Mobilizing the Patient's Immune System to Treat Serious Diseases

May 15, 2025

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Our Mission:
To Develop Therapies that Mobilize the Immune System

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Agenda and Speakers

Mobilizing the Immune System: Cue Biopharma's Novel Biologics Portfolio

Welcome – Cue's Progress and Promise	Dan Passeri, Chief Executive Officer (CEO)
CUE-401	Matteo Levisetti, MD, Chief Medical Officer
KOL Perspective – Tregs in Context	Dr. Richard DiPaolo Dr. Andrew Cope
Discussion & Q&A	Cue Management & KOLs
Closing Remarks	Dan Passeri, CEO

Dan Passeri, MSc, JD
Chief Executive Officer

Matteo Levisetti, MD
Chief Medical Officer

Richard DiPaolo, PhD
Professor and Chair, Department of Molecular Microbiology & Immunology, Saint Louis University,

Andrew Cope, MD, PhD
Head, Centre for Rheumatic Diseases, King's College, London

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Key Takeaways

Mobilizing the Immune System to Treat Serious Disease

- **Specific T cell populations** play a critical role in serious diseases
- **CUE's biologics platform** is designed to enable selective modulation of T cell populations to treat immune mediated diseases
- **Maturing clinical data show notable increases in survival for CUE-100 Series** and confirm the safety and efficacy of our IL-2 mutein in this setting
- **CUE-401 is a unique Treg inducer** with potential to become a new standard of care for autoimmune and inflammatory diseases
- **Recent Boehringer Ingelheim collaboration provides validation of our approach** to redirect anti-viral T cells to treat autoimmune disease with CUE-501

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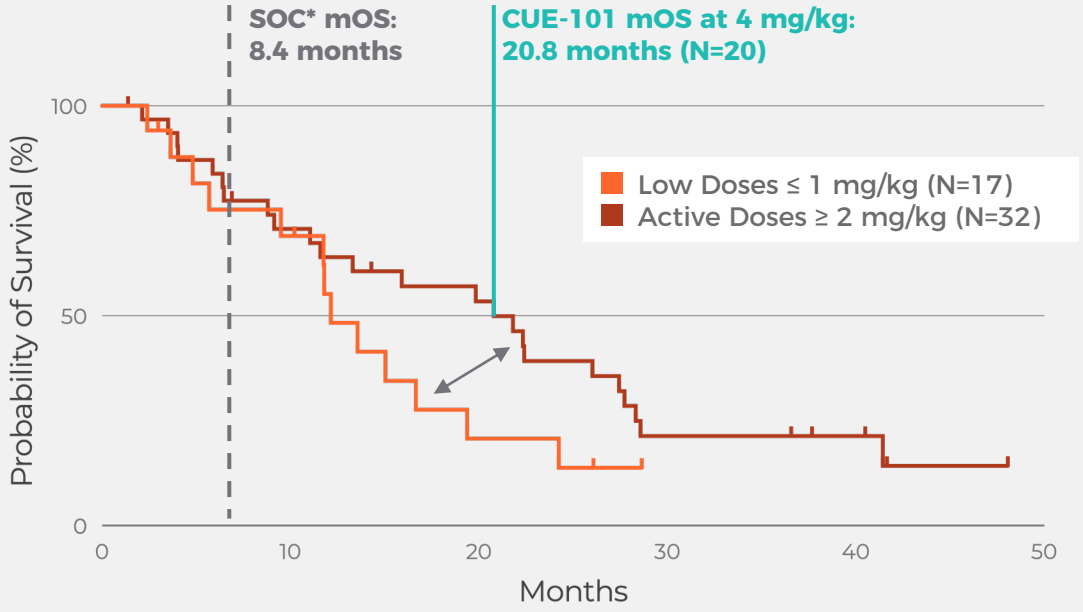
CUE-100 Series

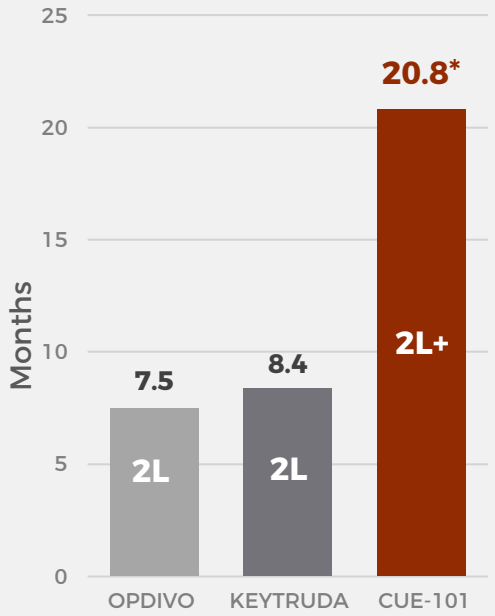
Maturing Data Shows Notable Increase in Survival

Overall Survival of CUE-101 Monotherapy



Data Extract: 04-Aug-24 from live database with active patients.
* Historic - Keytruda - Reported Median Overall Survival (mOS) for second line HNSCC patients
Cohen et al KEYNOTE-040 Lancet, 2018
Note:: Comparison to historic data is a cross-trial comparison and does not involve a head-to-head trial

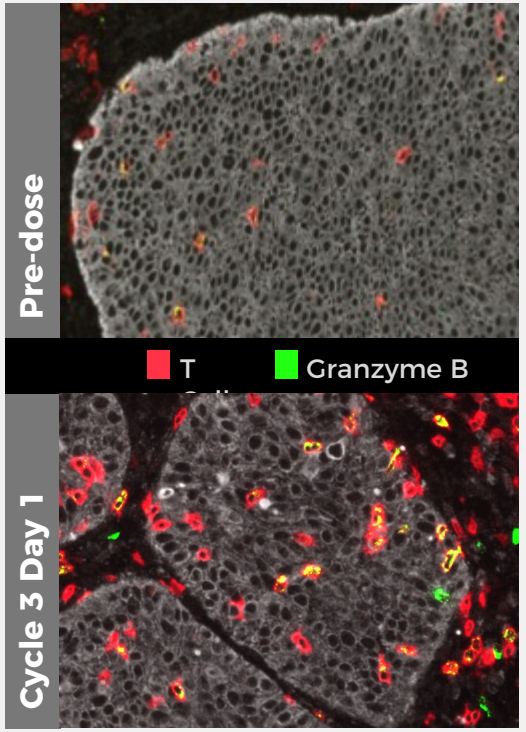
CUE-101 Median Overall Survival vs. Benchmarks[1,2]



1. Ferris et al Checkmate 141 *NEJM* 375;19, 2016
2. Cohen et al KEYNOTE-040 *Lancet*, 2018

Note:: Comparison to historic data is a cross-trial comparison and does not involve a head-to-head trial

Marked Increase in T Cell Infiltration Post-CUE-101



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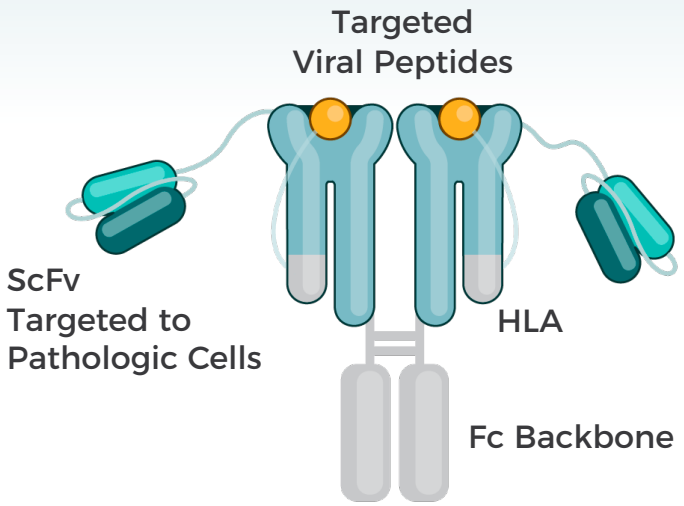
CUE-500 Series

Partnering with Boehringer Ingelheim to Validate and Accelerate Development of CUE-501



- Paints target cell with a virus-specific epitope
- Redirects anti-viral killer T cells to eradicate pathogenic B cells, cancer cells, mast cells, and others

CUE-501 Pre-IND Enabling Studies Underway

CUE-501 Deal Terms	
Upfront	$12 million
R&D Funding	Providing research support payments
Milestones and Royalties	Up to $345 million in success-based milestone payments, beginning with two preclinical development milestones, plus royalties on net sales
Focus	Autoimmune and inflammatory diseases with potential to expand into other applications where B cells play a key role

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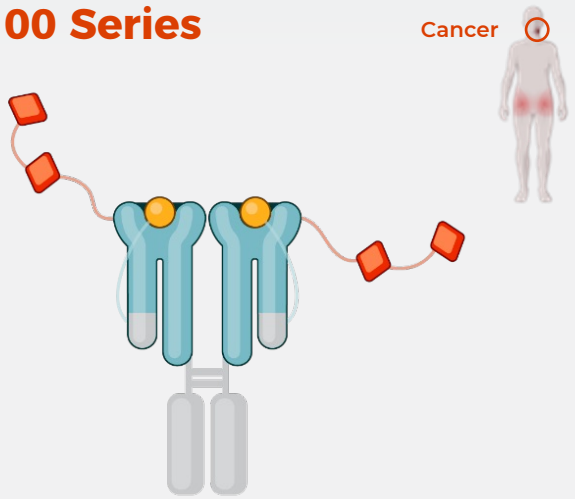
Innovative Growth Through our Platform

Redirecting Immune Repertoires as a Therapeutic Strategy

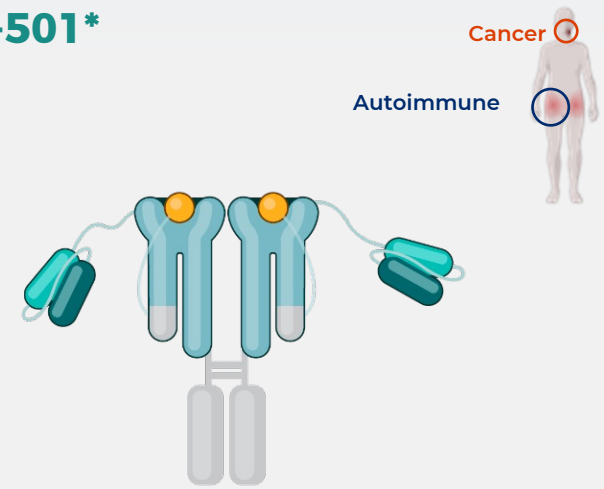
CUE-100 Series



Selectively Targeting Multiple Cancers

- Clinically de-risked
- Demonstrated Clinical Benefit: ORR and Survival
- Efficient Production

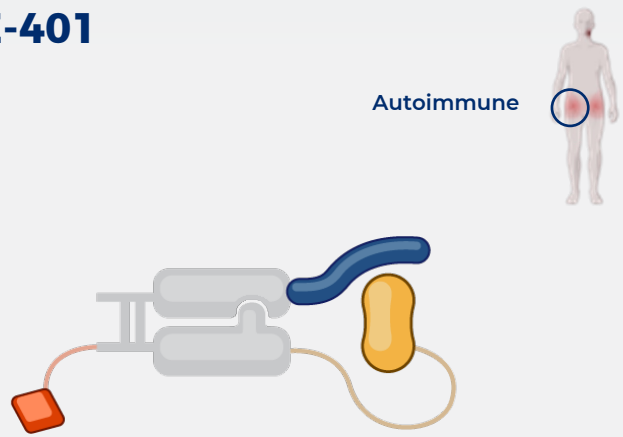
CUE-501*



Precision Approach to B Cell Depletion

- Recruitment and activation of virus specific killer T cells
- Platform opportunity for future expansion

CUE-401



Pipeline in a Product to Address Autoimmunity

- Re-establish immune balance/tolerance
- Potential to address multiple high-value indications
- Platform opportunity for future expansion

*Collaboration with Boehringer Ingelheim

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Matteo Levisetti, MD

CUE-401: A Disruptive Approach in Autoimmune Disease

First-in-class mechanism using cutting-edge technology

Possibility for new standard of care

No HLA restriction

CUE-401

Transforming Effector T Cells into Regulatory T Cells

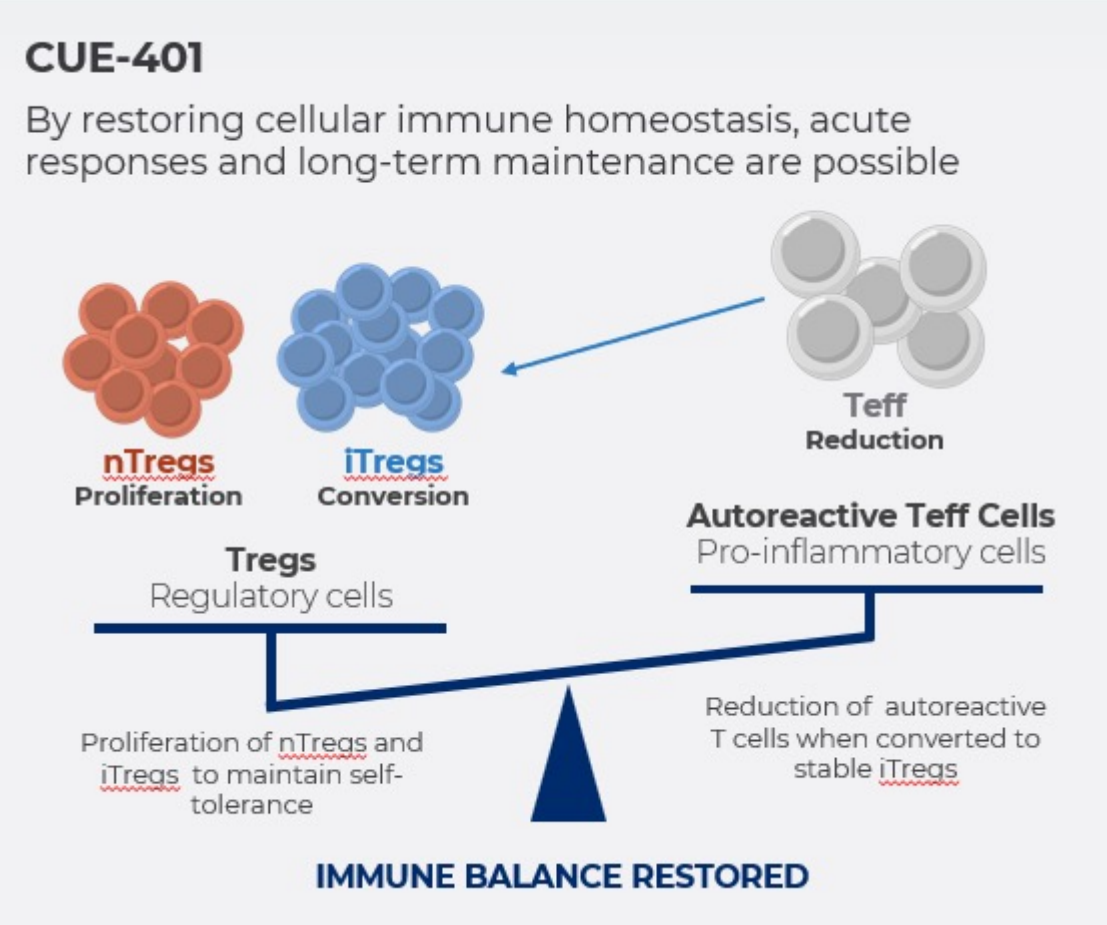
T Lymphocyte populations

- Effector T cells: Responsible for inflammatory activity in health and disease
- Regulatory T cells: Responsible for controlling unwanted/destructive inflammatory activity

Regulatory T cells are powerful anti-inflammatory mediators that are critical for maintenance of immune balance

CUE-401 has the potential to restore immune balance and tolerance by

- Transforming effector T cells into new Tregs, and
- Expanding existing Treg populations



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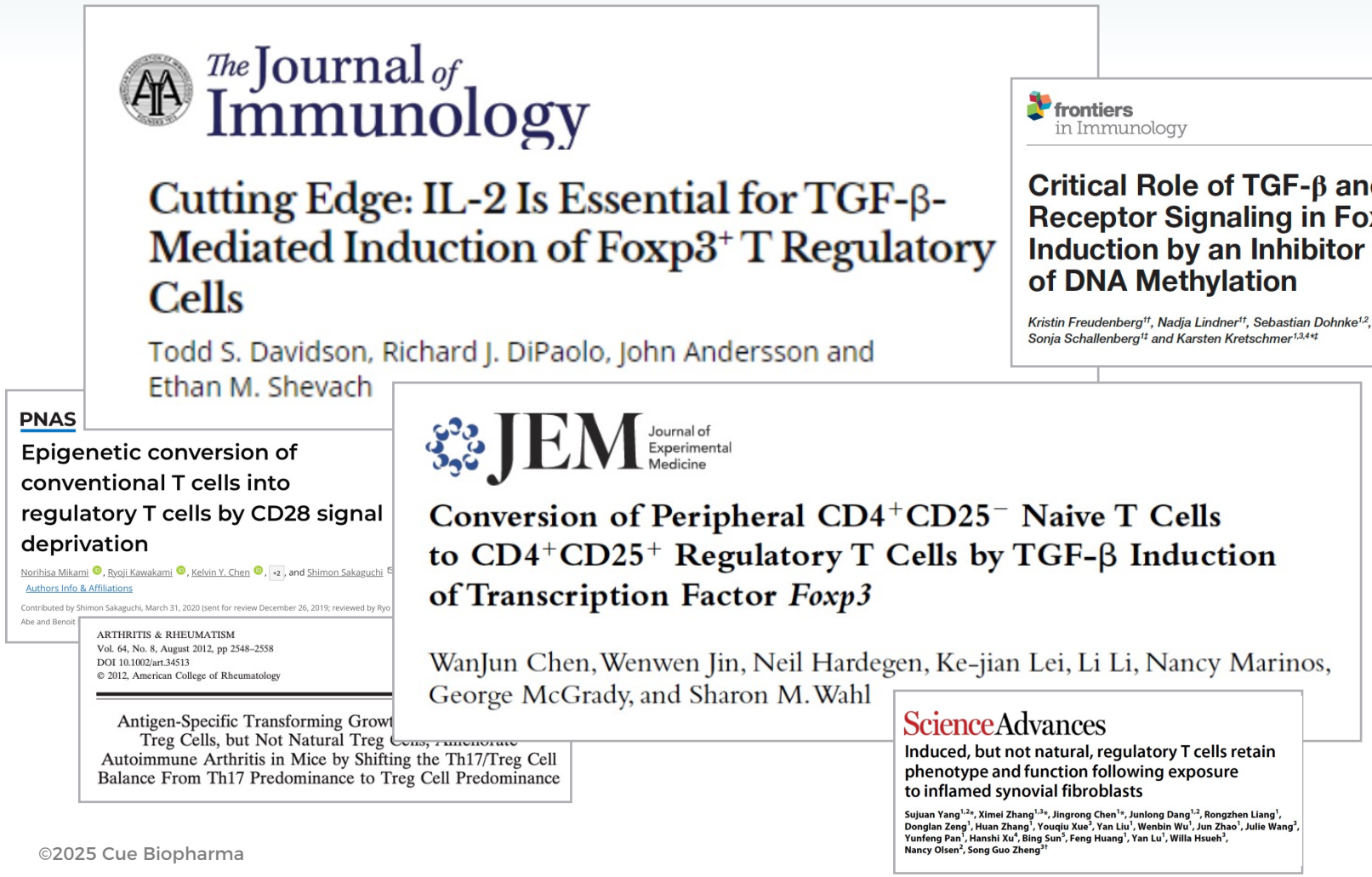
CUE-401: Harnessing the Power of TGF-β

Potent Immunosuppressive Cytokine Vital to the Maintenance of Immune Tolerance and Homeostasis



TGF-β *suppresses* the activity of multiple immune cell types while *inducing* immunosuppressive cell phenotypes.

TGF-β is regarded as one of the most potent immunosuppressive cytokines – vital to the maintenance of immune homeostasis and immunologic self-tolerance

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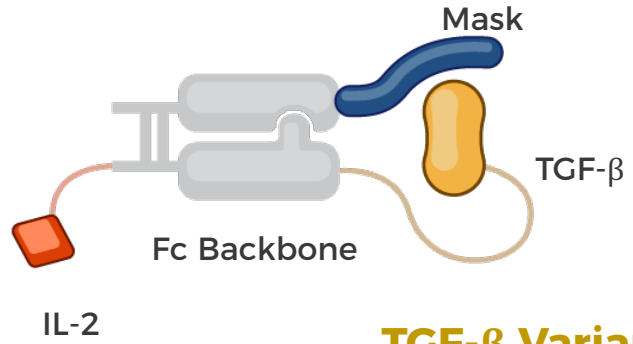
CUE-401: High-Value Opportunity

Potential for Disruptive Efficacy in Broad Range of Autoimmune Indications

Pipeline in a Product	• Transformative First-in-class mechanism • Cutting-edge biology compared with validated IL-2 approaches • Efficacy shown in multiple disease models suggests broad application in the autoimmune space
Components* Clinically Derisked	• No HLA restriction • Incorporates IL-2 and parts of Fc from CUE-100 series* • Favorable tolerability in multiple species, including non-human primates
Clear Path to Clinic	• Manufacturing and IND enabling studies underway
Near-term Value Inflection	• Phase 1 clinical data anticipated within 3-year time frame

Fc Backbone
Allows for simultaneous delivery of both IL-2 and TFG-β, along with ease of manufacturability



IL-2 Variant
Affinity attenuated; same as in CUE-100 series

TGF-β Variant
Affinity attenuated; improved safety and manufacturability

CUE-401 Masked Bispecific

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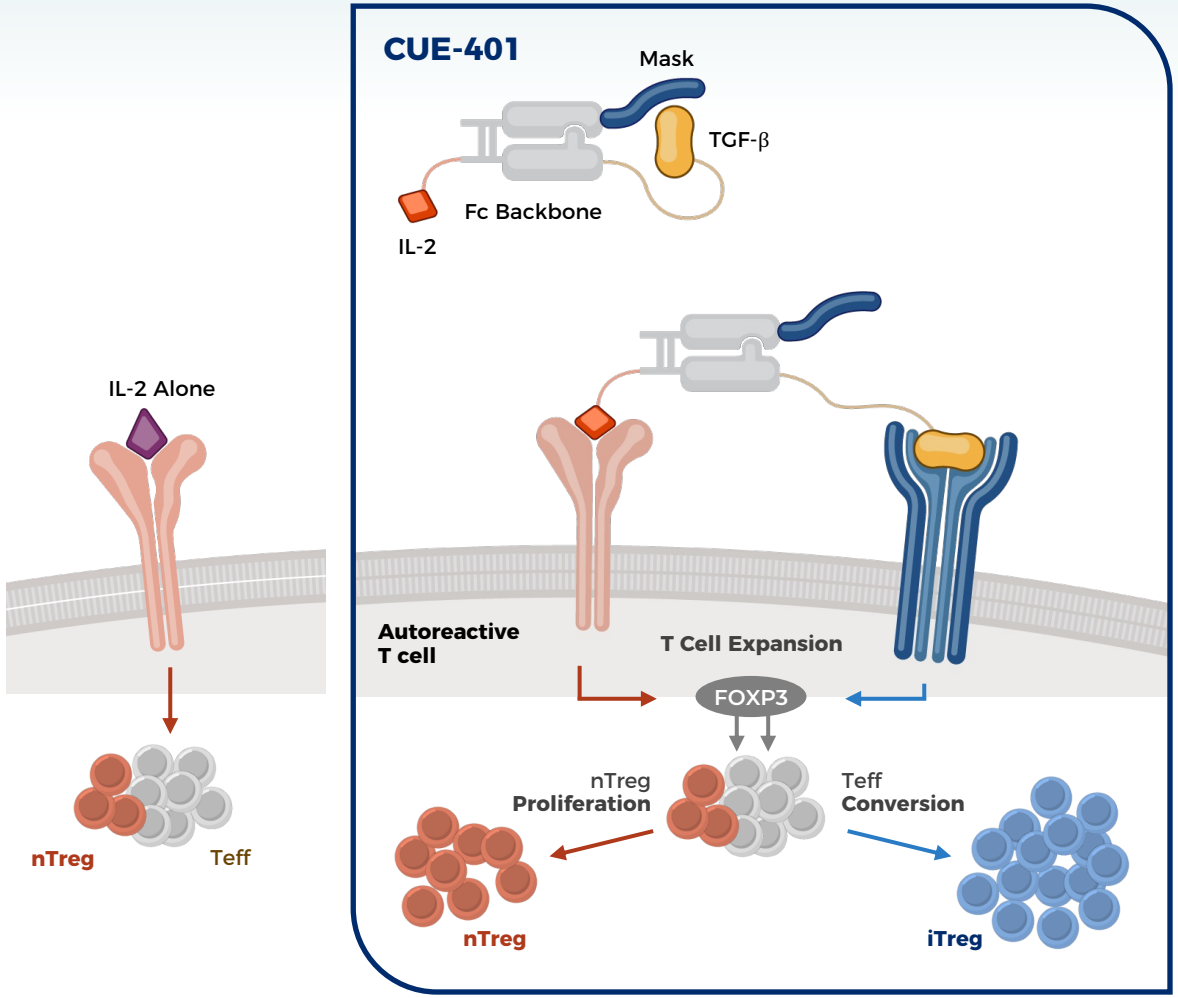
CUE-401: Differentiated Mechanism

Simultaneous IL-2 and TGF-β Signaling as the Essential Inducer for Treg Cells



"Breathing Mask" design enables active but attenuated TGF-β function and differentiated mechanism of action vs IL-2 muteins

Increased Quantity of Regulatory T Cells results from the proliferation of existing nTregs as well as induction of new iTregs from T effector pool

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Dr. Richard DiPaolo

Recognized Expert in Regulatory T Cells

Professor and Chair, Department of Molecular Microbiology & Immunology, Saint Louis University

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Background on Working with Tregs Induced by TGFβ and IL-2

Volume 178, Issue 7

1 April 2007

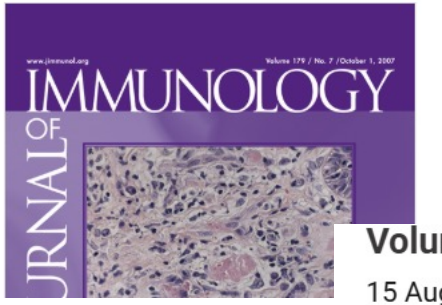
BRIEF REPORT | APRIL 01 2007

Cutting Edge: IL-2 Is Essential for TGF-β-Mediated Induction of Foxp3⁺ T Regulatory Cells FREE

Todd S. Davidson; Richard J. DiPaolo; John Andersson; Ethan M. Shevach

Volume 179, Issue 7

1 October 2007



RESEARCH ARTICLE | OCTOBER 01 2007

Autoantigen-Specific TGFβ-Induced Foxp3⁺ Regulatory T Cells Prevent Autoimmunity by Inhibiting Dendritic Cells from Activating Autoreactive T Cells[1] FREE

Richard J. DiPaolo; Carine Brinster; Todd S. Davidson; John Andersson; Deborah Glass; Ethan M. Shevach

+ Author & Article Information

J Immunol (2007) 179 (7): 4685–4693

Volume 187, Issue 4

15 August 2011

RESEARCH ARTICLE | AUGUST 15 2011

Antigen-Specific TGF-β–Induced Regulatory T Cells Secrete Chemokines, Regulate T Cell Trafficking, and Suppress Ongoing Autoimmunity

Thanh-Long M. Nguyen; Nicole L. Sullivan; Mark Ebel; Ryan M. Teague; Richard J. DiPaolo

+ Author & Article Information

J Immunol (2011) 187 (4): 1745–1753.

https://doi.org/10.4049/jimmunol.1004112 **Article history** Advertisement

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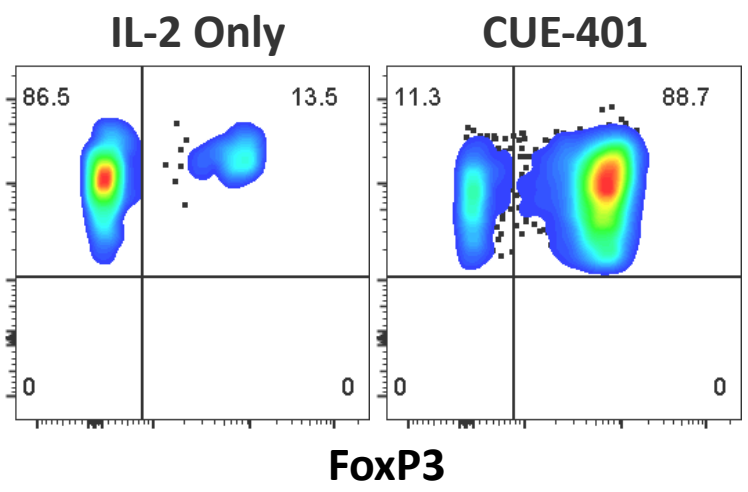
Testing Effects of CUE-401 on CD4$^+$ T cell Activation *in vitro*

CUE-401 Induces/Expands iTregs

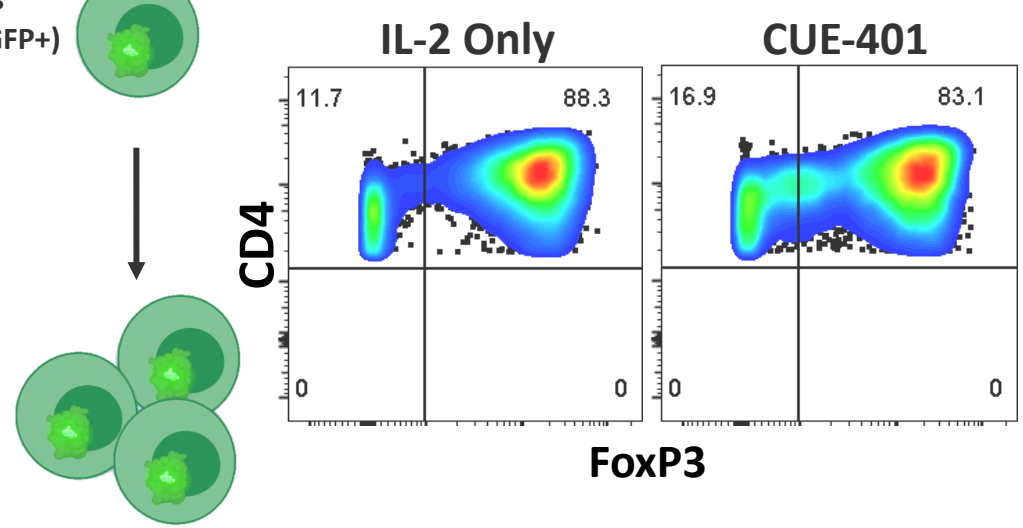
Naïve CD4 T cell (Foxp3.GFP-)

iTregs (Foxp3.GFP+)



CUE-401 Expands nTregs

Tregs (Foxp3.GFP+)



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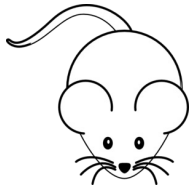
Testing the Effects of CUE-401 *in vivo*

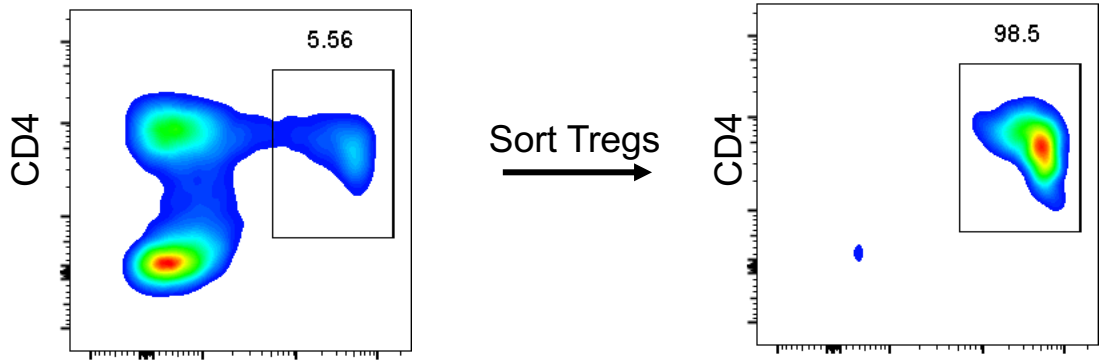


Vehicle Controls vs. Treated with CUE-401 (1 mg/kg)

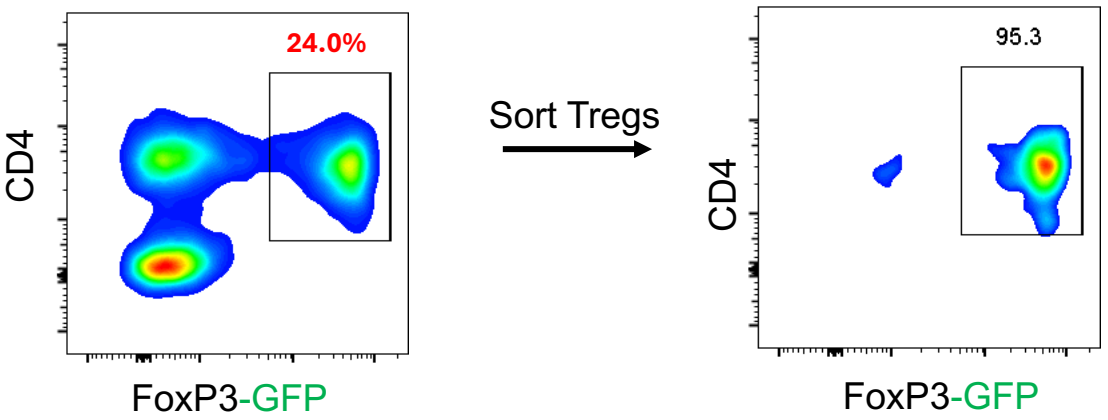
- **Flow Cytometry**
- **Cell sorting and Foxp3 promoter TSDR methylation**
- **Cell Sorting and re-activation**

CUE-401 Increases Tregs

Control



CUE-401



- Both had ~100% demethylated Foxp3 Treg-specific demethylated region (TSDR)
- Both retained Foxp3 expression after restimulation for 6 days in vitro.

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Summary of *in vivo* Studies with CUE-401

- **CUE-401 treatment is effective at increasing the frequency of $CD4^{+}Foxp3^{+}$ Tregs.**
- **Tregs induced/expanded by CUE-401 are stable.**
- **Tregs induced/expanded by CUE-401 have an activated phenotype ($CD25^{hi}CTLA4^{hi}GITR^{hi}$).**
- **CUE-401 did not induce the expansion/activation of effector T cells (unlike IL-2 complexes).**
- **A subset of the CUE-401 induced/expanded Tregs were transcriptionally distinct from Tregs expanded with IL-2 complexes**
  - **These Tregs shared transcripts previously associated with TGFβ + IL-2 induced Tregs *in vitro*.**

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Testing CUE-401's Ability to Suppress Autoimmunity

Isolate spleen cells.

Deplete CD25 expressing cells (Tregs).

Transfer $25x10^6$ into T cell deficient mice.

Transfer a small number of autoreactive T cells (gastritis-causing) to track effects on autoreactive T cells

BALB/c Nude Mice

After 2 months mice develop autoimmunity **due to lack of Tregs.**

100% of mice develop autoimmune gastritis.

JOURNAL ARTICLE

Pillars Article: Immunologic Self-Tolerance Maintained by Activated T Cells Expressing IL-2 Receptor α-Chains (CD25). Breakdown of a Single Mechanism of Self-Tolerance Causes Various Autoimmune Diseases. *J. Immunol.* 1995. 155: 1151–1164 Get access ›

Shimon Sakaguchi , Noriko Sakaguchi , Masanao Asano , Misako Itoh , Masaaki Toda

The Journal of Immunology, Volume 186, Issue 7, April 2011, Pages 3808–3821,
https://doi.org/10.1093/jimmunol/186.7.3808
Published: 01 April 2011

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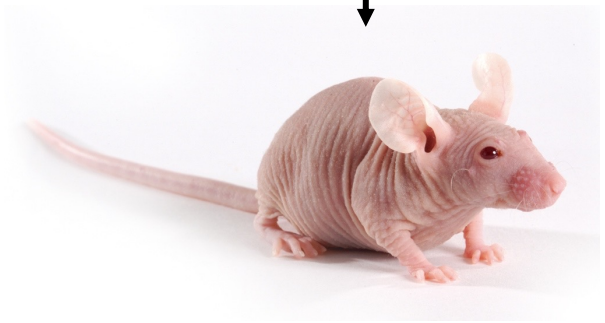
Can CUE-401 Suppress Autoimmunity?

25x10^6 CD25-(Treg) depleted splenocytes (Thy1.2)
+50,000 TxA23-Thy1.1 autoreactive tracer cells (0.2%)



Day +1, +14

- PBS (control)
- CUE-401 (1 mg/kg)

Take Down Day 62

- Analyze cells in: stomachs, spleens, LNs
- Analyze gastric pathology

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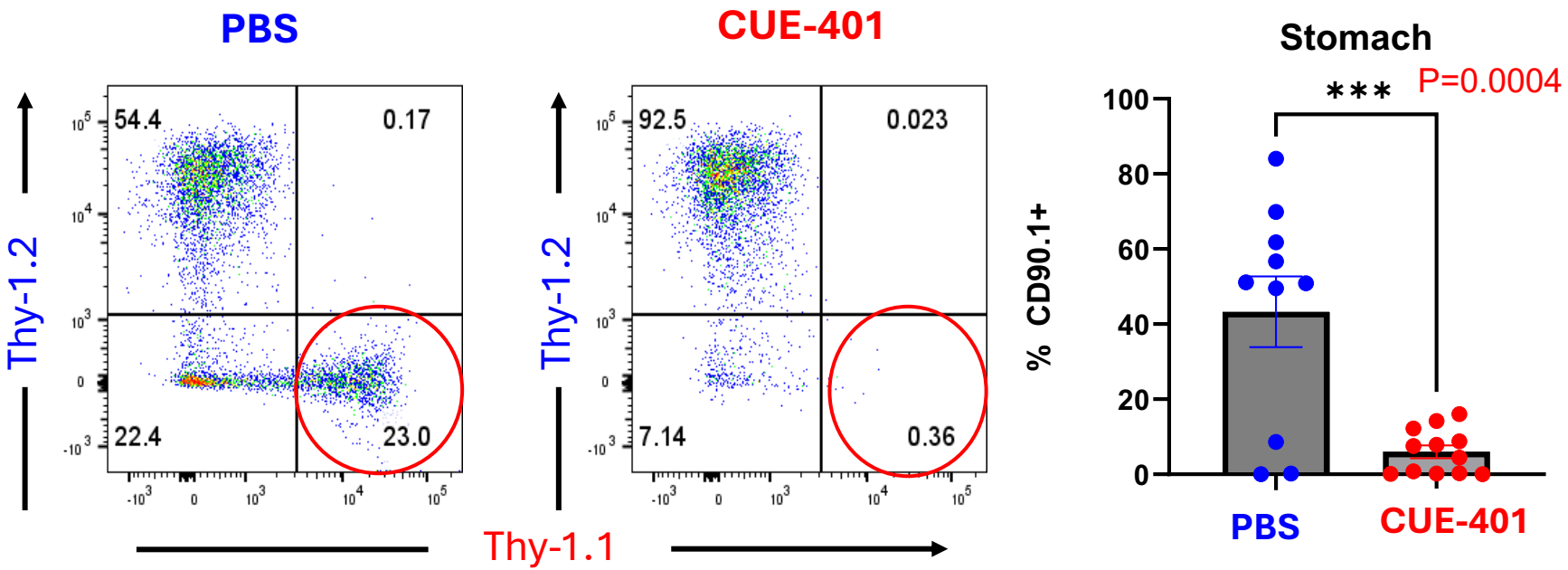
Significant Reduction of Autoreactive T cells in Stomach



- **CUE-401 treatment resulted in significant and durable reduction of autoreactive T cells in the target tissue (stomach) that was maintained long after treatment.**

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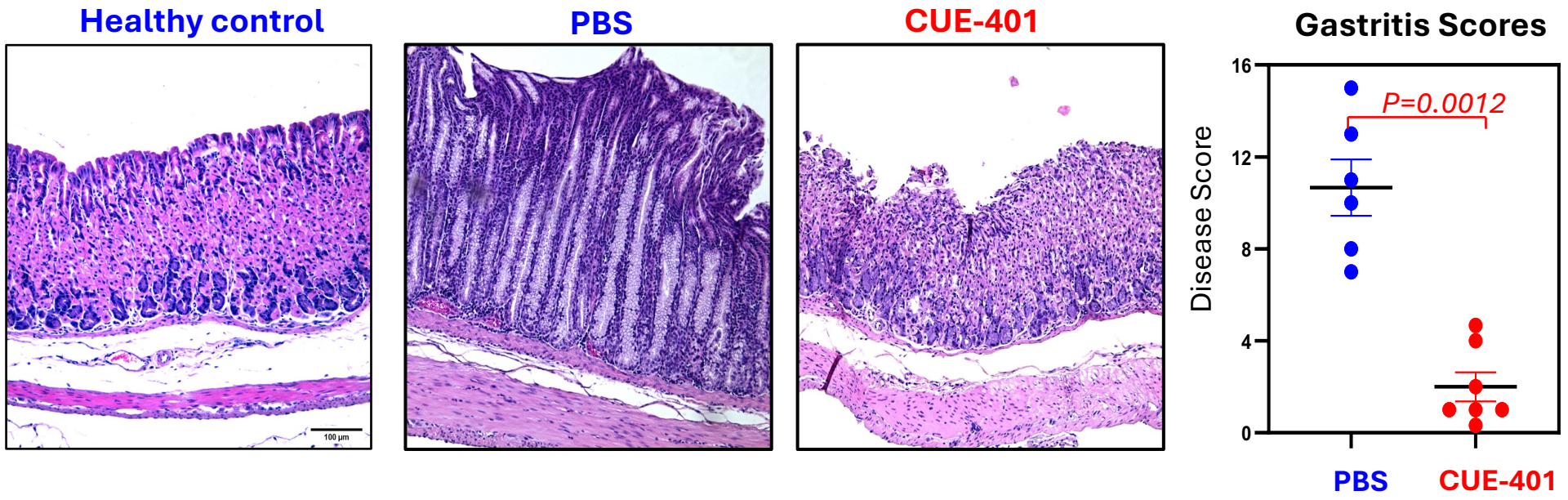
Significant Reduction of Gastric Pathology in Stomach



- **CUE-401 treatment results in significant protection from autoimmune gastritis caused by a deficiency in Tregs.**
- **Initial short-term treatment with CUE-401 resulted in long-term protection.**

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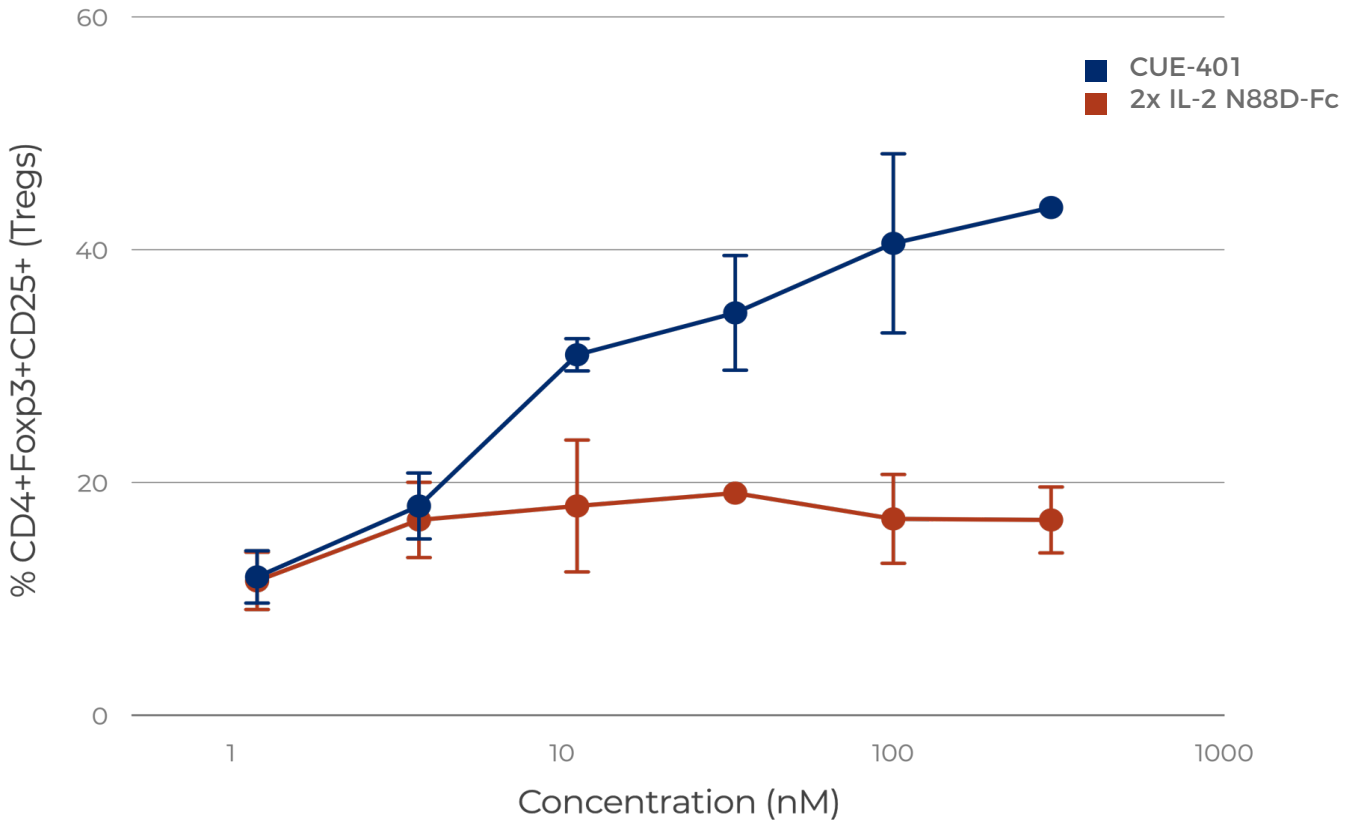
Conclusions

- **CUE-401 functions to induce new Tregs, expand existing Tregs, and suppress some effector T cell functions.**
- **In vivo administration of CUE-401 is effective at inducing/expanding and activating Tregs.**
- **Short term CUE-401 administration resulted in durable suppression of autoimmunity in a well-established model where autoimmunity develops as a result of a deficiency in Tregs.**

CUE-401 Harnesses Multiple Signals to Induce Tregs

Data Shows Evidence of Inducible Treg Expansion from Effector T Cells (Teff), Differentiating from IL-2 Muteins

Treg Generation from Effector T Cells (Teff) with CUE-401 vs IL-2 Mutein: Human MLR* (In Vitro GVHD)



*Mixed Lymphocyte Reaction

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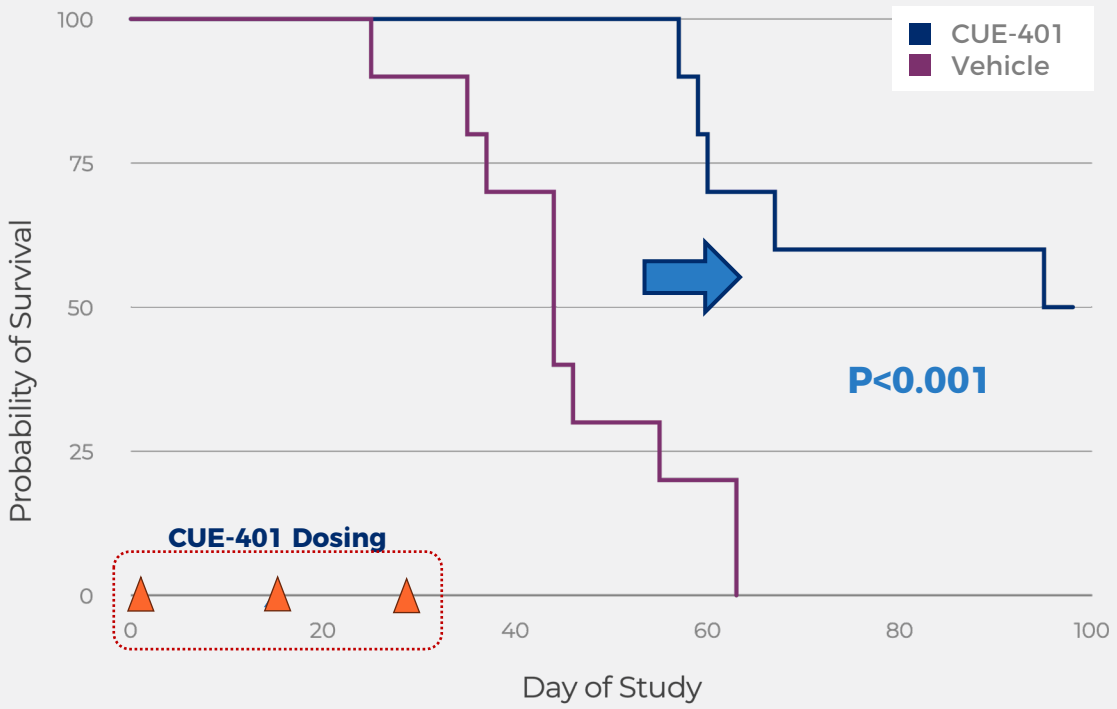
CUE-401: Differentiated Benefit in Pre-clinical Models of GVHD vs. IL-2 Mutein

CUE-401 Increased Survival 9+ Weeks Post-Treatment while IL-2 Mutein Alone Accelerated Disease

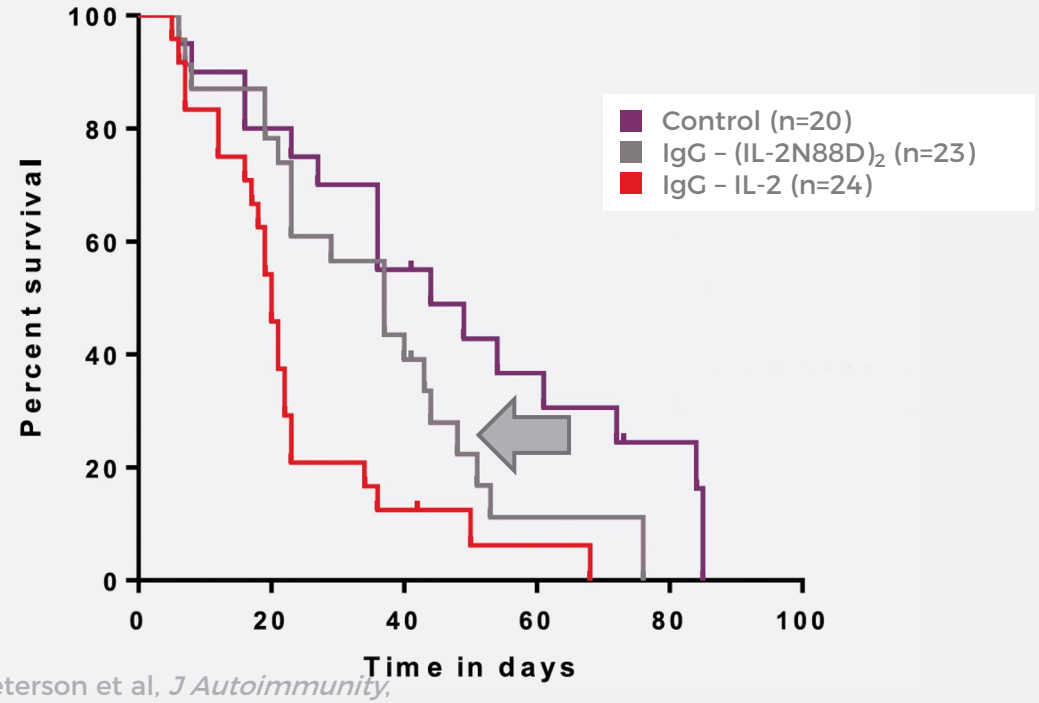
CUE-401

Significantly delayed development of GVHD & increased overall survival



IL-2 (wild type or a mutein)

Accelerated progression of GVHD & reduced overall survival



Peterson et al, *J Autoimmunity*, 2018

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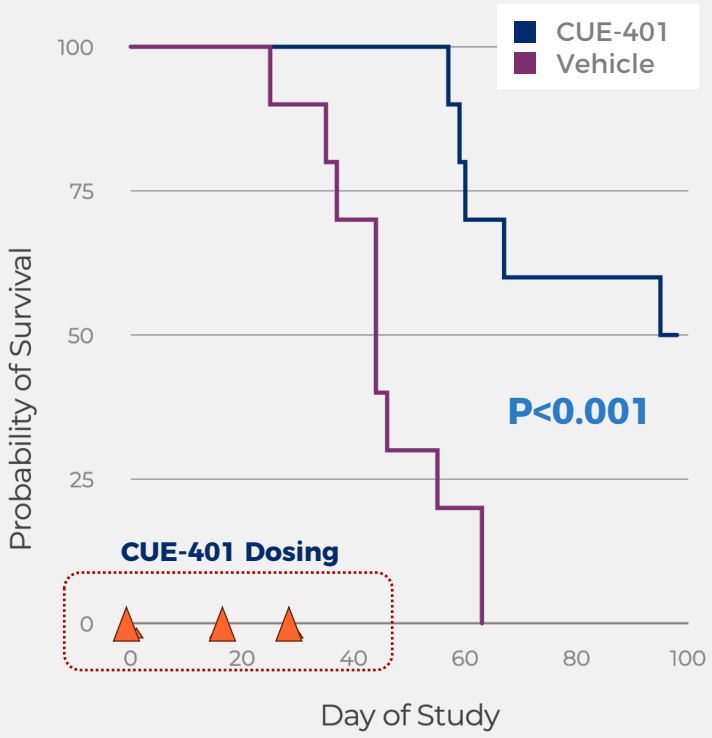
CUE-401: Durable Benefit & Pharmacodynamics in Model of Acute GVHD

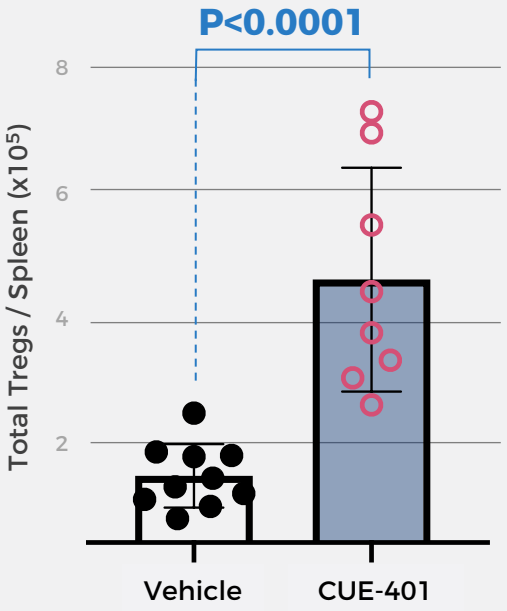
Increased Tissue Tregs, Reduced Proinflammatory Cytokines, and Increases Survival 9+ Weeks Post-Treatment

Delayed GVHD & Increased Overall Survival

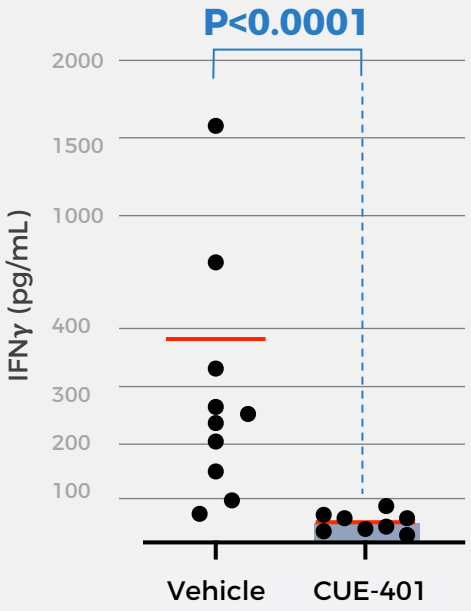


n=10 per treatment group

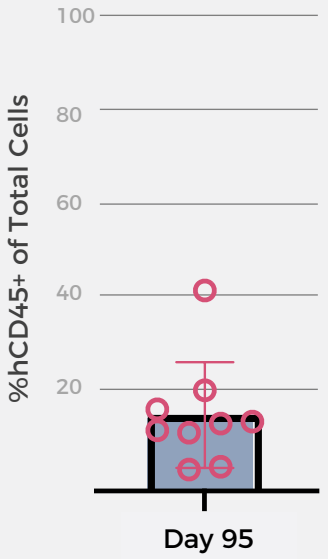
Increased Treg Numbers in GVHD Target Tissue



Reduced Systemic Inflammatory Cytokines



Persistence of Human T Cell Graft



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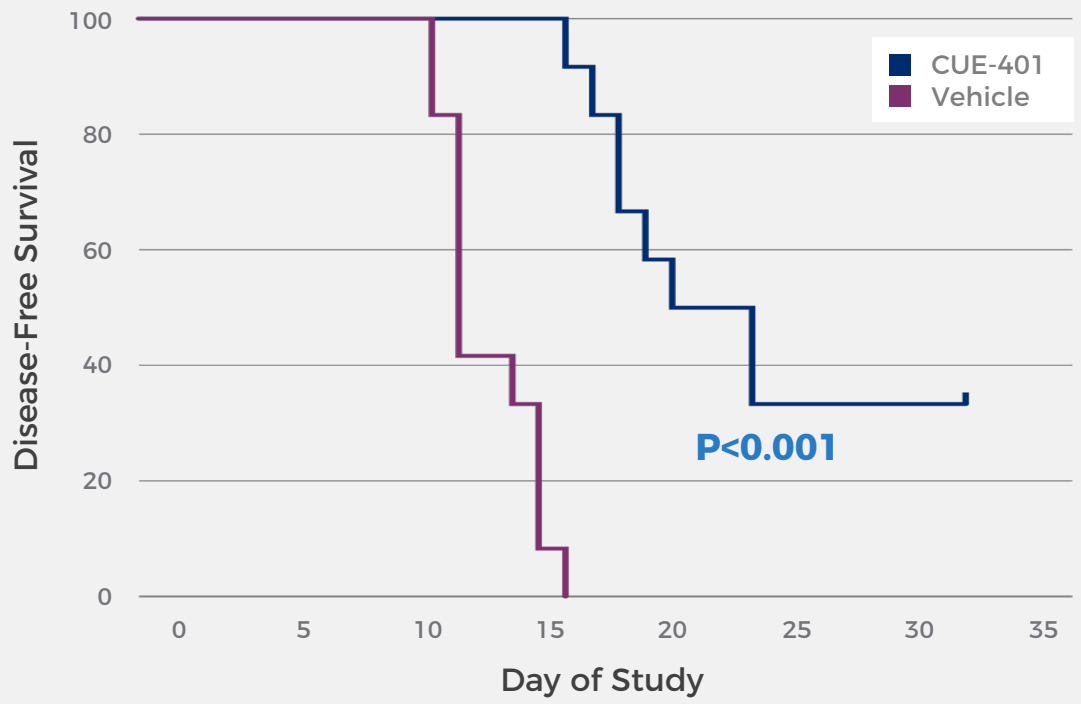
CUE-401: Efficacy Across Diverse Disease Mechanisms

Functional Suppression of Inflammation in Multiple Disease Models Supports Broad Applicability in the Clinic

EAE Model of Multiple Sclerosis

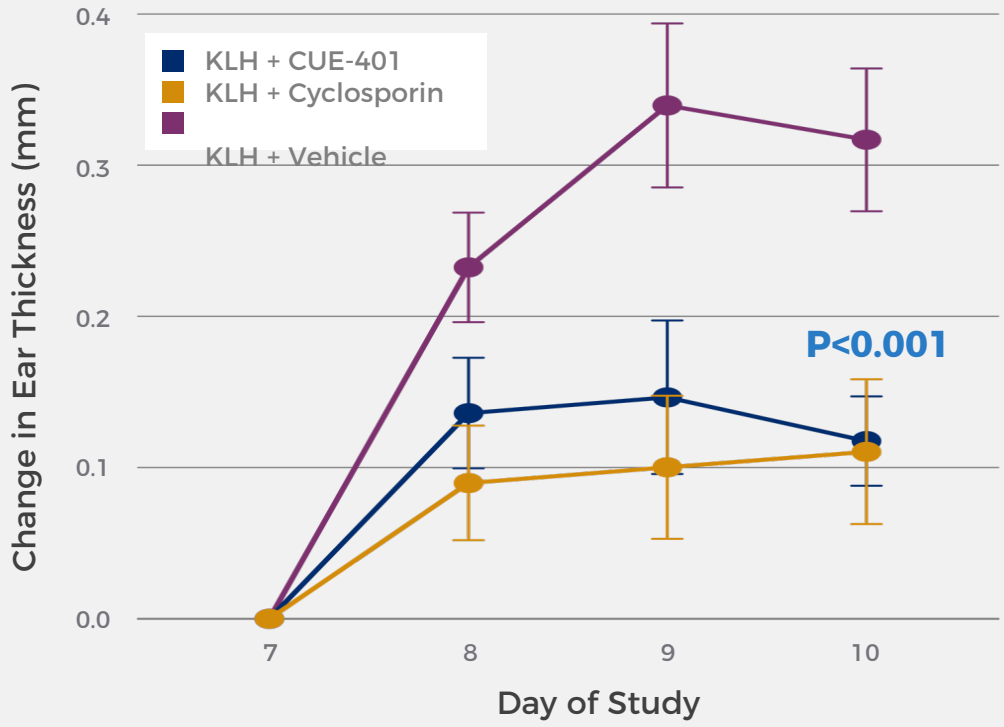
Significant inhibition and delay of disease onset



n=10 per treatment group

Delayed Type Hypersensitivity – T Cell Mediated

Significant inhibition of cutaneous inflammation



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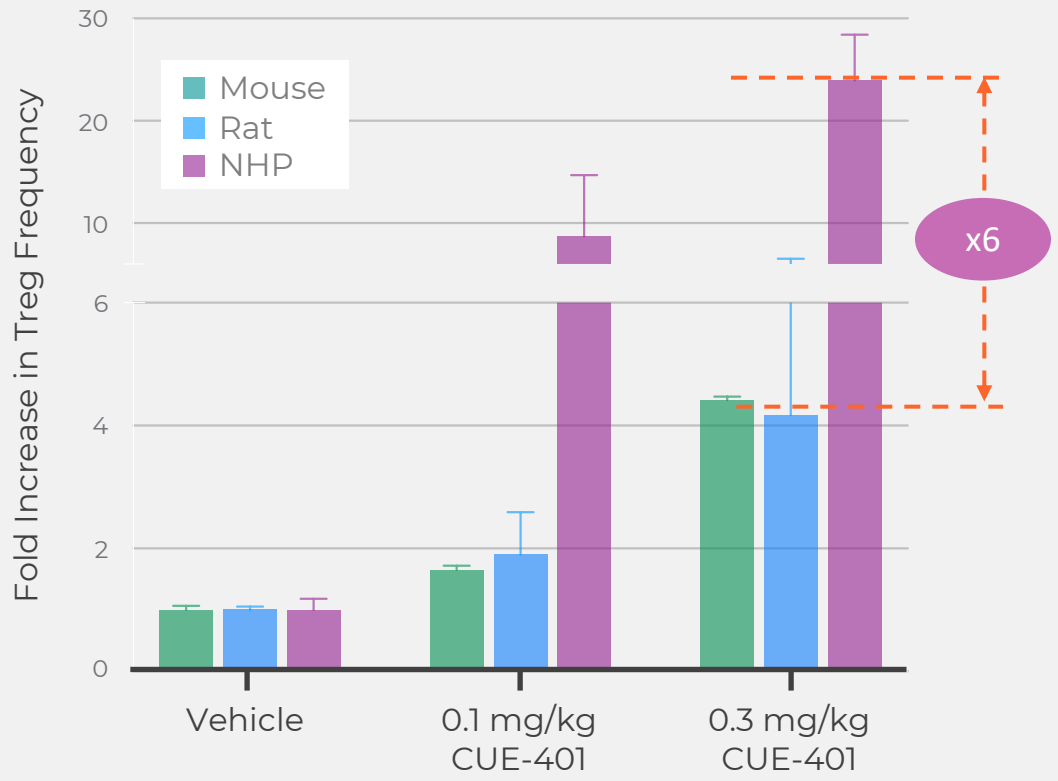
CUE-401: Significant Treg Expansion Across Species

Greatest Magnitude of Treg Expansion in Non-Human Primates (NHP) – Supports Strong Potential Activity in Humans



- CUE-401 is well tolerated across all species at dose levels that result in maximum Treg expansion

→ **Enables & derisks efficient IND-enabling GLP toxicology program**

- Even low doses in NHP drive significant Treg increases well beyond that observed in rodents

→ **CUE-401 has the potential to promote meaningful increases in Tregs in humans at low dose levels**

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CUE-401: Novel, First-in-Class T Cell Immunomodulator

Broad Potential Across Multiple Autoimmune Indications

CUE-401

Bispecific Mechanism of Action	**"Breathing Mask"** design enables active but attenuated TGF-β function and differentiated mechanism of action vs IL-2 muteins with potential to induce immune tolerance
Preclinical Data	**CUE has generated efficacy and tolerability data sets** in 3 species and 3 disease models in addition to data generated by Dr. DiPaolo
Pharmacokinetics	**Comparable exposure across species** sufficient to promote significant Treg expansion
Manufacturing	Productive cell line established, **stability demonstrated, and scale-up manufacturing ongoing**

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Dr. Andrew Cope
The Clinical Perspective

Head, Centre for Rheumatic Diseases, King's College, London

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A Commitment to T Cell Biology and "Immune Reset"

Chronic Exposure to Tumor Necrosis Factor (TNF) In Vitro Impairs the Activation of T Cells through the T Cell Receptor/CD3 Complex; Reversal In Vivo by Anti–TNF Antibodies in Patients with Rheumatoid Arthritis

Andrew P. Cope, Marco Londei, N. Randall Chu, Shara B. A. Cohen, Michael J. Elliott, Fionula M. Brennan, Ravinder N. Maini, and Marc Feldmann

The Mathilda and Terence Kennedy Institute of Rheumatology, Sunley Division, Hammersmith, London, W6 8LW, United Kingdom

nature immunology

Complement regulator CD46 temporally regulates cytokine production by conventional and unconventional T cells

John Cardone[1,8], Gaelle Le Friec[1,8], Pierre Vantourout[2,3], Andrew Roberts[2,3], Anja Fuchs[4], Ian Jackson[1,5], Tesha Suddason[1,5], Graham Lord[1,5], John P Atkinson[6], Andrew Cope[5,7], Adrian Hayday[1–3,5] & Claudia Kemper[1]

ARTICLE

https://doi.org/10.1038/s41467-019-08332-9 OPEN

The cholesterol biosynthesis pathway regulates IL-10 expression in human Th1 cells

Esperanza Perucha[1,2], Rossella Melchiotti[3], Jack A Bibby[1,2], Wing Wu[1,2], Klaus Stensgaard Frederiksen[4], Ceri A. Roberts[2,14], Zoe Hall[5], Gaelle LeFriec[6], Kevin A. Robertson[7], Paul Lavender[8], Jens Gammeltoft Gerwien[4,15], Leonie S. Taams[2], Julian L. Griffin[5], Emanuele de Rinaldis[3], Lisa G.M. van Baarsen[9,10], Claudia Kemper[6,11,12], Peter Ghazal[7,13] & Andrew P. Cope[1,2]

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Lessons from the Clinic:

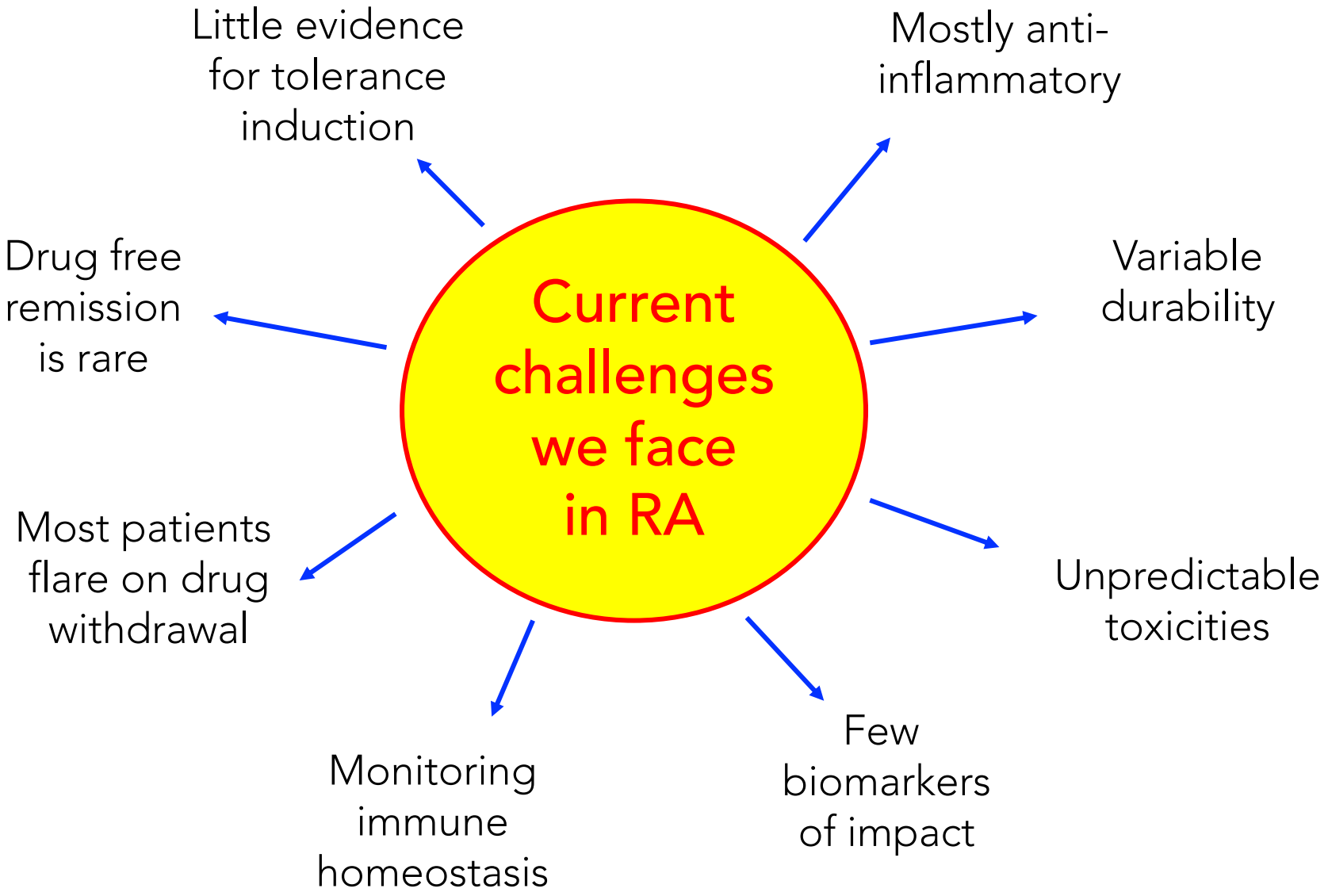
Co-stimulation modulation delays but does not prevent RA

Abatacept in individuals at high risk of rheumatoid arthritis (APIPPRA): a randomised, double-blind, multicentre, parallel, placebo-controlled, phase 2b clinical trial

*Andrew P Cope, Marianna Jasenecova, Joana C Vasconcelos, Andrew Filer, Karim Raza, Sumera Qureshi, Maria Antonietta D'Agostino, Iain B McInnes, John D Isaacs, Arthur G Pratt, Benjamin A Fisher, Christopher D Buckley, Paul Emery, Pauline Ho, Maya H Buch, Coziana Ciurtin, Dirkjan van Schaardenburg, Thomas Huizinga, René Toes, Evangelos Georgiou, Joanna Kelly, Caroline Murphy, A Toby Prevost, on behalf of the APIPPRA study investigators**

Cope et al, *Lancet* 2024;403:838-849

The Problem with Current Therapies for Rheumatoid Arthritis



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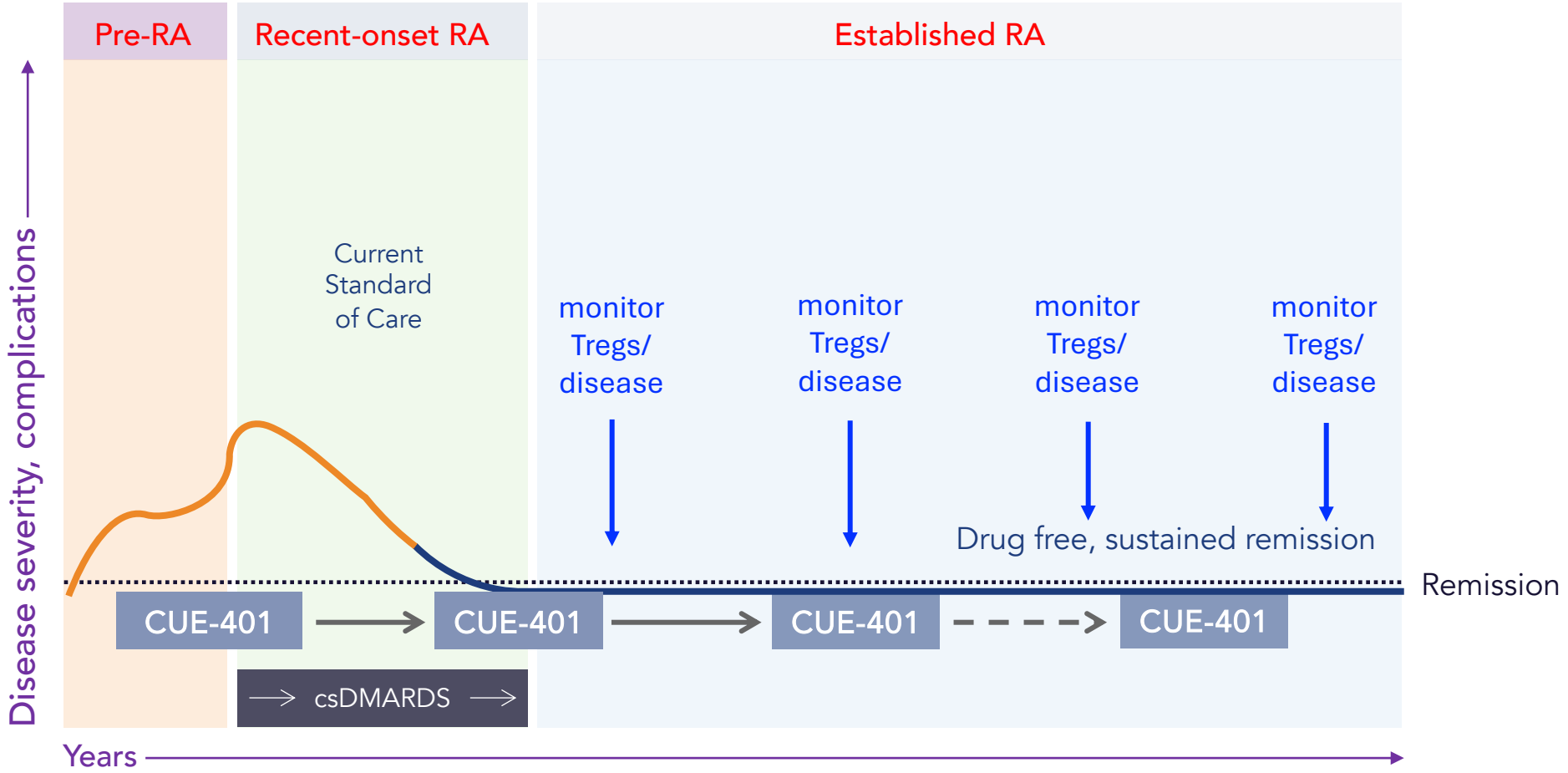
Future Vision for Immune Reset in RA with CUE-401



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CUE-401: Clinical Perspectives

- CUE-401 may be a transformative treatment for RA at several stages of disease, including pre-RA, recent-onset RA and established RA.
- Expansion and induction of Tregs by CUE-401 is an innovative approach to achieving an immune “reset” in patients with autoimmune and inflammatory disease.
- Mechanism of action of inducing and expanding Tregs is likely to be efficacious in many autoimmune diseases.
- Immune rebalance established by durable expansion of Tregs may lead to long-lasting tolerance.

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KOLs & Cue Management
Discussion and Q&A Session

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Dan Passeri, CEO
Key Take Aways

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Key Takeaways

Mobilizing the Immune System to Treat Serious Disease

- **Specific T cell populations** play a critical role in the immune response to serious diseases
- **CUE's unique biologics platform** enables selective modulation of select T cell populations for the treatment of autoimmune disease and cancer
- **New data suggest that CUE-401 could transform** the standard of care for autoimmune diseases and inflammation
- **Recent Boehringer Ingelheim collaboration showcases CUE-501:** potential ability to use anti-viral T cells to treat autoimmune disease
- **Maturing clinical data show notable increase in survival data for CUE-100 Series**
- **Current cash resources** expected to support key developments of our corporate strategy

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Thank you.

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