Arch Biopartners

TSXV: **ARCH.V**
OTCQB: **ACHFF.US**

Phase II Biotechnology Company Targeting Acute Kidney Injury (AKI)

July 2025

Notes about forward looking statements

This presentation contains forward-looking statements within the meaning of applicable Canadian securities laws regarding expectations of our future performance, liquidity and capital resources, as well as the ongoing clinical development of our drug candidates targeting the dipeptidase-1 (DPEP1) pathway, including the outcome of our clinical trials relating to LSALT peptide (Metablok) and cilastatin, the successful commercialization and marketing of our drug candidates, whether we will receive, and the timing and costs of obtaining, regulatory approvals in Canada, the United States, Europe and other countries, our ability to raise capital to fund our business plans, the efficacy of our drug candidates compared to the drug candidates developed by our competitors, our ability to retain and attract key management personnel, and the breadth of, and our ability to protect, our intellectual property portfolio. These statements are based on management's current expectations and beliefs, including certain factors and assumptions, as described in our most recent annual audited financial statements and related management discussion and analysis under the heading "Business Risks and Uncertainties". As a result of these risks and uncertainties, or other unknown risks and uncertainties, our actual results may differ materially from those contained in any forward-looking statements. The words "believe", "may", "plan", "will", "estimate", "continue", "anticipate", "intend", "expect" and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. We undertake no obligation to update forward-looking statements, except as required by law. Additional information relating to Arch Biopartners Inc., including our most recent annual audited financial statements, is available by accessing the Canadian Securities Administrators' System for Electronic Document Analysis and Retrieval ("SEDAR") website at www.sedar.com.

Arch is developing new treatments to prevent acute kidney injury (AKI).

Advancing LSALT peptide for inflammation-related AKI and repurposing cilastatin for toxin-related AKI.

- AKI caused by inflammation commonly occurs in patients who have on-pump cardiac surgery or septic shock.
- AKI caused by toxins, is a common negative side effect of certain antibiotics, steroids or antivirals.

- Each year, AKI affects approximately 14-16 million people in the US and Europe, yet no approved drug treatments exist—patients often require dialysis or a transplant to survive.[1,2]

Sources

Two drugs, two Phase II trials for AKI.

LSALT Peptide (ongoing) and cilastatin (pending) are being studied in Phase II trials, each addressing distinct causes of AKI.

LSALT Peptide

- Novel drug candidate for inflammation-related AKI.
- Ongoing international Phase II trial targeting cardiac surgery-associated AKI (CS-AKI).

Cilastatin

- A repurposed drug to prevent toxin-related AKI.
- Arch is providing the drug supply for the investigator-led Phase II PONTiAK* trial.

** Prevention Of NephroToxic Induced AKI.*

LSALT peptide is in an ongoing Phase II trial, targeting CS-AKI—an unmet medical need.

Approximately 30% of cardiac surgery (CS) patients who are on bypass machines experience acute kidney injury (AKI).[3,4]

- There are no approved drug treatments for CS-AKI.
- Over one million cardiac surgeries, including bypass procedures, are performed each year.[5]

- For more information about the Phase II CS-AKI Trial, please visit Clinicaltrials.gov. Learn more about LSALT peptide at archbiopartners.com.

Sources

LSALT peptide specifically targets Dipeptidase-1 (DPEP1).

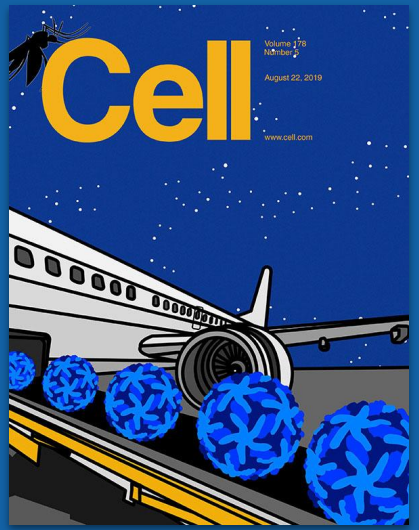
A new mechanism of action.

Arch scientists discovered that the enzyme DPEP1 mediates the recruitment of white blood cells and inflammation injury in the kidneys, lungs and liver.

Details of this discovery, including how LSALT peptide targets DPEP1 to block neutrophil adhesion and prevent inflammation-driven organ injury, were published in the journal *Cell*.[6]

Sources

Read the paper online:



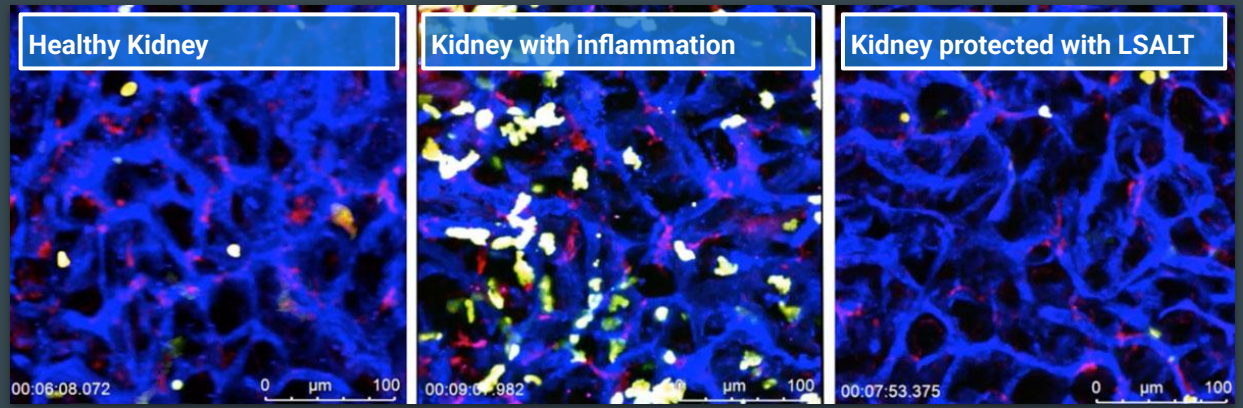
***Cell*, August 2019**

Read the full publication online or download the PDF version.
archbiopartners.com

See how LSALT peptide works.

LSALT peptide was shown to prevent inflammation in preclinical models of AKI, similar to the inflammation found in CS-AKI.

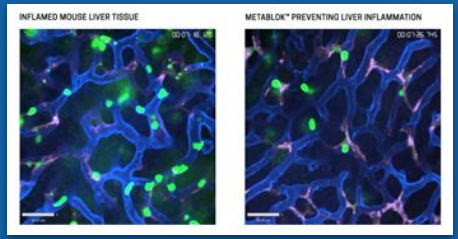
Intravital Kidney Tissue Microscopy Video – CLICK IMAGES TO WATCH THE VIDEOS ONLINE



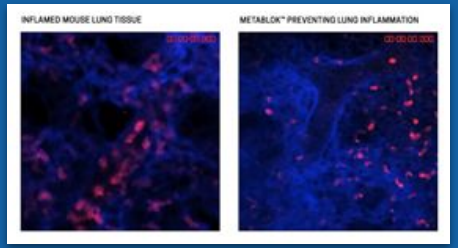
Inflammatory White Blood Cells Healthy Blood Vessels

See how LSALT works in other organs:

Preventing inflammation in the liver.



Preventing inflammation in the lungs.



Cilastatin is Arch's second drug asset targeting toxin-related AKI.

20-30% of AKI cases in hospitalized patients are caused by kidney-toxic drugs.[7,8] Cilastatin is being studied to prevent AKI caused by these toxins.

Originally developed by Merck for another indication, cilastatin is now off-patent.

Arch Biopartners has patents to repurpose cilastatin as a potential new treatment to prevent toxin-related AKI.

Sources

Advancing cilastatin to prevent drug toxin-related acute kidney injury (AKI).

Arch is supporting the PONTiAK Phase IIb trial by providing cilastatin drug supply for the study, which is funded and managed by third parties.

The PONTiAK trial is an investigator-led study using cilastatin to prevent drug toxin-induced AKI. The trial has received a no objection letter from Health Canada and will begin recruitment pending local ethics board approvals.

Led by researchers in Calgary, the study is independently funded, with Arch supplying cilastatin and contributing to trial design.

Cilastatin as a potential treatment for AKI.

Studies indicate that cilastatin has off-target effects that may help prevent AKI caused by nephrotoxic drugs, including radiographic contrast agents. The PONTiAK trial will evaluate its potential to prevent AKI from other drug toxins.

PONTiAK will build on research published by lead Arch scientists and their colleagues in *JCI (The Journal of Clinical Investigation)* in 2018, in which cilastatin was shown in preclinical models to inhibit leukocyte recruitment and reduce drug toxin uptake in the kidney, preventing AKI caused by radiographic contrast.[9]

Sources

Read the paper online:


JCI The Journal of Clinical Investigation

Renal immune surveillance and dipeptidase-1 contribute to contrast-induced acute kidney injury

Arthur Lau, … , Craig N. Jenne, Daniel A. Muruve

J Clin Invest. 2018;128(7):2894-2913. https://doi.org/10.1172/JCI96640.

Research Article | Inflammation | Nephrology

Radiographic contrast agents cause acute kidney injury (AKI), yet the underlying pathogenesis is poorly understood. Nod-like receptor pyrin containing 3–deficient (Nlrp3-deficient) mice displayed reduced epithelial cell injury and inflammation in the kidney in a model of contrast-induced AKI (CI-AKI). Unexpectedly, contrast agents directly induced tubular epithelial cell death in vitro that was not dependent on Nlrp3. Rather, contrast agents activated the canonical Nlrp3 inflammasome in macrophages. Intravital microscopy revealed diatrizoate (DTA) uptake within minutes in perivascular CX_3CR1^+ resident phagocytes in the kidney. Following rapid filtration into the tubular luminal space, DTA was reabsorbed and concentrated in tubular epithelial cells via the brush border enzyme dipeptidase-1 in volume-depleted but not euvolemic mice. $LysM\text{-}GFP^+$ macrophages recruited to the kidney interstitial space ingested contrast material transported from the urine via direct interactions with tubules. CI-AKI was dependent on resident renal phagocytes, IL-1, leukocyte recruitment, and dipeptidase-1. Levels of the inflammasome-related urinary biomarkers IL-18 and caspase-1 were increased immediately following contrast administration in patients undergoing coronary angiography, consistent with the acute renal effects observed in mice. Taken together, these data show that CI-AKI is a multistep process that involves immune surveillance by resident and infiltrating renal phagocytes, Nlrp3-dependent inflammation, and the tubular reabsorption of contrast via dipeptidase-1.

Find the latest version:

https://jci.me/96640/pdf

***JCI*, June 2018**

Read the full publication online or download the PDF version.

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A strong patent position.

LSALT peptide

- Composition and method of use patents, including protection for off-label applications, issued in the United States and PCT countries.
- Drug approval for CS-AKI could support off-label use for lung and liver inflammatory injury, other AKI indications, and septic shock.

Cilastatin

- Method of use patents to repurpose cilastatin as a treatment to prevent AKI.
- There is no commercial history of cilastatin as a stand-alone drug product.

Next Steps: focused on drug approvals.

The CS-AKI and PONTiAK Phase II trials target approximately 50% of AKI cases in hospitalized patients, advancing LSALT peptide and cilastatin as potential game-changers in global kidney care.

The CS-AKI Phase II trial supports LSALT peptide's approval as the first DPEP1 inhibitor to prevent inflammation related AKI.

The Phase IIb PONTiAK trial aims to establish cilastatin as a much needed new treatment to prevent toxin-related AKI.

Investor Information

Visit our Investor Hub to read the latest news releases and download financial reports and filings (also at SEDAR+). www.archbiopartners.com/investor-hub

Capitalization

Jul 4, 2025

$1.75 CAD **TSXV - ARCH.V**

$1.30 USD **OTCQB - ACHFF**

52 Week:

High $2.20 Low $1.27 CAD

Common shares outstanding:

65,906,366 March 31, 2025

Market Capitalization:

$115 M CAD

Options: 3,665,000

Exercisable Warrants:

None issued or outstanding

Latest news highlights, read and subscribe online.

April 22, 2025

…Toronto General Hospital Ethics Board Approval to Join the Phase II Trial for LSALT Peptide…

Read online

February 18, 2025

…Announces Health Canada No Objection Letter (NOL) Granted for Phase II PONTiAK Trial…

Read online

Executive Management

Richard Muruve

CEO, Director, Co-founder

Mr. Muruve co-founded the company with the Arch Inflammation team in 2010. Prior to his work at Arch, Mr. Muruve was a Vice President at Bank of Montreal where he spent 12 years in the Investment Banking Group.

Andrew Bishop

CFO, Director

Mr. Bishop is a Partner and Co-Founder of Bingley Capital Inc. and brings over 20 years of experience in advising biotech and health care companies.

Dr. Daniel Muruve MD

CSO, Co-Founder

A Professor in the Dept. of Medicine at the University of Calgary. Dr. Muruve has undertaken extensive post-graduate medical and scientific training at the University of Calgary, Harvard University and the University of Lausanne.

A committed board and advisors.

Claude Allary, Director
Co-founder, partner of Bionest Consulting, Sanofi, Pfizer, Glaxo

Farris Smith, Strategic Advisor
CFO, Leo Pharma (North America), former CFO of Novo Nordisk Canada.

Richard Rossman, Director
Gastroenterologist (retired), Asst. Professor at McMaster University, Helix Biopharma (Board)

Patrick Vink, Strategic Advisor
Former COO, Cubist Pharma (purchased by Merck for $10B)

Dr. David Luke, Strategic Advisor
Dr. Luke previously at Pfizer Inc (20+ years), as Senior Medical Director.

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About the company

archbiopartners.com/about-us

Investor information

archbiopartners.com/investor-hub

Latest news releases

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X (Twitter)

https://x.com/archbiopartners

LinkedIN

https://bit.ly/ArchBiopartners-LinkedIn

Sources

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8. Garcia, G., et al., **"Drug-Induced Acute Kidney Injury: A Cohort Study on Incidence and Risk Factors"**, *Frontiers in Medicine*, Volume 11, 2024. https://doi.org/10.3389/fmed.2024.1459170
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