

## Phase II Biotechnology Company Targeting Acute Kidney Injury (AKI)

April 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](http://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.<sup>1,2</sup>

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).<sup>3,4</sup>

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

- For more information about the Phase II CS-AKI Trial, please visit [Clinicaltrials.gov](https://www.clinicaltrials.gov). Learn more about LSALT peptide at [archbiopartners.com](https://www.archbiopartners.com).

## Sources

# LSALT peptide specifically targets Dipeptidase-1 (DPEP1).

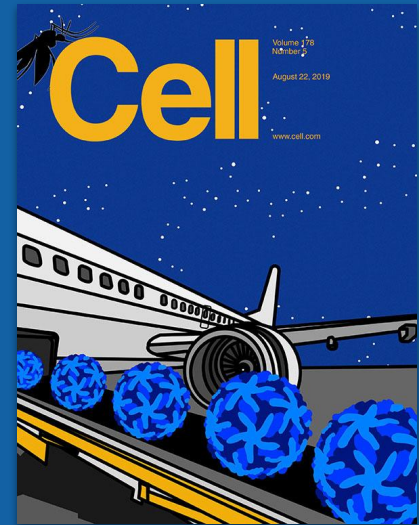
A new mechanism of action.

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*.<sup>6</sup>

Sources

Read the paper online:



*Cell*, August 2019

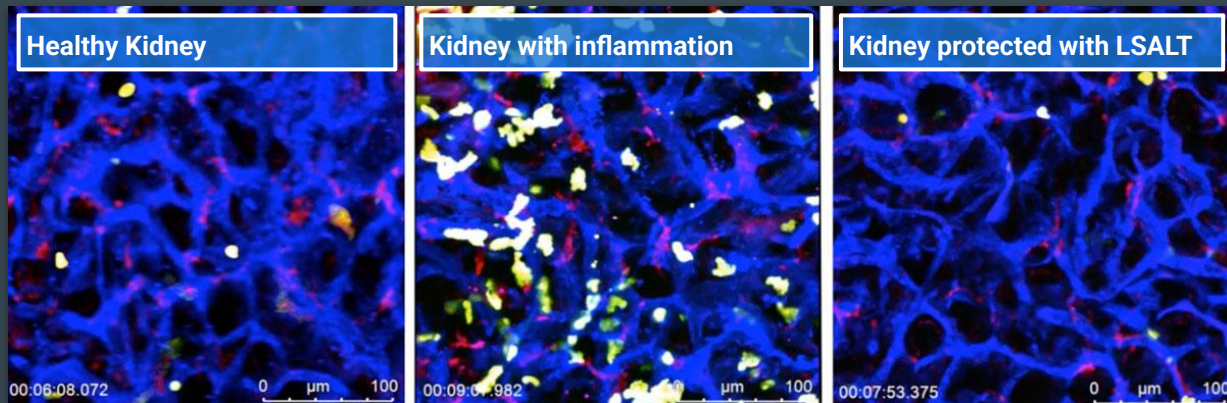
Read the full publication online or download the PDF version.

[archbiopartners.com](http://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.

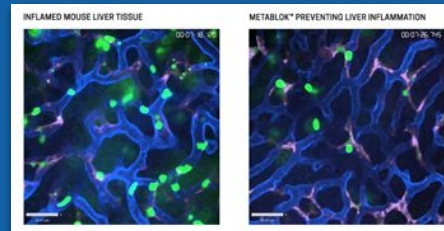
[Intravital Kidney Tissue Microscopy Video](#) – CLICK IMAGES TO WATCH THE VIDEOS ONLINE



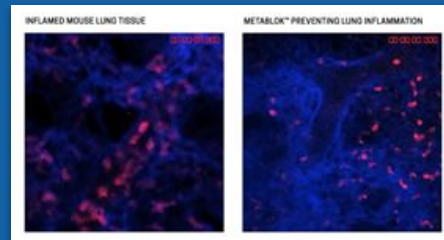
 Inflammatory White Blood Cells  Healthy Blood Vessels

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.<sup>7,8</sup> 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.<sup>9</sup>

[Sources](#)

Arch Biopartners

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. Not all AKI is caused by contrast-induced nephropathy (CIN), and the underlying pathogenesis is poorly understood. We investigated the role of renal immune surveillance 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 Nip2. Rather, contrast agents activated the conserved Nip2 effector caspase in macrophages. Intra-aortic microscopy revealed dendritic (DTA) uptake within minutes in perivascular CD45<sup>hi</sup> resident macrophages in the kidney. Following application into the tubular renal space, DTA was reabsorbed from the urine via direct interactions with tubules. CI-AKI was dependent on resident renal phagocytes, *Nip2*, leukocyte recruitment, and dipeptidase-1. Levels of the inflammation-related urinary biomarkers *IL-18* and *cap42* 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 multifactorial process that involves immune surveillance by resident and infiltrating renal phagocytes, Nip2-dependent inflammation, and the tubular reabsorption of contrast via dipeptidase-1.

Find the latest version:

<https://doi.org/10.96640/jci>



*JCI*, June 2018

Read the full publication online or download the PDF version.  
[archbiopartners.com](https://doi.org/10.96640/jci)

# 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](http://www.archbiopartners.com/investor-hub)

## Capitalization

Apr 4, 2025

\$1.69 CAD TSXV - ARCH.V

\$1.19 USD OTCQB - ACHFF

52 Week:

High \$2.41 Low \$1.36 CAD

## Common shares outstanding:

65,906,366 March 28, 2025

## Market Capitalization:

\$111 M CAD

Options: 3,865,000

Exercisable Warrants:

None issued or outstanding

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February 18, 2025

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

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January 8, 2025

...Ontario Research Ethics  
Board Approval to  
Proceed with Phase II  
Trial for LSALT Peptide...

[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, Assistant 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.

# Contact us for more information.

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## About the company

[archbiopartners.com/about-us](https://archbiopartners.com/about-us)

## Investor information

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## Latest news releases

[archbiopartners.com/investor-hub/press-releases](https://archbiopartners.com/investor-hub/press-releases)

## X (Twitter)

<https://x.com/archbiopartners>

## LinkedIn

<https://bit.ly/ArchBiopartners-LinkedIn>



# Sources

1. Sawhney, S., et al. "Harmonization of epidemiology of acute kidney injury and acute kidney disease produces comparable findings across four geographic populations", *Kidney International*, Volume 101, Issue 6, 1271 - 1281. <https://doi.org/10.1016/j.kint.2022.02.033>
2. Pavkov M.E., et al. "Trends in Hospitalizations for Acute Kidney Injury – United States, 2000–2014", *MMWR Morbidity Mortality Weekly Report* 2018;67:289–293. <http://dx.doi.org/10.15585/mmwr.mm6710a2>
3. Nadim, M., et al. "Cardiac and Vascular Surgery–Associated Acute Kidney Injury: The 20th International Consensus Conference of the ADQI (Acute Disease Quality Initiative) Group", *Journal of the American Heart Association*, 2018, 7(11). <https://doi.org/10.1161/JAHA.118.008834>
4. Scurt, F. G., et al. "Cardiac Surgery–Associated Acute Kidney Injury", *Kidney360* 5(6):p 909-926, June 2024. <https://doi.org/10.34067/KID.0000000000000466>
5. Vervoort, D., et al. "Global Cardiac Surgical Volume and Gaps: Trends, Targets, and Way Forward." *Annals of Thoracic Surgery Short Reports*, 2024, 2:320–324. <https://doi.org/10.1016/j.atssr.2023.11.019>
6. Choudhury, S.R., et al. "Dipeptidase-1 Is an Adhesion Receptor for Neutrophil Recruitment in Lungs and Liver", *Cell*, Volume 178, Issue 5, Pg1205-1221.E17, August 22, 2019. DOI: <https://doi.org/10.1016/j.cell.2019.07.017>
7. Yousif, Z. K., et al., "Clinical Characteristics and Outcomes of Drug-Induced Acute Kidney Injury Cases", *Kidney International Reports*, Volume 8, Issue 11, 2333 - 2344. <https://doi.org/10.1016/j.ekir.2023.07.037>
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>
9. Lau, A., et al., "Renal immune surveillance and dipeptidase-1 contribute to contrast-induced acute kidney injury", *JCI, The Journal of Clinical Investigation*, June 4, 2018. *J Clin Invest.* 2018 Jul 2;128(7):2894-2913. <https://doi.org/10.1172/JCI96640>