New Drugs for Acute Kidney Injury and Chronic Kidney Disease

September 2026

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.sedarplus.ca.

A critical gap in kidney treatment

Acute Kidney Injury (AKI) and Chronic Kidney Disease (CKD) affect millions of people, with limited drug treatment options available.

Patients with kidney failure must undergo dialysis while waiting for a kidney transplant in order to survive.

Arch Biopartners is pioneering a new pharmaceutical drug market targeting acute and chronic kidney injury.

Sources

Global scale and clinical burden of kidney disease

Millions face acute and chronic kidney disease every year.

Acute Kidney Injury (AKI)

- Affects approximately 13 million people globally each year, with a significant burden in the U.S. and EU.[1, 2, 3]
- Triggered by inflammation or toxins; once AKI occurs, patients are at increased risk of dialysis, complications, and mortality.

Chronic Kidney Disease (CKD)

- More than 800 million people globally have chronic kidney disease. In the U.S., diabetic kidney disease (DKD) is the leading cause of kidney failure.[10, 11, 17, 20]
- Current therapies may slow CKD progression, but they do not eliminate the significant residual risk of kidney failure or the need for renal replacement therapy.

Sources

A dual-platform approach to kidney disease

Arch Biopartners is developing novel, on-target drug candidates for acute kidney injury (AKI) and chronic kidney disease (CKD).

Two drugs in Phase II trials to prevent AKI:

- **LSALT peptide (Phase II)** – Targeting inflammation-related AKI
- **Cilastatin (Phase II)** – A repurposed drug to prevent toxin-induced AKI

Next-generation drugs for diabetic kidney disease (DKD), the leading cause of kidney failure in the U.S.: [20]

- **CKD Platform** – Developing new drug candidates that target DKD

Sources

LSALT peptide

Targeting acute inflammation in the kidneys.

LSALT peptide is in a Phase II trial to protect kidneys from cardiac surgery-associated acute kidney injury (CS-AKI).

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

- Over one million cardiac surgeries, including bypass procedures, are performed each year.[5]
- No drugs are currently approved to prevent AKI during cardiac surgery.

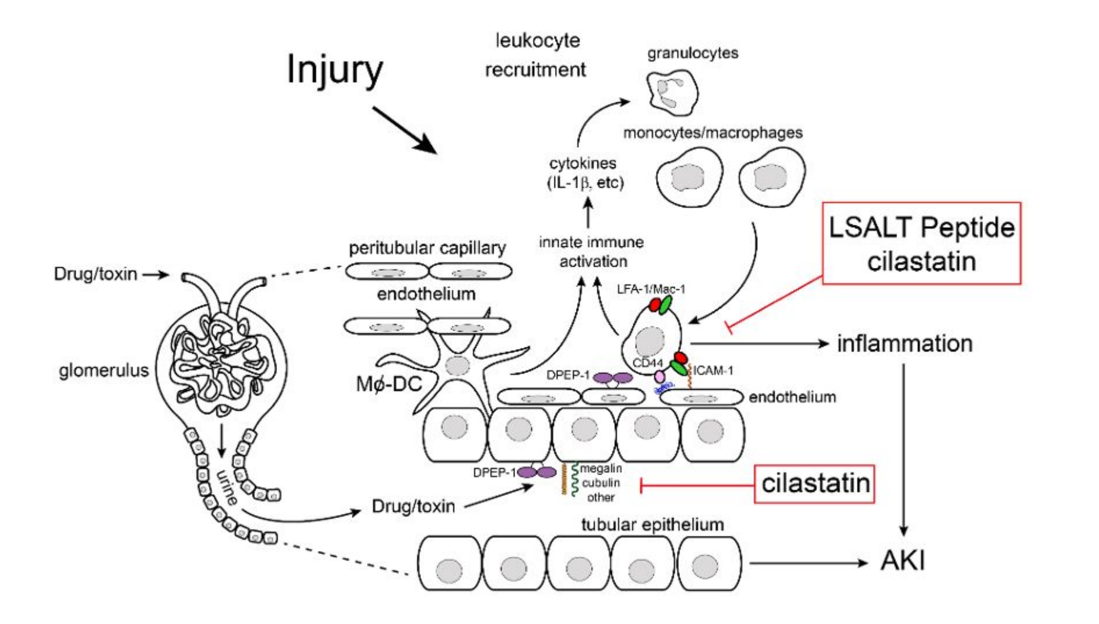
- Phase II trial, designed for up to 240 patients, currently recruiting across Canada. Trial details: ClinicalTrials.gov
- More about LSALT peptide at archbiopartners.com/lsaltpeptide

Sources

DPEP1: A therapeutic target for acute kidney injury.

Arch's AKI programs share a common biological target: dipeptidase-1 (DPEP1).

LSALT peptide is intended to reduce inflammation-driven injury by inhibiting DPEP1's role in leukocyte recruitment to the kidney, while **cilastatin** is intended to reduce toxin uptake-related injury relevant to nephrotoxic drugs.



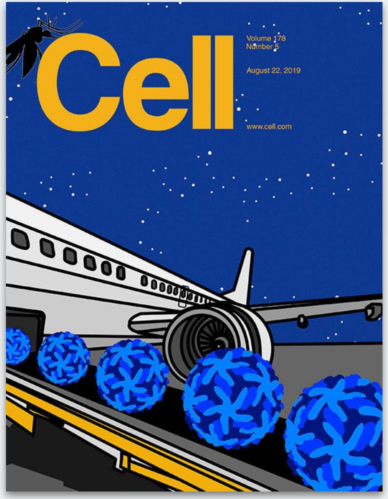
Caption: This diagram shows how DPEP1 contributes to kidney injury via two mechanisms: leukocyte-driven inflammation targeted by LSALT peptide, and toxin uptake in proximal tubules inhibited by cilastatin.

LSALT peptide targets Dipeptidase-1 (DPEP1).

A novel mechanism to block kidney inflammation.

In a 2019 *Cell* publication, Arch scientists reported that DPEP1 mediates white blood cell adhesion in the kidney, driving inflammation and AKI.

The published data show that DPEP1 inhibition by LSALT peptide reduced neutrophil recruitment and improved survival in models of systemic inflammation.[6] These findings established DPEP1 as a key therapeutic target for inflammation-driven organ injury, providing the preclinical rationale for current development of LSALT peptide to prevent AKI.



***Cell*, August 2019**

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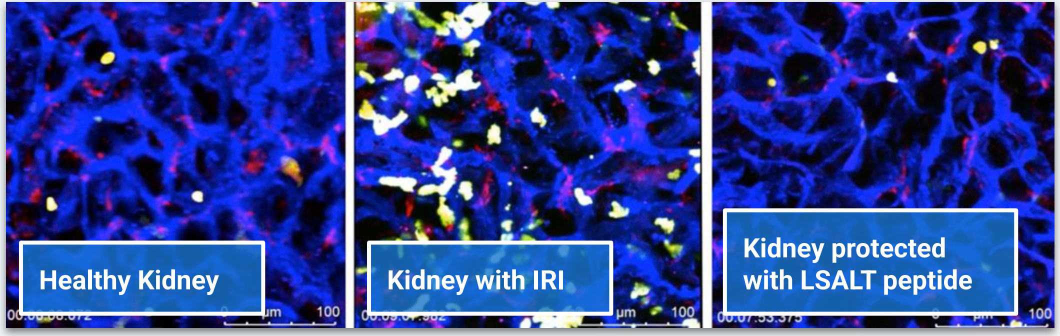
Sources

Peer-reviewed data in *Science Advances* shows LSALT peptide blocking the DPEP1 pathway to prevent kidney inflammation in mouse kidney models.

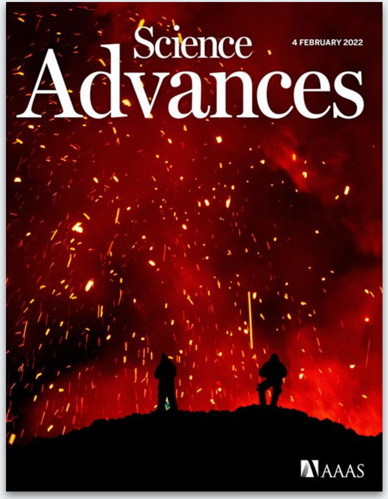
LSALT peptide reduced ischemia-reperfusion injury (IRI), an inflammation-driven form of AKI, supporting advancement to human clinical studies.[18]

Intravital Kidney Tissue Microscopy (click to open video)



Inflammatory White Blood Cells
Healthy Blood Vessels

Sources



Science Advances, February 2022

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Previous Phase II trial confirms DPEP1 targeting and inflammation reduction.

Biomarker results show a signal of reduced inflammation.

Arch scientists' 2024 *BMJ Open* publication reported results from a Phase II placebo-controlled trial of LSALT peptide in hospitalized patients.

LSALT peptide demonstrated a general trend towards declining inflammatory biomarkers, including a significant decrease in CXCL10 (p-value = 0.02), a chemokine that helps recruit white blood cells to sites of inflammation.

These findings support DPEP1 inhibition as a strategy to reduce inflammation and protect organs that express DPEP1, including kidneys, lungs, and liver.[19]

***BMJ Open*, March 2024**

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Sources

Cilastatin

Preventing kidney injury caused by toxic pharmaceutical drugs.

Cilastatin: a repurposed DPEP1 inhibitor with a long safety record, now in a Phase II clinical trial for drug toxin-induced AKI.

Drug toxin-induced AKI is a common complication of widely used antibiotics, chemotherapy, and other nephrotoxic drugs, with no approved treatments.

The Phase II PONTiAK trial is a 698-patient study evaluating cilastatin's ability to prevent AKI in hospitalized adults exposed to commonly used nephrotoxic drugs.

The trial enrolls patients receiving four types of kidney-damaging medications: chemotherapy agents, immunosuppressants, antibiotics, and contrast dyes.

Recruitment began in July 2025 at five hospital sites in Alberta, led by investigators at the University of Calgary.

The study is independently funded and managed, with Arch supplying cilastatin to support the trial.

- Trial details: ClinicalTrials.gov
- More info: archbiopartners.com/cilastatin

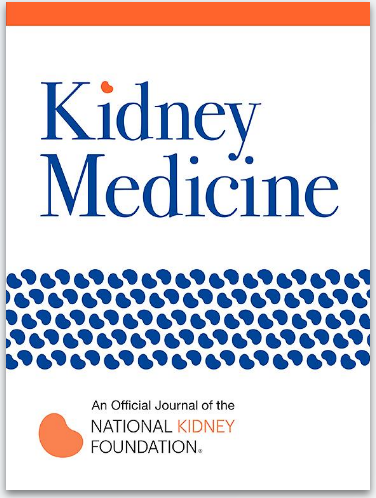
Cilastatin as a treatment for drug toxin-induced AKI.

Cilastatin has been shown to inhibit toxin uptake by kidney cells, a mechanism that may help prevent AKI caused by widely used antibiotics, chemotherapy drugs, and contrast agents used in medical imaging.

Laboratory studies published in the *Journal of Clinical Investigation* (2018) showed cilastatin blocks DPEP1-mediated toxin uptake and inflammatory cell recruitment in kidney tissue.[8]

A 2024 systematic review and meta-analysis published in *Kidney Medicine* analyzed 10 studies involving over 6,800 patients. Cilastatin demonstrated strong safety and nephroprotective potential, with up to a 74% reduced risk of AKI in clinical settings.[9]

Sources



***Kidney Medicine*, December 2024**

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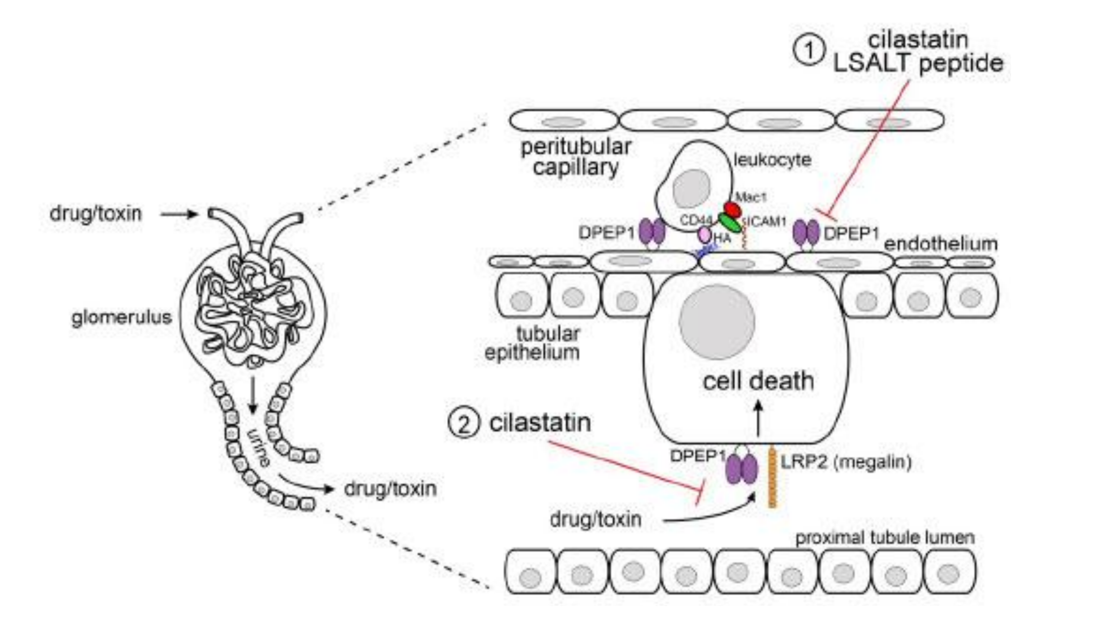
Cilastatin is an enzymatic DPEP1 inhibitor that blocks both endothelial and tubular DPEP1 in the kidney.

Cilastatin helps prevent toxin-related AKI by stopping the reabsorption of toxins and heme pigments, such as those seen in rhabdomyolysis or drug-induced injury.

LSALT peptide targets inflammation-driven kidney injury, while cilastatin is being evaluated for toxin-related injury in kidney tubules.



Caption: (1) DPEP1-mediated leukocyte adhesion/recruitment driving renal inflammation (targeted by LSALT peptide), and (2) DPEP1-associated proximal tubular toxin uptake contributing to tubular injury (inhibited by cilastatin).

Chronic kidney disease (CKD) platform

Targeting IL-32, a novel lipid droplet-associated cytokine linked to diabetic kidney disease (DKD).

- More than 800 million people are affected globally; 35 to 38 million in the U.S.[10, 11, 12]
- Diabetes drives up to 40% of CKD.[13]
- Current CKD treatments do not target kidney-specific disease mechanisms.

Arch's IL-32 strategy advances a therapeutic approach that is critical for preventing irreversible structural organ damage and slowing the progression toward kidney failure.

A next-generation drug platform for chronic kidney disease (CKD).

Arch's CKD platform targets interleukin-32 (IL-32), a human cytokine implicated in kidney inflammation and diabetic kidney disease (DKD).

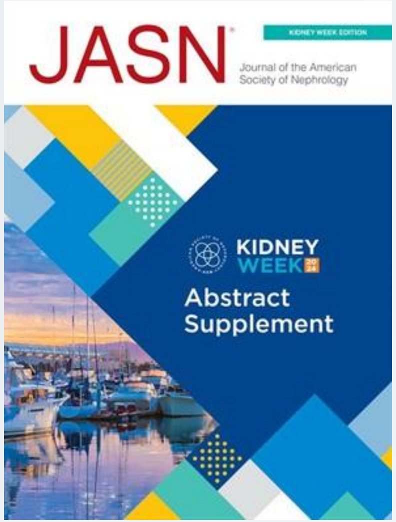
IL-32 has been identified as a potential mediator of lipid droplet accumulation and chronic inflammation in kidney cells, both recognized as key drivers of DKD, a leading cause of kidney failure worldwide.[13,14,15]

Video: American Diabetes Association Thought Leadership Series at the 2026 Scientific Session (click to watch on YouTube)

Early research identified IL-32 as a lipid-associated cytokine triggering inflammation and injury in diabetic kidney disease (DKD).

IL-32 was first identified as a potential mediator of lipid droplet accumulation and inflammation in DKD in findings presented at Kidney Week 2024 and published in the *JASN Abstract Supplement*.

Evidence from patient samples and disease models confirmed IL-32's role in kidney injury, establishing a mechanistic link between metabolic stress, inflammation, and tubular damage. These findings support the rationale for developing new DKD drugs that target IL-32.



***JASN*, October 2024**

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The potential role of IL-32 in diabetic kidney disease (DKD).

Peer-reviewed findings published in *Inflammation Research* (February 2026) reveal new mechanistic insights into IL-32's role in DKD, based on research led by Arch Biopartners scientists.

Studies of human kidney tissue show that during DKD, kidney cells accumulate lipid droplets coated with IL-32, a cytokine linked to inflammation and cell injury. This lipid and IL-32 association may connect metabolic stress with inflammation, driving kidney damage.

These findings highlight IL-32 as a promising new therapeutic target to preserve kidney structure and slow DKD progression.

Sources

***Inflammation Research*, February 2026**

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About Arch Biopartners

Developing new drugs for acute and chronic kidney disease.

Arch's leadership in kidney therapeutics is protected by strong patents.

LSALT peptide

Composition and method-of-use patents. Approval for the CS-AKI indication could also support use of LSALT peptide in the lungs, liver, other AKI indications, and sepsis.

Cilastatin

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

CKD Platform

Composition and method-of-use patents covering therapies that target IL-32. The platform includes new drug compositions and additional therapeutic approaches for CKD.

Next steps: Advancing three drug programs for acute and chronic kidney injury

Arch is advancing two Phase II programs in AKI and an IL-32 CKD platform toward lead optimization and IND planning.

The **LSALT peptide** and **cilastatin** Phase II trials target two major hospital settings associated with up to 60% of AKI cases: cardiac surgery and nephrotoxic drug exposure.[3,7]

Successful completion could establish two urgently needed preventive treatment options for global kidney care.

Arch's IL-32 **CKD platform** is advancing a patented drug library toward lead optimization.

Next steps include selecting lead candidates, IND-enabling work, and building future partnerships.

Sources

Investor Information

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

Capitalization

Sep 3, 2026
$1.08 CAD TSXV - ARCH.V
$0.76 USD OTCQB - ACHFF

52-week:
High: $1.72 Low: $0.39 CAD

Common shares outstanding:
67,933,289 April 2, 2026

Market Capitalization:
$73 M CAD

Options: 5,165,000

Exercisable Warrants: None

Latest news highlights, read and subscribe online.

August 31, 2026
Receives Central IRB Approval to Support U.S. Sites in Phase II CS-AKI Trial of LSALT Peptide
Read online

July 17, 2026
Arch Biopartners Engages Private Laboratory to Accelerate IL-32 Chronic Kidney Disease Program
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

Financial executive with 20+ years in biotech and healthcare. Co-Founder of Bingley Capital and CFO at Arch Biopartners and AmacaThera. Former senior healthcare investment banker at HSBC; has led over 100 financing, M&A and licensing transactions.

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, strategic advisors and scientific leaders.

Patrick Vink, Chair, Director

Patrick Vink, M.D., M.B.A., has been an advisor to the pharmaceutical industry since 2015 and has served as a director or chair of public and private companies. He was Executive Vice President and COO of Cubist Pharmaceuticals until its $9.5 billion acquisition by Merck in 2015.

Richard Rossman, Director

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

Farris Smith, Strategic Advisor

President, Vimy Pharma, Former CFO, Leo Pharma (North America), Novo Nordisk Canada.

Dr. David Luke, Strategic Advisor

Previously at Pfizer Inc (20+ years), as Senior Medical Director.

Dr. Justin Chun, Principal Scientist

Dr. Chun, MD, PhD, FASN is an Associate Professor and Royal College certified nephrologist at the University of Calgary.

Contact us for more information.

Contact us

+1 647 428-7031 office
info@archbiopartners.com

About the company

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https://x.com/archbiopartners

LinkedIn

https://bit.ly/ArchBiopartners-LinkedIn

Bluesky

https://bsky.app/profile/archbiopartners.com

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