New Drugs for Acute Kidney Injury and Chronic Kidney Disease

February 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.

Overview

Arch Biopartners is developing novel, on-target drugs to prevent 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 drug 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 drugs that specifically target DKD

Sources

A critical gap in kidney treatment.

AKI and CKD affect millions, with limited options that directly protect kidney function.

Arch is targeting these unmet needs with novel drug candidates that aim to prevent AKI and target CKD, with the goal of reducing the burden of dialysis and kidney transplantation.

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 surgery, sepsis, or toxic drugs. Once AKI occurs, many patients face a higher risk of dialysis or transplant.

Chronic Kidney Disease (CKD)

- More than 800 million people globally have chronic kidney disease, and in the U.S., diabetic kidney disease (DKD) is the leading cause of kidney failure.[10, 11, 17, 20]
- Current therapies slow progression; Arch is developing new drugs that target (DKD) to help address the underlying disease process.

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]

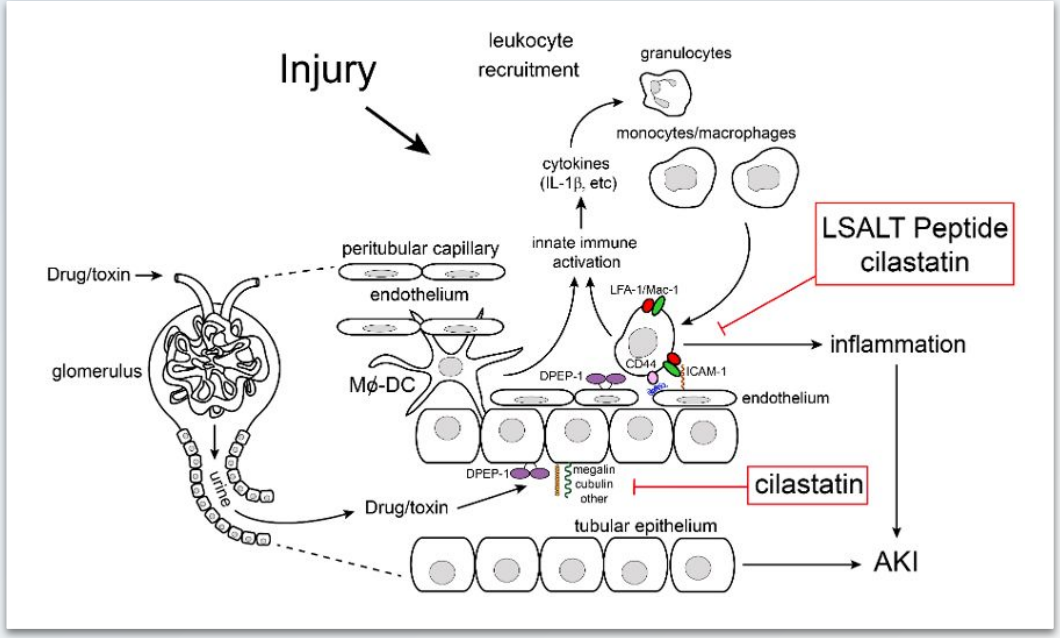
- Over one million cardiac surgeries, including bypass procedures, are performed each year.[5]
- No drugs are currently approved to prevent AKI during cardiac surgery.
- Phase II trial, designed for up to 240 patients, currently recruiting across Canada. Trial details: ClinicalTrials.gov
- More about 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.



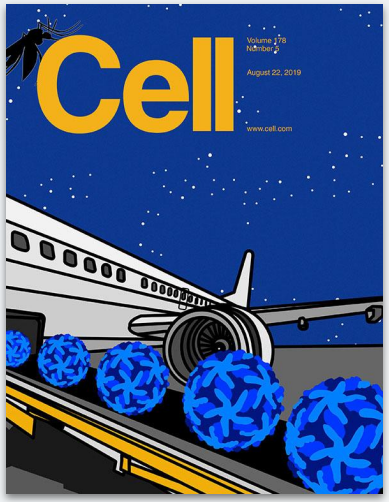
Caption: This diagram shows how DPEP1 contributes to kidney injury via two mechanisms: leukocyte-driven inflammation targeted by LSALT peptide, and toxin uptake in proximal tubules inhibited by cilastatin.

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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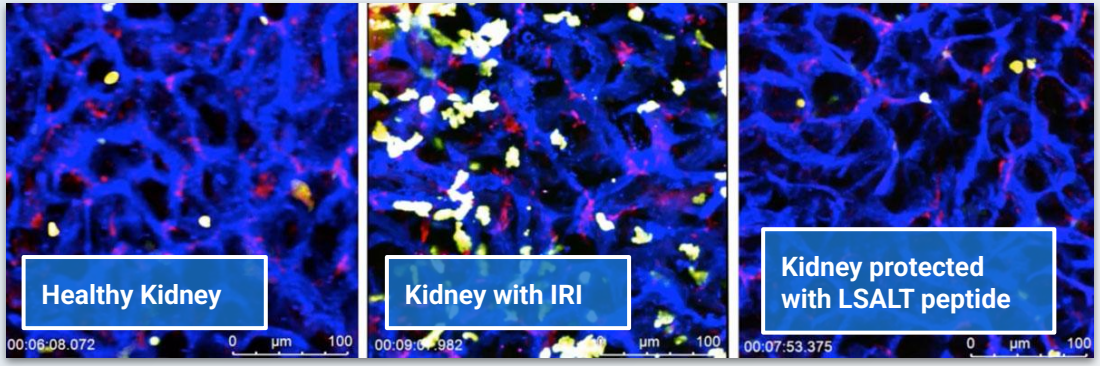
Sources

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

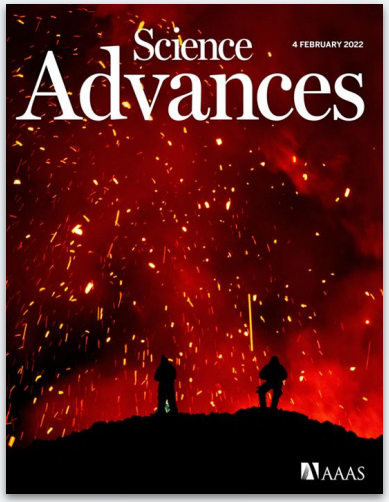
LSALT peptide reduced ischemia-reperfusion injury (IRI), an inflammation-driven form of AKI, supporting advancement to human clinical studies.[18]

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 linked to inflammatory and autoimmune disease activity.

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 developed by Merck with a long safety record, now in Phase II 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, among the millions who receive nephrotoxic drugs annually.

The trial enrolls patients receiving four types of kidney-damaging medications: chemotherapy agents, immunosuppressants, antibiotics, and contrast dyes. This allows researchers to evaluate the effectiveness of cilastatin across different drug exposures.

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

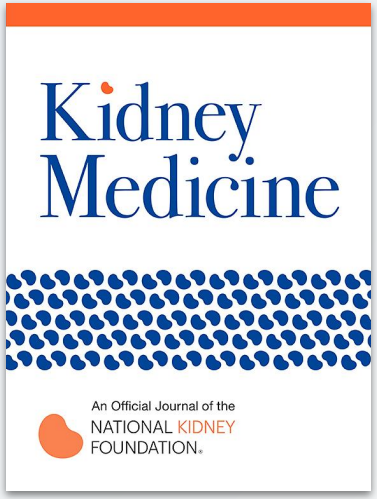
Cilastatin as a treatment for drug toxin-induced AKI.

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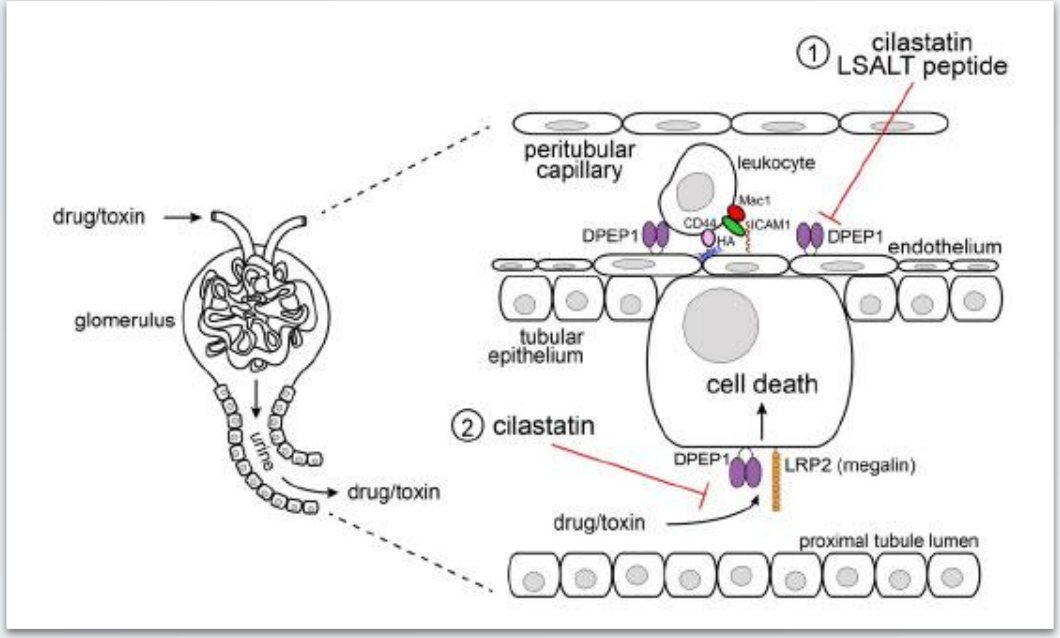
Cilastatin is an enzymatic DPEP1 inhibitor that blocks both endothelial and tubular DPEP1 in the kidney.

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.

By blocking DPEP1 on both kidney endothelium and tubules, cilastatin complements LSALT peptide, which targets only endothelial DPEP1.



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.

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]

- More than 800 million people 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 the IL-32 mechanism.

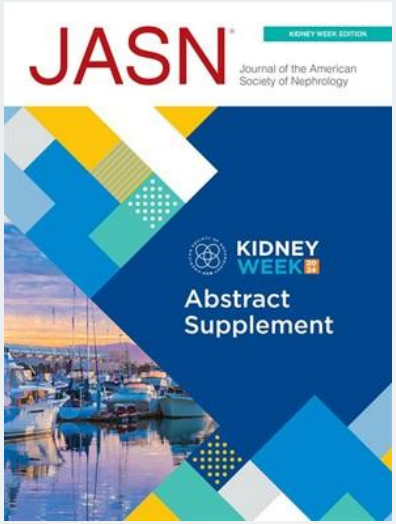
Arch's IL-32 strategy advances a therapeutic approach that is critical for preventing irreversible structural organ damage and slowing the progression toward kidney failure.

Sources

Early research identified IL-32 as a lipid-associated cytokine driving 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 laid the groundwork for further research to validate IL-32 as a disease-driving cytokine and potential drug target in DKD.



***JASN*, October 2024**

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Interleukin-32 (IL-32) as a novel driver of 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

Advancing an integrated program with new treatments targeting inflammation- and toxin-related kidney injury.

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

Patents covering both composition and method-of-use for targeting IL-32. Includes several therapeutic approaches to treat CKD and other metabolic disease indications.

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 address distinct forms of acute kidney injury, together representing up to 60% of AKI in hospitalized patients.[3,7] Successful completion could establish two urgently needed treatment options for global kidney care.

Arch's IL-32 **CKD platform** is advancing a patented compound 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

Feb 18, 2026
$0.93 CAD TSXV - ARCH.V
$0.64 USD OTCQB - ACHFF

52-week:
High: $1.95 Low: $0.67 CAD

Common shares outstanding:
66,933,289 January 28, 2026

Market Capitalization:
$62 M CAD

Options: 5,165,000

Exercisable Warrants: None

Latest news highlights, read and subscribe online.

February 9, 2025
Arch Scientists Publish New Data Linking the Cytokine IL-32 to Inflammation and Diabetic Kidney Disease
Read online

January 16, 2025
Announces Appointment of Dr. Patrick Vink as Chairman of the Board
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 non-executive board member or chair of several public and private companies in North America and Europe. He currently serves as Chair of the Board of F2G Ltd, Micreos B.V., and Secura Bio.

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.

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https://bit.ly/ArchBiopartners-LinkedIn

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

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