KEZAR LIFE SCIENCES

Selective Targets. Broad Impact.

Uniquely Powerful Approaches to Tackling the Toughest Diseases

Corporate Overview
September 2021

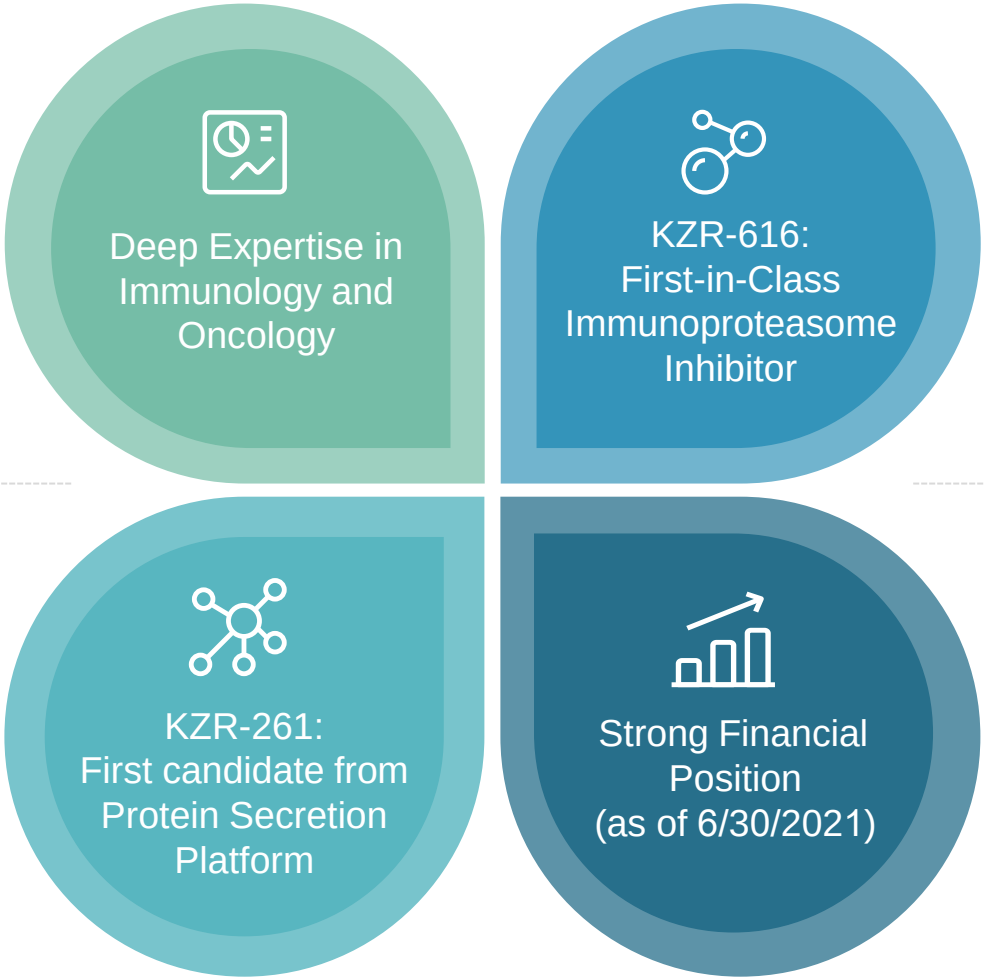
Forward-Looking Statements Disclaimer

This presentation contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Words such as “may,” “will,” “should,” “expect,” “plan,” “anticipate,” “target,” and similar expressions (as well as other words or expressions referencing future events or circumstances) are intended to identify forward-looking statements. Each of these forward-looking statements involves substantial risks and uncertainties that could cause actual results to differ significantly from those expressed or implied by such forward-looking statements. Forward-looking statements contained in this presentation include, but are not limited to, statements regarding the disruption of our business and clinical trials from the global outbreak of a novel strain of coronavirus (COVID-19), the potential use of our product candidates to treat patients, the association of data with treatment outcomes, the design, timing of initiation, progress, enrollment and scope of clinical trials for our product candidates, the expected timing of program updates and data disclosures, the timing of filing INDs and other regulatory documents, the timing and likelihood of seeking regulatory approval for our product candidates, and the patient prevalence, regulatory pathway and competitive landscape for our product candidates.

These forward-looking statements reflect Kezar's current beliefs and expectations. Many factors may cause differences between current expectations and actual results, including unexpected safety or efficacy data observed during preclinical or clinical studies, clinical site activation rates or clinical trial enrollment rates that are lower than expected, changes in expected or existing competition, changes in the regulatory environment, and unexpected litigation or other disputes. Other factors that may cause our actual results to differ from current expectations are discussed in Kezar’s most recent Form 10-K or Form 10-Q filed with the U.S. Securities and Exchange Commission (SEC), under the caption “Risk Factors” and elsewhere in such reports. Except as required by law, we assume no obligation to update any forward-looking statements contained herein to reflect any change in expectations, even as new information becomes available.

The Kezar Opportunity: Harnessing Master Regulators of Cellular Function to Tackle Immune-mediated Diseases and Cancer

Builds on 10+ years of R&D work in proteasome biology and protein secretion led by Chris Kirk and Jack Taunton



A novel approach to harmonizing the immune system; Potential to be a pipeline in a drug

First in class agent with broad anti-tumor activity; Potential to inhibit multiple targets with a single small molecule

$129M cash, cash equivalents, and marketable securities; 48.1M common shares outstanding

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Our Programs Inhibit Multiple Drivers of Disease via Selective Targets to Address a Diverse Pipeline of Indications

COMPOUND	THERAPEUTIC INDICATION	DEVELOPMENT STAGE				MILESTONES
		PRE-CLINICAL	PHASE 1	PHASE 2	PHASE 3	
Selective Immunoproteasome Inhibition						
KZR-616	Lupus Nephritis (LN)			MISSION		Q4 2021 (interim) 1H 2022 (top-line data)
	Dermatomyositis (DM) Polymyositis (PM)			PRESIDIO		1H 2022 (top-line data)
Protein Secretion Inhibition						
KZR-261	Oncology					IND Accepted
KZR-TBD	Oncology & Autoimmunity					N/A

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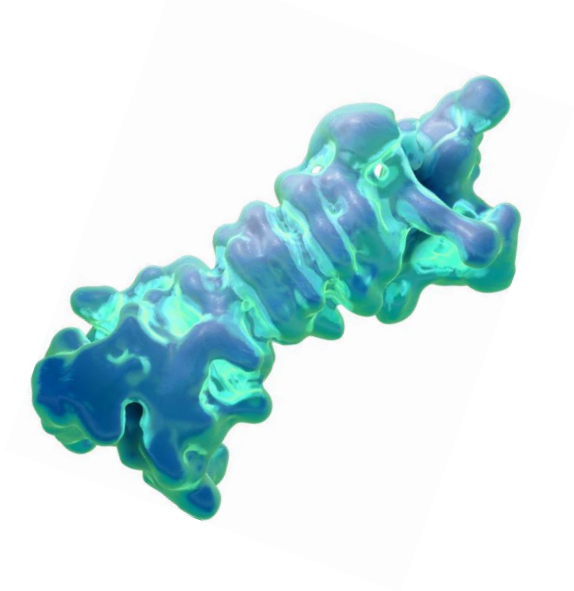
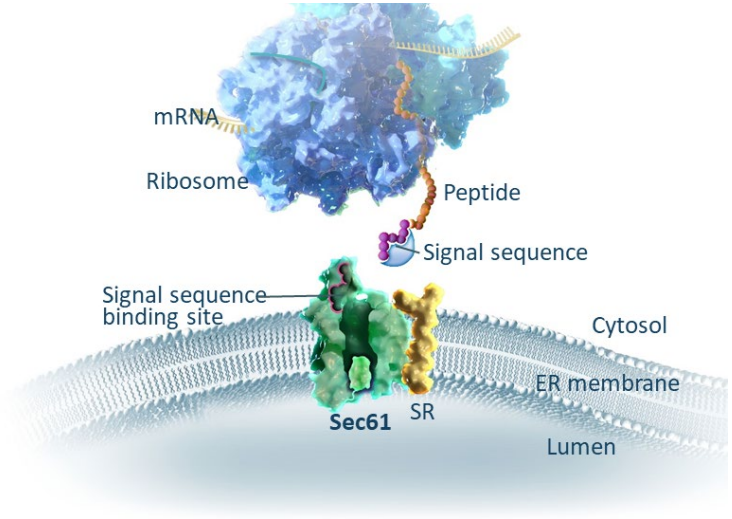
Kezar's Novel, Complementary Programs Target Master Regulators of Cellular Function to Achieve Broad Therapeutic Activity

PROTEIN DEGRADATION:
The Immunoproteasome



- Modulates multiple drivers of inflammation
- Restores normal immune responses, while potentially avoiding immunosuppression

PROTEIN SECRETION:
The Sec61 Translocon



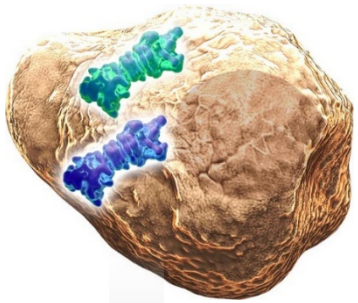
- Broad anti-tumor activity in preclinical models
- Applications in oncology, immuno-oncology, and autoimmunity
- Potential for small molecules to replace certain biologics

Selective Immunoproteasome Inhibition is not Cytotoxic and Results in Broad Immunomodulatory Activity Across the Adaptive and Innate Immune System

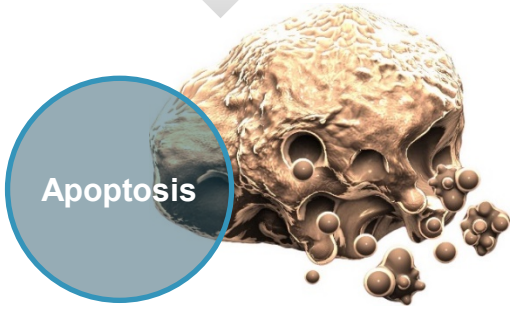
Dual-Targeting Proteasome Inhibitors

Myeloma Cell

Bortezomib
Carfilzomib

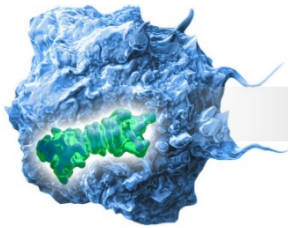


Dual inhibition required for cell death



Selective Immunoproteasome Inhibitors

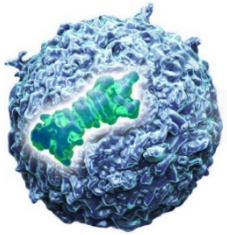
Macrophage



TNF- α
IL-23
IL-6

KZR-616
ONX-0914

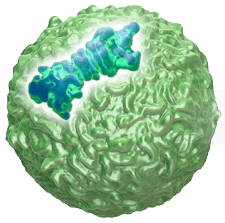
T-cell



Th1
Th17

Treg

B-cell



auto-Ab

Parlati et al. Blood 2009

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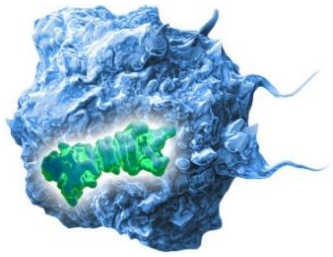
Inflammatory Disorders are Currently Treated One Cytokine or Cell at a Time, but the Immunoproteasome Covers Them All

Macrophages
Dendritic Cells
Monocytes



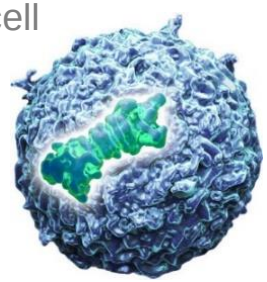
Cytokines
TNF-α
IL-6
IL-23

Humira, et al

Actemra

Skyrizi/Stelara

T-cell



Orencia

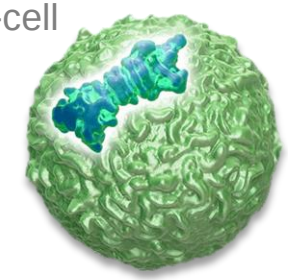
Lupkynis (voclosporin)

Xeljanz

Cytokines
IL-17

Cosentyx/Taltz

B-cell



Rituxan/Gazyva

Benlysta

KZR-616

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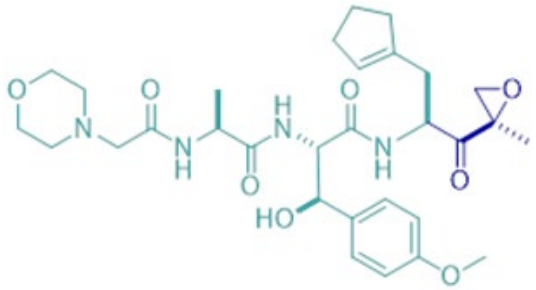
KZR-616 is Well-Positioned to be Used as a Chronic Therapy

KZR-616



EASY TO USE

Subcutaneous, weekly dosing
Amenable to patient self-administration

SAFE TO USE

Lack of immunosuppression
No off-target effects
Low DDI risk
Avoids steroid-related side effects

Selective Immunoproteasome Inhibition with KZR-616 has the Potential to Address Multiple Chronic Immune-Mediated Diseases

CURRENT TREATMENT PARADIGMS CAN BE INADEQUATE

- Existing therapies are ineffective in many patients
- Prolonged standard immunosuppressive treatment use results in significant complications (osteoporosis, muscle weakness, infections, bone marrow suppression, hepatoxicity, etc.)
- Adverse Events from immunosuppressive drugs contribute to high morbidity/mortality
- Targeted therapies (ie, biologics) may not address needs of all patients with diseases characterized by defects in multiple arms of the immune system

Indication	Clinical Data with Dual Proteasome Inhibitors	Preclinical Data w/Kezar Compounds (ONX 0914/ KZR-616)
Orphan and/or High Unmet Need		
Myositis	✓*	✓
Lupus Nephritis (LN)	✓	✓
Antibody-mediated transplant rejection	✓	✓
Graft versus host disease (GVHD)	✓	✓
Myasthenia gravis (MG)	✓	✓
ITP	✓	
AIHA	✓	
IgA nephropathy (IgAN)	✓	
IgG4-related disease	✓	
Neuromyelitis optica (NMO)	✓	
Pemphigoid	✓	
CIDP	✓	
Anti-NMDA encephalitis	✓	
ANCA-associated vasculitis (AAV)	✓	
Large Market		
Rheumatoid Arthritis (RA)	✓	✓
Systemic Lupus Erythematosus (SLE)	✓	✓
Multiple Sclerosis (MS)		✓
Crohn's disease (CD)		✓
Type 1 Diabetes		✓

**Comorbid myositis*

Almani S et al. CJASN 2017
Oddis C et al. Nat Rev Rheumatol 2018
Barcellini W et al. Expert Rev Clin Immunol 2018

Abbreviations: ITP, Immune Thrombocytopenia; AIHA, Autoimmune HemCIDP, Chronic Inflammatory demyelinating polyneuropathy (CIDP)

Kezar is Currently Focusing its Development Efforts with KZR-616 on "Medium-Rare" Immune-mediated Diseases of High Unmet Need

Strategic

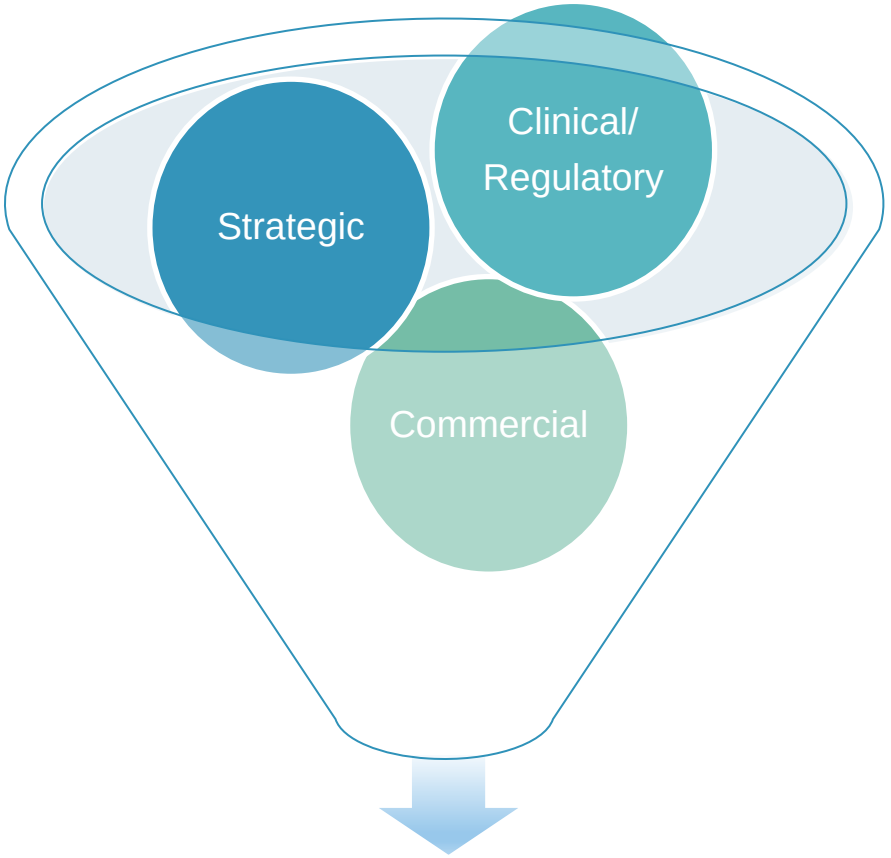
- High disease morbidity and/or mortality
- Limited effective treatment options available

Clinical/Regulatory

- Biologic fit based on MoA and disease characteristics
- Validated endpoints and expedited clinical and regulatory pathway
- Proof of principle with proteasome inhibitors

Commercial

- Clearly defined patient population
- Limited approved competition



Medium-Rare Immune-Mediated Diseases

(~30K-150K U.S. patients)

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KZR-616 has a Favorable Safety and Tolerability Profile for Use in Chronic Immune-Mediated Diseases

Safety

- Majority of treatment emergent adverse events (TEAEs) have been mild or moderate
- No clinically significant laboratory adverse events
- Preliminary data shows reduced risk for toxicities and AEs vs. standard of care and targeted small molecules in autoimmunity[1]
- Low rate of serious (≥ Grade 3) infections (2.2%) suggest lack of immunosuppression with KZR-616

Tolerability

- Injection site reactions are transient, manageable, and the most common TEAE
- Step-up dosing and lyophilized formulation contribute to a favorable tolerability profile

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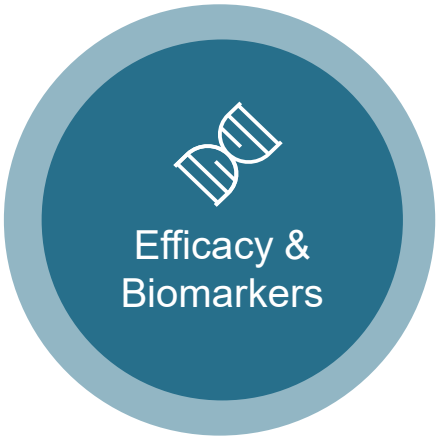
[1]AE rates for SOC from Oglesby et al. Int. J Rheum.2013

Key Attributes of KZR-616 Support Advancement into Phase 2 Trials

Based on data from 2 Healthy Volunteer Studies and MISSION Phase 1b data* in SLE patients (n>100)

- Well-tolerated for 13 weeks of treatment
- Safety profile does not indicate need for patient monitoring



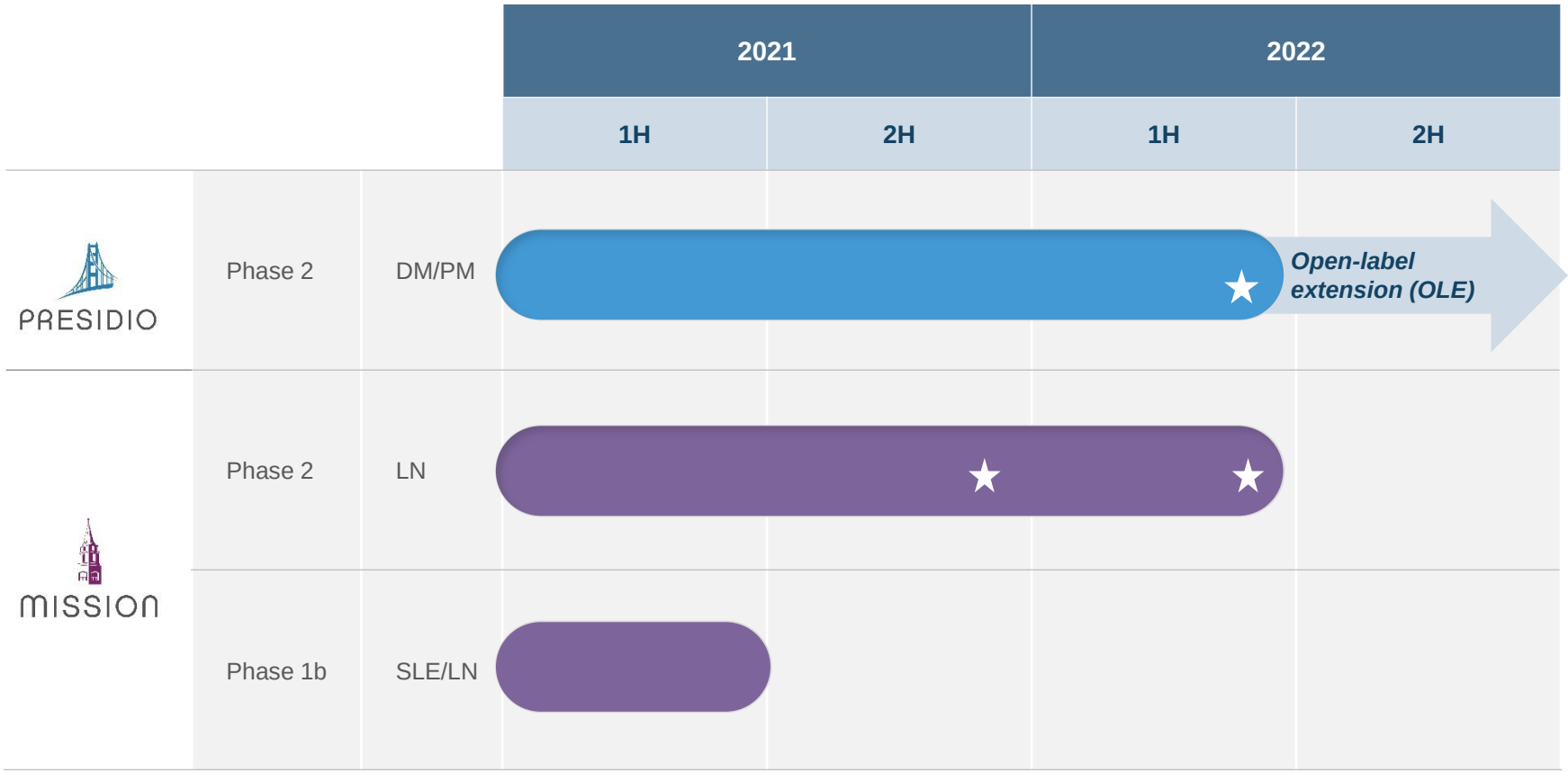
- Improvement across all measured parameters of disease activity
- Rapid and sustained immunomodulatory gene expression changes
- Reduction in key biomarkers of disease activity

- Consistent PK and PD across subjects and with repeat dosing
- Target levels of immunoproteasome inhibition at doses ≥ 30mg

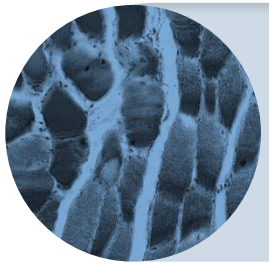
**MISSION Ph1 data as of 4/5/2021*

Furie et al, EULAR 2019, ACR 2019/2020, ASN 2020

KZR-616 is Now in Phase 2 Clinical Trials for the treatment of Dermatomyositis/Polymyositis and Lupus Nephritis, rare diseases with high morbidity and mortality



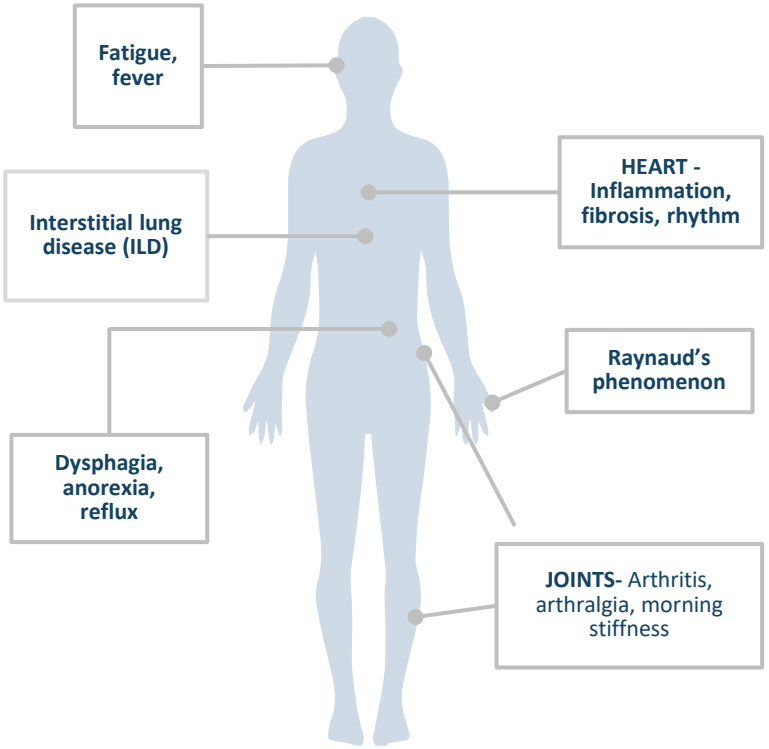
KZR-616 has the Potential to Address the Significant Unmet Need in Dermatomyositis (DM) and Polymyositis (PM)



DM and PM are chronic, severe, and often debilitating autoimmune diseases resulting in high morbidity and mortality. They are characterized by inflammation of the muscles and associated tissues.

Prevalence: up to ~120K US patients

- *Limited effective treatment options*
- *KZR-616 has been granted ODD by the US FDA for both indications*
- *Women 2x as likely to have DM/PM*
- *Prevalence is highest among the black population*



Morbidity

- Difficulty in activities of daily function which can lead to being wheel-chair bound
- Skin rash and ulcerations, itching and pain
- Dyspnea /Cough to respiratory failure
- Calcinosis
- Dysphagia
- Treatment side-effects

Mortality

- ILD, especially progressive ILD
- Cancer associated Dermatomyositis
- Dysphagia—aspiration
- Risk of treatment related infections

Abbreviations: DM, Dermatomyositis; PM, Polymyositis; ODD, Orphan Drug Designation, ILD-Interstitial Lung Disease

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KZR-616 Improved Muscle Function in a Mouse Model of Polymyositis and Dermatomyositis*

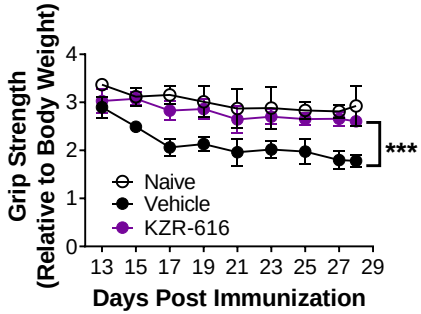
CIM MODEL

- Gold standard model for PM and DM (Sugihara 2007)
- Replicates multiple features of clinical disease

Tx
(2 weeks)

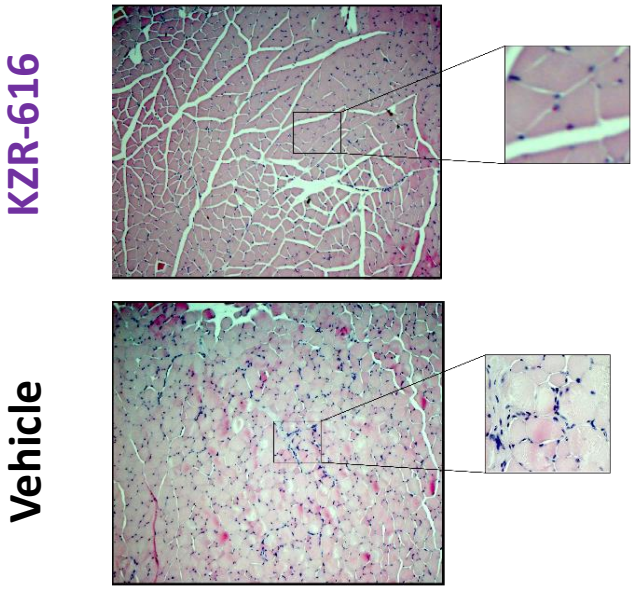
- KZR-616 treatment of diseased animals restored normal muscle function
- Significant reduction in tissue damage (histology and circulating enzyme levels)

Muscle Strength

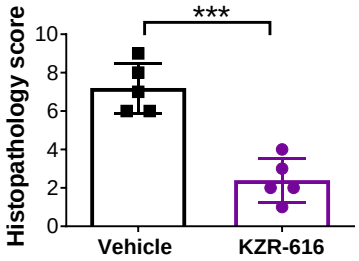


Triceps Histology

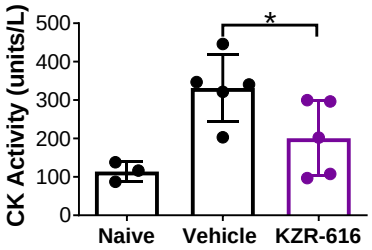
(H&E Staining)



Muscle Histology

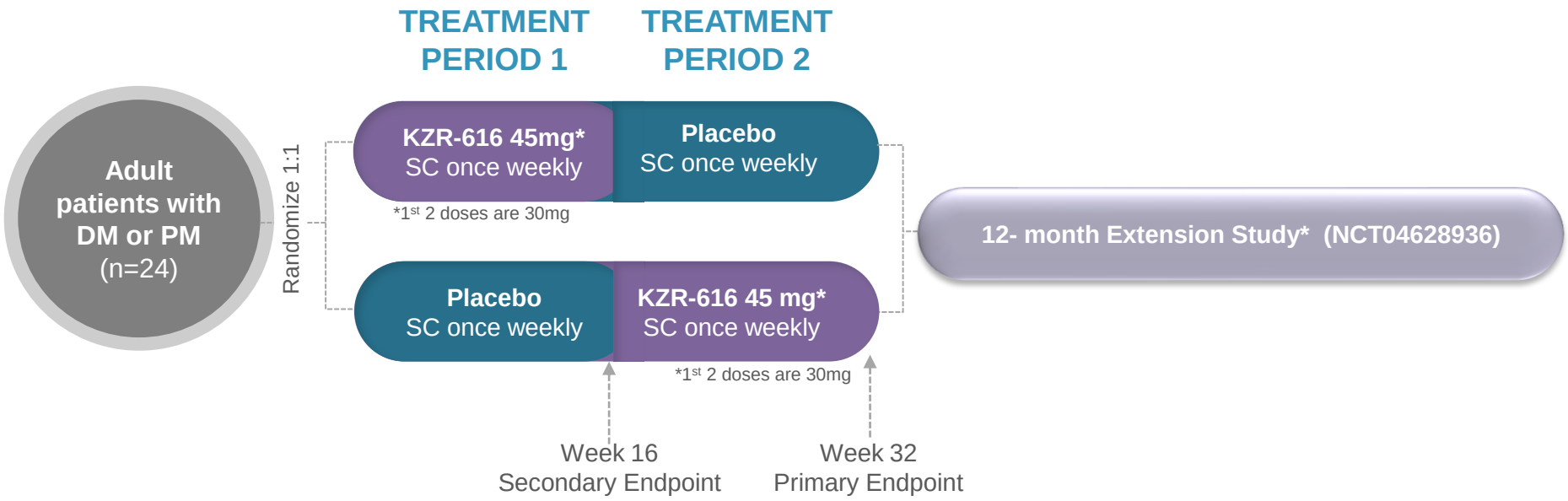


Muscle Enzymes



CIM=C protein-induced myositis
*Oliva MDR et al, ACR 2020

PRESIDIO Phase 2 Placebo-Controlled Cross-over Study for the Treatment of Dermatomyositis and Polymyositis Designed to Inform Late-Stage Studies



ENDPOINTS

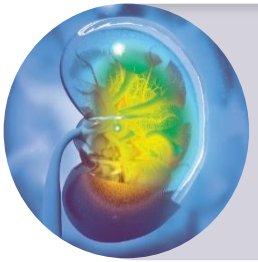
1º: Efficacy: Total Improvement Score (TIS)

2º: Safety and tolerability; Patient Reported Outcomes (PROs), PK

Exploratory: Biomarkers, PK/PD relationship

Study NCT04033926

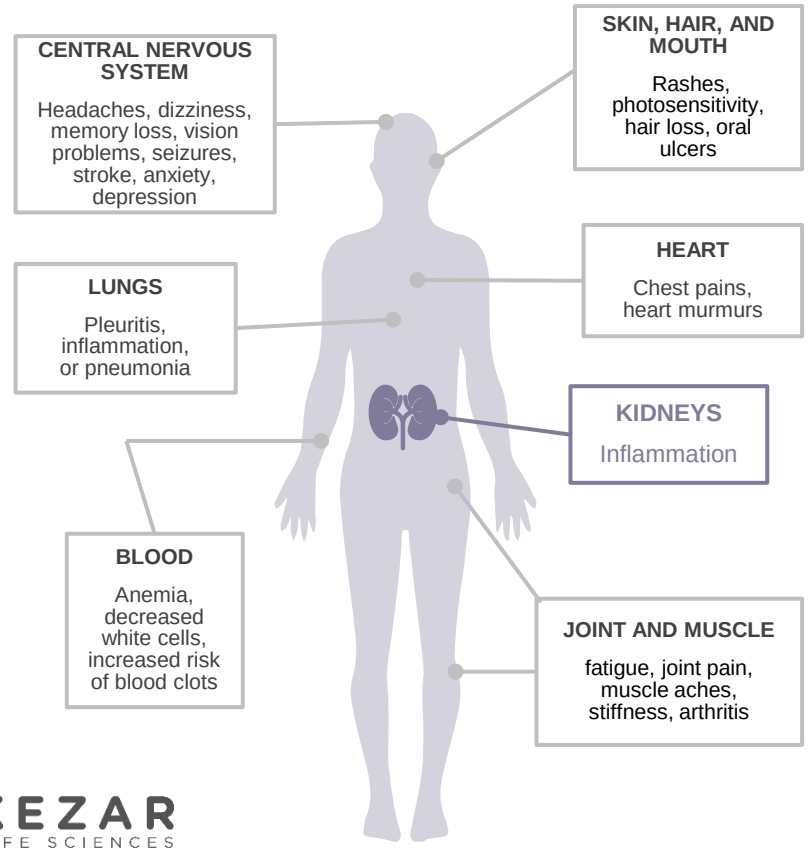
KZR-616 has the Potential to be a Fast-Acting and Broad Immunomodulatory Treatment for Lupus Nephritis



LN is a rare disease involving inflammation of the kidneys and represents a serious complication of Systemic Lupus Erythematosus (SLE). LN and SLE are chronic, debilitating, and progressive diseases that carry an increased risk of death.

Prevalence: *~150K US patients*

- *Limited effective treatments; immunosuppressive drugs carry challenging side effects*
- *Predominantly affects women (9/10) and women of color are disproportionately affected*



- Autoantibody induced immune complexes and autoreactive T-cells result in kidney damage, which can lead to kidney failure
- Significantly increased risk of kidney failure and death relative to non-LN SLE patients
- Quantitative disease measure (reduction in proteinuria) correlates with long-term outcomes
  - Rapidity and durability of response correlates with improved long-term outcomes

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MISSION Phase 2 Trial to Demonstrate Responder Rate of KZR-616 60 mg Weekly and Inform Late-Stage Studies in LN

KEY INCLUSION CRITERIA:

Biopsy-proven Class III or IV +/- Class V LN w/significant proteinuria (UPCR ≥1) despite standard therapy

Must be on stable therapy for at least 8 weeks prior to study entry

ENDPOINTS

1º: Efficacy
Number of patients with ≥ 50% reduction in UPCR

2º:
Safety and tolerability;
Additional renal response parameters (e.g. CRR);
Extra-renal SLE disease indices;
Patient Reported Outcomes

Exploratory:
Biomarkers

NCT03393013

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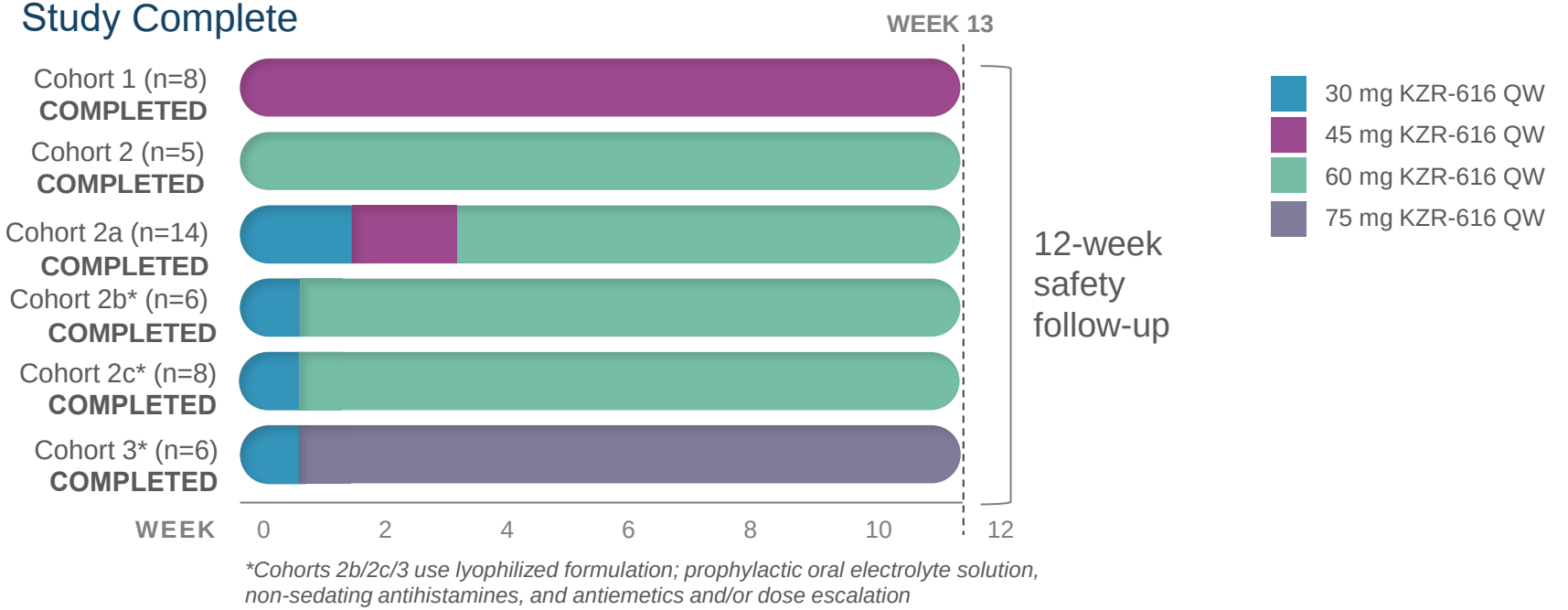
MISSION Phase 1b: Safety, Tolerability, and Exploratory Efficacy in SLE Patients

Study Complete



**Cohorts 2b/2c/3 use lyophilized formulation; prophylactic oral electrolyte solution, non-sedating antihistamines, and antiemetics and/or dose escalation*

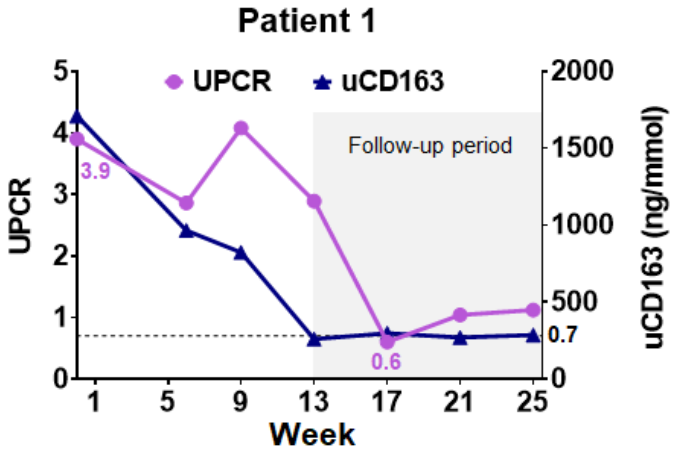
ENDPOINTS

1º: Safety

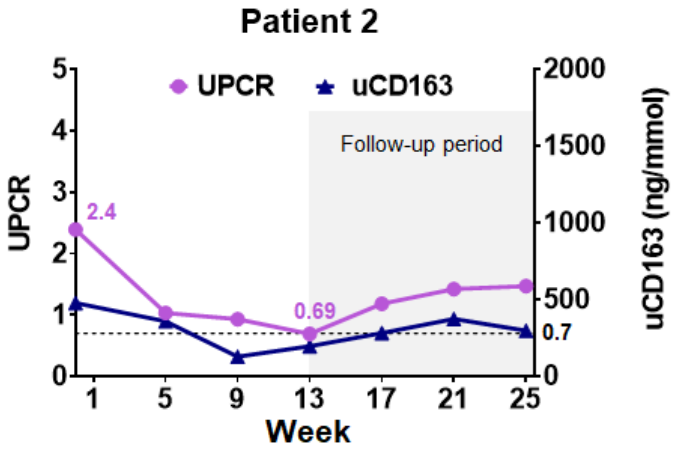
2º: Recommended Phase 2 doses, Plasma PK

Exploratory: Efficacy, PD, Biomarkers, Pharmacogenomics

Results Observed in 2 of 2 LN Patients Treated in the MISSION Phase 1b Showed Rapid Improvement in Renal Function



	SLEDAI-2K	anti-dsDNA(IU/ml)
Baseline	17	137
Week 13	8	53
Week 17	8	73
Week 25	8	61



	SLEDAI-2K	anti-dsDNA(IU/ml)
Baseline	14	123.5
Week 13	8	52
Week 17	8	40
Week 25	NA	53

- Baseline stable treatment regimen of leflunomide, hydroxychloroquine, and prednisone (10mg/day); failed prior tacrolimus
- Nephrotic range
- >50% reduction in UPCR at week 17
- Reduced anti-dsDNA at week 13
- Drug holiday due to AE W2-4 & W11

- Baseline stable treatment regimen of MMF (2g), hydroxychloroquine, and prednisone (10mg/day) Nephrotic range
- >50% reduction in UPCR at week 5
- Improve symptom scores at week 5
- Reduced anti-dsDNA at week 5

End of Treatment W13; End of Study=W25

KZR-616 Demonstrated Improvement on Exploratory Efficacy Measures of Disease Activity Across Organ Systems in SLE

Tool	Improvement	Mean Patient Score* (n=35)		
		Baseline	EOT -W13	EOS -W25
SLEDAI-2K	+	9.1	6.6	7.1
CLASI-A	+	4.3	2.3	2.3
Tender Joint Count	+	11.1	4.8	5.8
Swollen Joint Count	+	7.6	2.5	2.3
Physician Global Assessment Score	+	57.0	39.7	38.2
Patient Pain Assessment	+	58.3	38.2	42.7
Patient Global Assessment Score	+	58.5	43.1	41.7

BL=Baseline; EOT=End of Treatment; EOS=End of Study; W13=week 13; W25=week 25; CLASI-A, Cutaneous Lupus Erythematosus Disease Area and Severity Index-Activity; SD, standard deviation; SLEDAI-2K, Systemic Lupus Erythematosus Disease Activity Index 2000.

Data as of April 5, 2021

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All Patients Experienced Significant Reduction in Anti-dsDNA Levels

Patients Completing Study w/Elevated anti-dsDNA at BL*

Individual	Mean anti-dsDNA level, IU/mL (baseline)	% Change from baseline, week 13 (end of treatment)	% Change from baseline, week 25 (end of study)
Patient A	1015	−64.0	−82.0
Patient B[a]	87	−20.7	−33.3
Patient C	32	−6.3	−18.8
Patient D[b]	134	−60.4	−54.5
Patient E[a]	90	−76.7	−68.9
Patient F[b]	98	−46.9	−45.9
Patient G	29	−17.2	−24.1
Patient H[a]	162	-42.6	-33.3

[a]History of nephritis. [b]Active nephritis.
Abbreviation: anti-dsDNA, anti–double-stranded DNA antibody

**Elevated levels of anti-dsDNA antibodies are highly specific markers of SLE disease activity (>20 IU/mL considered elevated)*

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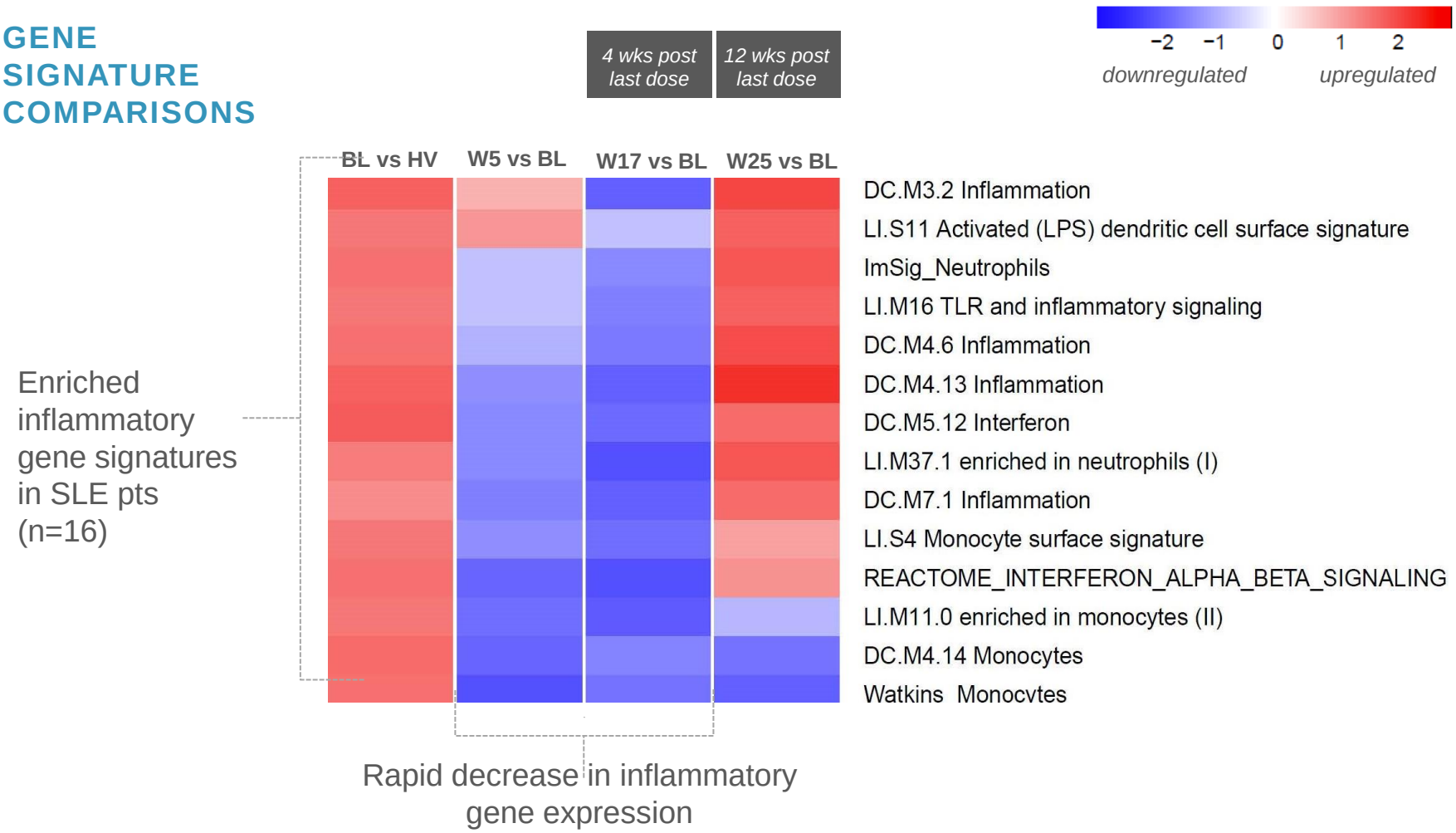
KZR-616's Broad Immunomodulatory Activity is Supported by Whole Blood RNASeq Data from MISSION Phase 1b in SLE Patients

GENE SIGNATURE COMPARISONS



*Fan et al.,*ACR 2019

KZR-616 Continues to Demonstrate a Favorable Safety and Tolerability Profile For Use in Chronic Diseases

Measures, N% of patients	Cohort 1 (n=8)	Cohort 2 (n=5)	Cohort 2a (n=14)	Cohort 2b (n=6)	Cohort 2c (n=8)	Cohort 3 (n=6)	All patients Cohorts 1-3 (n=47)
Target dose, mg	45	60	60	60	60	75	45-75
Mean compliance, %	69.2	52.3	70.3	92.3	100.0	76.9	76.9
≥1 Treatment Emergent Adverse Event (TEAE)	8 (100.0)	5 (100.0)	12 (85.7)	4 (66.7)	7 (87.5)	3 (50.0)	39 (83.0)
Most Common TEAEs							
Injection Site Erythema	5 (62.5)	2 (40.0)	5 (35.7)	2 (33.3)	6 (75.0)	0 (0)	20 (42.6)
Nausea	2 (25.0)	4 (80.0)	5 (35.7)	1 (16.7)	4 (50.0)	3 (50.0)	19 (40.4)
Vomiting	1 (12.5)	5 (100.0)	3 (21.4)	1 (16.7)	2 (25.0)	1 (16.7)	13 (27.7)
Infections and Infestations TEAEs	1 (12.5)	0 (0)	5 (35.7)	2 (33.3)	2 (25.0)	1 (16.7)	11 (23.4)
Serious TEAEs	0 (0)	1 (20.0)	2 (14.3)	1 (16.7)	0 (0)	0 (0)	4 (8.5)
TEAEs leading to discontinuation	3 (37.5)	3 (60.0)	2 (14.3)	0 (0)	0 (0)	2 (33.3)	10 (21.3)
Patients receiving prednisone	5 (62.5)	5 (100.0)	10 (71.4)	4 (66.7)	5 (62.5)	3 (50.0)	31 (66.0)

Cohorts 2b, 2c, and 3 received a lyophilized formulation of KZR-616, prophylactic oral electrolyte solution, nonsedating antihistamines, and antiemetics and/or dose escalation.
Furie et al, EULAR 2021 and Data on File.

Abbreviations: TEAE, treatment-emergent adverse event

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KZR-261, Our First Protein Secretion Candidate, is Anticipated to enter a Phase 1b Trial in Solid Tumors in 2021

COMPOUND	THERAPEUTIC INDICATION	DEVELOPMENT STAGE				MILESTONES
		PRE-CLINICAL	PHASE 1	PHASE 2	PHASE 3	
Selective Immunoproteasome Inhibition						
KZR-616	Lupus Nephritis (LN)			MISSION		Q4 2021 (interim) 1H 2022 (top-line data)
	Dermatomyositis (DM) Polymyositis (PM)			PRESIDIO		1H 2022 (top-line data)
Protein Secretion Inhibition						
KZR-261	Oncology					IND Accepted
KZR-TBD	Oncology & Autoimmunity					N/A

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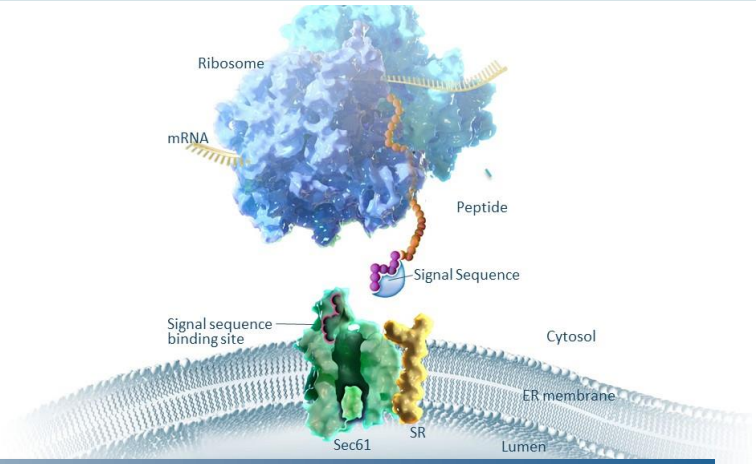
Kezar's Novel Platform for Drug Discovery Targets the Sec61 Translocon and the Protein Secretion Pathway

- Unique drug discovery engine developed with applications in multiple diseases
- Opportunity for orally bioavailable inhibitors of 1 or more high value targets with a single compound



Multi-Target → Target Selective

Multi-Protein Secretion Inhibitors

- Inhibition of **multiple** secreted/membrane proteins
- Combination therapy in a single molecule
- **Multiple oncology indications (tumor agnostic)**

KZR-261: 1st clinical candidate

Subset Protein Secretion Inhibitors

- Inhibition of relevant **subset** secreted/membrane proteins
- Non-cytotoxic agents
- **Indications: oncology, immuno-oncology, autoimmunity**

Single Protein Secretion Inhibitors

- Inhibition of a **single** secreted/membrane protein
- Non-cytotoxic agents
- **Indications: Many…**

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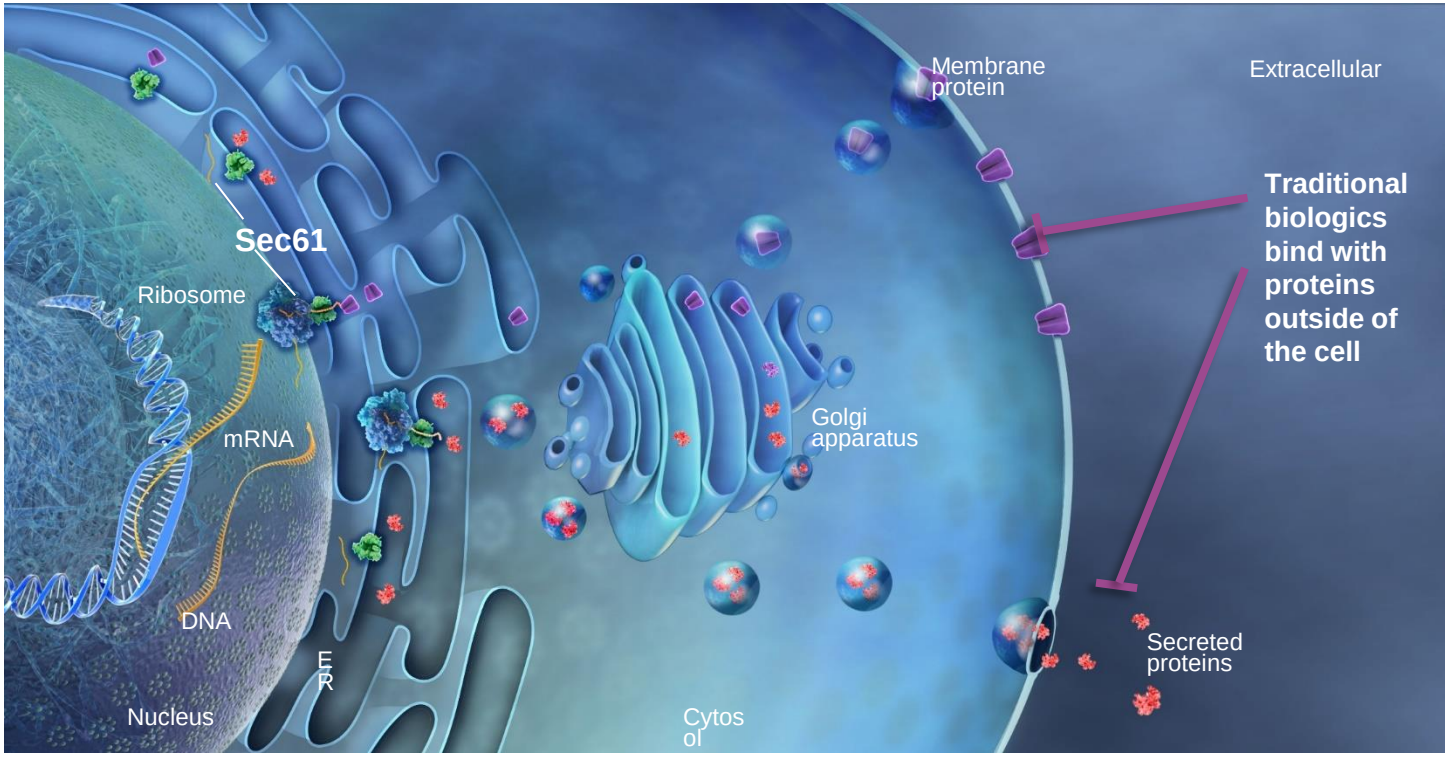
Targets of Most Biologics Utilize Sec61 for Secretion or Membrane Expression

A plethora of validated targets utilize the Sec61 translocon. Kezar's program holds the potential for superior small molecule therapeutics against these targets.

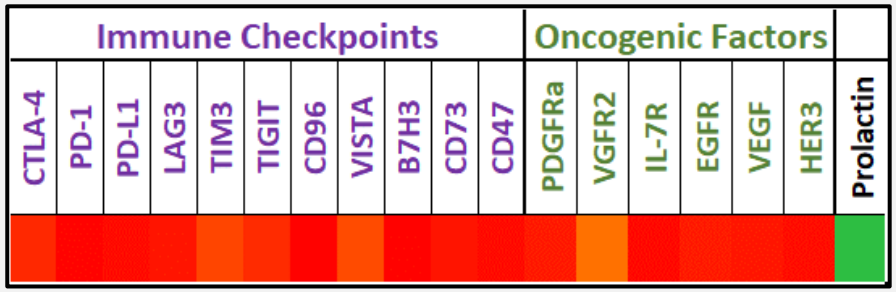


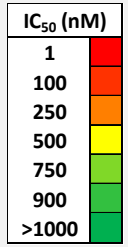
Membrane Proteins (partial list)
EGFR (ERBITUX)
IL-6R (ACTEMRA)
PD-1 (OPDIVO)
PDL1 (TECENTRIQ)
CTLA4 (YERVOY)

Secreted Proteins (partial list)
TNF-α (HUMIRA)
IL-17 (COSENTYX)
PCSK9 (REPATHA)
IL-6 (SYLVANT)
BAFF (BENLYSTA)

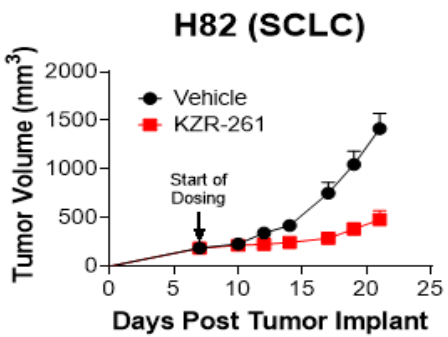
KZR-261, our First-in-Class Protein Secretion Inhibitor, Blocked Expression of Therapeutically Relevant Targets and Inhibited Tumor Growth In Vivo

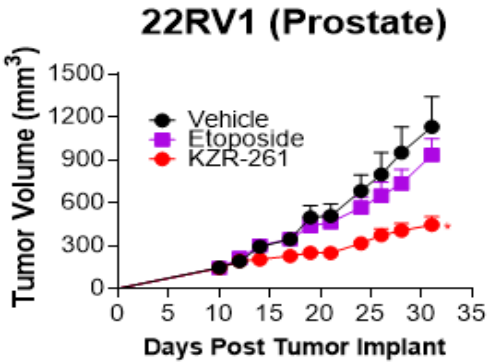
In vitro Protein Secretion Assays

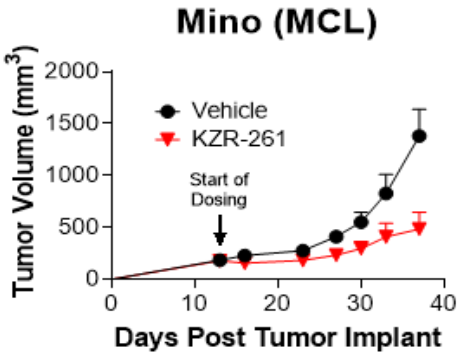




In vivo Tumor Xenografts







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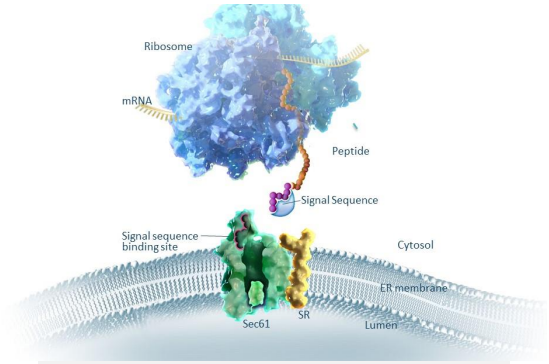
Lowe et al. ASCO 2020

Kezar has Demonstrated Proof of Concept for Discovery of Highly Selective Protein Secretion Inhibitors



Single Protein Secretion Inhibitors

- Inhibition of a **single** secreted/membrane protein
- Non-cytotoxic agents

	Target	834
Immuno-Oncology Checkpoints	CTLA-4	
	PD-1	
	PD-L1	
	LAG3	
	TIM3	
	TIGIT	
	CD96	
	VISTA	
	B7H3	
	CD73	
	CD47	

K_{ss} (nM): 1 | 100 | 250 | 500 | 1000 | 2500 | >5000

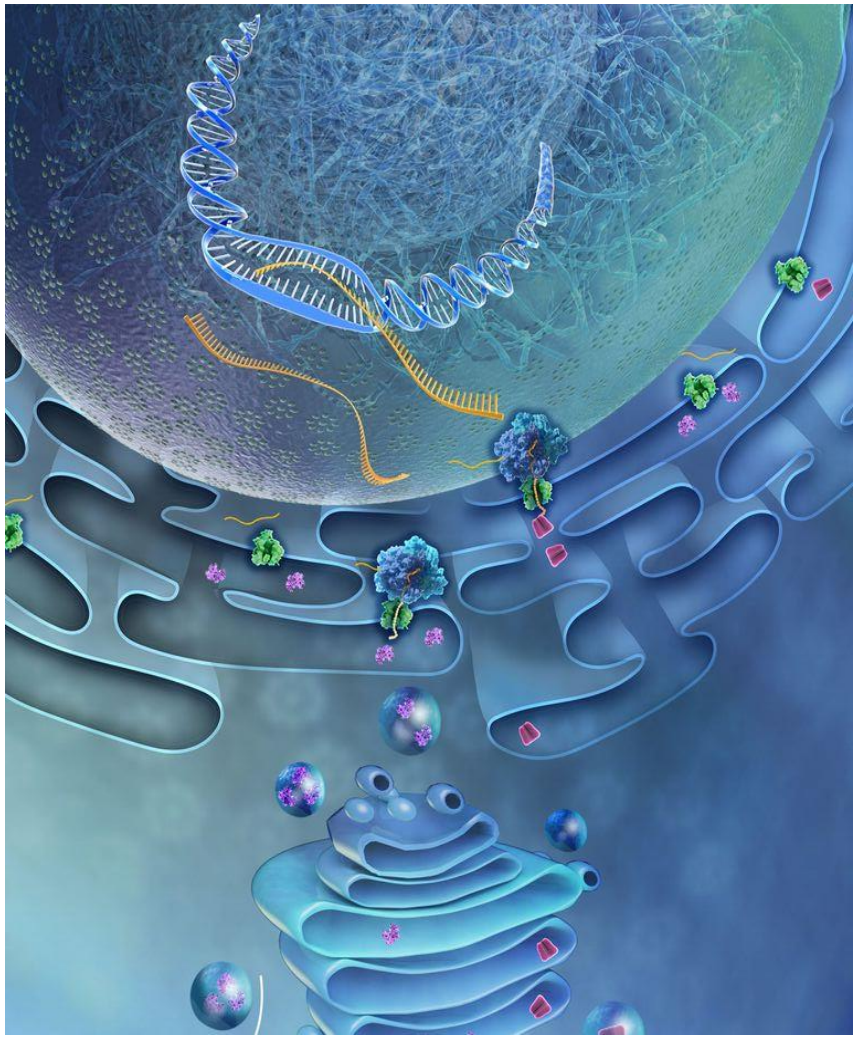
PD-1 Selective Sec61 Inhibitor Program

	9640
CTLA-4	
PD-1	
PD-L1	
LAG3	
TIM3	
TIGIT	
CD96	
VISTA	
B7H3	
CD73	
CD47	
Cytotoxicity (H929)	

KZR-261 Represents Kezar's First of Multiple Opportunities to Bring Protein Secretion Inhibitors Into Clinical Development

Combination Therapy in a Single Drug

- Sec61 is the gateway to the cell surface for multiple therapeutic targets across many indications
- Kezar has generated a unique and powerful engine for exploitation of Sec61 as a multivariate drug target
- Multi-protein targeting Sec61 inhibitors are potent, broad-based anti-cancer agents
- Multiple chemical series can be tuned to selectively target the interaction between unique protein signal sequences and Sec61
- Protein secretion inhibitors have the potential to replicate the activity of biologic therapy, provide multi-target inhibition and oral bioavailability



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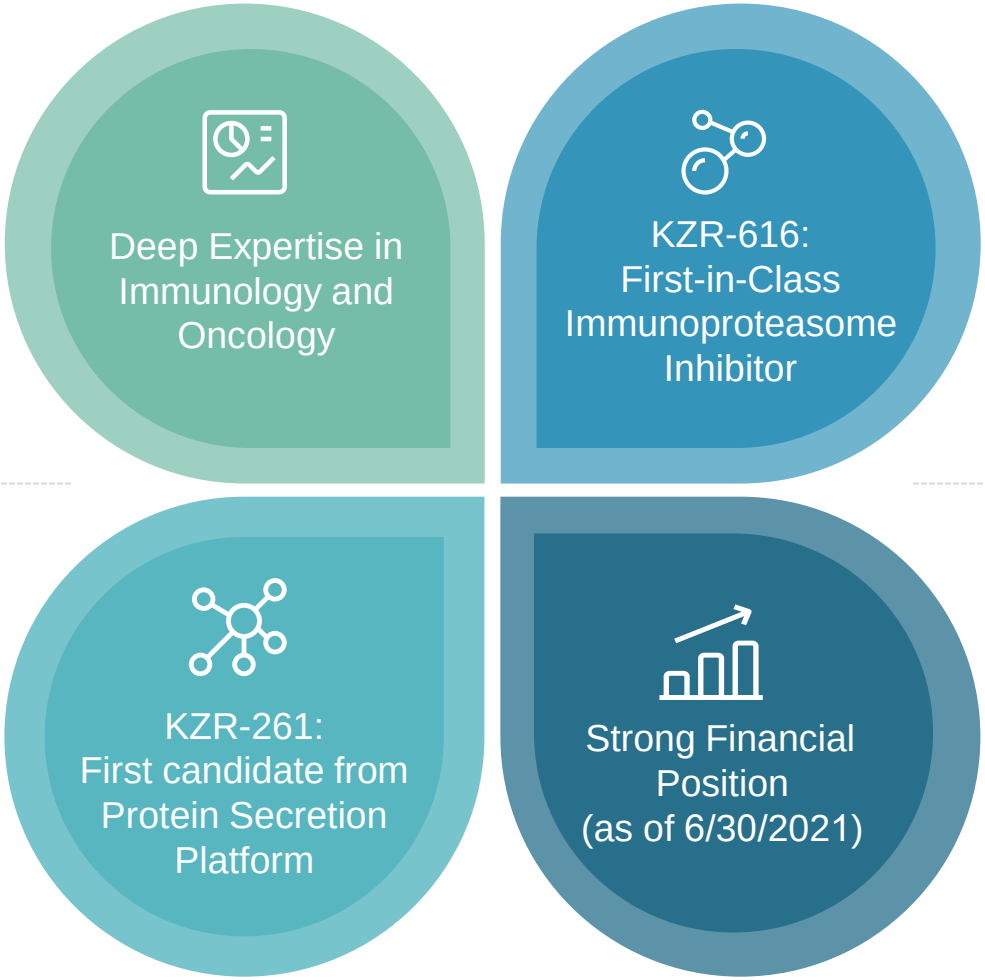
The Kezar Opportunity: Harnessing Master Regulators of Cellular Function to Tackle Immune-mediated Diseases and Cancer

Builds on 10+ years of R&D work in proteasome biology and protein secretion led by Kezar's Scientific Co-founders, Chris Kirk & Jack Taunton



A novel approach to harmonizing the immune system via immunomodulation; Potential to be a pipeline in a drug

First in class agent with broad anti-tumor activity; Potential to inhibit multiple targets with a single small molecule

$129M cash, cash equivalents, and marketable securities; 48.1M common shares outstanding

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