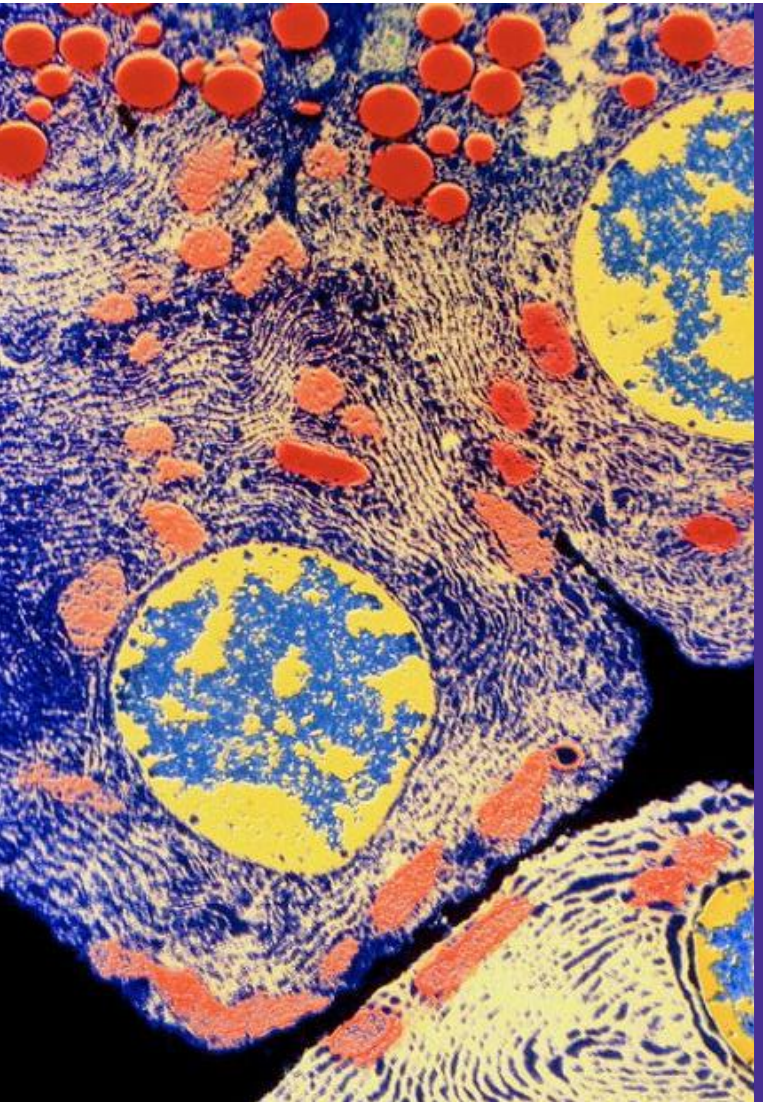
CalciMedica



Novel Therapies for Acute and Chronic Inflammatory and Immunologic Diseases

March 2026

Forward-Looking Statements

This presentation contains forward-looking statements which include, but are not limited to, statements regarding CalciMedica's business strategy and clinical development plans; the design and potential benefits of CalciMedica's product candidates; CalciMedica's ongoing and planned clinical trials; expected IP protections; the timing for CalciMedica's receipt and announcement of data from its clinical trials and other clinical milestones; the estimated patient populations and addressable market for CalciMedica's product candidates; and expectations regarding CalciMedica's cash runway. These forward-looking statements are subject to the safe harbor provisions under the Private Securities Litigation Reform Act of 1995. CalciMedica's expectations and beliefs regarding these matters may not materialize. Actual outcomes and results may differ materially from those contemplated by these forward-looking statements as a result of uncertainties, risks, and changes in circumstances, including but not limited to risks and uncertainties related to: the impact of fluctuations in global financial markets on CalciMedica's business and the actions it may take in response thereto; CalciMedica's ability to execute its plans and strategies; the ability to obtain and maintain regulatory approval for CalciMedica's product candidates; results from clinical trials may not be indicative of results that may be observed in the future; potential safety and other complications from CalciMedica's product candidates; economic, business, competitive, and/or regulatory factors affecting the business of CalciMedica generally; CalciMedica's ability to protect its intellectual property position; expected length of IP protection for CalciMedica's product candidates; the impact of government laws and regulations; and CalciMedica's cash runway and need for additional capital. Additional risks and uncertainties that could cause actual outcomes and results to differ materially from those contemplated by the forward-looking statements are included under the caption "Risk Factors" in CalciMedica's most recently filed periodic report, and subsequent periodic reports filed by CalciMedica, under the Securities Exchange Act of 1934, as amended, from time to time and available at www.sec.gov. These documents can be accessed on CalciMedica's web page at calcimedica.com.

These forward-looking statements are based on information available to, and expectations of, CalciMedica of the date of this presentation. CalciMedica disclaims any obligation to update these forward-looking statements, except as may be required by law.

CalciMedica: First-in-class CRAC-channel inhibition for acute and chronic inflammatory and immunologic diseases

Lead Program: Auxora (AP, AKI)

- First-in-class **CRAC-channel inhibitor** for acute illness
- **Positive Ph2b data** in acute pancreatitis
- Ph2 acute kidney injury data being discussed with FDA
- **Clinically meaningful effects on organ failure** and other endpoints
- **IP protection** exp. to 2041+[1]

Growth Program: CM5480 (PAH)

- Next gen. **CRAC-channel inhibitor** optimized for chronic dosing
- **Compelling preclinical efficacy in PAH model**, including on right ventricular function
- **Mechanistically distinct** from existing PAH therapies

Large Unmet Needs

- AP and AKI have no **approved therapies**; high morbidity and mortality in severe diseases
- PAH remains a **progressive, fatal disease** despite current SOC
- AP, AKI, and PAH are **>$1B potential markets**

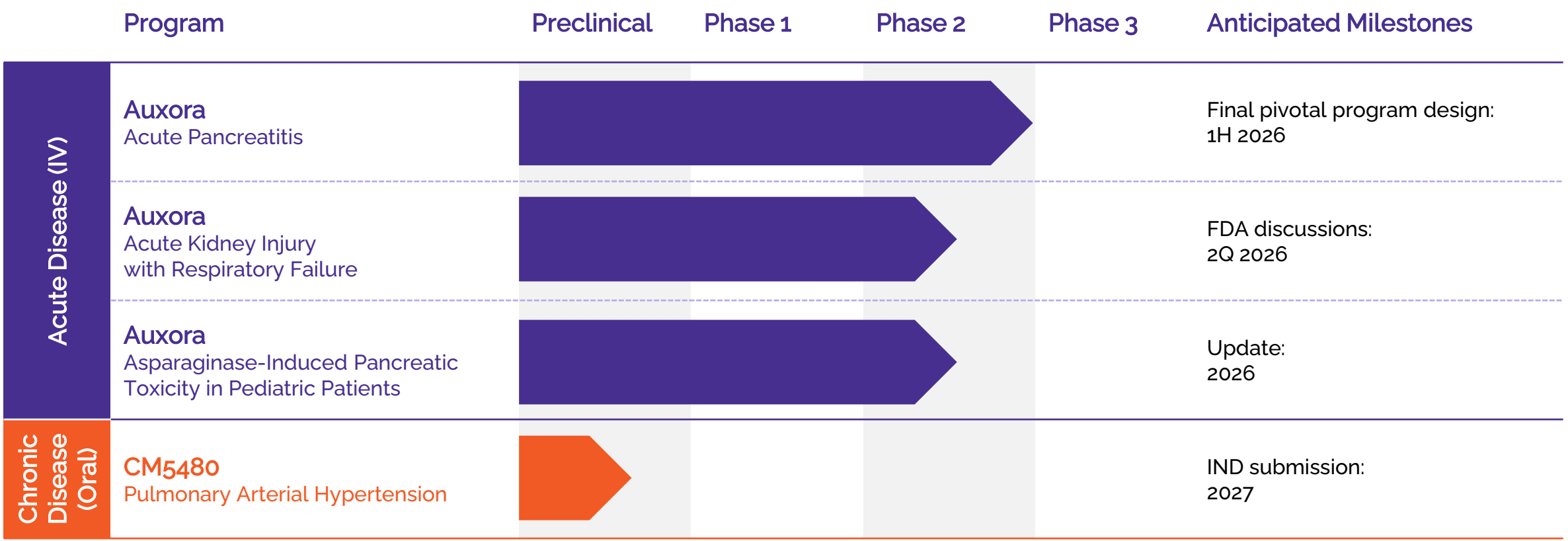
Anticipated Catalysts

- **AP:** Finalize pivotal program design – 1H 2026
- **AKI:** FDA discussions – Q2 2026
- **PAH**: IND submission – 2027
- **Cash runway** into 4Q 2026

CalciMedica

1. Composition of matter (2036), formulation (2038), and methods of use (2036-2041+) worldwide patent protection without extensions
AP: Acute Pancreatitis **AKI**: Acute Kidney Injury **PAH**: Pulmonary Arterial Hypertension

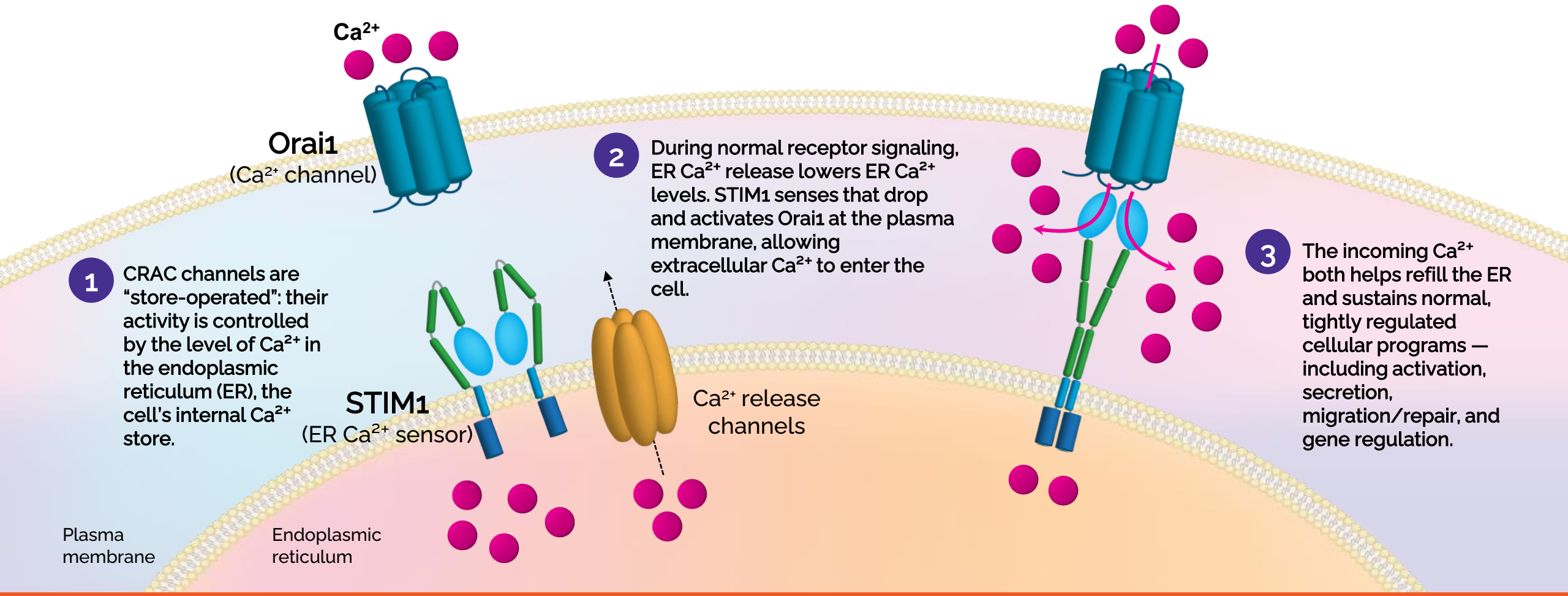
Late-stage acute programs (AP, AKI) with emerging PAH opportunity



CalciMedica

AP: Acute Pancreatitis **AKI**: Acute Kidney Injury **PAH**: Pulmonary Arterial Hypertension

CRAC channels: A store-operated calcium signaling system



In healthy cells, CRAC signaling is tightly regulated, localized, and self-limited

CalciMedica

Adapted from Roos,…Stauderman, J. Cell Bio. 2005; Zhang…Stauderman, Cahalan, Nature, 2005; Zhang…Stauderman, Cahalan, PNAS, 2006.

Disease can overdrive CRAC channel signaling, creating a persistent, pathologic calcium signal

DISEASE OVERDRIVES CRAC CHANNEL SIGNALING

Persistent receptor signaling in inflammatory settings
Repeated ER Ca^{2+} depletion can keep STIM1/Orai1 engaged

Cell stress
Oxidative and other cellular stressors can dysregulate STIM1/Orai1 signaling

Phenotypic change
Diseased cells can adopt an exaggerated SOCE-dependent state

Persistent Orai1-mediated Ca^{2+} influx generates a disease-driving calcium signal

Cells decode this signal through calcineurin/NFAT, NF-κB, mitochondrial stress pathways, and barrier/remodeling machinery

PERSISTENT CRAC CHANNEL SIGNALING RESULTS

Inflammatory activation
Drives cytokine production and immune-cell activation

Direct tissue injury
Promotes mitochondrial stress, enzyme dysregulation, and necrosis

Barrier / microvascular dysfunction
Can increase vascular leak and endothelial dysfunction

Vascular & RV remodeling
Promotes pulmonary vascular remodeling and RV dysfunction

Our approach: Selectively reduce disease-overdriven CRAC channel activity toward the physiologic range

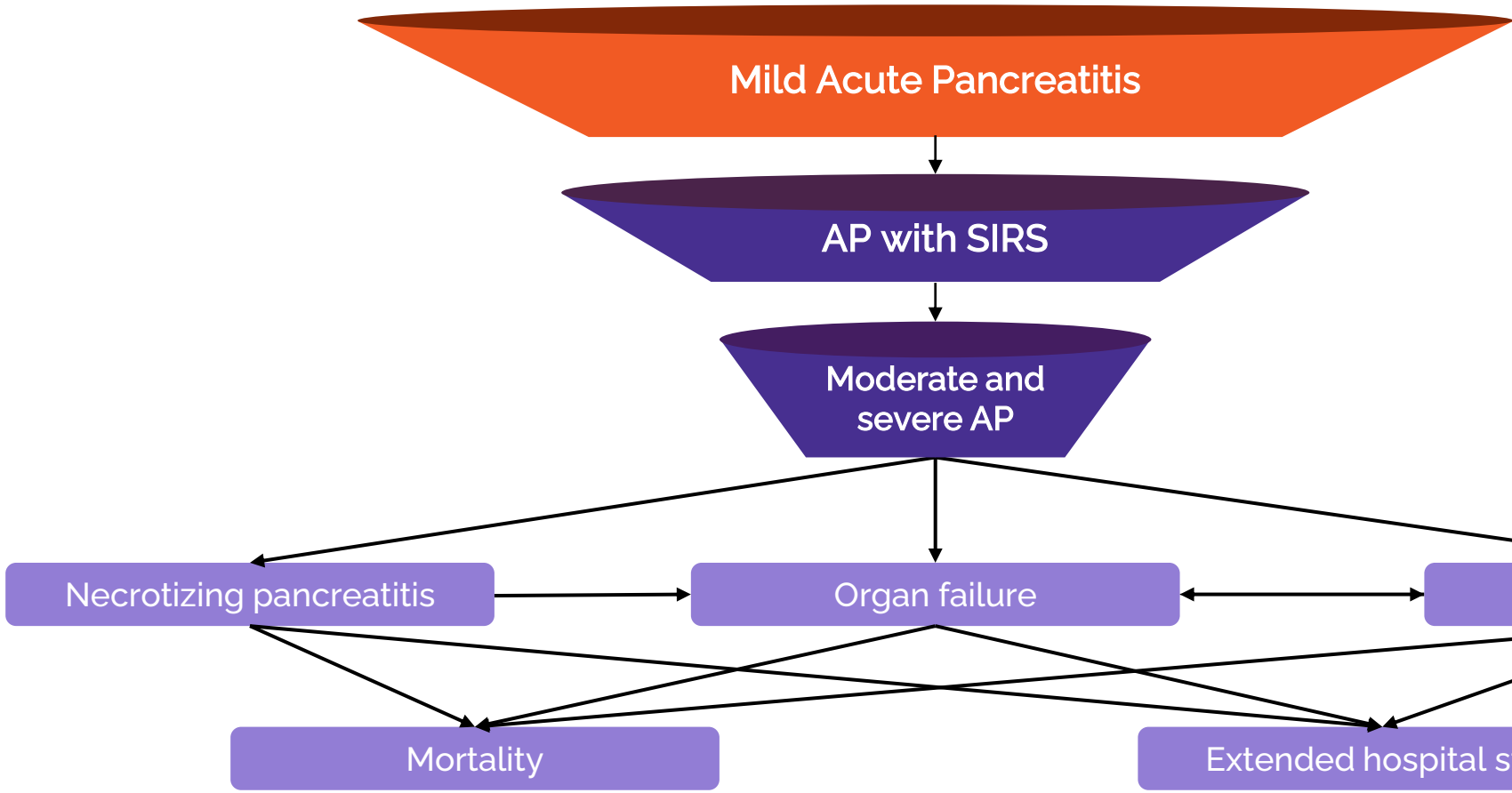
Too little CRAC channel activity	Physiologic CRAC channel activity range	Disease-overdriven CRAC channel activity
• Biallelic LoF in CRAC channels • Immunodeficiency, myopathy, ectodermal defects	• Normal, tightly regulated Ca^{2+} signaling • ER store refill + controlled cellular responses	• Persistent, pathologic Ca^{2+} signaling • Inflammation, injury, remodeling

Auxora / CM5480: Selective, partial Orai1 inhibition designed to restore CRAC channel activity toward the physiologic range

Auxora for Acute Pancreatitis (AP)

CalciMedica

Acute Pancreatitis: common, costly, and without approved treatments



US patients per year

~300K[1]
Hospitalized for AP

~170K[2,3]
Develop SIRS

~60K[4,5]
Progress in disease severity

Economic burden: >1M hospital days and ~$3B in U.S. costs annually

CalciMedica

1, Peery AF et al. Gastroenterology. 2022;162(2):621–44; 2. Machicado JD et al. United European Gastroenterol J. 2021;9(2):139–49.; 3. Singh VK et al. Clin Gastroenterol Hepatol. 2009;7(11):1247–51.; 4. Boxhoorn L et al. Lancet. 2020;396(10252):726–34; 5. Metri A et al. Surg Open Sci. 2024;19:109–17.; SIRS = Systemic Inflammatory Response Syndrome

Auxora targets key pathways driving AP and respiratory failure

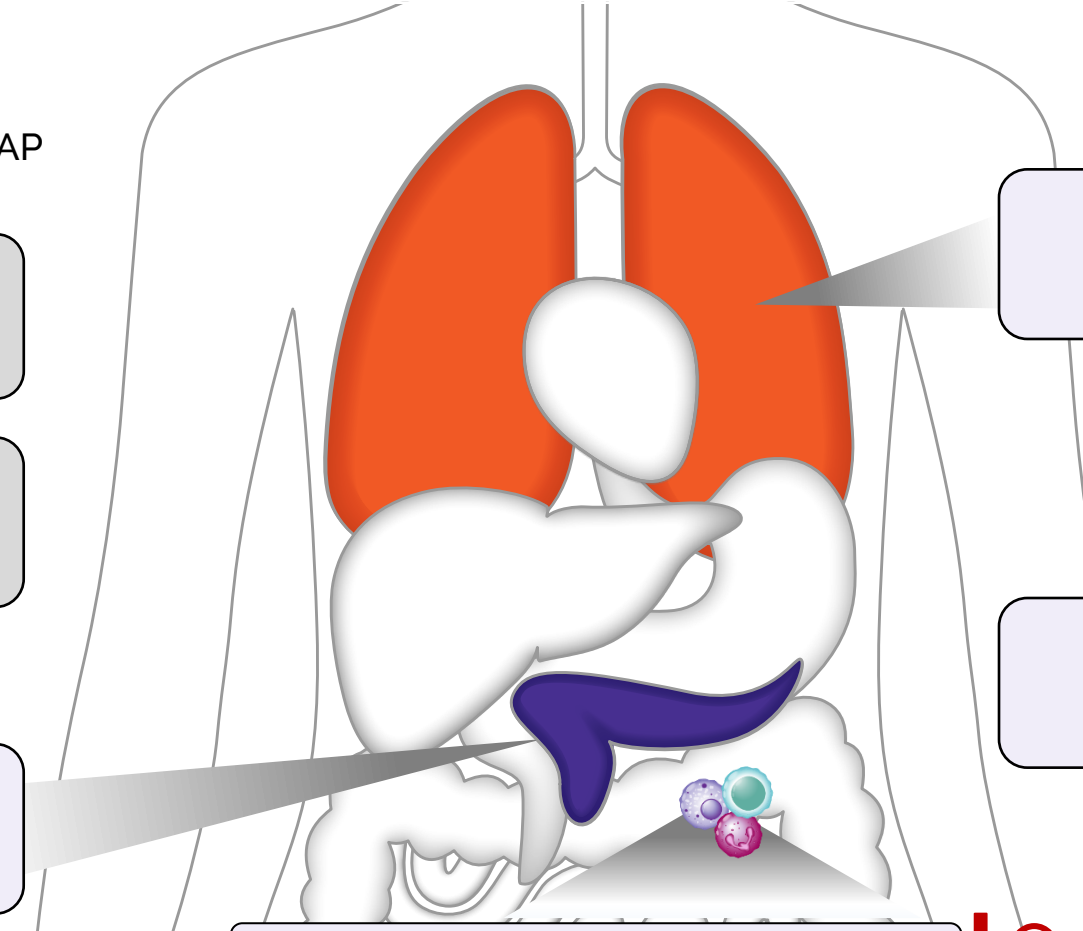
Auxora

Gallstones and excessive alcohol use are the most common causes of AP

Excessive release of Ca^{2+} in pancreatic acinar cells

Cellular Ca^{2+} overload and dysfunction due to CRAC overactivation

Pancreatic cell necrosis and dysfunction



Cascade of pro-inflammatory mediators

Systemic Inflammatory Response Syndrome (SIRS)

Acute lung injury and multiple organ failure

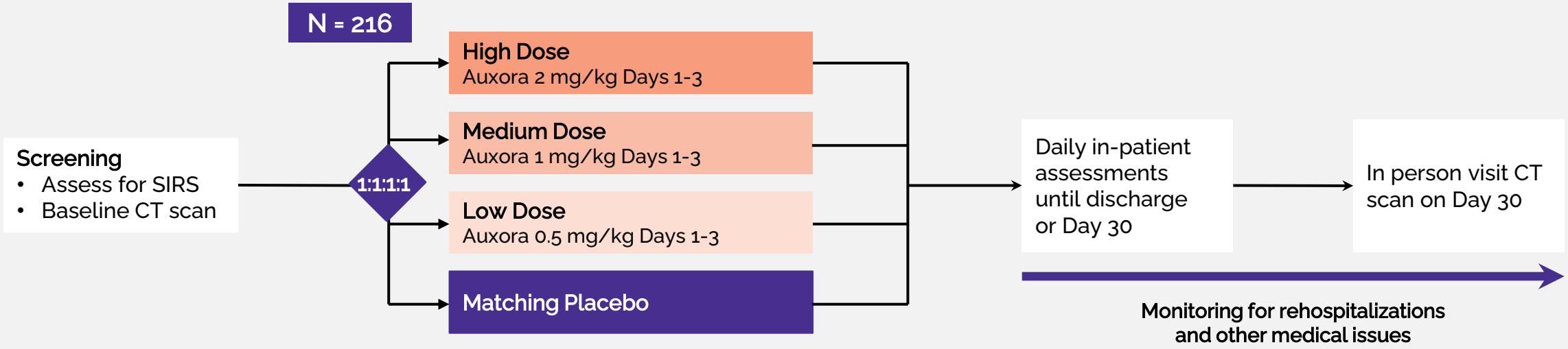
CalciMedica

1) Peng S, Ke L, Li W., ORAI1 CRAC Channel in Immune Cell is a Therapeutic Target for Pancreatitis-Associated Acute Lung Injury. 2023; 2) CalciMedica Inc. CRACing inflammatory disease. 2021.

CARPO Phase 2b clinical trial in AP with SIRS

Endpoints

- Time to solid food tolerance (primary endpoint)
- Severe organ failure
- Respiratory failure
- Length of hospital stay
- Time to medically indicated discharge
- Necrosis



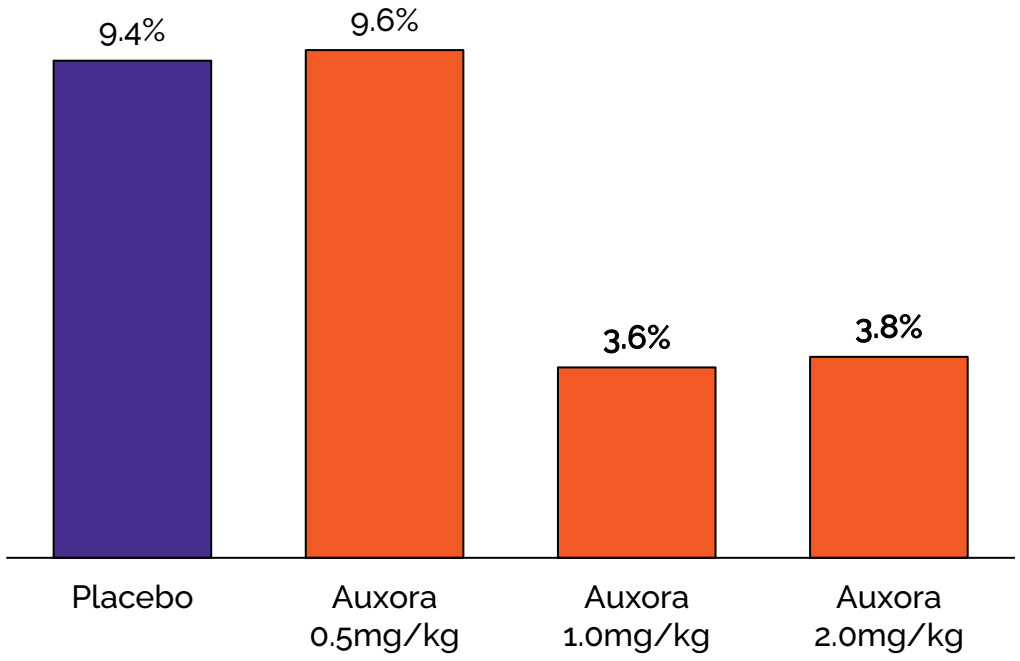
Primary Objective: Dose Response on Primary and Secondary Endpoints

CalciMedica

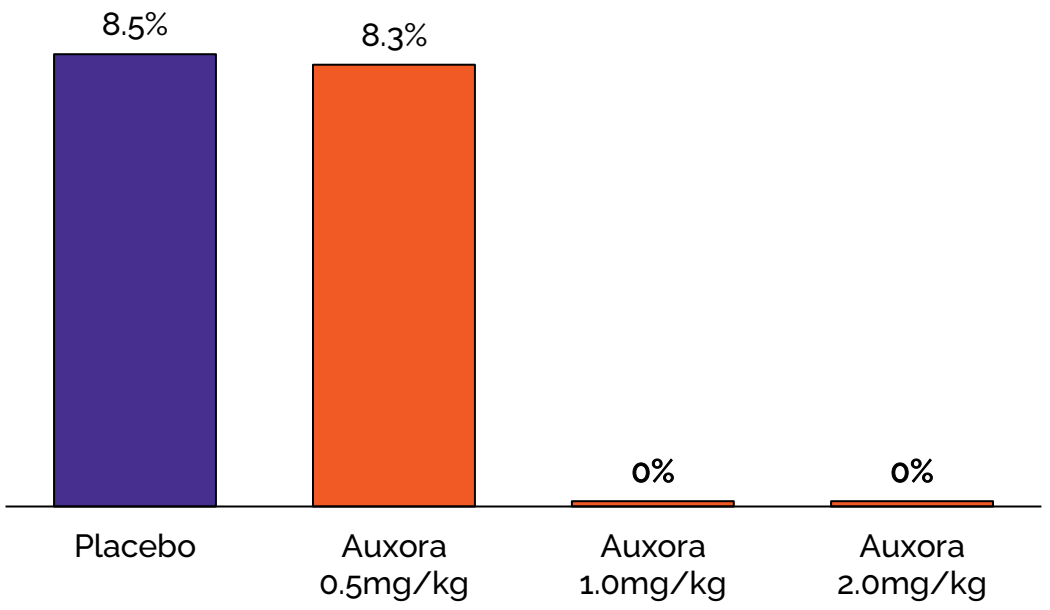
SIRS: Systemic Inflammatory Response Syndrome

Auxora reduced severe organ failure and prevented new severe respiratory failure

60% reduction in severe organ failure[1]



100% reduction in new severe respiratory failure[2]



p<0.05

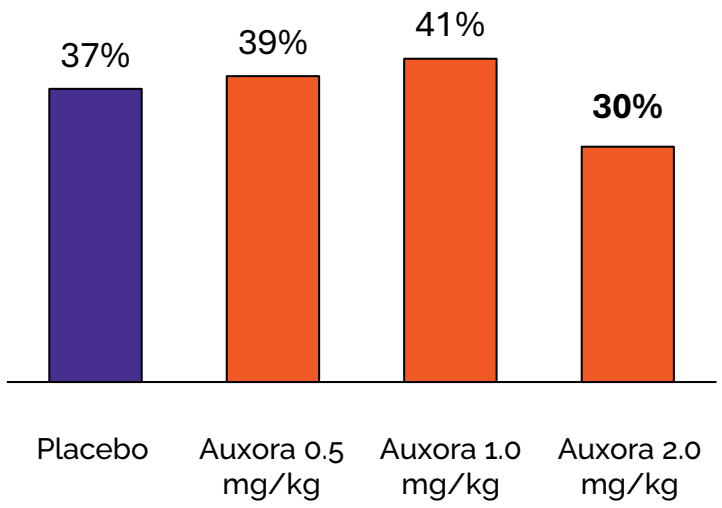
CalciMedica

1. mITT (N=214); severe organ failure includes severe respiratory failure, severe renal failure, and severe cardiovascular failure
2. mITT excluding baseline respiratory failure (N=197)

Auxora High-Dose improved key secondary endpoints

New Necrotizing Pancreatitis

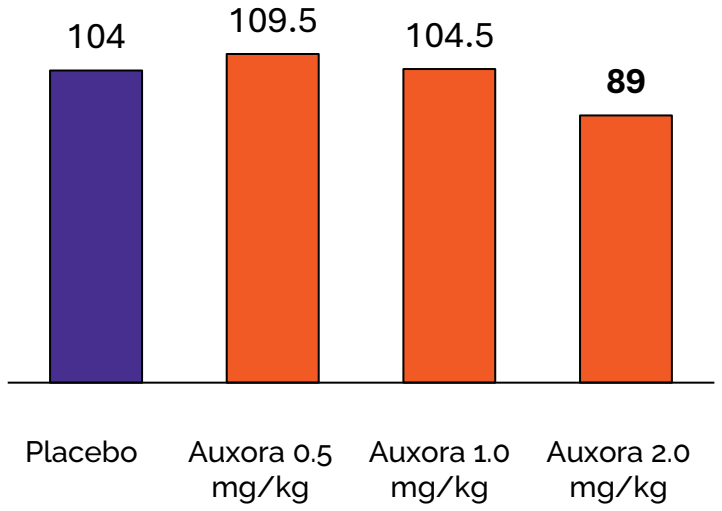
~20% relative reduction



Percentage[1] is based on the number of subjects without Necrotizing Pancreatitis at Screening and non-missing Day 30 Visit or post-treatment unscheduled visit CECT reading results

Time to Medically Indicated Discharge (median hours)

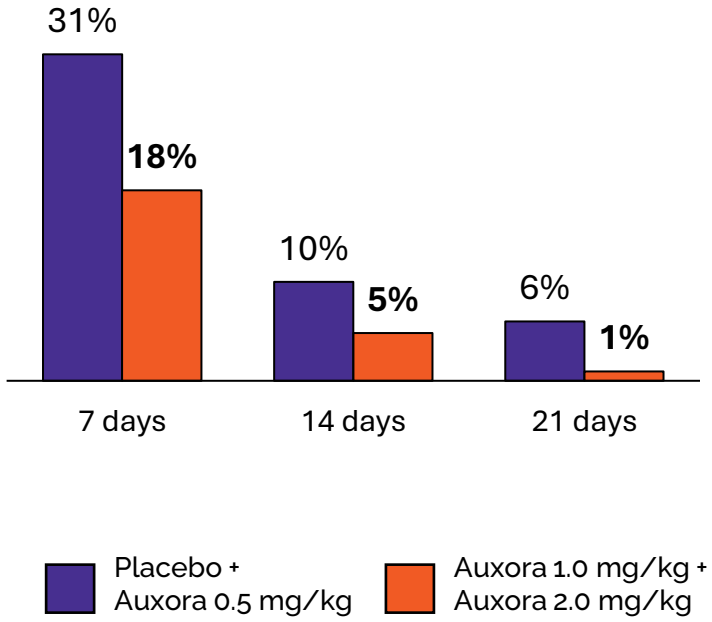
~15-hour improvement



Defined as: 1) No clinical evidence of infection necessitating continued hospitalization; 2) Solid food tolerance; 3) Abdominal pain has resolved or controlled with medications (non-opiate)

Patients Remaining Hospitalized

1% vs 6% (Day 21)



CalciMedica

Note: mITT (N=214) for all data

1. Percentages and times calculated by CMH test stratified by gender and HCT risk; LOCF used for missing data.

Win-ratio analysis shows Auxora High-Dose improved outcomes across multiple endpoints

	% of all patient-pair comparisons won (win-ratio analysis)		
Endpoint	Placebo (N = 53)	Auxora (N = 52)	Favored arm
All-cause mortality	-	-	(no events)
New severe respiratory failure	-	7.5%	✓ Auxora
New necrotizing pancreatitis	13.6%	22.3%	✓ Auxora
Time to medically indicated discharge	19.8%	26.5%	✓ Auxora
Across all hierarchical comparisons	33.4%	56.3%	✓ Auxora

Win-Ratio: 1.640 (95% CI: 1.03–2.61, p = 0.037)

CalciMedica

Win-ratio (Finkelstein–Schoenfeld): Patients are paired across treatment arms and compared hierarchically on prespecified outcomes. Each pair yields a win, loss, or tie. Percentages shown represent the proportion of all patient pairs won by each arm. CI: confidence interval
Note: Totals <100% because 10.3% of comparisons were ties

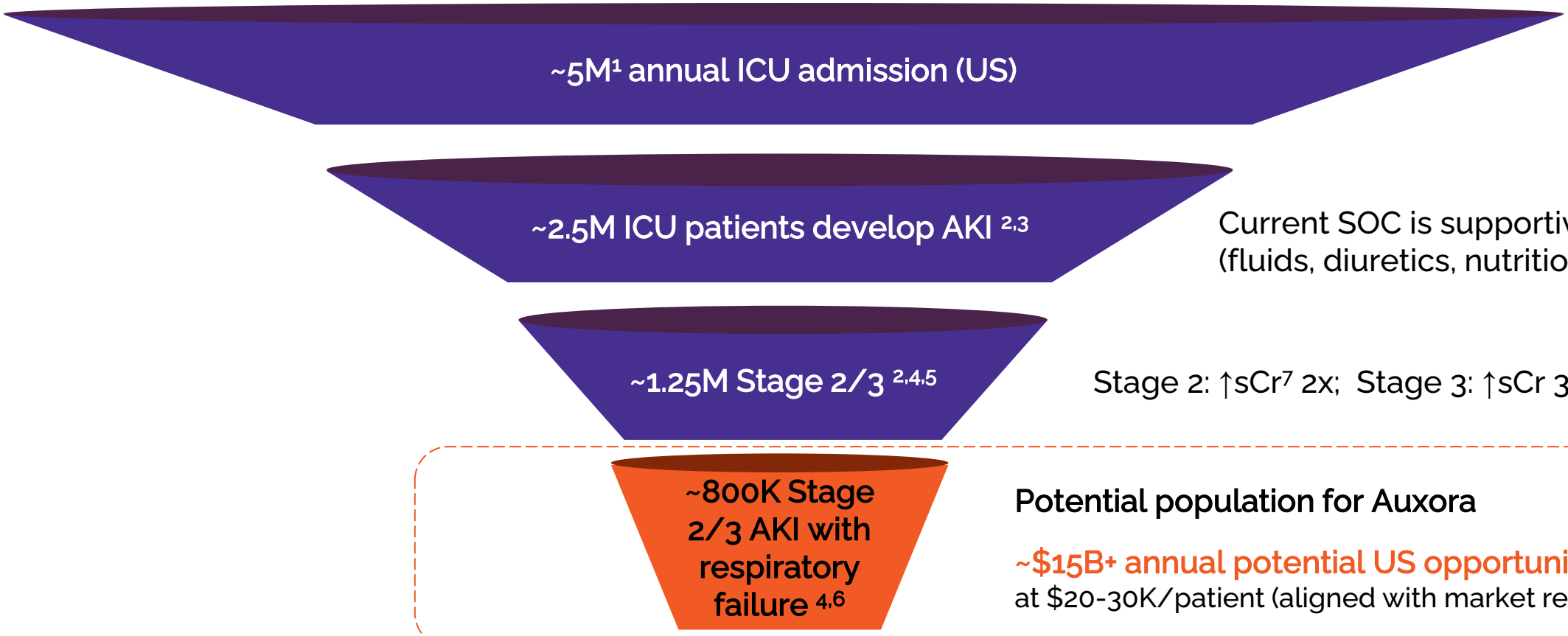
Advancing towards a potential pivotal program in AP

- Ongoing **constructive discussions with the FDA** regarding the design of a pivotal program in AP
- Leveraging AI-enabled analyses with Telperian to refine patient selection and endpoint strategy, with the goal of an **enriched, efficient study design**
- Final pivotal program design expected in **1H 2026**

Auxora for Acute Kidney Injury (AKI) with Respiratory Failure

CalciMedica

AKI with respiratory failure: large patient population, high mortality, no approved therapies



Current SOC is supportive therapy (fluids, diuretics, nutrition)

Stage 2: ↑sCr[7] 2x; Stage 3: ↑sCr 3x

Potential population for Auxora

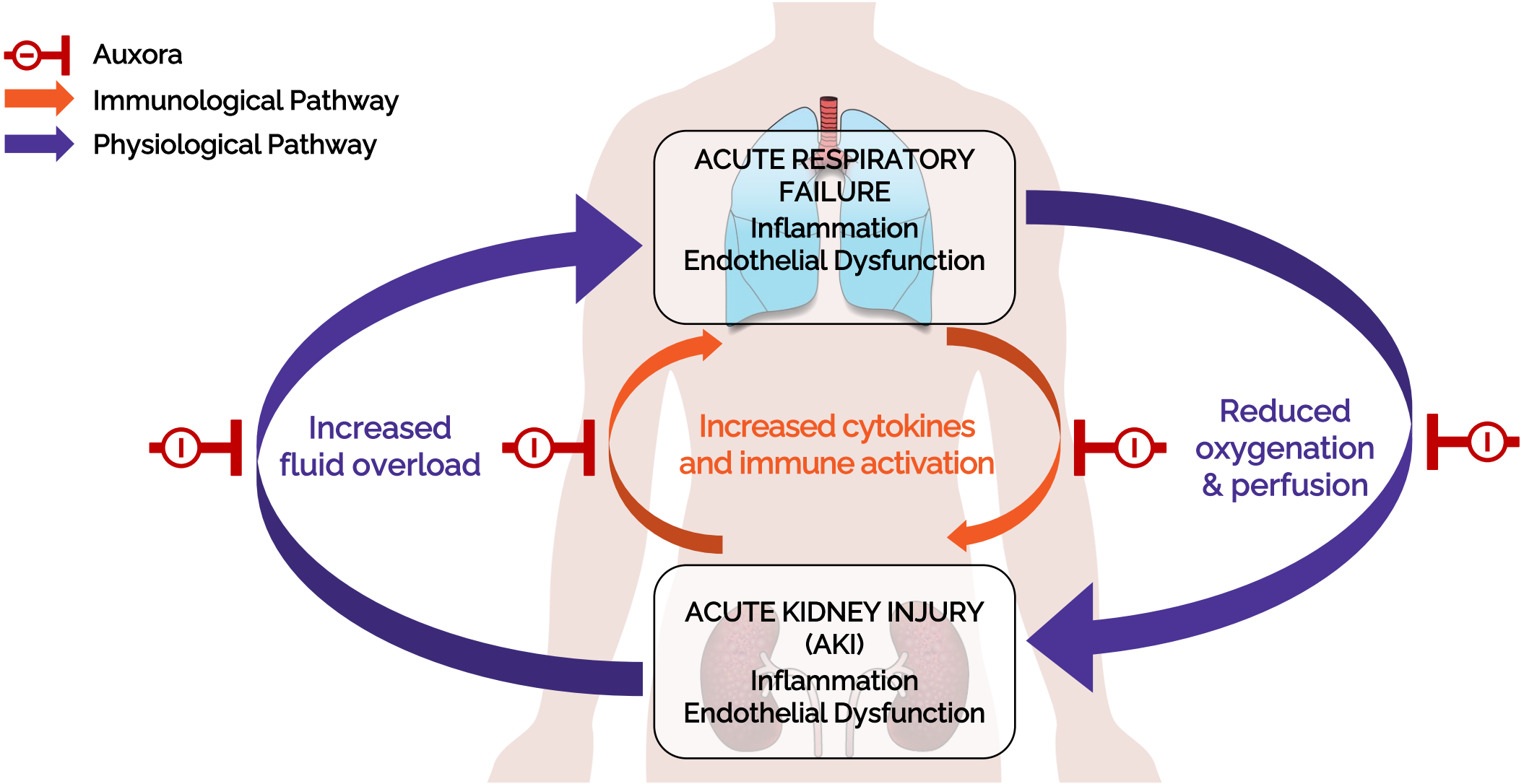
~$15B+ annual potential US opportunity
at $20-30K/patient (aligned with market research)[8]

No approved therapies; 90-Day mortality: 40-50%+[9,10,11]

CalciMedica

1. Critical Care Statistics; 2. Assante W et al. Proc (Bayl Univ Med Cent). 2025;38(3):266–71.; 3. Hoste EA et al. Intensive Care Med. 2015;41(8):1411–23.; 4. Vemuri SV et al. Crit Care Explor. 2022;4(7):e0720.; 5. McNicholas BA et al. Crit Care. 2023;27(1):3.;6. STARRT-AKI Investigators. N Engl J Med. 2020;383(3):240–51; 7. sCr: Serum Creatinine; 8. Note: market opportunity and pricing estimates are based on preliminary and high-level assumptions; 9. McNicholas BA et al. Crit Care. 2023;27(1):3.; 10. Panitchote A et al. Ann Intensive Care. 2019;9(1):74.; 11. Demirjian S et al. Clin J Am Soc Nephrol. 2011;6(9):2114–20.

Auxora targets key pathways driving AKI with respiratory failure

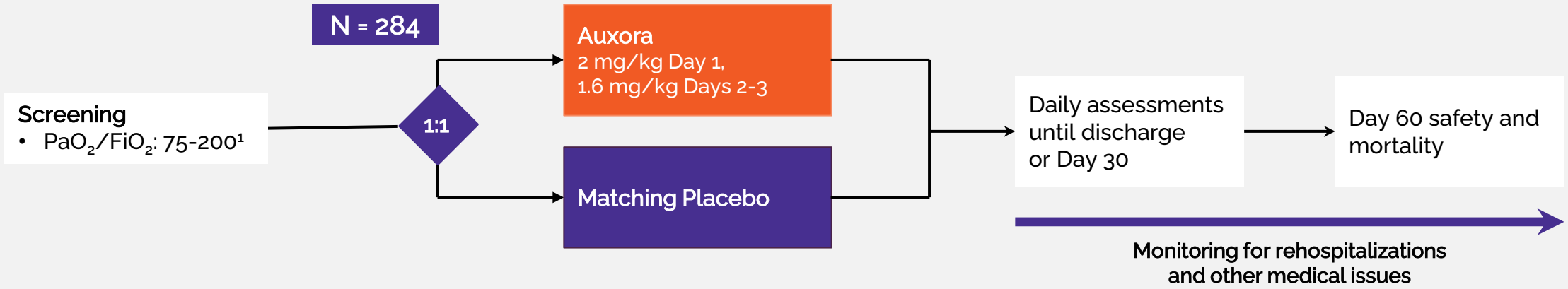


References: Faubel S, et al. Mechanisms and Mediators of Lung Injury After Acute Kidney Injury. Nat Rev Nephrol. 2016;12(1):48-60. doi: 10.1038/nrneph.2015.158.2); Alge J, et al. Two to Tango: Kidney-Lung Interaction in Acute Kidney Injury and Acute Respiratory Distress Syndrome. Front Pediatr. 2021;9:744110. doi: 10.3389/fped.2021.744110; Hou P, et al. Reduction in D-dimer Levels After Treatment with Auxora in Patients with Severe Covid-19 Pneumonia Reflects Endothelial Stabilization, 18 September 2023,

CARDEA Phase 2 trial in severe COVID-19 pneumonia evaluated Auxora in a population relevant to our AKI program

Endpoints

- Days to Recovery
- Mortality
- Ventilation use
- Days in the hospital



Relevance to AKI with Respiratory Failure

- Severe, hospitalized patients with **moderate-to-severe respiratory failure**
- **38 patients with AKI** at enrollment
- **190 patients** with daily biomarker sampling over 4 days

CalciMedica

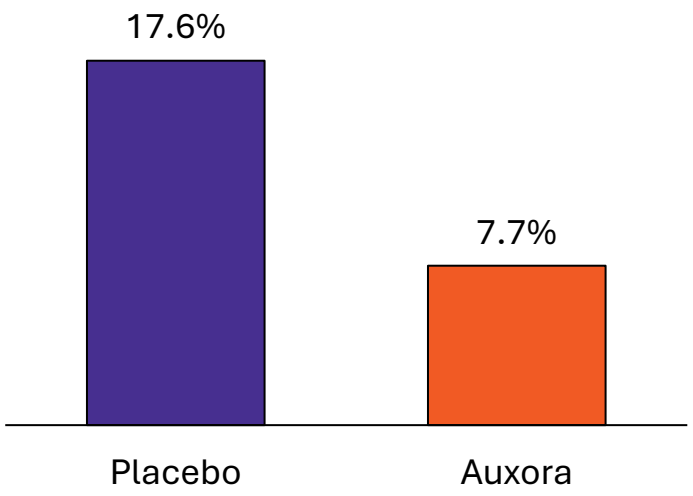
1. After 23 patients with PaO2/FiO2 >200 were enrolled

CARDEA results support Auxora's potential to improve outcomes in patients with respiratory and kidney impairment

CARDEA: Ph2 in Severe and Critical COVID-19 Pneumonia Patients

56% ↓ mortality (total)

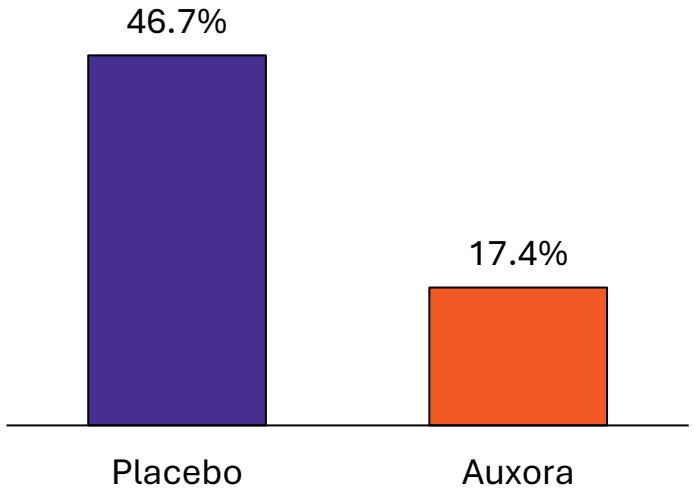
30-Day Mortality



Efficacy set (N=261); **p = 0.017**

63% ↓ mortality (AKI subgroup)

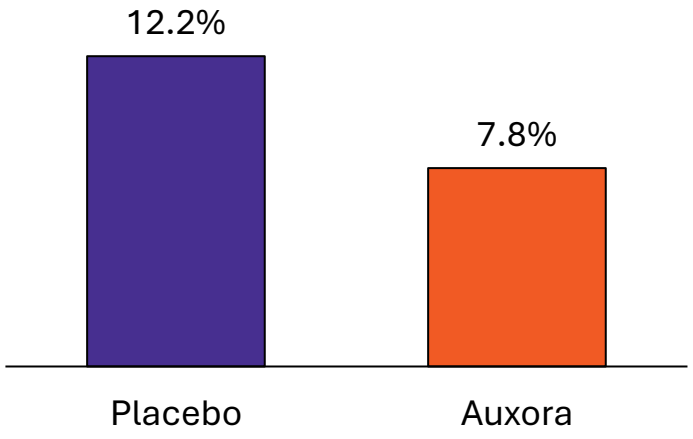
30-Day Mortality



AKI subgroup (N=38; eGFR ≤60)

40% ↓ new AKI (total)

Newly reported events of AKI



Safety set (N=281)

CalciMedica

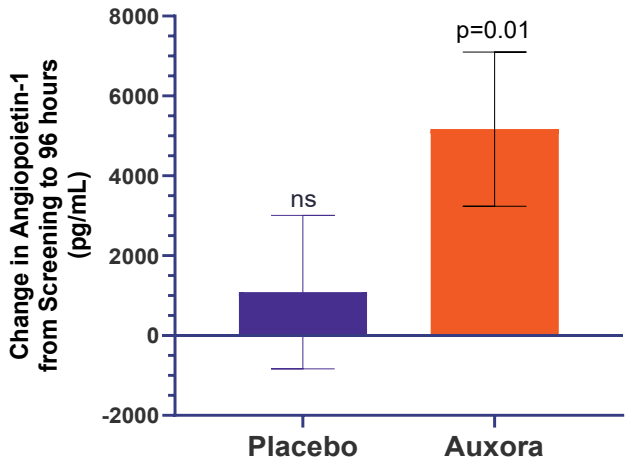
Publication: Bruen et al., Crit Care 2022.

In CARDEA, Auxora improved key biomarkers linked to vascular integrity and organ protection

72-Hour Changes in Cardiorenal Biomarkers from Ph2 in Severe and Critical COVID-19 Pneumonia Patients

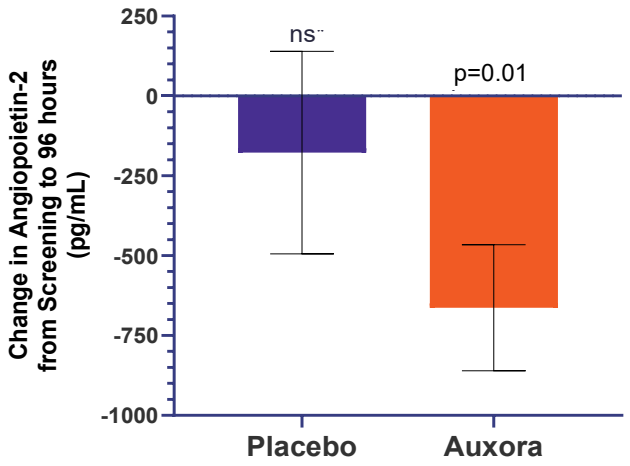
↑ Ang-1 = vascular stability and less leakage

Angiopoietin-1 Levels Increase Significantly with Auxora
(Means ± SEM)



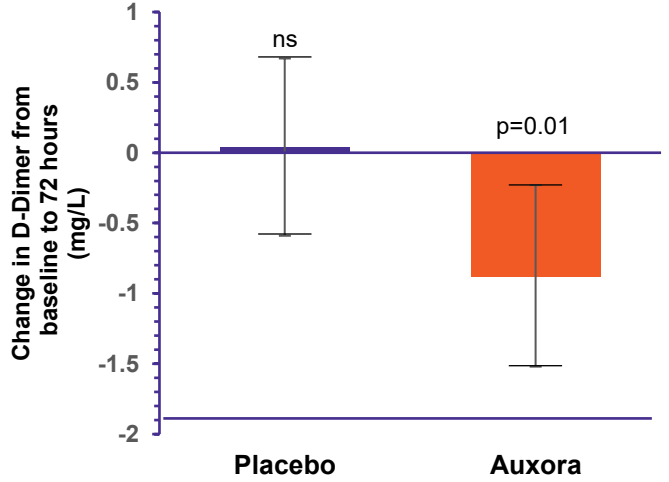
↓ Ang-2 = less inflammation and permeability

Angiopoietin-2 Levels Decrease Significantly with Auxora
(Means ± SEM)



↓ D-Dimer = less clotting, better microvasculature

D-Dimer Levels Decrease Significantly with Auxora
(Means ± SEM)



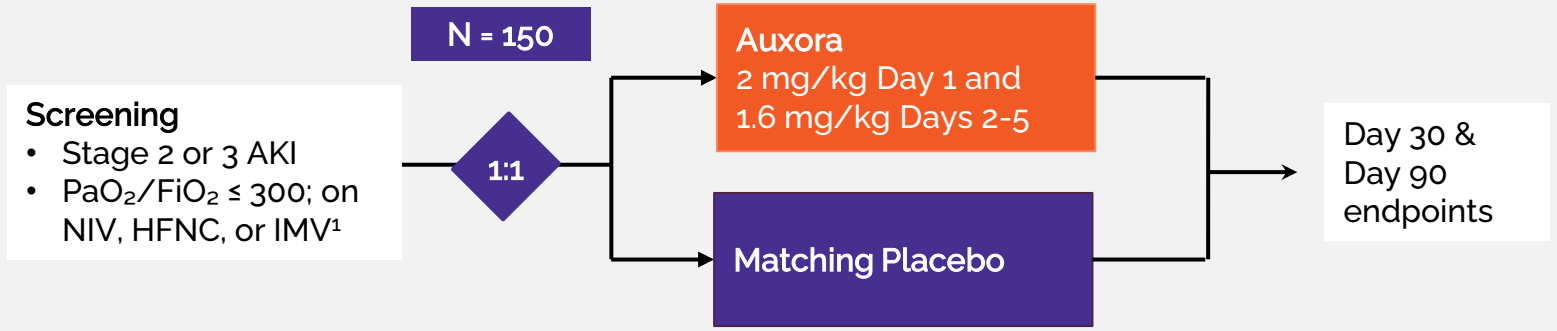
Between-group difference: p=0.046

Biomarker improvements associated with better outcomes in CARDEA

CalciMedica

D-dimer: N=261; Ang-1 / Ang-2: N=191 (subset consenting to blood draws)
Reference: Hou, et al. (2023). Reduction in D-dimer Levels After Treatment with Auxora in Patients with Severe Covid-19 Pneumonia Reflects Endothelial Stabilization. 10.21203/rs.3.rs-3349602/v2.

KOURAGE Phase 2 trial design (discontinued following IDMC review)



Primary Endpoint

- Hierarchical composite: Days-alive free from ventilation and dialysis (win-ratio analysis)

Secondary Endpoints

- Mortality
- Ventilator use
- Dialysis use
- Kidney outcomes at Day 90

March 2026 update:

Study Discontinuation

The trial was discontinued in January 2026 following a recommendation from the trial's IDMC regarding safety concern relating to a mortality imbalance that warranted reevaluation of the study design.

Safety Review

The IDMC did not identify evidence of drug-related toxicity.
The Company's comprehensive review, performed in conjunction with external experts, reached the same conclusion.
Imbalances severity of disease at baseline may have contributed to the observed safety concern.

Next step

The Company plans to discuss the KOURAGE data and potential future development in AKI with the FDA in 2Q 2026.

CalciMedica

1. non-invasive mechanical ventilation, high-flow nasal cannula, or invasive mechanical ventilation
IDMC: Independent Data Monitoring Committee

CM5480 for Pulmonary Arterial Hypertension

CalciMedica

Orai1 / CRAC channel biology is linked to pulmonary vascular remodeling and cardiac dysfunction

Orai1 contributes to pulmonary vascular remodeling and RV dysfunction in PAH

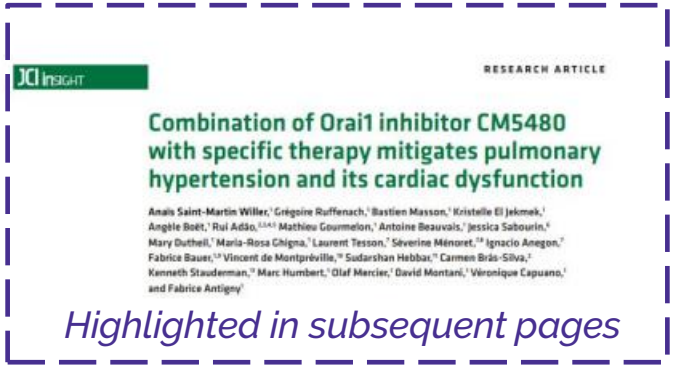
Circulation Research

ORIGINAL RESEARCH

Orai1 Inhibitors as Potential Treatments for Pulmonary Arterial Hypertension

In This Issue, see p 725
Meet the First Author, see p 726
Editorial, see p 728

Bastien Masson, Hélène Le Ribeuz, Jessica Sabourin, Loann Laubry, Emily Woodhouse, Richard Foster, Yann Ruchon, Mary Dutheil, Angèle Boët, Maria-Rosa Ghigna, Vincent Thomas De Montpreville, Olaf Mercier, David J. Beech, Jean-Pierre Benitah, Marc A. Bailey, Marc Humbert, David Montani, Véronique Capuano, and Fabrice Antigny



JCI insight

RESEARCH ARTICLE

Combination of Orai1 inhibitor CM5480 with specific therapy mitigates pulmonary hypertension and its cardiac dysfunction

Anais Saint-Martin Willer, Grégoire Ruffenach, Bastien Masson, Kristelle El Jekmek, Angèle Boët, Rui Adão, Mathieu Gourmelon, Antoine Beauvais, Jessica Sabourin, Mary Dutheil, Maria-Rosa Ghigna, Laurent Tesson, Séverine Ménoret, Ignacio Anegon, Fabrice Bauer, Vincent de Montpréville, Sudarshan Hebbar, Carmen Brás-Silva, Kenneth Stauderman, Marc Humbert, Olaf Mercier, David Montani, Véronique Capuano, and Fabrice Antigny

Highlighted in subsequent pages

Enhanced store-operated calcium entry (SOCE) in RV cardiomyocytes in a porcine model of RV dysfunction

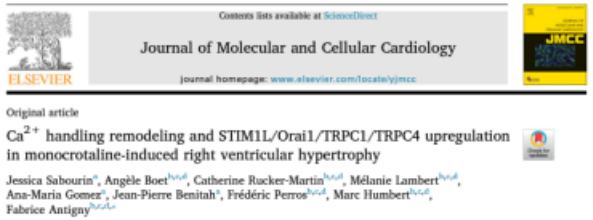
Circulation: Heart Failure

ORIGINAL ARTICLE

Ca^{2+} Cycling Alteration in a Porcine Model of Right Ventricular Dysfunction

Fabrice Antigny, PhD; Rui Luo, PhD; Romain Perrier, PhD; Bastien Masson, PhD; Guillaume Fadel, MD; Grégoire Ruffenach, PhD; Anaïs Saint-Martin Willer, PhD; Ali Akamkam, MD; Julien Grynblat, MD, PhD; Xavier Jais, MD; Jérôme Le Pavec, MD, PhD; Simon Dang Van, MD; Dorothée Brunet, MD; Florence Lefebvre, MSc; Garance Gérard, PhD; Séverine Domenichini, PhD; Angèle Boët, MD, PhD; Julien Guihaire, MD, PhD; Ana-Maria Gomez, PhD; David Montani, MD, PhD; Jean-Pierre Benitah, PhD; Marc Humbert, MD, PhD; Olaf Mercier, MD, PhD; Jessica Sabourin, PhD

Upregulation of Orai1 in RV cardiomyocytes in PAH model



Contents lists available at ScienceDirect

Journal of Molecular and Cellular Cardiology

journal homepage: www.elsevier.com/locate/yjmcc

ELSEVIER

JMCC

Original article

Ca^{2+} handling remodeling and STIM1L/Orai1/TRPC1/TRPC4 upregulation in monocrotaline-induced right ventricular hypertrophy

Jessica Sabourin, Angèle Boet, Catherine Rucker-Martin, Mélanie Lambert, Ana-Maria Gomez, Jean-Pierre Benitah, Frédéric Perros, Marc Humbert, Fabrice Antigny

Orai1 contributes to LV dysfunction

Circulation

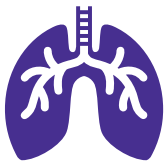
ORIGINAL RESEARCH ARTICLE

Orai1 Channel Inhibition Preserves Left Ventricular Systolic Function and Normal Ca^{2+} Handling After Pressure Overload

Fiona Bartoli, PhD, Marc A. Bailey, PhD, MBChB, MRCS, Baptiste Rode, PhD, Philippe Mateo, PhD, Fabrice Antigny, PhD, Kaveen Bedouet, BS, Pascale Gerbaud, MSc, Rajendra Gosain, PhD, Jeffrey Plante, PhD, Katherine Norman, MChem, Susana Gomez, MSc, Florence Lefebvre, MSc, Catherine Rucker-Martin, PhD, Justin F.X. Ainscough, PhD, Mark T. Kearney, MD, MBChB, Alexander-Francisco Bruns, PhD, Jian Shi, PhD, Hollie L. Appleby, PhD, Richard S. Young, PhD, MBChB, Heba M. Shawer, MRes, Marjolaine Debant, PhD, Ana-Maria Gomez, PhD, David J. Beech, PhD, Richard Foster, PhD, Jean-Pierre Benitah, PhD, and Jessica Sabourin, PhD

CM5480 reverses PAH-mediated genetic changes in both the lung and right ventricle of the heart

Transcriptomic profiling of CM5480 Treatment in the MCT Rat Model of PAH



305 dysregulated genes in the lung were restored by CM5480 in MCT-treated rats

- Pathways restored include metabolism and inflammation/immune response

2,358 dysregulated genes in the right ventricle were restored by CM5480 in MCT-treated rats

- Restoration of gene expression was most striking in the RV
- Pathways restored include inflammation and heart contraction

CM5480 is potentially disease-modifying and restores RV function in PAH

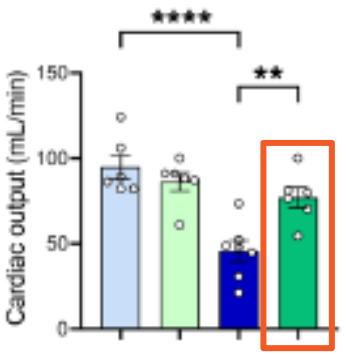
Publication: Saint-Martin Willer et al., *JCI (Journal of Clinical Investigation) Insight* 2025

CM5480 directly improves cardiac output and RV remodeling in preclinical PAH model as monotherapy and in combo with SOC

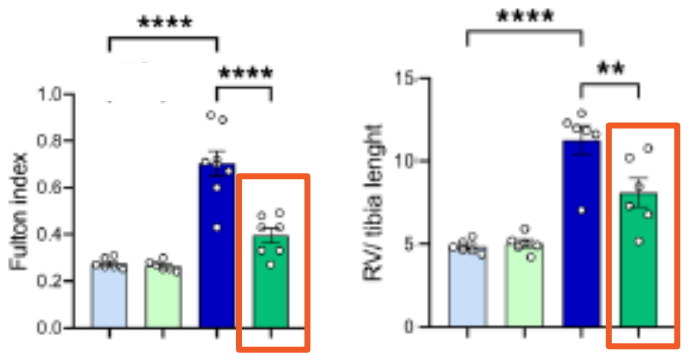
CM5480, a proprietary preclinical-stage CRAC channel inhibitor, was evaluated in a monocrotaline (MCT) rat model of established pulmonary arterial hypertension (PAH). The study was conducted at Inserm in the laboratory of Marc Humbert, M.D., Ph.D., a leading investigator in PAH.

CM5480 as Monotherapy

Improved Cardiac Output

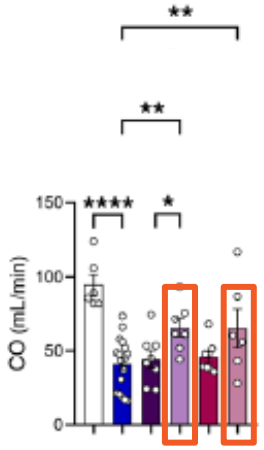


Reduced Right Ventricular (RV) Remodeling

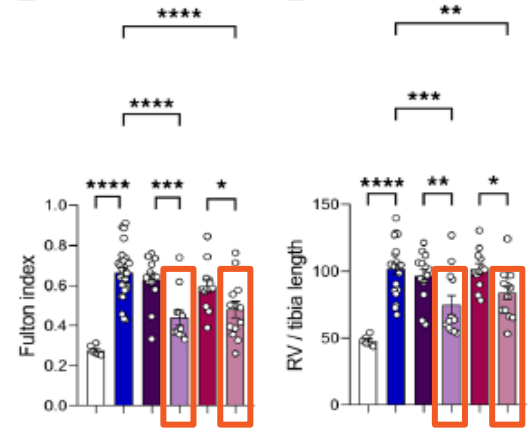


CM5480 in Combination with SOC

Improved Cardiac Output



Reduced Right Ventricular (RV) Remodeling[1]



CalciMedica

1. Fulton index and RV/tibia length measure RV hypertrophy; Fulton index = weight ratio of RV to the sum of the LV + septum
Publication: Saint-Martin Willer et al., *JCI (Journal of Clinical Investigation) Insight* 2025

CM5480 addresses both RV function and pulmonary vascular disease

Study Findings	Monotherapy (Sildenafil or Ambrisentan Alone)	CM5480 Alone	Combination Therapy (CM5480 + Sildenafil or Ambrisentan)
Reduced Elevated Right Ventricle Systolic Pressure (**RVSP**)	✓	✓	✓✓
Reduced Pulmonary Vessel Remodeling	✓[1]	✓	✓✓
Reduced Pulmonary Vascular Resistance (**PVR**)	✓	✓	✓✓
Improved Cardiac Output (**CO**)	✗	✓	✓
Reduced Right Ventricle (**RV**) **Remodeling**	✗	✓	✓

CalciMedica

1. Effect observed with Sildenafil only
Publication: Saint-Martin Willer et al., *JCI (Journal of Clinical Investigation) Insight* 2025

CRAC channel pathway does not interfere with and is not modulated by pathways targeted by existing PAH therapies

Effect of Orai1 Knockdown on mRNA Expression of Targeted Pathways in Human PAH-PASMCs

mRNA	Effect of Orai1 Knockdown
Endothelin Receptor Type A	**No change**
Endothelin Receptor Type B	**No change**
Phosphodiesterase 5 (PDE5)	**No change**
BMPR2	**No change**
BMPR1A	**No change**
TGFBR2	**No change**
TGBR3	**No change**
TGFBR1	**Decreased**

Effect of PAH Drugs on Orai1 Protein Expression in Human PAH-PASMCs

Drug Class	Drug	Orai1 Protein Expression
PDE5 inhibitor	Sildenafil	**Increased**
Endothelin inhibitor	Ambrisentan	**No effect**
Monoclonal antibody inhibitor of activin type II receptors	Bimagrumab	**No effect**
Tyrosine kinase inhibitor	Imatinib	**No effect**

CalciMedica

PASMC = pulmonary artery smooth muscle cell
Publication: Saint-Martin Willer et al., *JCI (Journal of Clinical Investigation) Insight* 2025

Anticipated Milestones

Anticipated milestones

AP	Final pivotal program design expected in 1H 2026
AKI	Discuss the KOURAGE data and potential future development in AKI with the FDA expected in 2Q 2026
PAH	IND submission expected in 2027
Cash Runway	Cash expected to fund current operations into 4Q 2026

Appendix

Large ICU/Hospital market opportunity in life-threatening conditions

Condition	US Prevalence
Acute Pancreatitis	~300K[1]
Acute Kidney Injury	~2.5M[2]
Acute Respiratory Distress Syndrome	~190K[3]
Traumatic Brain Injury	~215K[4]
Acute Ulcerative Colitis	~60K[5]

Initial Focus

Potential Expansions

Millions of ICU admissions annually in US for organ or respiratory failure – hundreds of thousands of deaths

CalciMedica

Note: Patient counts on this slide reflect either hospitalizations or ICU admissions in the US per year
1. See AP section for references 2. See AKI section for references; development program currently targeting Stage 2/3 AKI with respiratory failure
3. Yale Medicine 4. CDC 5. AJG 2024, El-Dallal et al.

Accelerating ICU recovery can drive significant hospital cost savings

Hospital Cost Driver	Potential Savings in Hospital Charges[1,2]
ICU stay	$26K / day
Mechanical Ventilation	$24K / day
Vasopressors	$28K / day

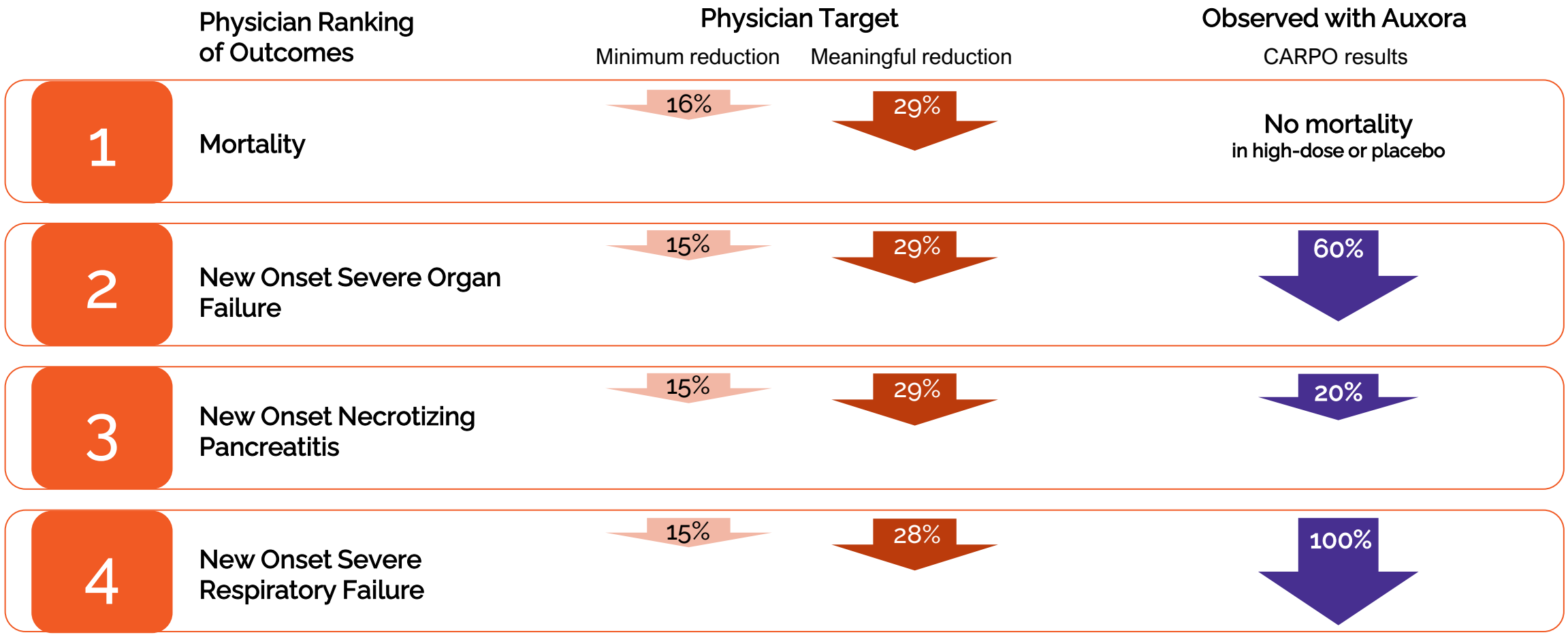
Reduce just one ICU or organ support day → tens of thousands in hospital savings

Data based on adults treated with vasopressors for septic shock in an ICU offering a good analog to other acute critical conditions like acute kidney injury and acute pancreatitis; values are hospital charge data, not direct costs; Each estimate is derived from a separate model and represents a marginal (non-additive) association with hospital charges

1. Hospital charges from Self et al., *CHEST* 2019; 155(2):315-321 2. Inflation adjustment of 55% on 2015 hospital charges to 2025 in line with BLS data, series ID: CUUR0000SEM

CalciMedica

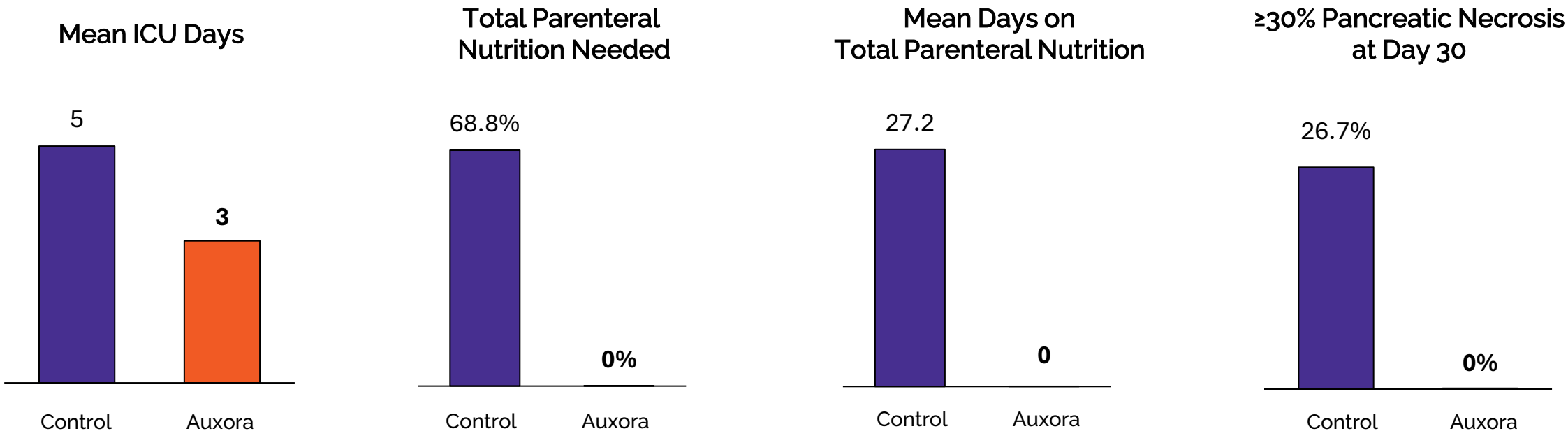
Auxora addresses top treatment objectives in AP



Source: Third-party market research with 20 U.S. HCP interviews and 151 HCP survey responses on AP management (February 2025).

In CRSPA, Auxora improved outcomes in pediatric severe pancreatitis patients receiving chemotherapy for leukemia

Ongoing CRSPA Phase 1/2 trial[1] is evaluating Auxora[2] in pediatric patients with asparaginase-induced pancreatitis (AIPT). Initial data at ASH compared eight Auxora-treated patients with 16 matched historical controls.



1. Seth Karol et al., Zegocractin to reduce the severity of asparagine-associated pancreatitis in children with acute lymphoblastic leukemia: results of the Phase 1 portion of the CRSPA study, ASH Poster #2837, December 2023
2. Auxora administered as 4-hour infusion: 30mg/m2 on day 1, 42mg/m2 on days 2-4; Receive all 4 doses if clinical condition permits and no DLT occurs
3. TPN: Total Parenteral Nutrition

Broad preclinical efficacy supports expansion into add'l I&I indications

Indication	Intended Formulation	Preclinical Observations
Pulmonary Arterial Hypertension	Oral	In vivo efficacy of CM5480 in a rat model of PAH (Saint-Martin Willer et al, 2025)
Rheumatoid Arthritis	Oral	In vivo efficacy of zegocractin and CM5480 in rat RA models (CalciMedica unpublished data)
Chronic Pancreatitis	Oral	In vivo efficacy of CM5480 in a mouse model of CP (Szabo et al, 2023)
Acute Ulcerative Colitis	IV	In vivo efficacy of zegocractin in a mouse model of inflammatory bowel disease (Letizia et al., 2022)
Traumatic Brain Injury	IV or Oral	In vivo efficacy of CM5480 in a mouse model of TBI (Mizuma et al., 2018)
Allergic Asthma	IV or Inhaled	In vivo efficacy of zegocractin in a mouse model of allergic asthma (Kahlfuss et al., 2022)