

NEW HUMAN DATA STRENGTHENS BIOLOGICAL RATIONALE FOR DMX-200 IN FSGS

Highlights

- New data presented at Kidney Week 2026 provides additional biological evidence supporting the inflammatory mechanism of action of DMX-200 which targets CCR2¹ in FSGS²
- Independent researchers conducted a study of 261 patients to determine whether CCR2 activation is linked to biomarkers associated with kidney outcomes in FSGS patients
- The study concluded CCR2 is a biologically relevant therapeutic target in FSGS, reinforcing the mechanism of action rationale for DMX-200
- Findings were generated from the internationally recognised NEPTUNE cohort, one of North America's largest nephrotic syndrome research networks, providing external validation of the CCR2 pathway in FSGS
- CCR2 activity, an indicator of inflammation, was found to be significantly elevated in kidney samples from patients with FSGS, compared with healthy controls
- Higher CCR2 activity was associated with worsening kidney function, while elevated urinary MCP-1³ levels were associated with poorer clinical outcomes in FSGS patients, further supporting the rationale for targeting the CCR2 pathway (MCP-1 (also known as CCL2) is a key inflammatory signalling molecule that activates the CCR2 receptor)
- Outcomes are consistent with Dimerix's Phase 2 trial where DMX-200 reduced MCP-1 levels, compared with increases observed in placebo-treated patients⁴
- Taken together, the independent findings and Dimerix's Phase 2 biomarker data provide converging evidence that DMX-200 targets a pathway associated with disease progression in FSGS
- Findings are the subject of a newly filed Dimerix provisional patent application (provisional patent application number 2026904579) and may provide additional support for potential regulatory filings for DMX-200, subject to success in the ACTION3 Phase 3 trial

MELBOURNE, Australia, 23 October 2026: Dimerix Limited (ASX: DXB, "Dimerix"), a clinical-stage biopharmaceutical company developing treatments for kidney disease, is pleased to announce new translational research findings presented at Kidney Week 2026 on 22 October 2026 in Denver USA, that provide further independent evidence in support of Dimerix's Phase 3 investigational therapy, DMX-200, in focal segmental glomerulosclerosis (FSGS) kidney disease. The findings demonstrate that activation of the MCP-1/CCR2 inflammatory pathway is associated with disease progression and poorer outcomes in patients with FSGS, further supporting CCR2 as a therapeutic target in the Company's fully recruited ACTION3 Phase 3 study.

The research, presented by Dr Sean Eddy from the University of Michigan, utilised data from the internationally recognised Nephrotic Syndrome Study Network (NEPTUNE), one of the largest and most comprehensive nephrotic syndrome research cohorts in North America.

The study identified elevated activation of the MCP-1/CCR2 inflammatory signalling pathway in patients with FSGS, and demonstrated associations between CCR2 pathway activation, kidney function decline and disease progression. Importantly, the findings support the scientific rationale for targeting CCR2, the receptor inhibited by DMX-200, and are the subject of a newly filed Dimerix provisional patent application (provisional patent application number 2026904579).

“These findings are significant because they provide independent human data linking activation of the CCR2 pathway to poorer outcomes in patients with FSGS. Importantly, they align with biomarker findings from Dimerix’s Phase 2 study, where DMX-200 reduced urinary MCP-1 levels while placebo-treated patients experienced increases. Together, these data further strengthen the scientific rationale for targeting CCR2 in FSGS as we continue progressing our fully recruited ACTION3 Phase 3 study.”

Dr Nina Webster, CEO & Managing Director, Dimerix

Biological Plausibility for DMX-200

The new research presented at Kidney Week 2026 identified that patients with FSGS who had higher levels of MCP-1 experienced poorer kidney outcomes, highlighting the importance of the CCR2 pathway which DMX-200 targets. MCP-1 (also known as CCL2) is a key inflammatory signalling molecule that activates the CCR2 receptor. Activation of the MCP-1/CCR2 pathway recruits inflammatory cells into the kidney, promoting ongoing damage and fibrosis. These findings align with Dimerix's Phase 2 clinical trial, in which DMX-200 reduced MCP-1 levels while placebo-treated patients showed increases.⁴

Taken together, the independent findings and Dimerix’s Phase 2 biomarker data provide converging evidence that DMX-200 targets a pathway associated with disease progression in FSGS. While clinical efficacy remains subject to the outcome of the ongoing ACTION3 Phase 3 study, these data further support the scientific rationale underpinning DMX-200’s development program.

These findings may also provide supportive biological evidence for potential regulatory submissions, subject to positive outcomes from the ACTION3 Phase 3 study.

About the Research

The presentation entitled "Intrarenal CCL2 and CCR2 Activation Profiles Are Linked to Non-invasive Urinary Biomarkers Associated with Outcomes in Patients with FSGS" was presented at Kidney Week 2026 by Dr Sean Eddy of the University of Michigan. The study utilised data generated through the NEPTUNE consortium and was funded by Dimerix. A copy of the presentation is attached.



The Phase 3 study, which is titled “Angiotensin II Type 1 Receptor (AT1R) & Chemokine Receptor 2 (CCR2) Targets for Inflammatory Nephrosis”, or ACTION3 for short, is a pivotal (Phase 3), multi-centre, randomised, double-blind, placebo-controlled study of the efficacy and safety of DMX-200 in patients with FSGS who are receiving a stable dose of an angiotensin II receptor blocker (ARB). Once the ARB

dose was stable, patients were randomised to receive either DMX-200 (120 mg capsule twice daily) or placebo.

The ACTION3 adult cohort is fully recruited with 333 patients randomised and dosed globally. Recruitment has been completed across 219 sites in 21 countries including the United States, Europe, Japan, China, Australia and New Zealand. The final adult patient commenced treatment in March 2026, transitioning the study from recruitment into treatment and follow-up. The final adult patient is expected to complete dosing in March 2028. Recruitment of paediatric patients aged 12-17 years continues as an independent cohort within the ACTION3 program and may support future expansion into adolescent indications in key territories.

The single Phase 3 trial in FSGS patients is designed to capture evidence of proteinuria and kidney function (eGFR slope) during the trial, aimed at generating sufficient evidence to support marketing approval. Further information about the study can be found on ClinicalTrials.gov (Study Identifier: NCT05183646) or Australian New Zealand Clinical Trials Registry (ANZCTR) (Study Identifier ACTRN12622000066785).

Potential catalysts

As Dimerix advances its pivotal, fully recruited ACTION3 Phase 3 trial for DMX-200 toward what is anticipated to be a major value-creating event if successful, the Company has simultaneously strengthened its growth profile through the recent acquisition of DMX-652 and its ongoing advancement into Phase 2 clinical development. Progress and positive outcomes from the DMX-652 development program have the potential to create substantial additional shareholder value, diversify Dimerix's clinical pipeline, and deliver multiple near- and medium-term catalysts for investors.

Authorised for lodgement by the Board of Dimerix

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About Dimerix

Dimerix Limited (ASX: DXB) is a clinical-stage biopharmaceutical company focused on developing new therapies for kidney diseases with significant unmet medical need. The Company's lead program, DMX-200, is currently in Phase 3 clinical development for focal segmental glomerulosclerosis (FSGS), a serious and life-threatening rare kidney disease with limited treatment options. In addition, Dimerix is advancing a second asset, DMX-652, via a Phase 2 program in acute kidney injury (AKI), further strengthening its pipeline in high-need renal indications. With a growing network of five commercial partners supporting global development and future market access, Dimerix is committed to expanding its reach and delivering innovative treatments to as many

patients as possible worldwide while creating long-term value for shareholders. For more information, please visit the company's website at www.dimerix.com.

About DMX-200 in FSGS

FSGS is a rare, serious kidney disorder characterised by progressive scarring (sclerosis) in parts of the glomeruli - the kidney's filtering units. This scarring leads to proteinuria, progressive loss of kidney function, and often end-stage renal disease. FSGS is increasingly understood to have an inflammatory component, with monocyte and macrophage activation contributing to glomerular injury. In the United States, more than 40,000 people are estimated to be living with FSGS, including both adults and children.⁵ There are no therapies specifically approved for FSGS in the US, and disease management relies on non-specific immunosuppressive and supportive therapies. In patients with progressive or treatment-resistant FSGS, the average time from diagnosis to end-stage kidney disease can be as short as five years. Even among those who undergo kidney transplantation, disease recurrence occurs in up to 60% of cases,⁶ underscoring the urgent need for new, disease-modifying treatments.

DMX-200 is a chemokine receptor (CCR2) antagonist administered to patients already receiving an angiotensin II type I receptor (AT1R) blocker, the standard of care treatment for hypertension and kidney disease. DMX-200 is protected by granted patents in various territories until 2032, with patent applications submitted globally that may extend patent protection to 2042, in addition to Orphan Drug Designation granted in the United States, Europe, UK and Japan⁷.

About DMX-652 in Acute Kidney Injury

Acute kidney injury (AKI) is a prevalent and severe clinical syndrome of substantial unmet need. It can arise from several high-risk clinical settings including, most notably, cardiac surgery and sepsis and is characterised by a rapid decline in kidney function (within hours), often resulting in high morbidity and mortality. The current addressable patient population is estimated to be approximately 260,000 across the major markets of United States, Germany, France, Italy, Spain and United Kingdom per annum,^{8,9} making this indication a potential orphan designation. AKI represents a significant clinical burden. There are no approved therapies for AKI, which is associated with increased risk of chronic kidney disease and long-term kidney complications.

DMX-652 is a selective inhibitor of USP30 - a mitochondrial enzyme that slows the removal of damaged mitochondria. DMX-652, which is a small molecule delivered as an oral once daily capsule, is designed to support mitochondrial quality control in injured kidney cells, a mechanism indicated in the progression of AKI caused by ischaemia (lack of blood flow resulting in oxygen starvation), toxins or sepsis related causes, as well as in other renal and non-renal indications. The DMX-652 acquisition includes the composition of matter patent, an open IND in the US with a Phase 2 clinical trial protocol which has received approval to proceed by the US Food and Drug Administration (FDA), as well as sufficient pharmaceutical grade (GMP) drug product for the Phase 2 trial and all manufacturing methodology. The Phase 2 trial is designed to investigate DMX-652's potential to prevent kidney injury and preserve renal function.

Dimerix Forward Looking Statement

This release includes forward-looking statements that are subject to risks and uncertainties. Although management believes that the expectations reflected in the forward-looking statements are reasonable at this time, Dimerix can give no assurance that these expectations will prove to be correct. Readers are cautioned not to place undue reliance on forward-looking statements. Actual results could differ materially from those anticipated. Reasons may include risks associated with drug development and manufacture, risks inherent in the regulatory processes, delays in clinical trials, results of clinical trials, contractual risks, risks associated with patent protection, future capital needs or other general risks or factors, including but not limited to those factors outlined in the most recent Dimerix Limited Annual Report.

References

- 1 *CCR2 (C-C chemokine receptor type 2, also known as CD192 or MCP-1 receptor) is a protein-coding G protein-coupled receptor (GPCR)*
- 2 *Focal segmental glomerulosclerosis (FSGS) is a rare kidney disease characterised by scarring (sclerosis) in parts of some of the kidney's tiny filtering units, known as glomeruli*
- 3 *MCP-1 (Monocyte Chemoattractant Protein-1), also known as CCL2 (Chemokine (C-C motif) ligand 2), is a small cytokine belonging to the CC chemokine family*
- 4 ASX release 27 October 2020
- 5 Nephcure FSGS Facts (<https://nephcure.org/>)
- 6 *Front. Immunol.*, (July 2019) | <https://doi.org/10.3389/fimmu.2019.01669>
- 7 ASX releases: 14 December 2015, 21 November 2018, 07 June 2021, 30 September 2025
- 8 Demirjian S, Bashour CA, Shaw A, et al. Predictive Accuracy of a Perioperative Laboratory Test–Based Prediction Model for Moderate to Severe Acute Kidney Injury After Cardiac Surgery. *JAMA* 2022;327:956–64. <https://doi.org/10.1001/jama.2022.1751>.
- 9 Hobson C, Ruchi R, Bihorac A. Perioperative Acute Kidney Injury. *Crit Care Clin* 2017;33:379–96. <https://doi.org/10.1016/j.ccc.2016.12.008>



KIDNEY
WEEK 20
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Intrarenal CCL2 and CCR2 Activation Profiles Are Linked to Noninvasive Urinary Biomarkers Associated with Outcomes in Patients with FSGS

Sean Eddy, PhD

University of Michigan

Khun Zaw Latt, Carl White, John Hartman, Viji Nair, Phillip J. McCown, Virginia Vega-Warner, Yuee Wang, Wenjun Ju, Matthias Kretzler, David Fuller

CCL2-CCR2 axis

- Chemokine (C-C motif) ligand 2 (CCL2, also known as monocyte chemoattractant protein 1 (MCP-1)) activates chemokine (C-C motif) receptor 2 (CCR2) on monocytes/macrophages
- CCR2 activation promotes cell migration, adhesion and macrophage chemotaxis
- Attracts immune cells (mostly monocytes and macrophages) to the fibrotic areas
- Contributes to inflammation and fibrosis in various organs including kidneys
- ACTION3, a pivotal Phase 3 clinical trial is currently evaluating efficacy of CCR2 inhibition with DMX-200 in participants with FSGS
- *Hypothesis:* A CCR2 activation profile may characterize heterogeneity in CCR2 pathway activity among patients with FSGS, and inform the biological rationale for CCR2 targeting therapies

NEPphrotic syndrome sTUDy NEtwork

Precision Medicine for Nephrotic Syndrome



John Sedor
Cleveland Clinic



Larry Holzman
University of Pennsylvania



Crystal Gadegbeku
Cleveland Clinic



Akinlolu Ojo
University of Kansas



Laura Barisoni
Duke University



Chrysta Lienczewski
University of Michigan

NEPTUNE provides a collaborative, investigational infrastructure across 32 North American sites for translational research in:

- Focal Segmental Glomerulosclerosis (FSGS)
- Minimal Change Disease (MCD)
- Membranous Nephropathy (MN)
- Pediatric Non-biopsy Cohort
- Alport Syndrome

Cleveland Clinic, Cleveland, OH
Cohen Children's Medical Center, Manhasset, NY
Children's Mercy Hospital, Kansas City, MO
Columbia University, New York, NY
Duke University, Raleigh, NC
Emory University, Atlanta, GA
Harbor UCLA Medical Center, Torrance, CA
Johns Hopkins Medical Institute, Baltimore, MD
John H. Stroger, Jr. Hospital of Cook County, Chicago, IL
Kansas University, Kansas City, KS
Levine Children Atrium Health, Charlotte NC
Medical University of South Carolina, Charleston, SC
Montefiore Medical Center, Bronx, NY
New York University School of Medicine, New York, NY
Providence Medical Research, Spokane, WA
Seattle Children's Hospital, Seattle, WA
Stanford University, Stanford, CA
The Mayo Clinic, Rochester, MN
The Ohio State University College of Medicine, Columbus, OH

Temple University, Philadelphia, PA
Texas Children Hospital, Baylor College of Medicine, Houston, TX
University of California as San Francisco, San Francisco, CA
University Health Network, Toronto, Ontario, Canada
University of Miami Medical Center, Miami, FL
University of Michigan, Ann Arbor, MI
University of Minnesota, Minneapolis, MN
University of North Carolina at Chapel Hill, Chapel Hill, NC
University of Pennsylvania, Philadelphia, PA
University of Southern California, Children's Hospital of LA, CA
University of Texas Southwest Medical Center, Dallas, TX
University of Washington, Seattle, WA
Wake Forest, Winston-Salem, NC
RDCRN III DMCC – Cincinnati Children's Hospital, Cincinnati, OH
NEPTUNE DACC – University of Michigan, Ann Arbor, MI

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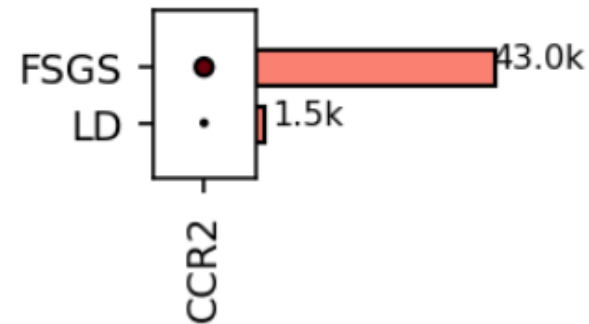
Demographics of patients with FSGS in NEPTUNE

Demographics	NEPTUNE enrolled (n=261)	With RNAseq (n=118)	With snRNAseq (n=33)
Age (mean ± SD, yrs)	34 ± 21	34 ± 21	34 ± 22
Female (%)	42	37	42
eGFR (mean ± SD, mL/min/1.73m ² , CKD-Epi)	70 ± 34	72 ± 31	64 ± 36
UPCR (median [IQR], mg/mg)	3.6 [1.6, 7.6]	2.8 [1.2, 6.2]	2.5 [4.7]
% APOL1 high risk	22	22	46

Developing CCR2 transcriptional gene activity network in patients with FSGS

- Differential expression of gene profiles from CCR2(+) compared to CCR2(-) immune cells and fibroblasts of patients with FSGS

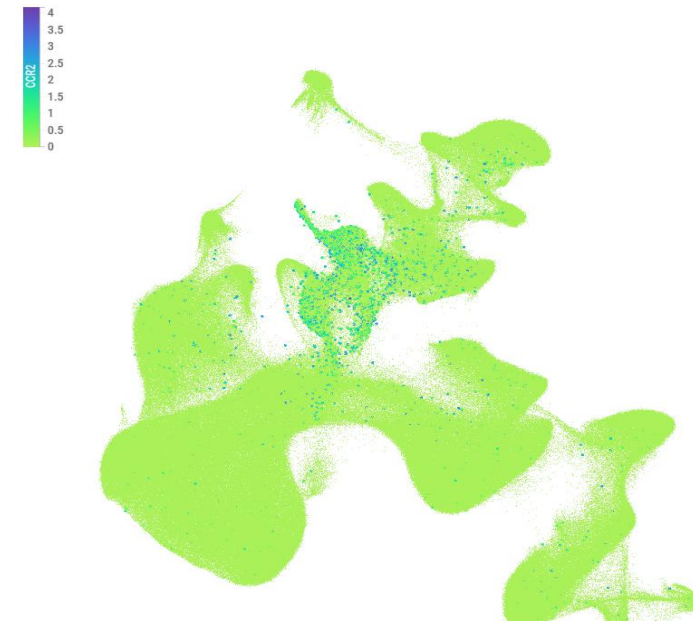
CCR2 expression in snRNAseq profiles (immune cells and fibroblasts)



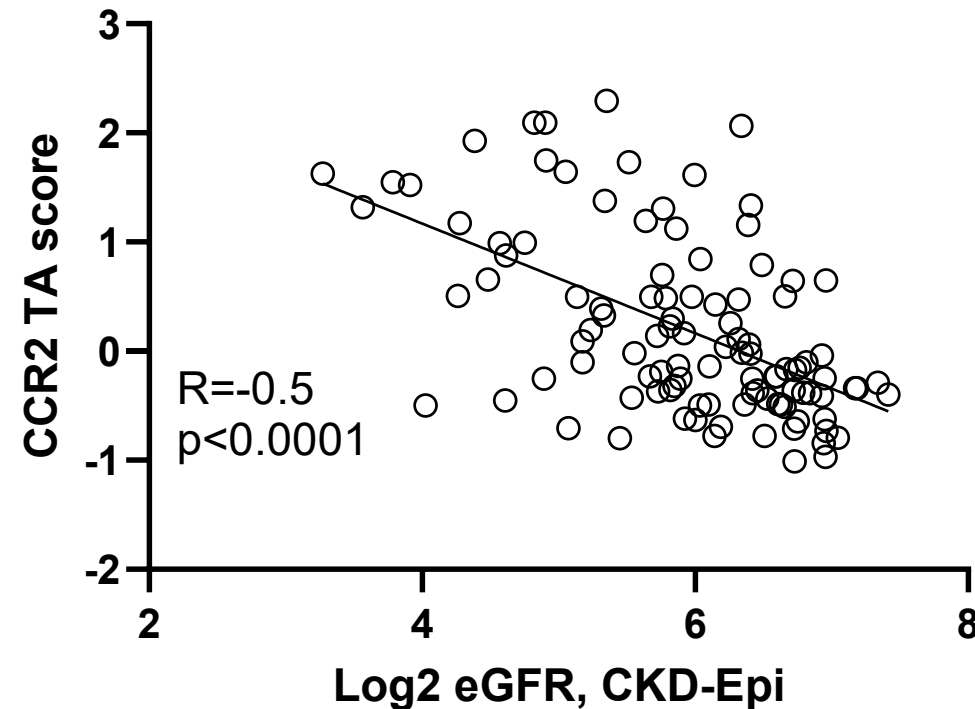
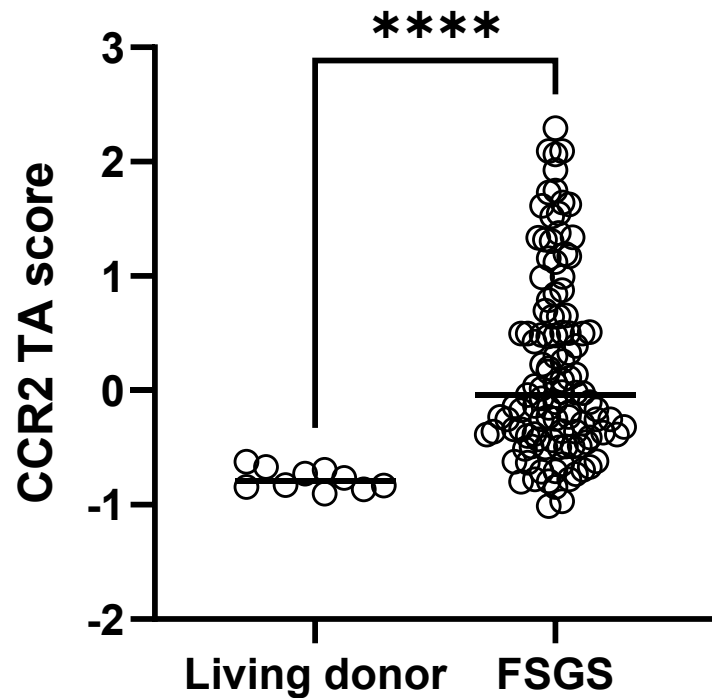
Developing CCR2 transcriptional gene activity network in patients with FSGS

- Differential expression of gene profiles from CCR2(+) compared to CCR2(-) immune cells and fibroblasts of patients with FSGS
- CCR2 co-expression analysis from tubulointerstitial (TI) bulk RNAseq profiles from patients with FSGS from NEPTUNE ($r>0.6$, $q<0.05$)
- 206 genes were shared and identified as CCR2-associated gene network and used to create a CCR2 transcriptional activity (TA) score

UMAP of CCR2+ cells (blue)



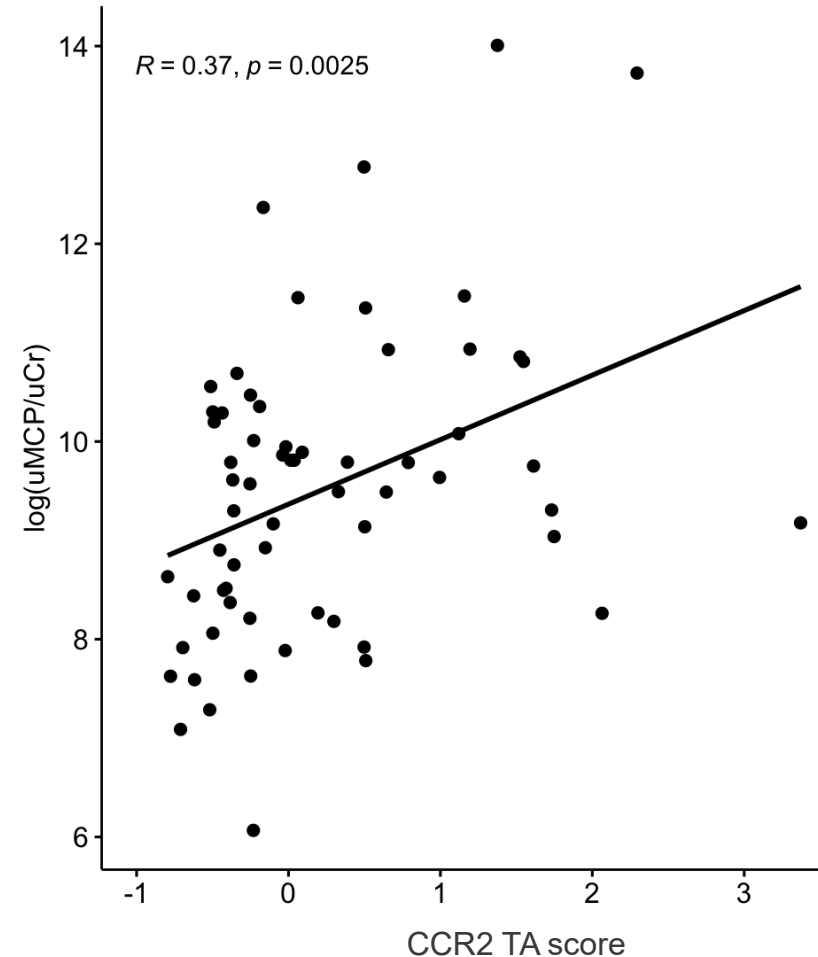
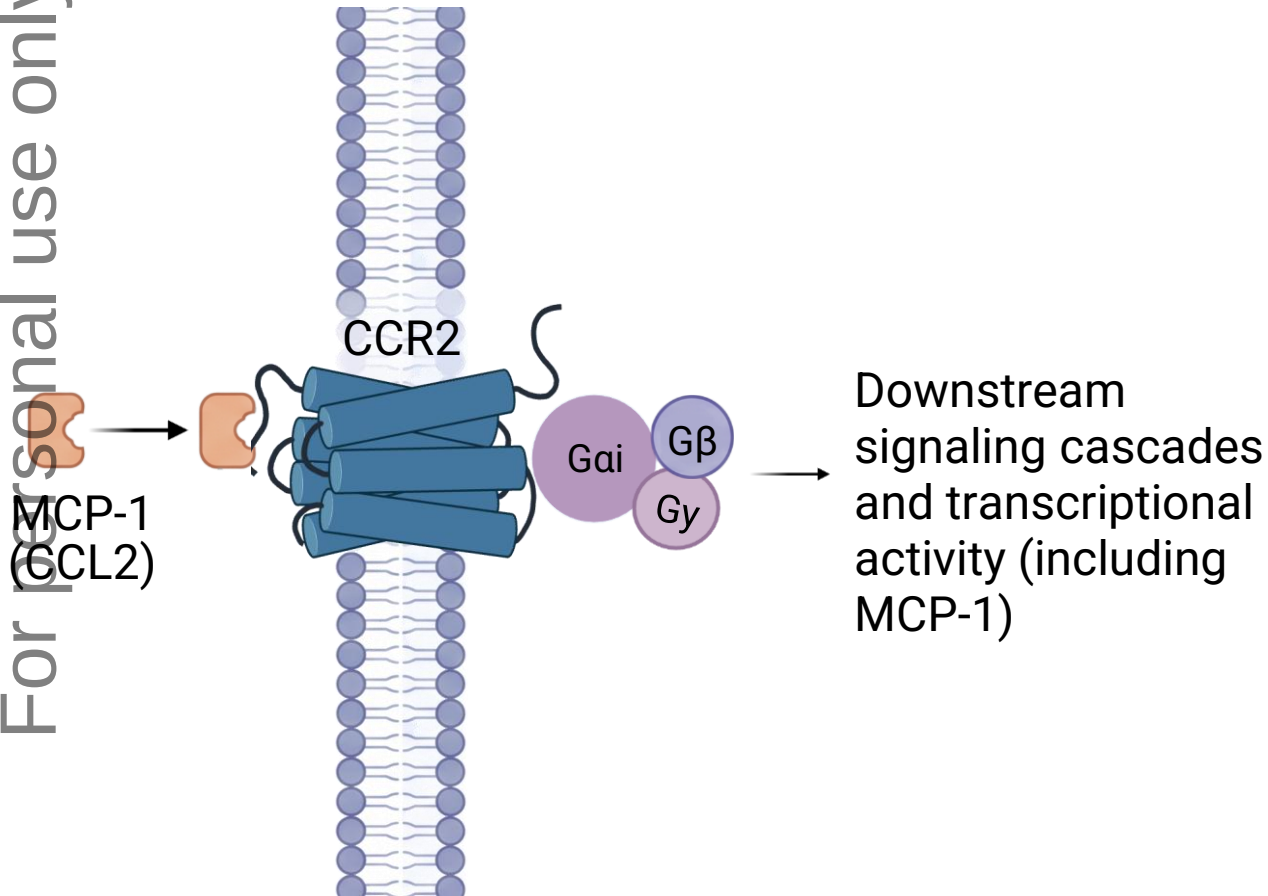
TI CCR2 TA was elevated in patients with FSGS and negatively correlated with eGFR



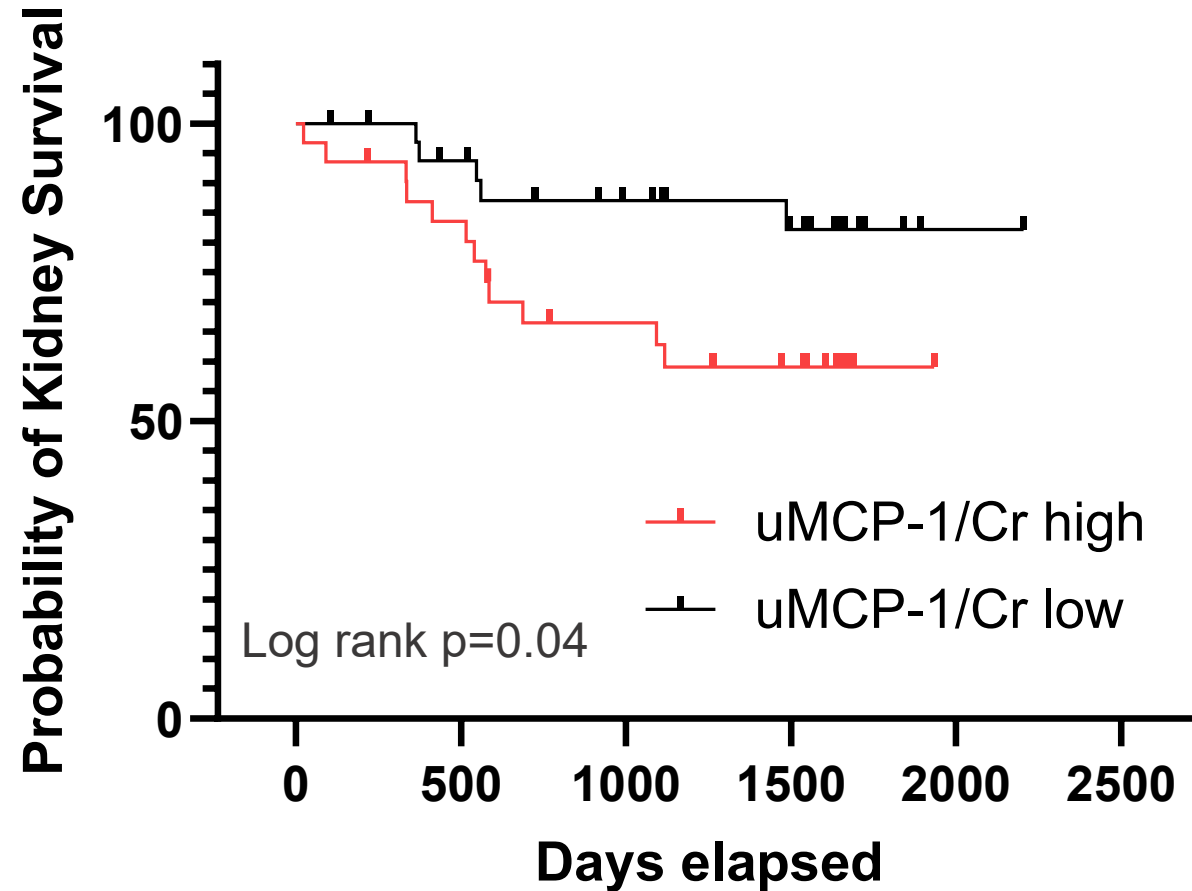
Similar associations were observed in the microdissected glomerular compartment

Could a urine MCP-1 biomarker profile serve as a non-invasive surrogate of CCR2 activation in the kidney?

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uMCP-1/Cr was associated with a composite endpoint (40% loss of eGFR or ESRK) in patients with FSGS



Conclusions

- A transcriptional profile associated with CCR2 activation was elevated in patients with FSGS and associated with a urine biomarker profile of MCP-1
- The urine profile was associated with a composite kidney endpoint in patients with FSGS
- Taken together, these results suggest that an intrarenal CCR2 TA score validates targeting CCR2 using DMX-200 in the ongoing ACTION3 study and that its activity may be monitored by urinary MCP1

Acknowledgements

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Questions