal NCT03361748)	Carvykti (pivotal NCT03548207)	CART-ddBCMA (pivotal NCT05396885)
Prior BCMA therapy	No exclusion	BCMA targeted therapy	Have received any therapy that is targeted to B-cell maturation antigen (BCMA)	Prior B-cell maturation antigen (BCMA) directed therapy
Prior cell therapy	Investigational cellular therapies within 8 weeks prior to the start of lymphodepletion	Investigational cellular therapy for cancer	Have received prior treatment with chimeric antigen receptor T (CAR-T) therapy directed at any target	Prior treatment with any gene therapy or gene-modified cellular immune-therapy

Source: Clinicaltrials.gov; Asherie N, et al. Haematologica. 2023

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

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

CAR T-cell Therapy



Patient Specific

Personalized treatment using patient's own T cells

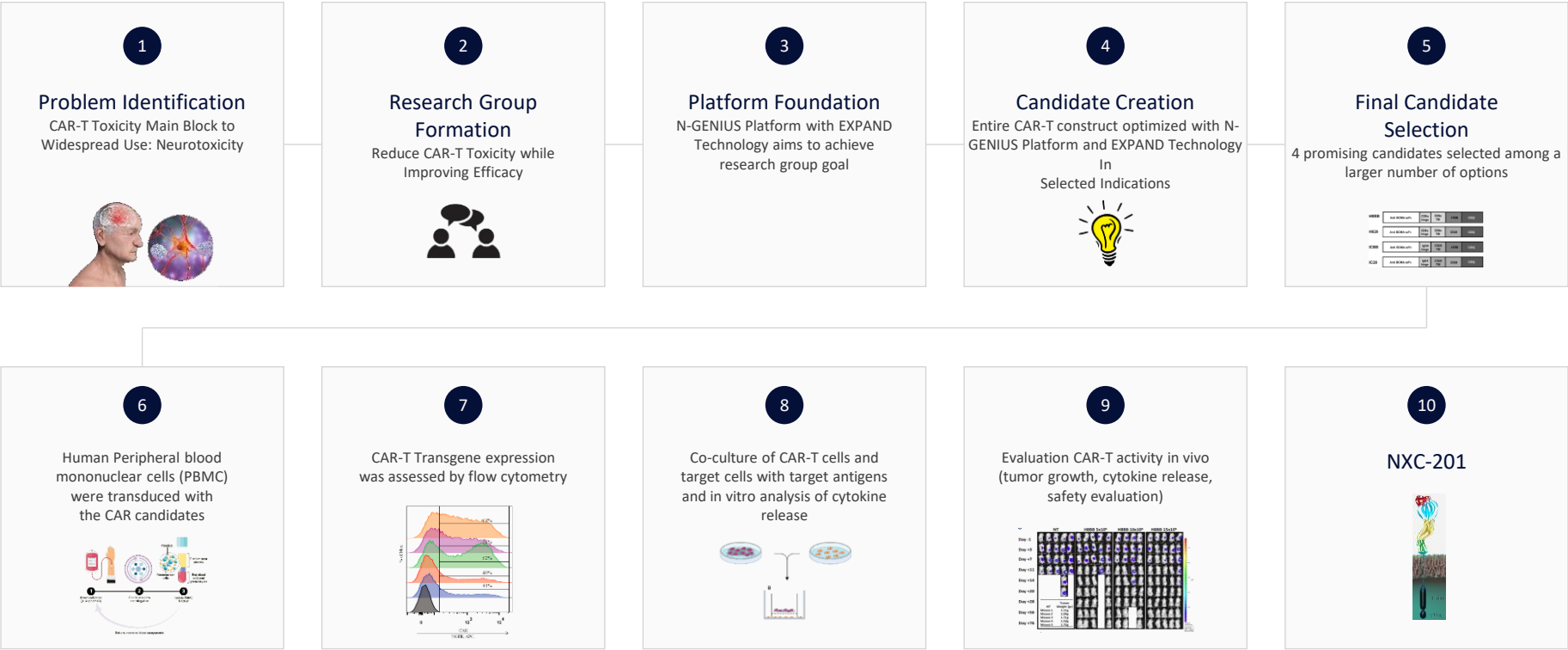
Genetic Modification

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

Targeted Therapy

Target cells that express antigens recognized by CARs

N-GENIUS Platform Process



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

Clinical Stage CAR-T for AL Amyloidosis and Select Immune-Mediated Diseases

November 2024

