AUTOIMMUNITY
BioSolutions

Next Generation, *Immuno-Corrective* Therapy for Autoimmune Disease Treatment

Company Overview

Eugene Williams, MBA

CEO

Email: eugene@abstherapeutics.com

Gaddiel Galarza-Munoz, PhD

Co-Founder and CSO

Email: gaddiel@abstherapeutics.com

Location: *K2-biolabs Incubator, Houston, Texas*

Autoimmunity BioSolutions (ABS): Overview

Driven by Strong Science

- Targeting novel, fundamental **autoimmune** disease pathway: **soluble Interleukin 7 Receptor (sIL7R)**
- **Genetically-Defined Disease Population** – 50% of Autoimmune Disease patient with SNP and elevated sIL7R suffer increased **risk and severity of autoimmune disease due to enhanced T cell expression**

Differentiated Approach

- **Immuno-corrective therapy in genetically-defined subpopulation corrects root cause of pathology to restore normal immune function without causing immunosuppression**

Focused Strategy for Value Creation

- **Efficient and Compelling Clinical POC in Lupus Nephritis and MS with mAb therapy to reduce sIL7R activity to normal levels and provide compelling clinical benefit**
- Significant genetic, scientific & biomarker data support sIL7R as a critical target

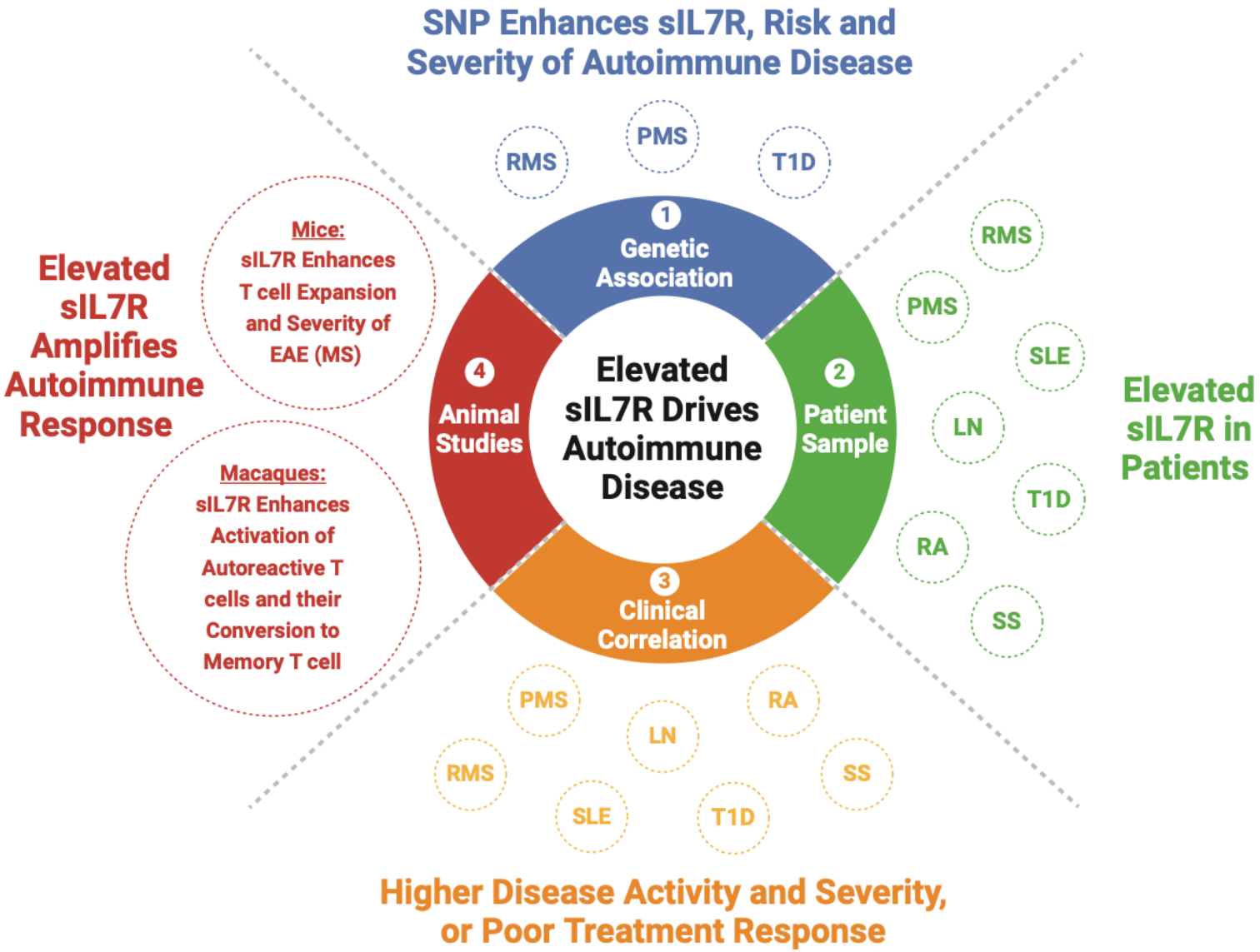
Broad Potential

- **Indication expansion** to other diseases supported by genetic, scientific and/or biomarker data:
  - Correlation of sIL7R with disease activity: **Type 1 diabetes, RA, Sjogren's**
  - Involvement of IL7R pathway: **UC, Crohn's, others**

Strengths

- **Recently closed initial $2M Seed financing – active discussions for second tranche**
- **Experienced leadership team with capital-efficient structure and development strategy**
- **Strong Intellectual Property Position**

ABS Therapy: Broad Potential for Genetically Targeted *Immuno-Corrective* Therapy in Various Autoimmune Diseases



RMS = Relapsing MS

PMS = Progressive MS

T1D = Type 1 diabetes

SLE = Systemic lupus erythematosus

LN = Lupus nephritis

RA = Rheumatoid arthritis

SS= Sjogren's syndrome

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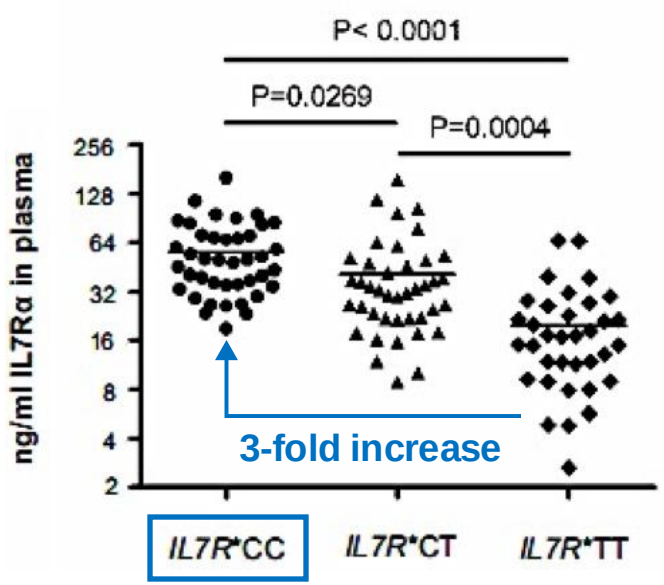
ABS Science:

Elevated soluble IL7 Receptor (sIL7R) Amplifies Autoimmune Disease Pathology

SNP Elevates sIL7R and Amplifies Autoimmune Disease Pathology:
Highly prevalent SNP, estimated over 50% of autoimmune disease

SNP rs6897932 upregulates sIL7R by 3-fold in MS patients



The risk 'C' allele of rs6897932 increases the expression of sIL7R in MS patients by 3-fold.

Lundstrom et al., 2013, ***PNAS*** *110, E1761-1770.*
PMID: 23610432

Genetics:

The SNP (IL7R*CC) is genetically linked to autoimmune diseases:

- It increases risk and severity of autoimmune diseases.
- It elevates sIL7R expression by 3-fold in MS and other autoimmune diseases.
- It is highly prevalent being present in 60% of patients of MS and other autoimmune diseases, such as SLE and T1D

Molecular and Clinical Correlation:

- sIL7R is elevated in numerous autoimmune diseases, including MS (relapsing & progressive), LN, SLE, RA, T1D, SS, and likely others.

- sIL7R correlates with higher disease activity, severity or poor response to treatment in numerous autoimmune diseases, including MS (relapsing & progressive), LN, SLE, RA, T1D, SS, and likely others.

MS = multiple sclerosis; LN = lupus nephritis; SLE = systemic lupus erythematosus;
RA = rheumatoid arthritis; T1D = type 1 diabetes; SS = Sjogren's syndrome

Strong Evidence of Causality: Elevated sIL7R Expands T cells and Enhances Disease Severity in Mouse Model of MS

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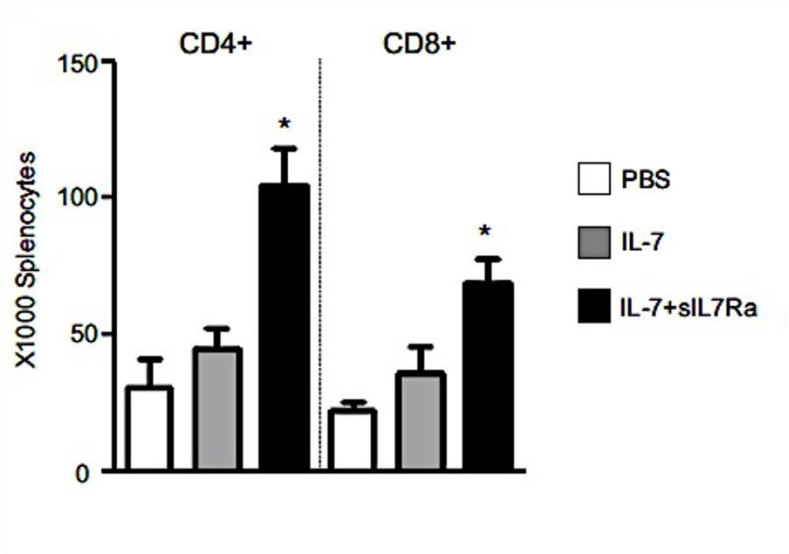
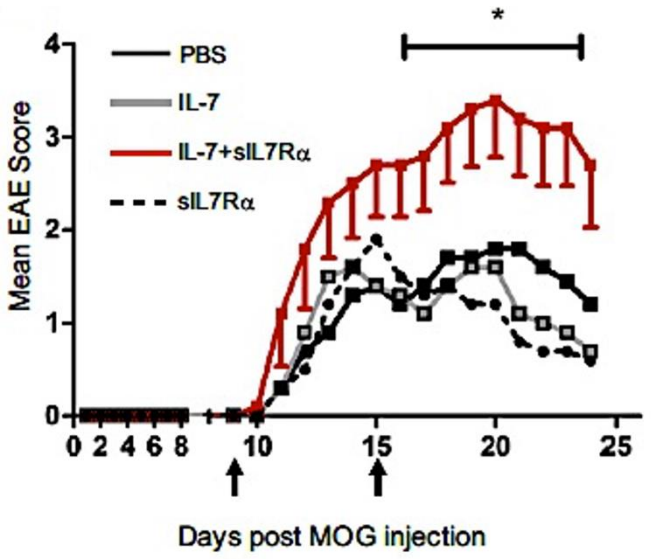
Injection of sIL7R protein in IL7 knock-out mice enhances expansion of T cells



sIL7R enhances expansion of T cells both *in vivo* and *in vitro*.

Injection of sIL7R in EAE mouse model of Progressive MS enhances disease severity

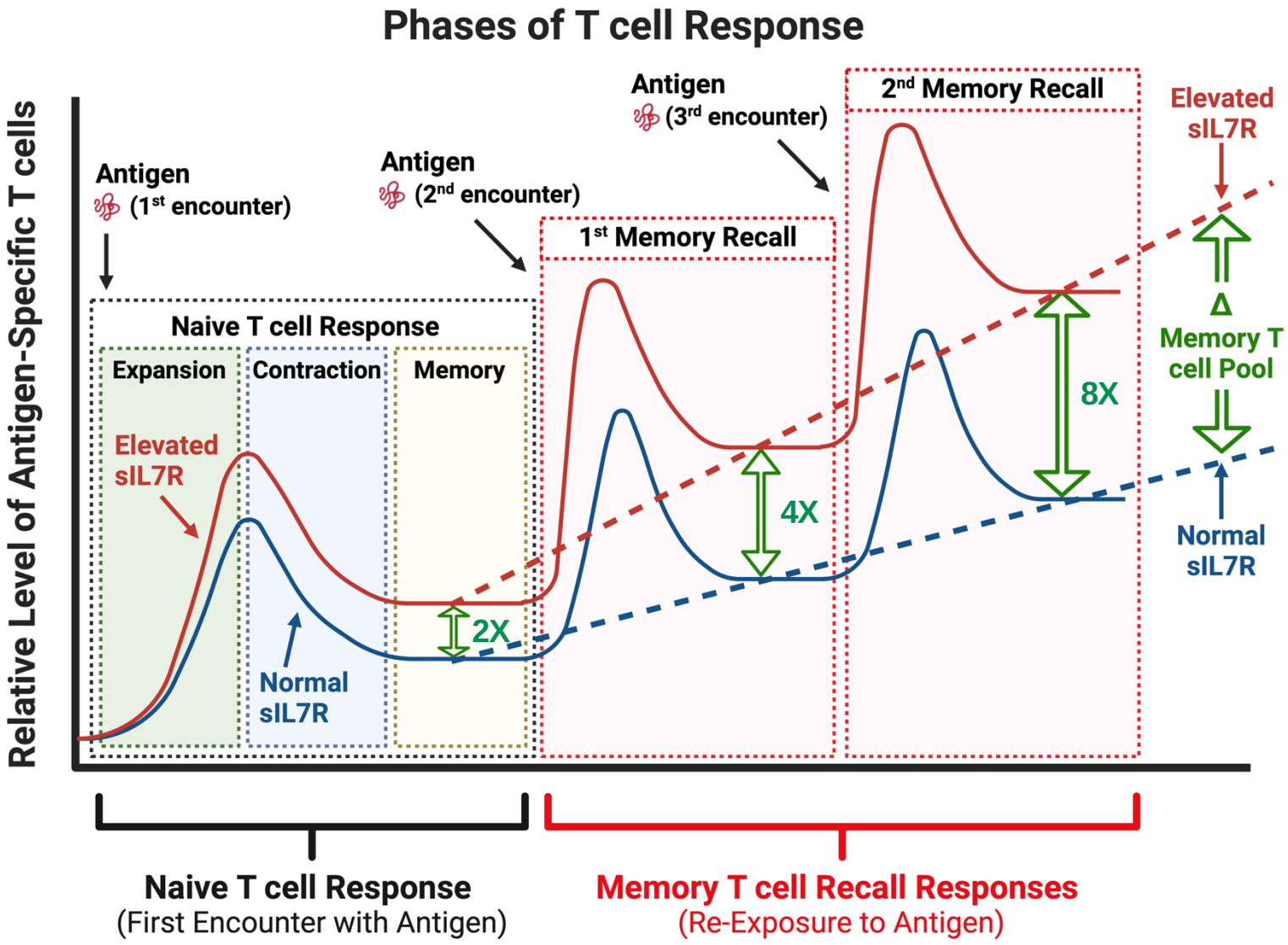


sIL7R enhances disease severity in EAE mouse model of Progressive MS.

Animal model to be used to establish initial proof of concept of top mAbs

*Lundstrom et al., 2013, **PNAS** 110, E1761-1770. PMID: 23610432*

Elevated sIL7R Drives Risk of More Severe Pathology: sIL7R Enhances the Growing Pool of Autoreactive Effector and Memory T cells



Elevated sIL7R

↓

Enhanced T cell response to auto-antigen

↓

Higher formation of autoreactive memory T cells

↓

Perpetuate autoimmune responses and exacerbate severity of pathology

SNP Elevates sIL7R and Amplifies Disease Pathology in Three Ways: ABS Strategy to Demonstrate Benefit in Each Area

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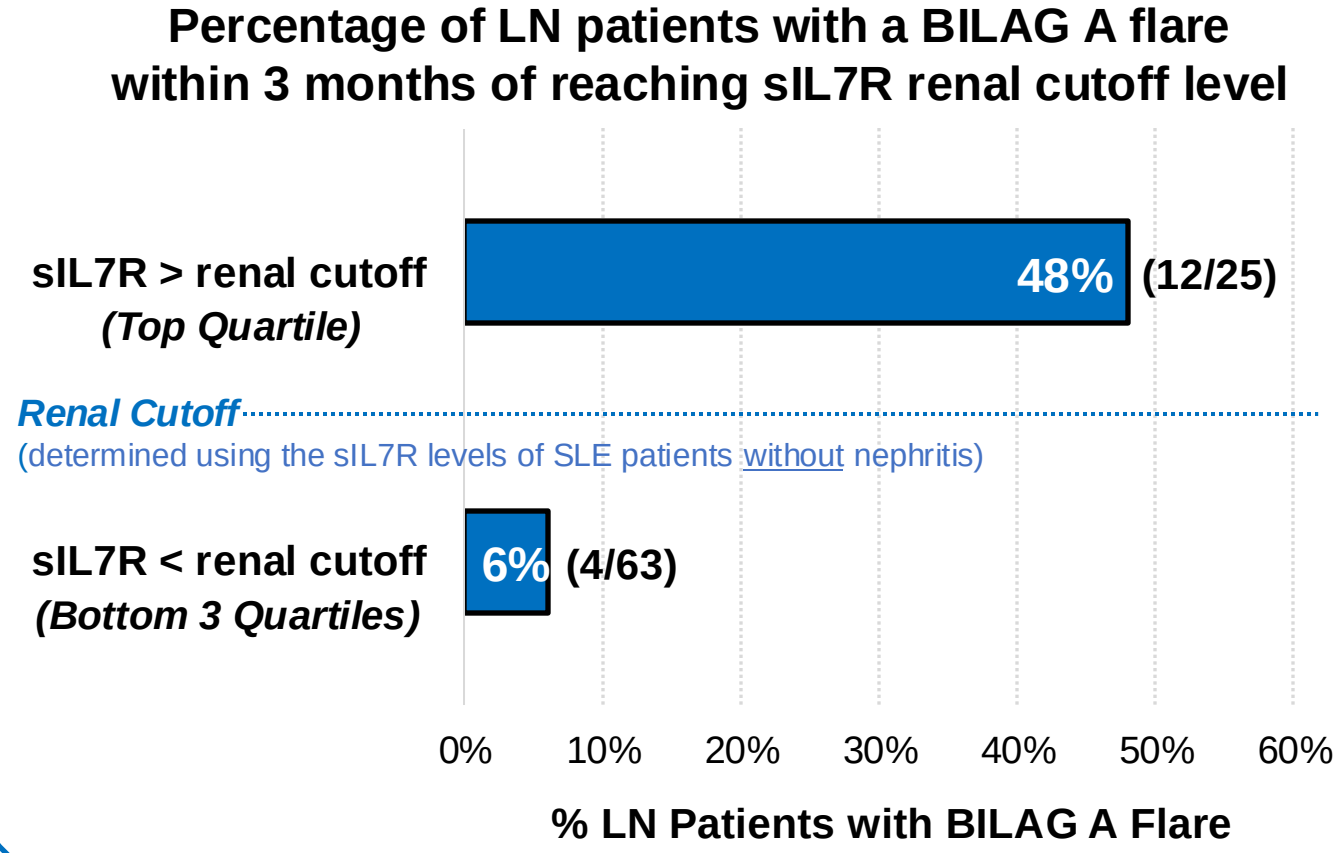
The Science	Disease Impact	Examples
▻ SNP elevates sIL7R expression by 3-fold. ▻ Elevated sIL7R extends and expands the impact of IL7 on T cells. ▻ Greater activation and proliferation of autoreactive T cells. ▻ Growing pool of effector and memory T cells leads to growing disease severity.	**More Severe Flares**	***Lupus Nephritis (LN)*** **LN patients with highest sIL7R levels have an *8-fold higher* incidence of severe flares (48% vs 6%).**
	Greater Risk of Progression	***Progressive MS (PMS)*** **MS patients with the SNP and elevated sIL7R have a 37% higher risk of developing Progressive MS.**
	Poor Response to Therapy	***Rheumatoid arthritis (RA)*** **RA patients with highest sIL7R were most likely to be non-responders to anti-TNF therapy.**

Lupus Nephritis (LN): The Top Quartile of LN Patients by sIL7R Levels Had an *8-fold Higher* Risk of a Severe Flare (BILAG A) Within 3 Months

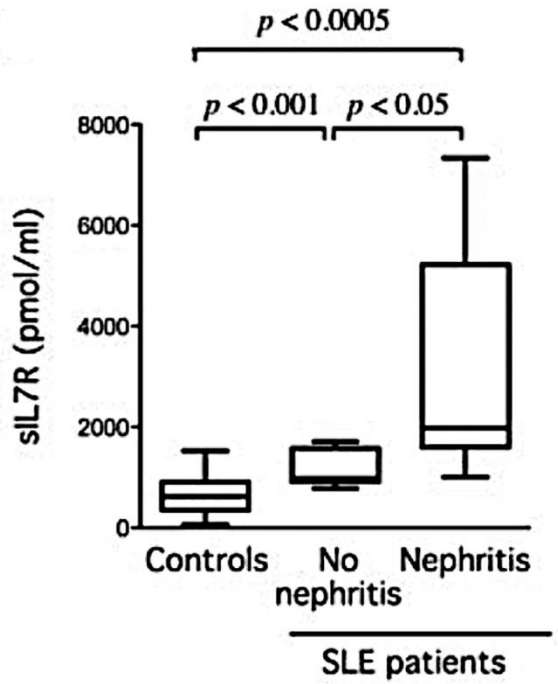
- 88 LN patients with normal kidney function (GFR >60) were assessed for levels of sIL7R.
- 25 of the 88 subjects had sIL7R levels above this renal cutoff level.
- ***12 of the 25 with the highest sIL7R (48%) had a BILAG A flare within 3 months of reaching renal cutoff value, as compared to 4 of the 63 (6%) with lower sIL7R.***
- Urinary Protein/Creatinine levels also higher in patients with elevated sIL7R (p=0.01)



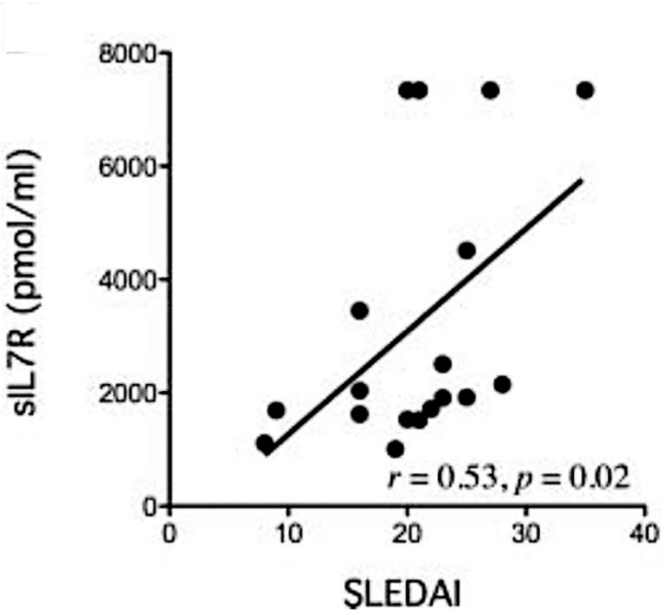
Lauwerys et al., 2014, **Lupus Science and Medicine,** 1:e000036

Lupus Nephritis (LN): Strong Correlation of sIL7R with the Development of LN and Disease Severity

Elevated sIL7R in SLE, particularly in LN



Strong Correlation of sIL7R with Disease Activity in LN



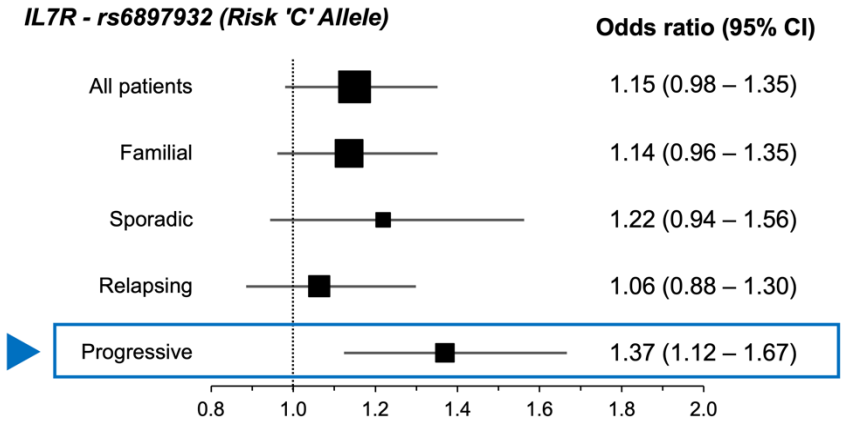
The levels of sIL7R are the highest in LN and correlate with disease activity:

Healthy controls < SLE (w/o nephritis) < **Lupus Nephritis**

*Badot et al., 2013, **Ann Rheum Dis.** 72 (3): 453-456.PMID: 23264357*

Progressive MS: The SNP Enhances Risk to Develop Progressive MS, sIL7R is Elevated in Primary Progressive MS

The sIL7R-enhancing SNP rs6897932 increases the risk of Progressive MS

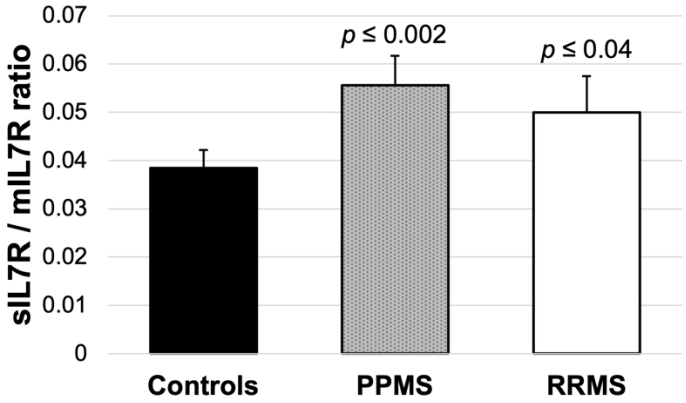


37% higher risk to develop Progressive MS

Adapted from: Traboulsee et al., 2014, ***Neurogenetics*** *15(3): 165-9.*
PMID: 24770783

(The SNP data in this publication was analyzed for the protective 'T' allele and is shown here transformed to the risk 'C' allele for clarity.)

sIL7R RNA is elevated in Primary Progressive MS (PPMS)



sIL7R expression is the highest in PPMS:

[measured as the ratio of sIL7R to mIL7R]

Healthy Controls < RRMS < **PPMS**

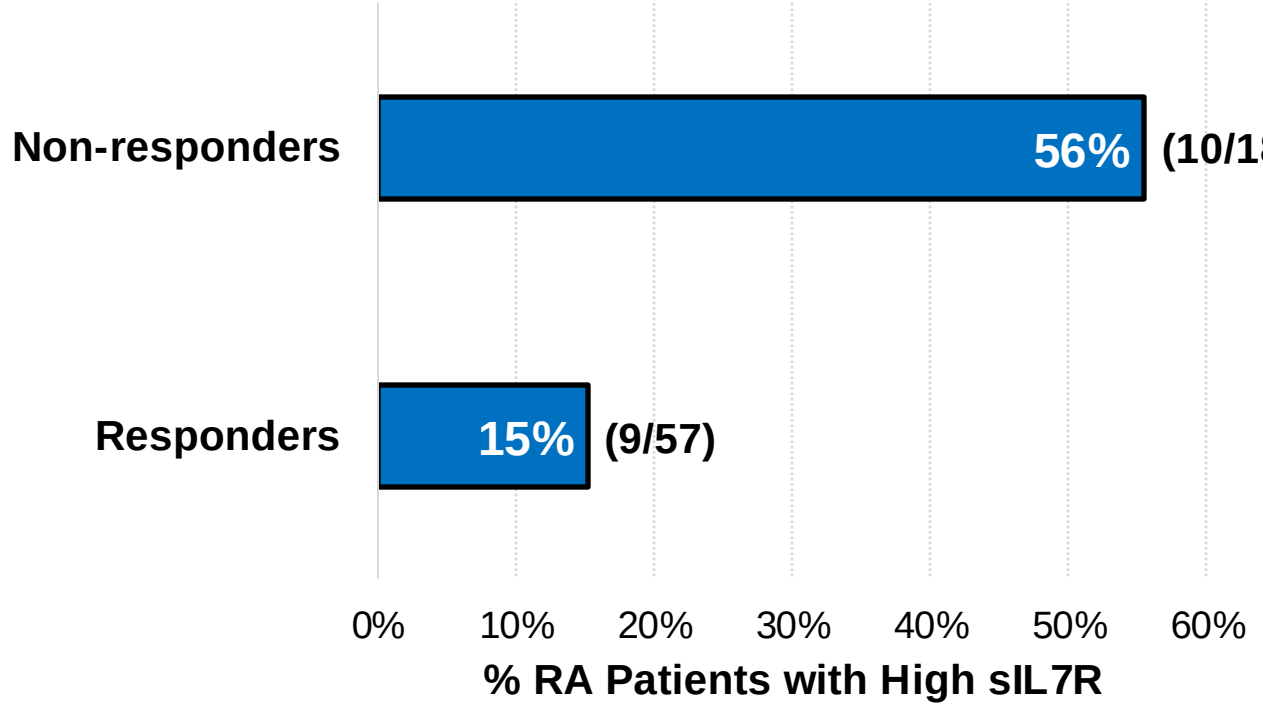
Adapted from: McKay et al., 2008, ***Genes Immun.*** *9(1): 1-6.*
PMID: 17928869

(The published data was shown as mIL7R/sIL7R.
For clarity, it is shown here transformed to sIL7R/mIL7R.)

Rheumatoid arthritis (RA): Patients with high sIL7R Levels Had a *3-fold Higher* Risk of Failure in Response to Treatment

- In DMARD-resistant RA patients, elevated sIL7R levels strongly correlated with poor response to subsequent anti-TNF therapy (infliximab):
  - High sIL7R strongly correlated with poor response to infliximab, whereas low sIL7R strongly correlated with adequate response.
  - **56% of non-responders had high sIL7R**, whereas **only 15% of responders had high sIL7R**.
- sIL7R is an accurate predictor of response to infliximab therapy:
  - **Low levels of sIL7R** = **87%** probability of being responders.
  - **High levels of sIL7R** = **29%** probability of being responders.

Percentage of LN patients with high levels of sIL7R among responders and non-responders to treatment



Badot *et al., 2011,* ***J Cell Mol Med*** 15, 2335-2342.

ABS Development Plan:

Monoclonal Antibody to Normalize sIL7R and Provide Clinical Benefit

SNP Elevates sIL7R by 3-fold in MS Driving Disease Severity:
Goal of ABS Therapy – Normalize sIL7R with a Selective Antibody

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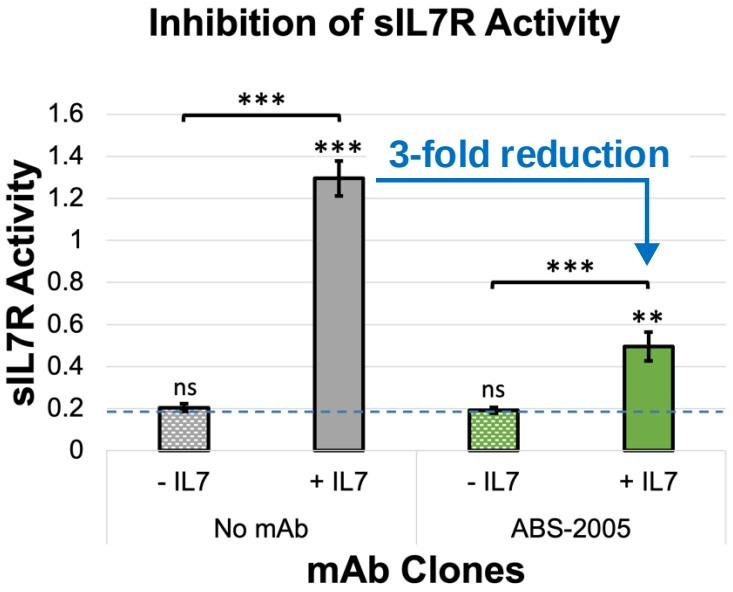
- **Goal of ABS therapy:** 3-fold reduction in sIL7R levels and/or function to normalize immune response.
- **ABS mAb therapy achieves this normalization goal *ex vivo*.**
- 20 selective α-sIL7R antibody candidates in evaluation now.
- **Seed round** will generate lead candidate and back-ups.

Anti-sIL7R candidate mAb (ABS-2005) achieves 'normalization' goal



ABS mAb therapy reduces sIL7R activity by 3-fold in functional *ex vivo* assay with human T cells.

Galarza-Munoz et al., unpublished results.

CDI

Collaboration with CDI Laboratories

(spinoff of John Hopkins University and pioneers of developing of highly selective, mono-specific monoclonal antibodies)

Ongoing Selection and Validation of Murine α-sIL7R mAb Leads: Leads for Both Therapeutic and Diagnostic Reagents are Expected in Q2 2025

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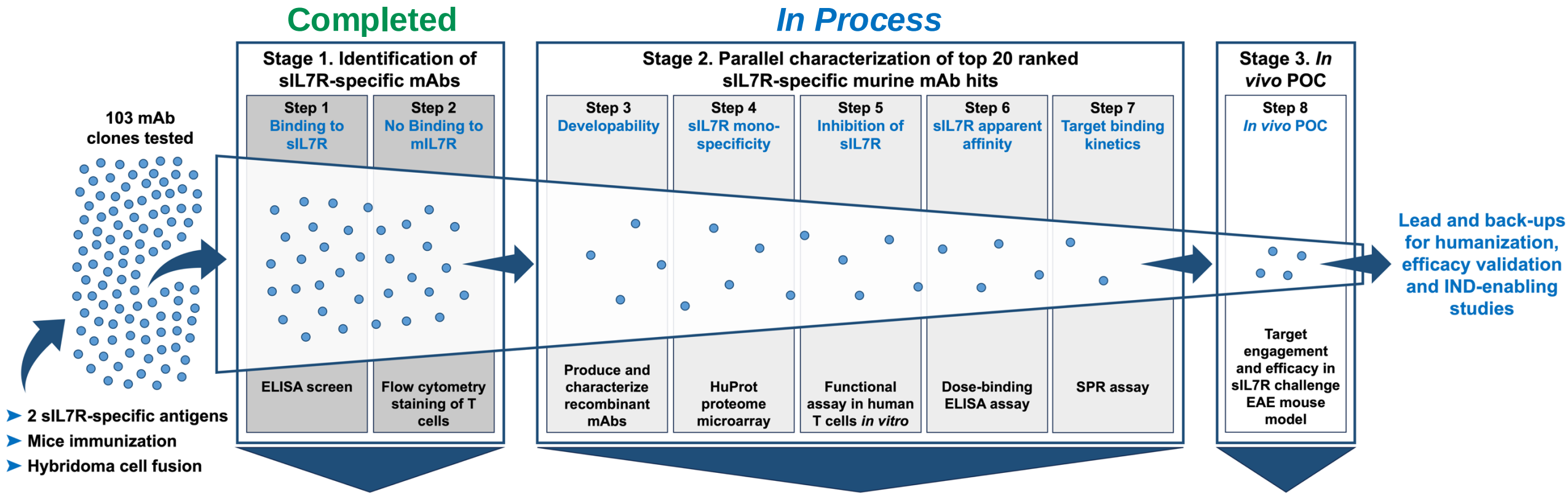

34 mAb clones bound the target with 100% specificity

Top 60% (20 hits) will be prioritized for further characterization based on sIL7R binding affinity.

sIL7R specificity is critical since the SNP only upregulates sIL7R and mAbs against mIL7R cause immunosuppression.

All 20 top ranked mAbs will be characterized in parallel and the top 4 ranked mAbs will be tested *in vivo*

So far, all mAb hits tested have the desired activity:

- ➤ 8/8 hits bound sIL7R with nM apparent affinity.
- ➤ 2/2 hits inhibited sIL7R activity (current leads).

The top 4 *in vivo* validated mAb leads will be humanized for further efficacy testing in appropriate animal models of various indications, including EAE and LN.

Development Strategy: From Preclinical Candidate Selection to Initial Clinical POC

	2025	2026	2027	2028
Indication	**Series Seed: $4-5M**	**Series A: $30M - $40M (varies based on scope)**		
	Selection and Validation of Preclinical Lead Candidate	IND-enabling work for lead program: GLP Tox; GMP manufacturing	**Clinical POC**	
LN	*In vivo* models; patient bio-sample assessment for support of biology, trial design input; KOL and Association Collaborations		➤ Enrich for baseline sIL7R ➤ Time to event – BILAG A ➤ Urine Biomarkers	
MS	*In vivo* models; patient bio-sample assessment for support of biology, trial design input; KOL and Association Collaborations		➤ Slowing indicators of progression or POC in SPMS	
Dx	Collaboration with Large Diagnostics Player Assays to quantify sIL7R in plasma by ELISA and genotype the SNP			
Others		Additional *in vitro* / *in vivo* evaluation in other indications (e.g., T1D, SLE, RA)		

Intellectual Property: Broad Protection of sIL7R Pathway

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Filed Patent Applications

Patent Coverage: Exclusive License from University of Texas (UT) covers:
- Various drug modalities (mAbs and ASOs).
- Numerous indications in autoimmune disease and cancer.
- U.S. and International rights, with other applications in drafting.

5
Patent Families

- mAbs for autoimmune diseases (Composition of Matter, and Methods)
- ASOs for autoimmune diseases (Composition of Matter, and Methods)
- ASOs for cancer (Composition of Matter, and Methods)
- Companion diagnostic (multiplex assay for risk assessment)
- Companion diagnostic (mAbs for specific quantification of sIL7R)

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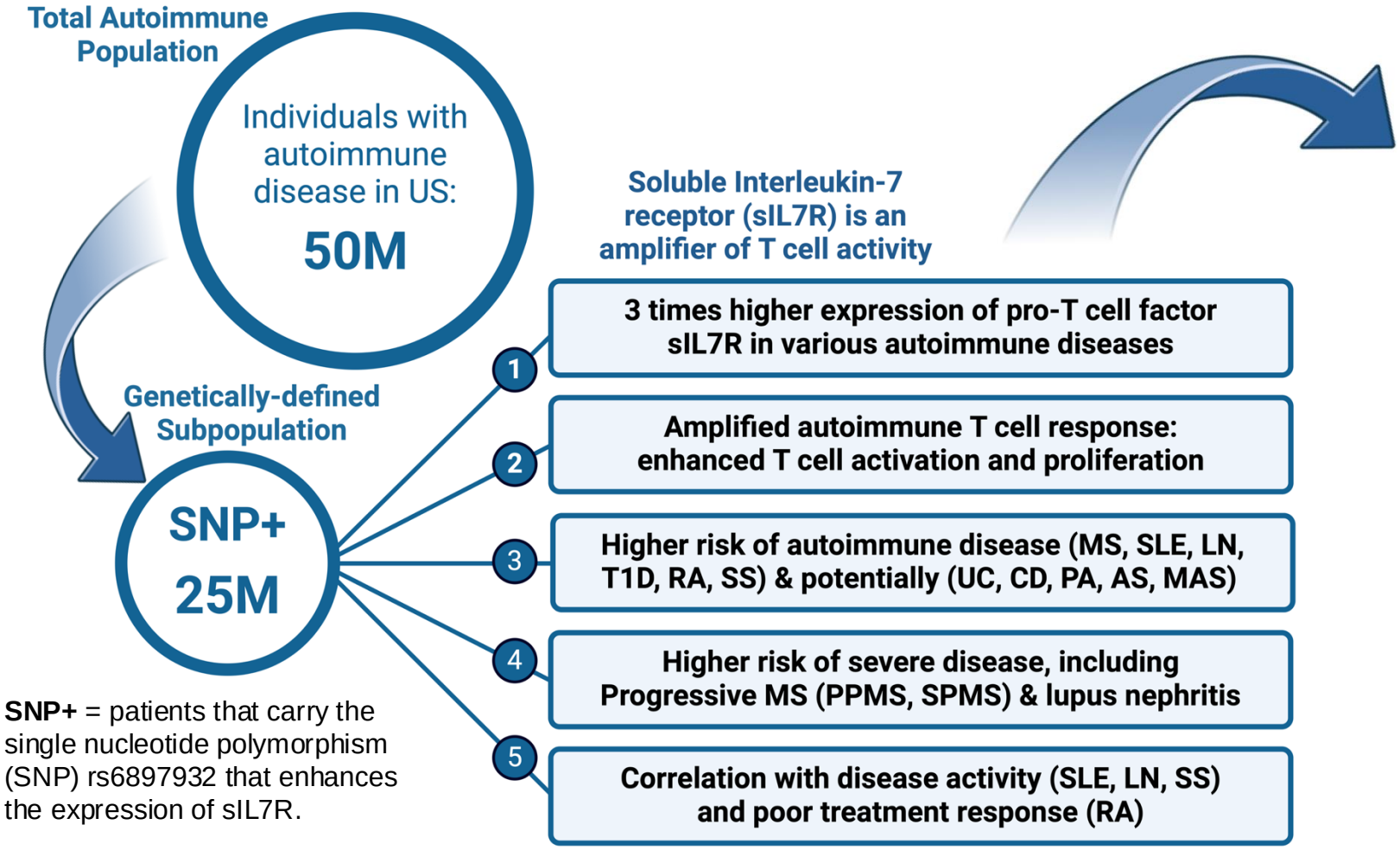
Issued Patents

Patents Issued / Allowed: 2 Families
- ASOs for autoimmune diseases (U.S. 11,118,186 B2; U.S. 11,807,853 B2; and US20240093203A1)
- Companion diagnostic (U.S. 10,961,581 B2; and US20210180133A1)

Experienced IP Attorney

General Counselor: Peter Weinstein, PhD, JD (Entralta Law Firm)
- Accomplished attorney on Patent Litigation, Prosecution, Transaction & Corporate Law.
- Founder & CEO, Entralta; Former Senior Counsel, Baxter Healthcare Corporation; Former Attorney, Brobeck, Phleger & Harrison, and Fish & Richardson; Former Examiner, US Patent and Trademark Office.

ABS Vision: Broad Potential for Genetically Targeted *Immuno-Corrective* Therapy in Autoimmune Diseases



SNP+ = patients that carry the single nucleotide polymorphism (SNP) rs6897932 that enhances the expression of sIL7R.

ABS Therapeutic Goal:

To normalize sIL7R levels in patients with the SNP to restore immune balance (immune homeostasis), and thereby reduce or eliminate the need for immunosuppressive therapy.

ABS Vision:

To establish anti-sIL7R therapy as a safe and effective first line / base line therapy for patients with the SNP.

MS = multiple sclerosis; SLE = systemic lupus erythematosus; LN = lupus nephritis; T1D = type 1 diabetes; RA = rheumatoid arthritis; SS = Sjogren's syndrome; UC = ulcerative colitis; CD = Crohn's disease; PA = psoriatic arthritis; AS = ankylosing spondylitis; MAS = multiple autoimmune syndrome; PPMS = primary progressive MS; SPMS = secondary progressive MS.

ABS Investment Thesis: **Summary**

- The SNP leads to 3-fold higher sIL7R leading to greater risk of autoimmune disease and greater disease severity.
  - **SNP role discovered by ABS Scientific Founders.**
- Highly prevalent SNP – over 50% of Autoimmune Disease patients.
  - Addressable market of ~25M in the U.S., ~60M Global
- Potential to address area of great unmet need in Lupus Nephritis
  - Elevated sIL7R leads to an 8-fold higher rate of severe flares.
  - Goal of ABS antibody therapy – normalize sIL7R, reduce severity of disease and slow progression to dialysis.
  - High unmet need, potential for efficient clinical POC and rapid route to approval.
- Potential in MS to demonstrate prevention of progression, supporting first line or baseline treatment for all patients with the SNP.
  - Easily diagnosable biomarkers with proprietary assays: SNP and circulating levels of sIL7R.
  - ABS seeking collaboration with large diagnostics partner to roll out diagnostics.
- Clinical benefit in Lupus Nephritis + First line treatment to slow progression for all autoimmune patients with the SNP equals **revenue potential in the $ Billions**.
- Large pharma is very actively looking to acquire next generation autoimmune disease therapies.

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ABS Team:

Management Team, BOD, SAB, and Key Consultants

Management Team: CEO & CSO

Eugene Williams, MBA

CEO

- **Senior life science executive and serial entrepreneur (>40 ys experience)**
- **Significant Experience in founding, R&D management, commercialization, deal making, etc., in biotech and pharma**
- Chair & Co-Founder, ProMIS Neurosciences
- Former SVP & General Manager, Genzyme (Immune Mediated Diseases)

Gaddiel Galarza-Muñoz, PhD

Founder and CSO

- **Principal Investigator, ABS**
- **Discoverer of sIL7R pathway, developer of sIL7R-targeting drugs & awarded $2.7M in grants for ABS**
- Adjunct Assistant Professor, Neurology, University of Texas Medical Branch (UTMB)
- PhD Neurobiology, University of Puerto Rico
- Postdoc (Molecular Genetics & RNA Biology), Duke University and UTMB

Board of Directors

Brock Reeve, M.Phil, MBA – *Board Chairman*

- **Co-founder & CEO, Eos BioInnovation**
- Former Executive Director, Harvard Stem Cell Institute (2006-2022) & Former CEO, IVIVA Medical
- Director: Axonis Therapeutics; HDAX Therapeutics; Regatta Bio; Thrive Bioscience

Eugene Williams, MBA – *CEO & Board Director*

- **Senior life science executive and serial entrepreneur with >40 years of experience in biotech & pharma.**
- Chairman and Co-Founder, ProMIS Neurosciences
- Former SVP & General Manager, Genzyme (Immune Mediated Diseases)

Mariano A. Garcia-Blanco, MD, PhD – *Founder, SAB Chair & Board Director*

- **Chair & Professor, Microbiology, Immunology & Cancer Biology, University of Virginia**
- Founder of five companies
- Member of American Academy of Arts & Science; 190+ peer-reviewed publications

Jimmy Lu, JD, MBA – *Board Director*

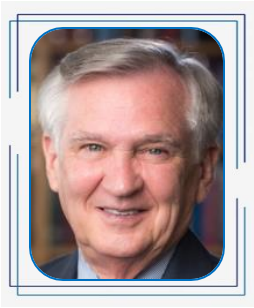
- **Co-founder & Managing Director, Eos BioInnovation**
- Venture Partner, Panacea Venture; Former Managing Director, WI Harper
- Director, Window Therapeutics; Former Director, StemCyte

Scientific Advisory Board: Key Expertise

Mariano A. Garcia-Blanco, MD, PhD – SAB Chair

RNA Biology and Immunology

- Chair & Professor, Microbiology, Immunology & Cancer Biology, University of Virginia; **Founder of five companies.**
- **Member of American Academy of Arts & Science with over 190 peer-reviewed publications**



Edward J. Benz, Jr., MD

Oncology and RNA splicing

- Emeritus CEO, Dana-Farber Cancer Institute
- Richard & Susan Smith Distinguished Professor of Medicine, Harvard Medical

Adrian R. Krainer, PhD

Therapeutic ASOs (academic dev. Spinraza®)

- St. Giles Professor & Cancer Center Research Deputy Director, Cold Spring Harbor Laboratory
- Co-founder & Director, Stoke Therapeutics

George J. Hutton, MD

Principal investigator in > 40 MS trials

- Professor and Vice Chair for Clinical Affairs, Neurology, Baylor College of Medicine
- Medical Director, Maxine Mesinger Multiple Sclerosis Clinic

Sir Richard Roberts, PhD

Nobel Prize in Medicine for RNA splicing

- Chief Scientific Officer, New England Biolabs
- Expert on RNA splicing and scientific product innovation

Consultants/Advisors: Key Business and R&D Expertise

Business

R&D

Thomas F. Farb-Horch

Financing

- Senior life science executive and serial entrepreneur (18 companies, 7 multi-billion exits, >35 years experience)
- **Early-stage financing: >$750 million**

Takashi Kiyoizumi, MD, PhD, MBA

Business Development

- Senior life science executive, serial entrepreneur and angel investor (>40 years experience)
- **Significant BD Experience: >25 strategic and licensing deals**

Bobby W. Sandage, PhD

Drug Development & Regulatory Affairs

- Senior life science executive, investor and serial entrepreneur (>40 years experience)
- **Significant Drug Dev. & Regulatory Experience: >14 approved drugs**

Patrick Hextall

mAb Drug Development & CMC

- Serial mAb development project manager in biotech and as consultant (>20 years experience)
- **Extensive experience in therapeutic mAb development: >15 mAbs developed (phase I-III)**

Mark Maginn, PhD, MBA

NHP Pharmacology & GLP Toxicology

- Significant drug development experience with CROs, pharma and biotech (>25 years experience)
- **Extensive preclinical drug development experience: >25 INDs, 4 approved drugs**