Dimerix

Investor Presentation

September 2026

Developing new therapies to treat kidney diseases with unmet clinical needs

Authorised for lodgement by the Board of the Company

Forward looking statements

This presentation includes forward-looking statements that are subject to risks and uncertainties. Although we believe 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.

DMX-200: Phase 3 Global Opportunity

5 commercial partners
DMX-200 licensed USA, EU, Canada, Australia, NZ, Japan, China, S.Korea, SEA and GCC[1]

up to $1.9 billion in total development and sales milestone payments **plus** royalties on net sales[1]

Phase 3 trial **recruitment complete** in trial of DMX-200 in focal segmental glomerulosclerosis (FSGS)[2]

FSGS indication is a **rare disease** that causes scarring of the kidney, leading to irreversible damage[3]

Reduced risk
- Proteinuria endpoint **passed blinded interim** (futility) assessment[1]
- Blinded review confirmed ACTION3 **statistically powered (>90%)** to demonstrate statistical significance of predicted proteinuria treatment effect of DMX-200[4]

Orphan drug designations regulatory, marketing exclusivity and pricing **benefits** in key territories[5]

~$1.9 billion*

5 commercial licensing partners

*total upfront and potential milestone payments + royalties on net sales

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1. Based on ASX releases 05 October 2023, 27 May 2024, 07 January 2025, 01 May 2025 and 16 June 2026; SEA = Southeast Asia, GCC = Gulf Cooperation Council; 2. AS release 10 March 2026; 3. ASX release 28 April 2026; 3. Guruswamy Sangameswaran KD, Baradhi KM. Focal Segmental Glomerulosclerosis (July 2021), online: https://www.ncbi.nlm.nih.gov/books/NBK532272/; 4. ASX release 11 March 2024 and 28 April 2026; 5. ASX releases: 14 December 2015, 21 November 2018, 07 June 2021 and 30 September 2025

Cycle of damage in glomerular diseases

What is FSGS?

Focal	**= some**
Segmental	**= sections**
Glomerulo	**= of the kidney filtering units**
Sclerosis	**= are scarred**

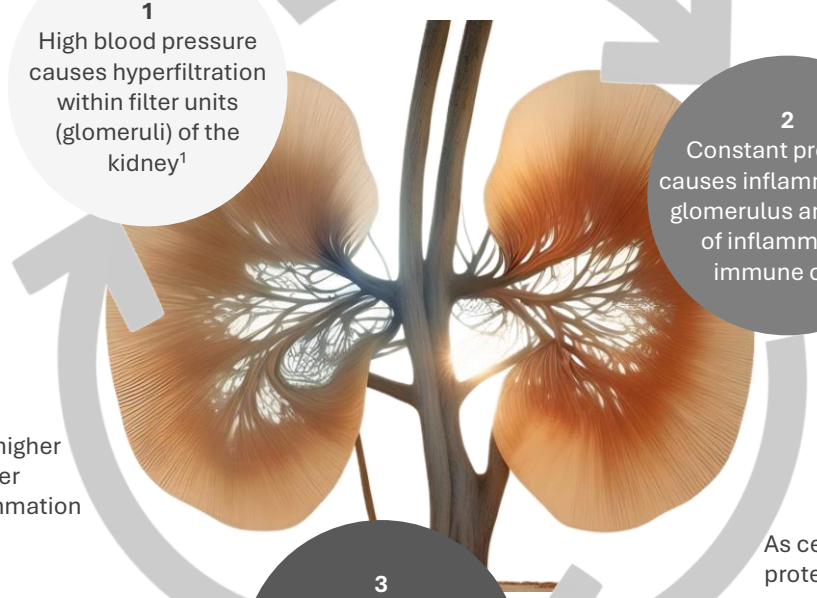
Existing blood pressure medication

DMX-200

Glomeruli exposed to stress from high blood pressure

1
High blood pressure causes hyperfiltration within filter units (glomeruli) of the kidney[1]

2
Constant pressure causes inflammation of glomerulus and influx of inflammatory immune cells



3
Pro-inflammatory environment drives sclerosis and fibrosis that is permanent and non-reversable

Fewer kidney cells drive higher blood pressure and further hyperfiltration and inflammation

As cells die, glomerular become scarred and protein leaks into the urine (**proteinuria**)

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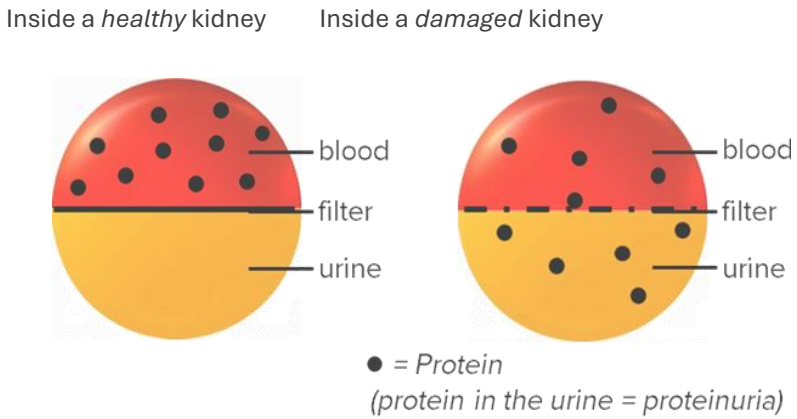
1. Lewis, E. J. et al. (2001), New Engl J Medicine 345, 851–860

Interpreting proteinuria as a surrogate endpoint

Proteinuria is the quantity of protein in the urine

A healthy kidney is a good filter and allows little to no protein into the urine[1]



- When kidneys are damaged, protein can leak into the urine causing proteinuria
- Proteinuria represents an important early marker of kidney function[2]

Proteinuria as a predictor of kidney disease[3]

Dip test	*uPCR g/g*	
Neg	<0.13	*normal*
Trace	0.13	
+	0.62	
	0.88	*proteinuric*
++	1.24	
+++	2.30	
++++	>3.09	*nephrotic*

NORMAL
MICROALBUMINURIA
HEAVY ALBUMINURIA

Minimal uPCR for ACTION3 inclusion

Proteinuria is typically less variable and easier to measure than eGFR[4]

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1.Guruswamy Sangameswaran KD, Baradhi KM. Focal Segmental Glomerulosclerosis (July 2021), online: https://www.ncbi.nlm.nih.gov/books/NBK532272/; 2. Nephcure FSGS living with the disease (2024) at https://nephcure.org/livingwithkidneydisease/ns-and-other-glomerular-diseases/understanding-fsgs/ ; 3. Adapted from graphics prepared by Renal Unit at the Royal Infirmary of Edinburgh and the University of Edinburgh; 4. PARASOL outcomes 2024, a working group made up of: NephCure, ISGD, NKF, KHI are co-sponsors of the project, FDA and EMA involvement, University of Michigan is data coordinating center, Industry invited as participants

ACTION3 phase 3 clinical trial

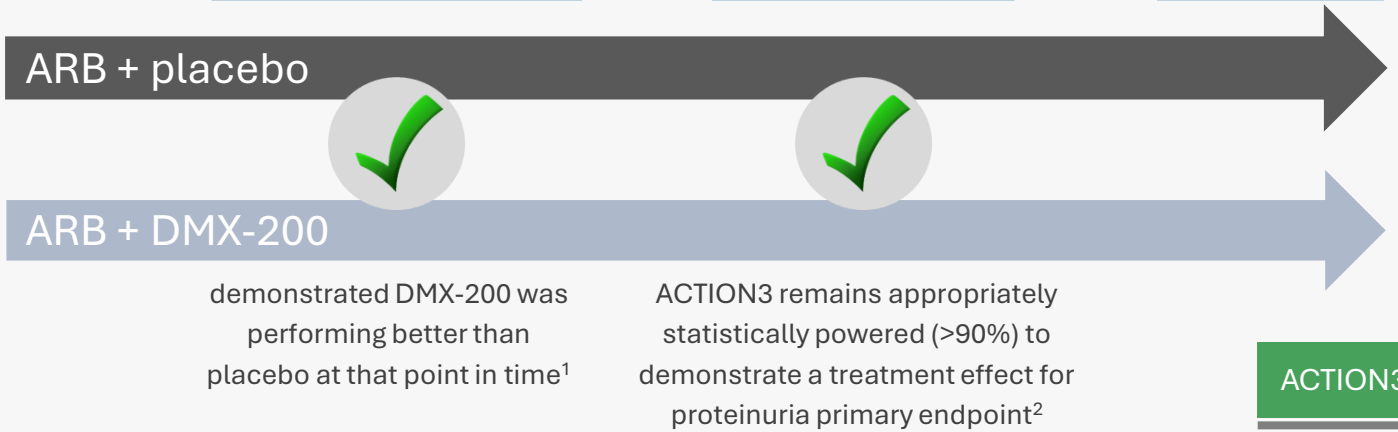
FSGS CLINICAL STUDY

A randomised, double-blind, multi-centre, placebo-controlled study of renal outcomes of DMX-200 in patients with FSGS receiving an ARB (n=≥286)

Background

- Patients recruited, then screened and stabilised on background medications
- Patients randomised to receive drug or placebo
- DXB remains blinded at all times during study

Phase 3 Trial Timeline



Open Label Extension

DMX-200
75/81 (93%)[3] patients enrolled in open label extension study to date

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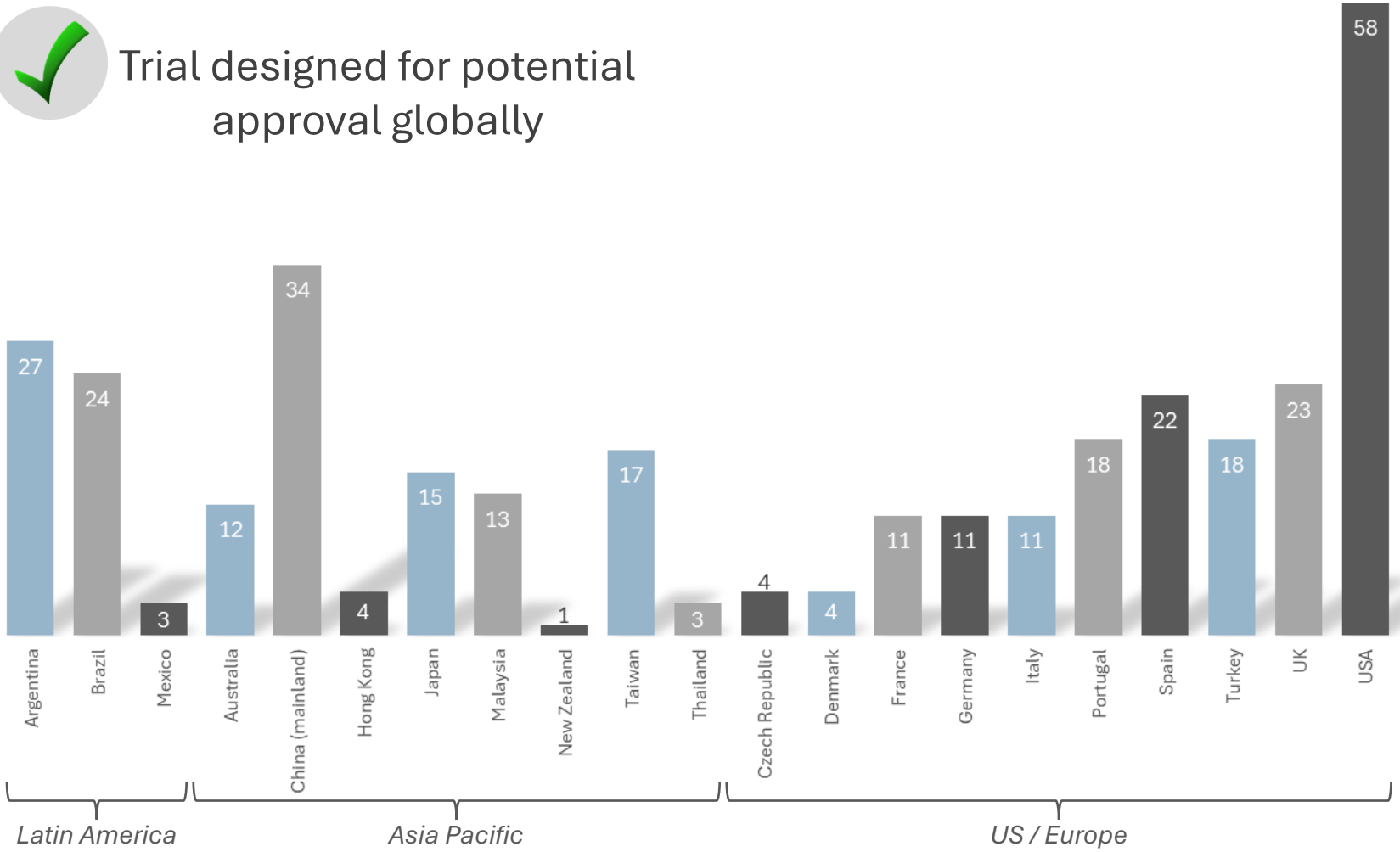
1. ASX release 11 March 2024, Predictive Power statistical model using industry standard as set by the independent renal biostatistician consultant for Dimerix, blinded interim Phase 3 analysis data does not guarantee a statistically significant outcome at the end of the trial; 2. ASX release 28 April 2026; 3. number and % of eligible patients who have completed 2 years treatment and elected to enter the OLE as at 16 June 2026; ARB = angiotensin receptor blocker; uPCR = urinary proteinuria; eGFR = estimated glomerular filtration rate (kidney function);

ACTION3 adult patient recruitment by territory

FSGS CLINICAL STUDY

Trial designed for potential approval globally



Recruitment completed

(adult population)[1]

333

Adult patients recruited, randomised and dosed (target ≥286)[2]

1. ASX release 15 December 2025; 2. Final numbers of adult patients, ASX release 10 March 2026; paediatric patients will continue to recruit, and will not impact final analysis timelines

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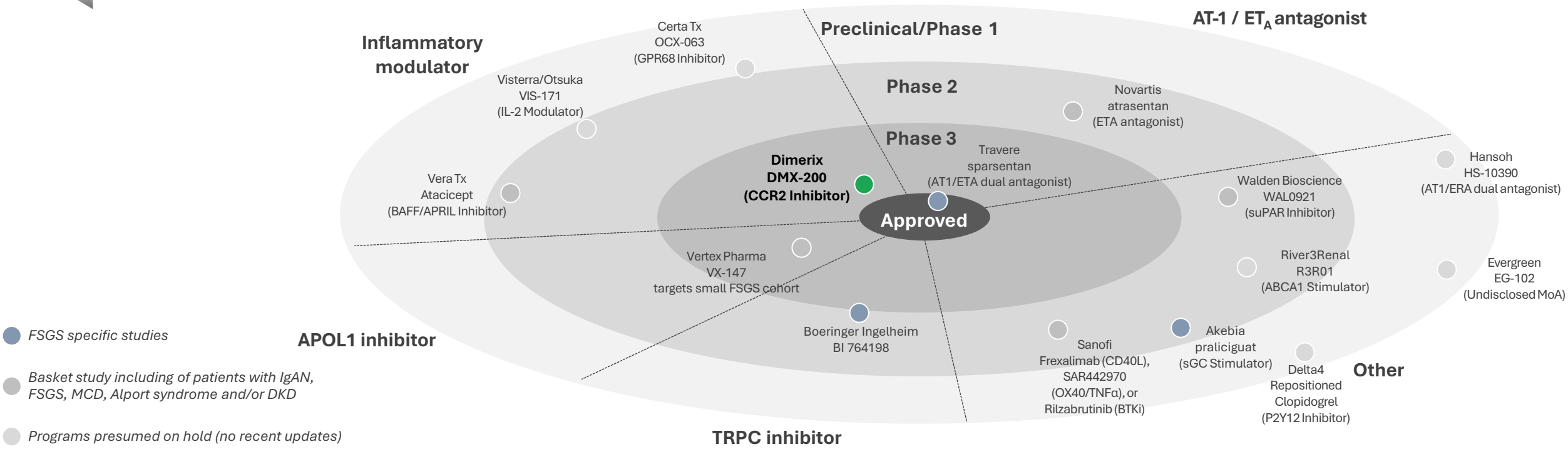
Competitive/complementary trial landscape in FSGS

- Low competition in inflammatory treatment options, large unmet medical need
- DMX-200 is the only inflammatory modulator in development specifically for FSGS
- DMX-200 has potential for use in conjunction with other drugs in development if approved



Source: Company information and clinicaltrials.gov

Summary of licensing deals for DMX-200 to date

Dimerix has successfully partnered DMX-200 across multiple key markets

Licensing deals collectively valued up to

~AU$1.9 billion

in total upfront and potential milestone fees plus royalties[1-5]

>AU$80 million

in total upfront payments[1-5]

	1 BIOMARIN	2 EVEREST MEDICINES	3 ADVANZ PHARMA	4 FUSO	5 taiba
	United States	China,S.Korea.SEA	EU, CA, AU, NZ	Japan	GCC, Iraq
Upfront	AU$48 million[1]	AU$14.1 million[2]	AU$10.8 milllion[3]	AU$7.2 million[4]	AU$0.5 million[5]
Milestones	Up to AU$892 million[1]	Up to AU$467 million[2]	Up to AU$219 million[3]	Up to AU$100 million[4]	Up to AU$120 million[5]
Royalties	Escalating low-teen-low twenties%	Between 10-15%	Escalating mid-teen-20%	Between 15-20%	Starting at 30%

1. Based on US conversions & further terms outlined in ASX Announcement on 01 May 2025; 2. Based on US conversions & further terms outlined in ASX Announcement on 16 June 2026; ; 3. Based on Euro conversions & further terms outlined in ASX Announcement on 5 October 2023; 4. Based on Japanese ¥ Yen conversions & further terms outlined in ASX Announcement on 7 January 2025 ; 5. Based on US dollar conversions & further terms outlined in ASX Announcement on 27 May 2024

DMX-200 - geographic diversification of fee revenue

Collectively, five commercial agreements valued up to **A$1.9 billion** across upfront + milestone fees:[1]

On signing		Short Term Potential		Longer Term Potential			Diversified revenue & risk sharing with licensing partners
Upfront fees		Milestone Payments		Milestone Payments	Royalties		
A$81 million	+	**A$237 million**	+	**A$1.56 billion**	**10 – 30 %**	=	
In licensing fees		Between now and commercial launch		Post commercial launch	Post commercial launch		

*Upfront and development milestones collectively **A$318 million***

Based on ASX releases 05 October 2023, 27 May 2024, 07 January 2025, 01 May 2025 and 16 June 2026

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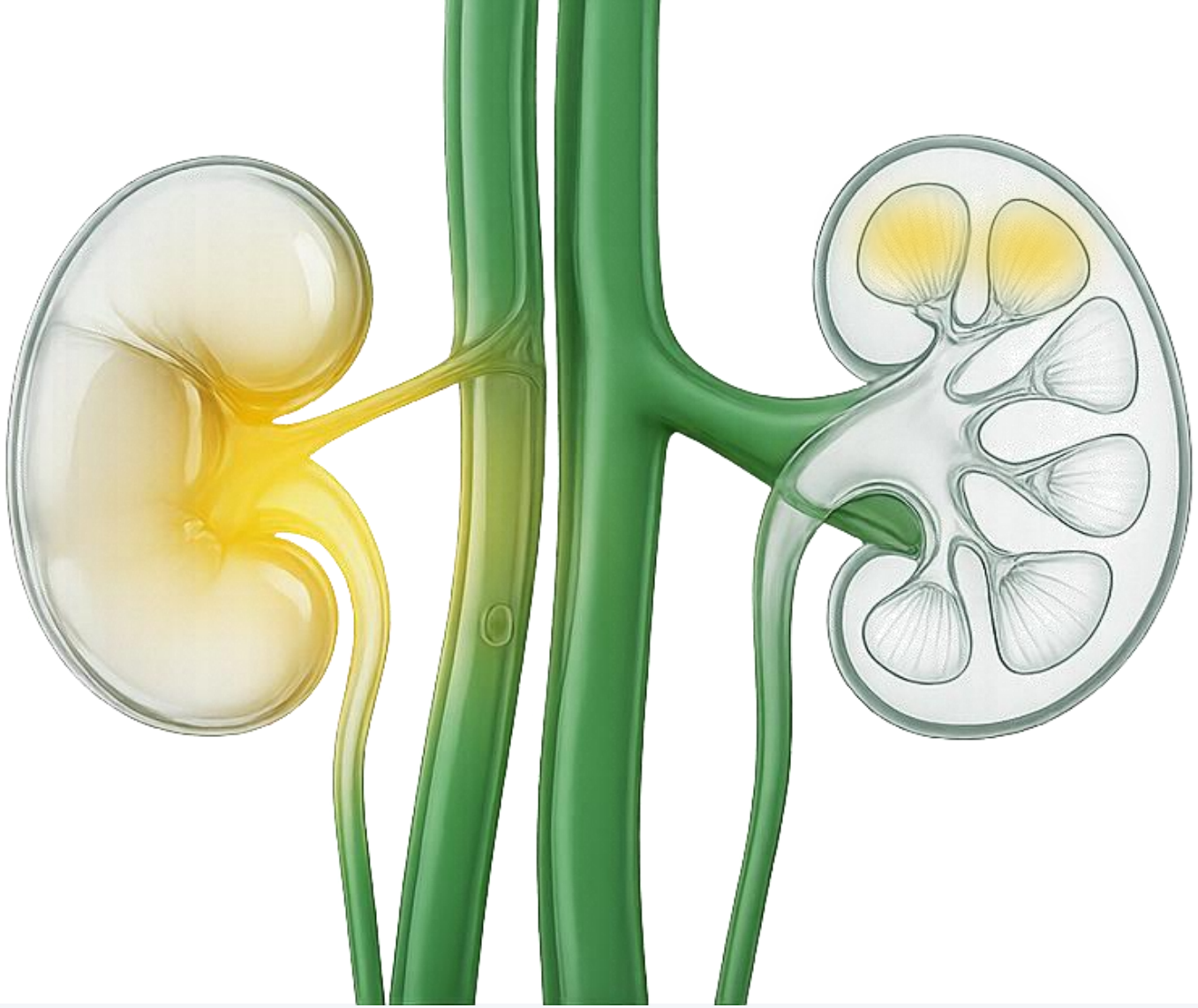
DMX-652 in

Acute Kidney Injury



DMX-652: Phase 2 in acute kidney injury

A complementary, clinical-stage pipeline addition

Large value potential

- Secured a Phase 2 ready asset, which is already FDA cleared & manufactured
- Substantial work to date undertaken by originator means significant early risk already removed[1]

First and best in class

- First-in-class USP30 target that promotes renewal of mitochondria
- DMX-652 targets one of the emerging key drivers of kidney injury and reduces both cellular energy failure and inflammation

Leverages existing expertise

- Dimerix team are experts in kidney disease; capable of identifying high value assets
- Experienced in-house team with proven track record of advancing high value assets through clinical development and into commercialisation

Large and growing market

- No approved therapy for high-risk acute kidney injury patients and growing demand for new therapies[2]
- US$3.5 Billion in 2026
- est. US$7.5 Billion by 2036

Strategic value

- Dimerix has significant optionality over the development and commercialisation direction of DMX-652, as the outright asset owner
- DMX-652 has the potential to be used for a variety of other indications, many of which may also have large markets and unmet medical needs

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1. Dimerix internal analysis; Mission Therapeutics MTX-652 development file; 2. Zarbock et al, Intensive Care Med 2023; Vervoort et al, Ann Thorac Surg 2023; 3. Dimerix internal market analysis consensus figure based on meta-analysis of 21 market reports (2026) ; Includes China and Japan markets, label expansion into sepsis-associated AKI (SA-AKI) and contrast-associated AKI

Introducing DMX-652

A first-in-class, oral, once-daily USP30 inhibitor for the prevention of acute kidney injury in high-risk patients undergoing cardiac surgery

Small molecule

New chemical entity; oral capsule formulation; GMP-grade API available to support Phase 2 clinical trial

Once-daily dosing

Convenient dosing schedule to provide pre-operative and post operative USP30 inhibition

Well tolerated

Phase 1 multi-part study completed in 109 healthy volunteers, (85 volunteers administered DMX-652) no serious adverse events and wide therapeutic margin

Phase 2 ready

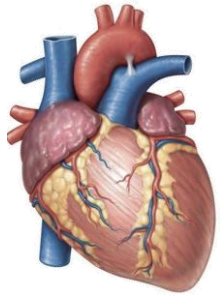
IND open, approved protocol and established global medical advisory board

Key value proposition with encouraging safety profile

What is AKI associated with cardiac surgery

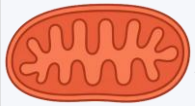
Currently no approved disease-modifying therapies

1 Cardiac Surgery



Cardiopulmonary bypass starves the kidney of oxygen (ischaemia) and causes oxidative stress in kidney cells

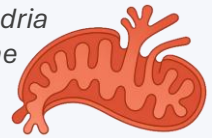
2 Ischaemia



Healthy mitochondria

Kidney starved of oxygen

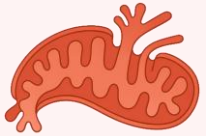
Damaged mitochondria causes rupture of the mitochondrial membrane



~26% of patients undergoing cardiac surgery suffer kidney injury; increases to 30-50% in high-risk patients

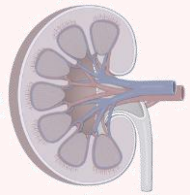
3 Reperfusion: restores blood flow to kidney

Mitochondria rupture



kidney damage caused by a massive influx of reactive oxygen species (ROS) that leads to cell death

AKI



Sudden decline in kidney function (hours)

Drives ICU stays, dialysis, mortality and/or progression to Chronic Kidney Disease

Mitochondria = "batteries" for the kidney cell
The kidney has the second-highest mitochondrial content and oxygen consumption in the body to fuel intense energy demands

Source: Scurt FG et al, Kidney360 2024; Zarbock A et al, Intensive Care Med 2023; KDIGO clinical practice guidelines for AKI

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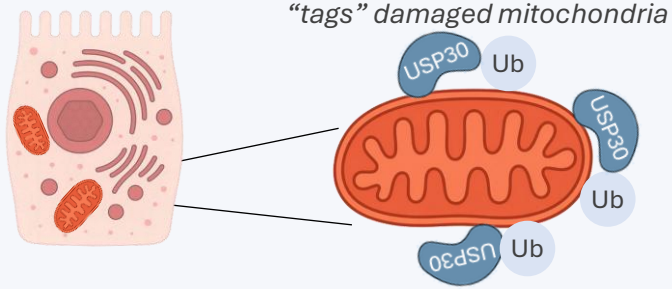
How DMX-652 works – unique, novel mechanism

DMX-652 aims to enable the body's natural quality-control system to remove damaged mitochondria (a process called mitophagy) before they trigger kidney cell death

Healthy kidney

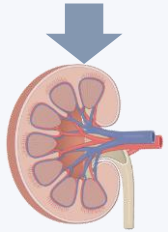
Kidney tubule cell

USP30 regulates Ub which "tags" damaged mitochondria



- Ubiquitin-specific protease 30 (USP30) enzyme expressed on mitochondria
- **USP30 regulates Ubiquitin (Ub)** accumulation to remove damaged mitochondria whilst also preventing premature clearance of healthy mitochondria

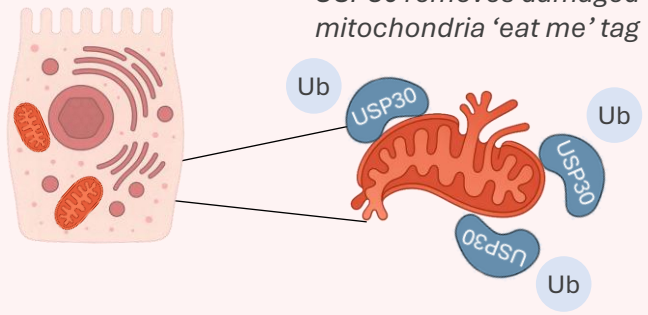
Maintain kidney function



Acute Kidney Injury (AKI)

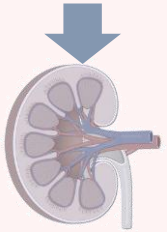
Kidney tubule cell

USP30 removes damaged mitochondria 'eat me' tag



- USP30 opposes Ub tagging, removing the 'eat me' tag
- **Damaged mitochondria are not effectively removed**
- Drives inflammation and cell death

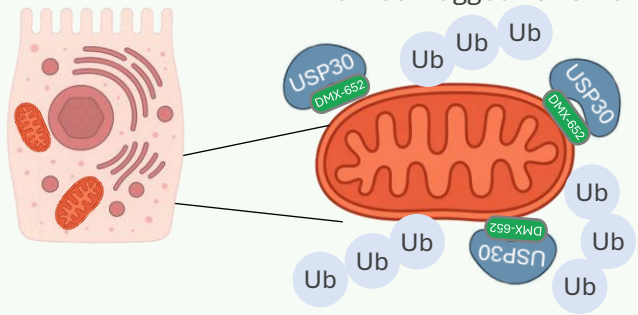
Sudden decline in kidney function (hours)



DMX-652 targets USP30

Kidney tubule cell

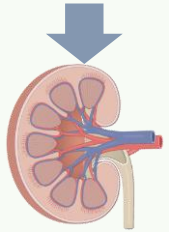
Damaged mitochondria can now be "tagged" & removed



- DMX-652 blocks USP30, enabling normal ubiquitination
- selective degradation of damaged mitochondria, **leaving only healthy mitochondria**

Aims to:

- *Preserve kidney function*
- *Reduced AKI severity and incidence*
- *Prevent dialysis*
- *Shorter ICU and hospital stays*
- *Reduced risk of progression to CKD*



Source: Tang C et al, Nat Rev Nephrol 2021; Bhatia D, Choi ME, J Life Sci 2019; Mission Therapeutics MTX-652 preclinical package (2024)

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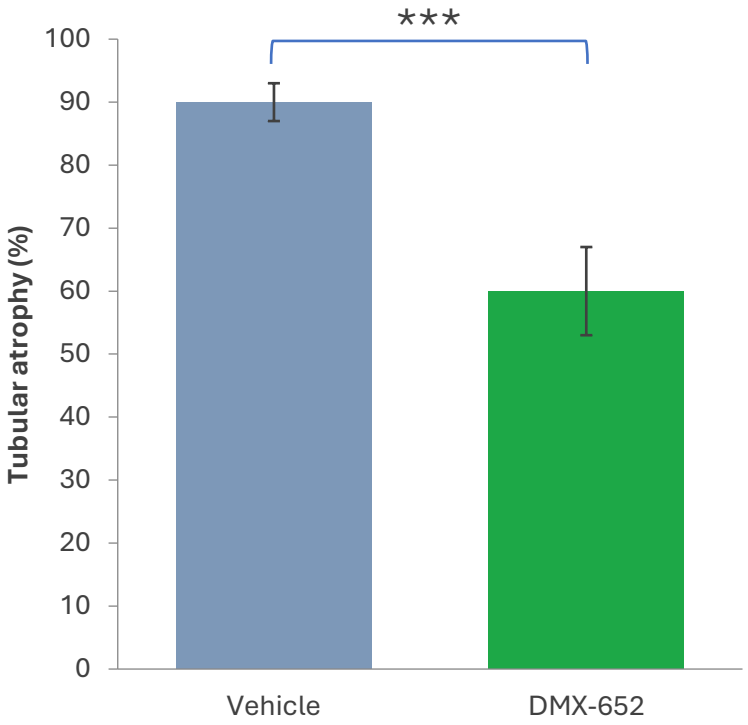
DMX-652 Pre-clinical data demonstrates efficacy signal

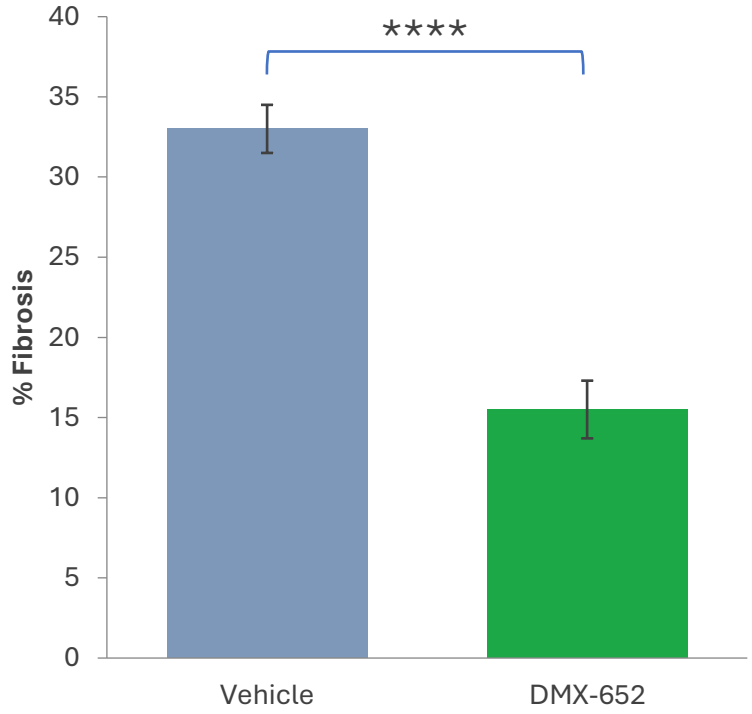
DMX-652 reduces kidney damage

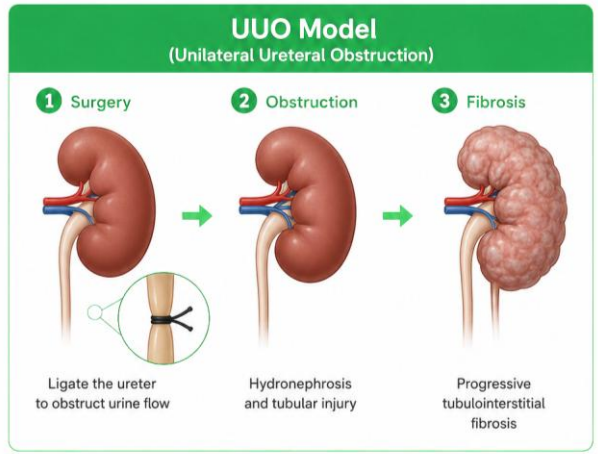
- The uIRI model replicates the processes of cardiac surgery that cause AKI
- DMX-652 significantly reduces the number of kidney tubules damaged when blood is restricted

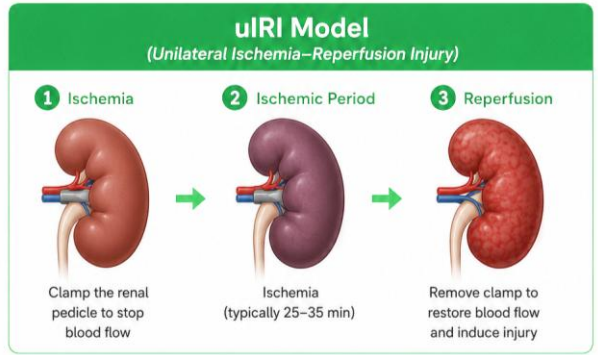


DMX-652 reduces scarring (fibrosis)

- The UUO model predicts acute to chronic kidney disease progression.
- DMX-652 significantly reduces the scarring that drives chronic disease







*uIRI - Unilateral renal ischemia-reperfusion model, UUO - Unilateral ureteral obstruction model. Bars = mean ± error; values, ****<0.0001, ***<0.001. Mission Therapeutics unpublished data*

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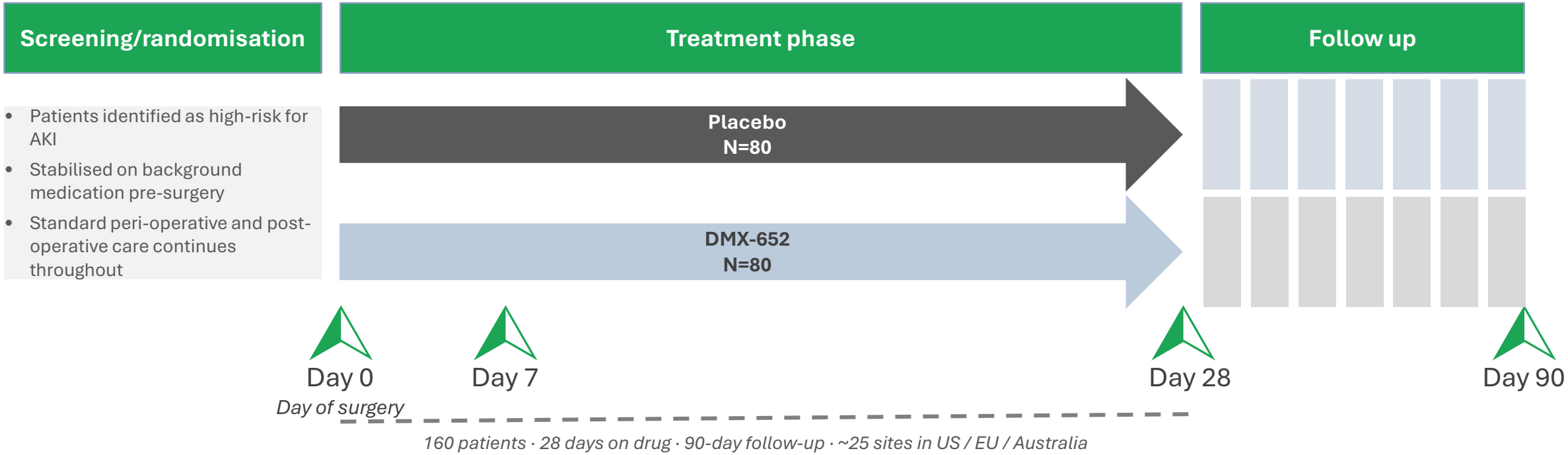
Proposed phase 2 clinical trial design

A multicentre, double-blind, randomised, placebo-controlled study evaluating the safety and efficacy of DMX-652 in patients at high risk of AKI following cardiac surgery (n=160)



Primary endpoint

AKI incidence by 7 days post-surgery

Secondary endpoints

Safety and tolerability

PK

MAKE30

MAKE90

Source: Mission Therapeutics MTX-652 Phase 2 protocol (FDA Study-May-Proceed, 2023); Dimerix MTX-652 development plan (2026); MAKE 30 & MAKE 90 = Major Adverse Kidney Events occurring at 30 or 90 days

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Significant unmet need in AKI

With no approved disease-modifying therapies, severe consequences for patients, surgeons, and health systems

0	1 in 4	30-50%
No approved therapies for the prevention of cardiac surgery-associated AKI	Cardiac surgery patients develop AKI after on-pump surgery[1]	AKI rate in high-risk patients *(e.g. age, diabetes, low cardiac function, prior kidney disease)*[1]

Consequences of CS-AKI[2]:

- Prolonged intensive care and hospital length of stay
- Higher rates of renal replacement therapy (dialysis)
- Increased short- and long-term mortality
- Substantially elevated risk of progression to chronic kidney disease and end-stage renal disease

"CS-AKI is a predictable, frequent and devastating complication of cardiac surgery. Effective preventive therapies are a critical unmet need."
Consensus position, 2024 ADQI Conference

1. Alshaikh HN et al, Ann Thorac Surg 2018; Zarbock A et al, Intensive Care Med 2023; 2. Scurt FG et al, Kidney360 2024; Lau D et al, J Thorac Cardiovasc Surg 2021

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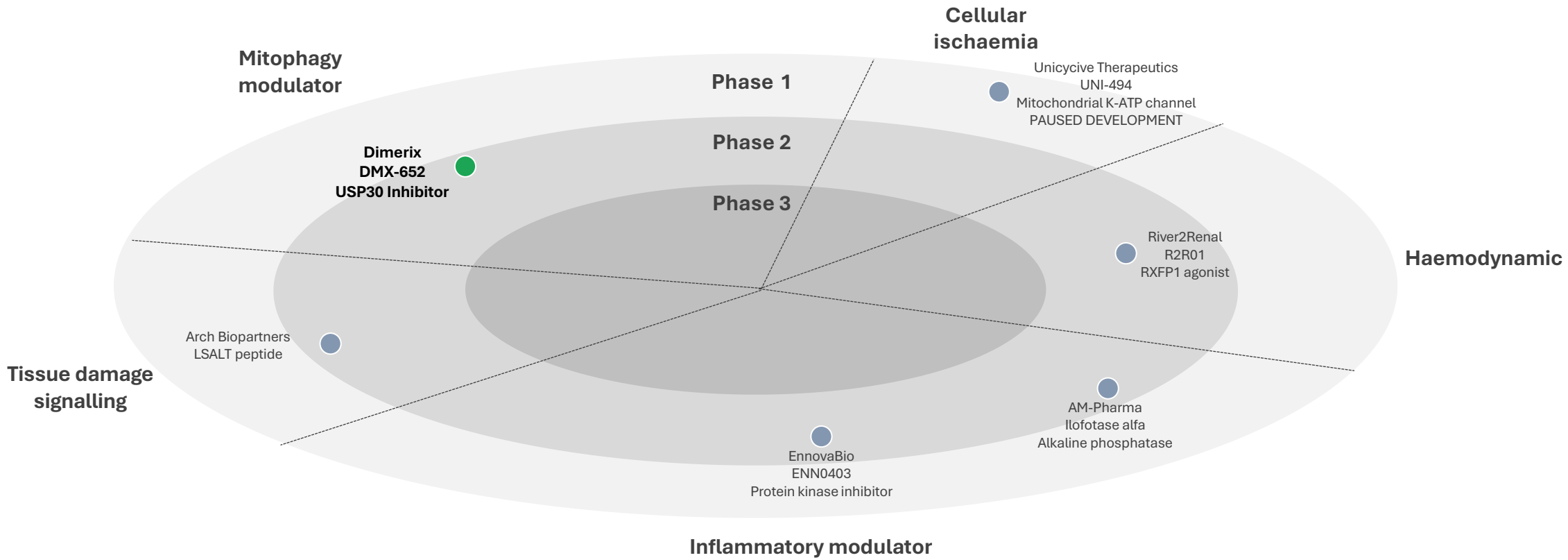
Competitive/complementary trial landscape in AKI

DMX-652's differentiated mechanism of action supports complementary use and the potential for superior or additive outcomes



Source: Company information and clinicaltrials.gov

DMX-200 and DMX-652: complementary growth pillars

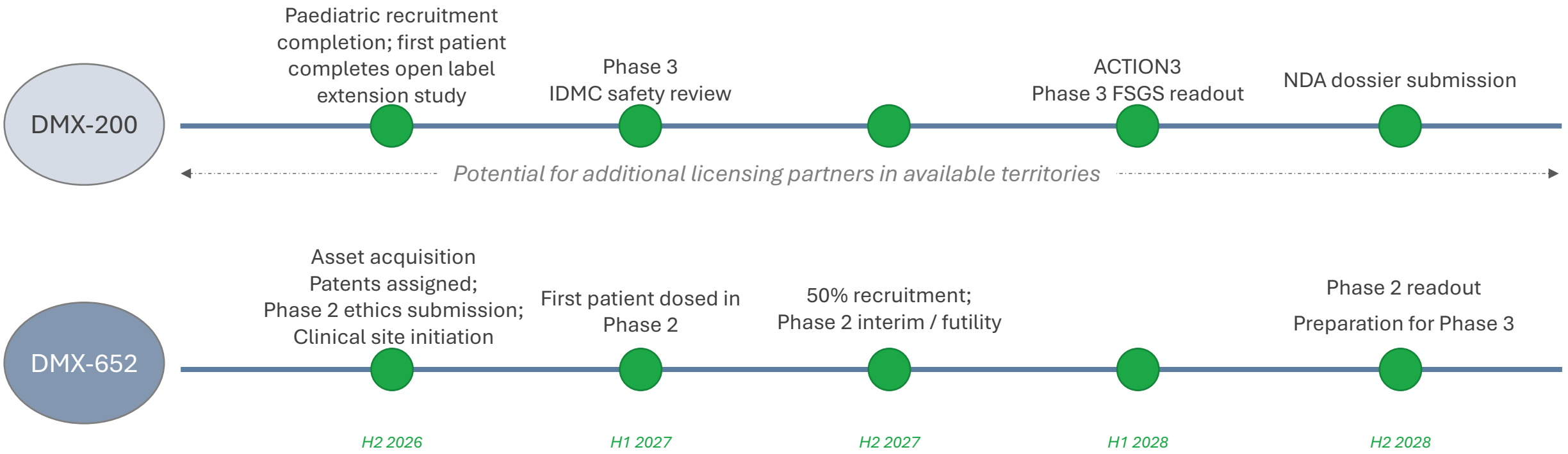
Strategic fit: shared renal capabilities, shared regulatory/clinical infrastructure, shared small-molecule know-how

DMX-200		DMX-652
Focal Segmental Glomerulosclerosis (FSGS) rare **kidney** disease	1 **Shared kidney disease focus**	1 Acute **kidney** injury associated with cardiac surgery
CCR2 inhibitor that modulates inflammatory pathways involved in kidney damage	2 **Different Mechanisms of Action**	2 **USP30 inhibitor** designed to enhance mitophagy and mitochondrial quality control in damaged cells
existing in-house infrastructure and global network	3 **Shared Development Infrastructure**	3 Can **leverage existing** expertise in clinical, regulatory, manufacturing, nephrology networks, know-how, and commercial relationships
more advanced, **near-commercialisation** asset with Phase 3 fully recruited and commercial partnering activity	4 **Balanced Risk and Timing**	4 provides a **new growth engine**, with an open IND, FDA clearance to proceed to Phase 2, and completed Phase 1 safety studies

1. ASX release 10 March 2026; 2. ASX releases March 2024 and April 2026; 3. ASX releases 05 October 2023, 27 May 2024, 07 January 2025; 01 May 2025 and 16 June 2026; 4. ASX release 17 July 2026

Key potential value inflection points

Disciplined, stage-gated capital deployment focused on the clinical value inflection[1]



Two clinically differentiated assets in renal disease, addressing two separate high unmet need indications with independent timelines; building a steady cadence of value-creating milestones

1. Based on current anticipated activities timeline

Corporate overview

Ticker Symbol	ASX: DXB
Cash Balance (Jun26)*	$16.2 million
Market Capitalisation[1]	$174 million
Share price[1]	$0.29
Total ordinary shares on issue[1]	600,396,776
Average Daily Liquidity by value for past 90 trading days[2]	$0.85 million

**excluding ~$48.1million:*

- *AU$14.1 million Everest upfront payment received; [3] and*
- *$34 million available loan facility [4]*

SHARE PRICE

SUBSTANTIAL SHAREHOLDERS[3]

Position	Holder Name	Holding	% IC
1	Mr P Meurs	91,090,374	15.3%
TOTAL (TOP 5) Shareholders		**155,204,214**	**25.9%**

1. As at 3 September 2026; 2. Past 90 trading days liquidity as at 5 August 2026; 3. Shareholder register as at 3 September 2026; 3. ASX release 28 August 2026; 4. ASX release 3 September 2026

Dimerix

(ASX:DXB)

A biopharmaceutical company developing innovative new therapies in areas with unmet medical needs, with a core focus on kidney diseases.

SCAN ME

WELL POSITIONED **TO DELIVER** AGAINST STRATEGIC PLAN

ESG Statement

Dimerix is committed to integrating Environmental, Social and Governance (ESG) considerations across the development cycle of its programs, processes and decision making. The Dimerix commitment to improve its ESG performance demonstrate a strong, well-informed management attitude and a values led culture that is both alert and responsive to the challenges and opportunities of doing business responsibly and sustainably.

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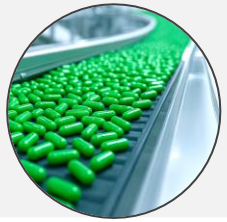
DMX-200 – inflammatory modulator

A CCR2 inhibitor working synergistically alongside the current standard of care (AT1R blocker): G protein-coupled receptor (GPCR)

Commercial manufacturing sites established in USA[3]	1 x 120mg capsule BID	Well tolerated with no safety concerns to date (>400 patients dosed)[3]	4 clinical studies completed to date: positive safety and efficacy signals across studies[3]
			
Small molecule	**Easy & convenient dosing**	**No safety concerns noted[3]**	**Positive efficacy signals[3]**

1. ASX release and investor presentation 29Jul20 and ASX release 11Mar24 BID: bis in die/ twice a day

DMX-200: unique pharmacology

- CCR2 activation promotes recruitment of inflammatory monocytes to the kidney

  DMX-200 **inhibits** CCR2[1]

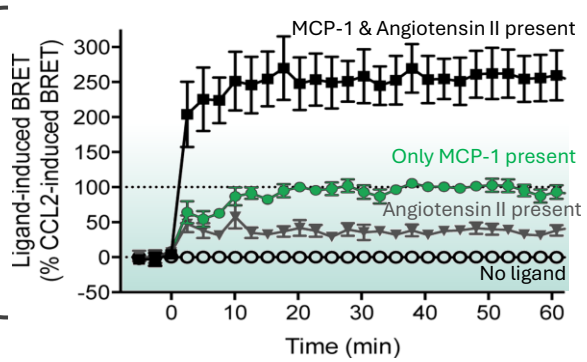
- Monocytes promote sclerosis and fibrosis of the kidney

  DMX-200 **reduces** inflammatory cells[1,2,3]

- Podocytes are the essential filter cells of the kidney

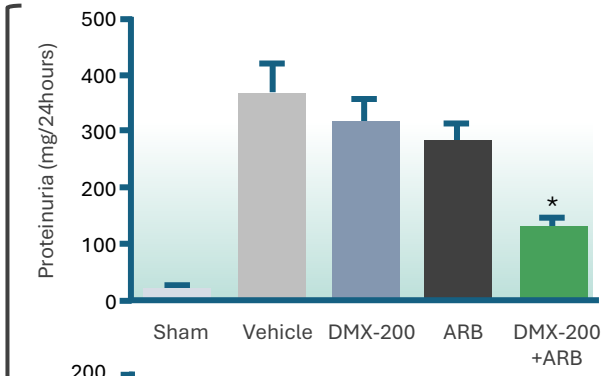
  DMX-200 **preserves** podocytes[1]

in-vitro

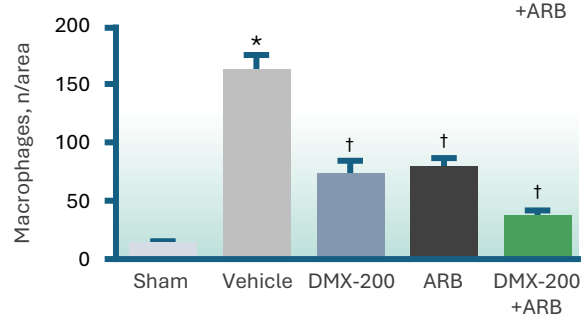


Complex of CCR2 and AT1R increases aberrant signaling when both receptors activated[1]

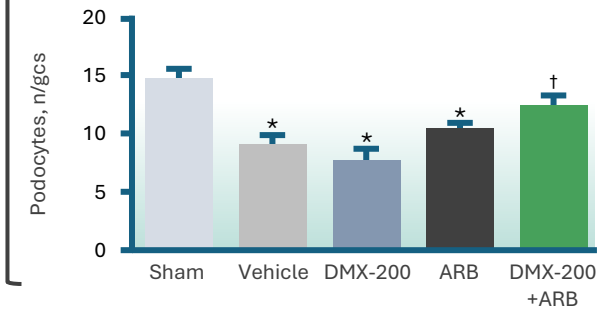
in-vivo[1]



Simultaneous inhibition of CCR2 and AT1R reduces proteinuria an important early marker of kidney function[1]



Simultaneous inhibition of CCR2 and AT1R reduces recruitment of monocytes to the kidney[1]



Simultaneous inhibition of CCR2 and AT1R preserves the number of essential filter cells (podocytes) in the kidney[1]

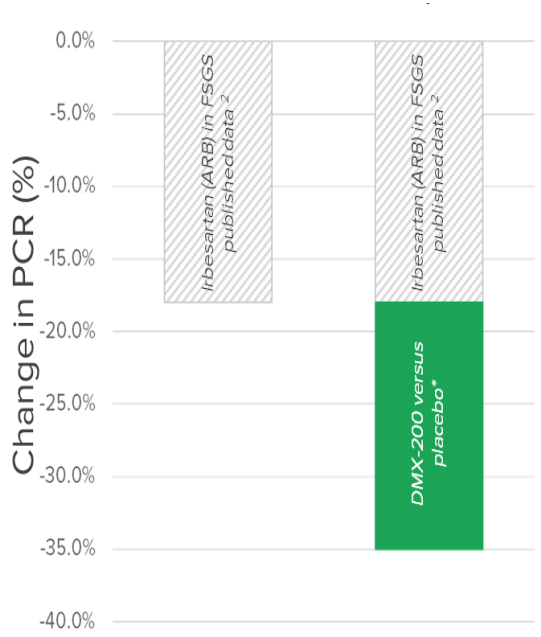
Dimerix

1. Ayoub MA, et al. (2015) PLoS One; doi.org/10:e0119803; 2. ASX presentation 27 October 2020; 3. Liu Y. et al (2024) Role of MCP-1 as an inflammatory biomarker in nephropathy, Front. Immunol., Sec. Inflammation doi.org/10.3389/fimmu.2023.1303076; CCR2: C-C chemokine receptor type 2; AT1R: Angiotensin II type 1 receptor; MCP1: Monocyte chemoattractant protein-1 (also known as CCL2, the ligand for CCR2); BRET: Bioluminescence Resonance Energy Transfer Assay; PCR = protein creatinine ratio; *P<0.05 vs sham STNx rats ; P<0.05 vs vehicle STNx rats;

DMX-200: Phase 2 met primary endpoint

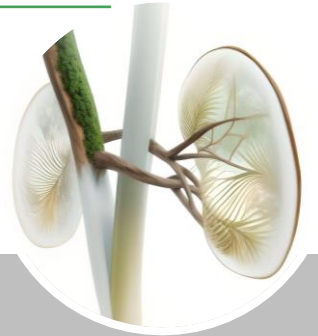
Clinically encouraging outcomes achieved for patients,[1,2] with no safety concerns noted[3]

Average reduction of **17%** in proteinuria after 16 weeks treatment on DMX-200 versus placebo[3]



"Any reduction in proteinuria could yield years of preserved native kidney function and delay the onset of kidney failure and its attendant morbidity and mortality"

Kidney survival study – Troost et al, August 2020[2]



EFFICACY

- 86% of patients demonstrated **reduced proteinuria** when administered DMX-200 compared to when administered placebo
- DMX-200 reduced inflammatory biomarker by **39%** vs placebo

SAFETY

- No safety concerns noted – reduced development risk

PCR = protein creatinine ratio; ARB = angiotensin receptor blocker; 1. Trachtman, et al., 2018. J Amer Soc Nephrology 29(11):2745-2754; 2. Troost JP et al (August 2020); doi.org/10.1053/j.ajkd.2020.04.014; 3. Repeated measures mixed model analysis per protocol; top line data was reported as grouped analysis on 29 July 2020, study not designed for statistical significance;

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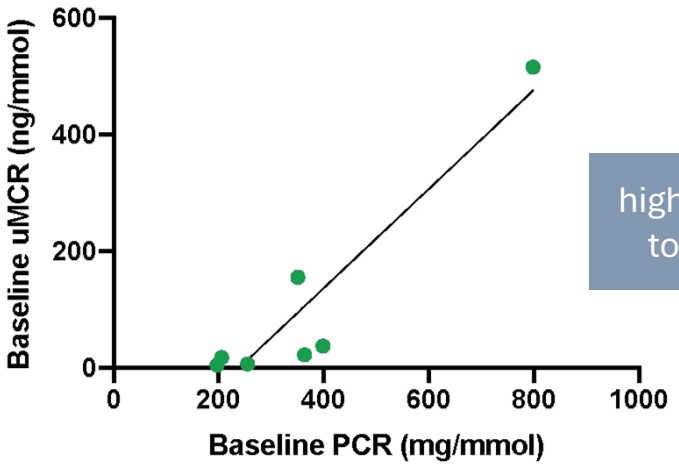
DMX-200 Phase 2 effect on inflammatory biomarker[1]

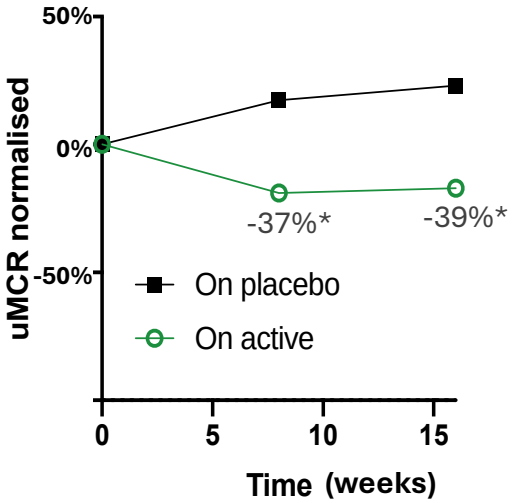
Unlike other CCR2 antagonists investigated to date, treatment with DMX-200 reduces the urine concentration of the pro-inflammatory ligand of CCR2 called MCP-1[2]

Average baseline MCP-1 versus average baseline proteinuria



high MCP-1 correlates to high proteinuria

Change in MCP-1 over time on DMX-200 versus placebo



MCP-1 levels reduced when on DMX- 200 treatment

- **16 weeks treatment with DMX-200 vs placebo reduced inflammatory biomarker by 39%:**
  - DMX-200 blocks receptor responsible for inflammation
  - Translates to reduced inflammation and subsequent fibrosis (scarring) in the kidney[2]

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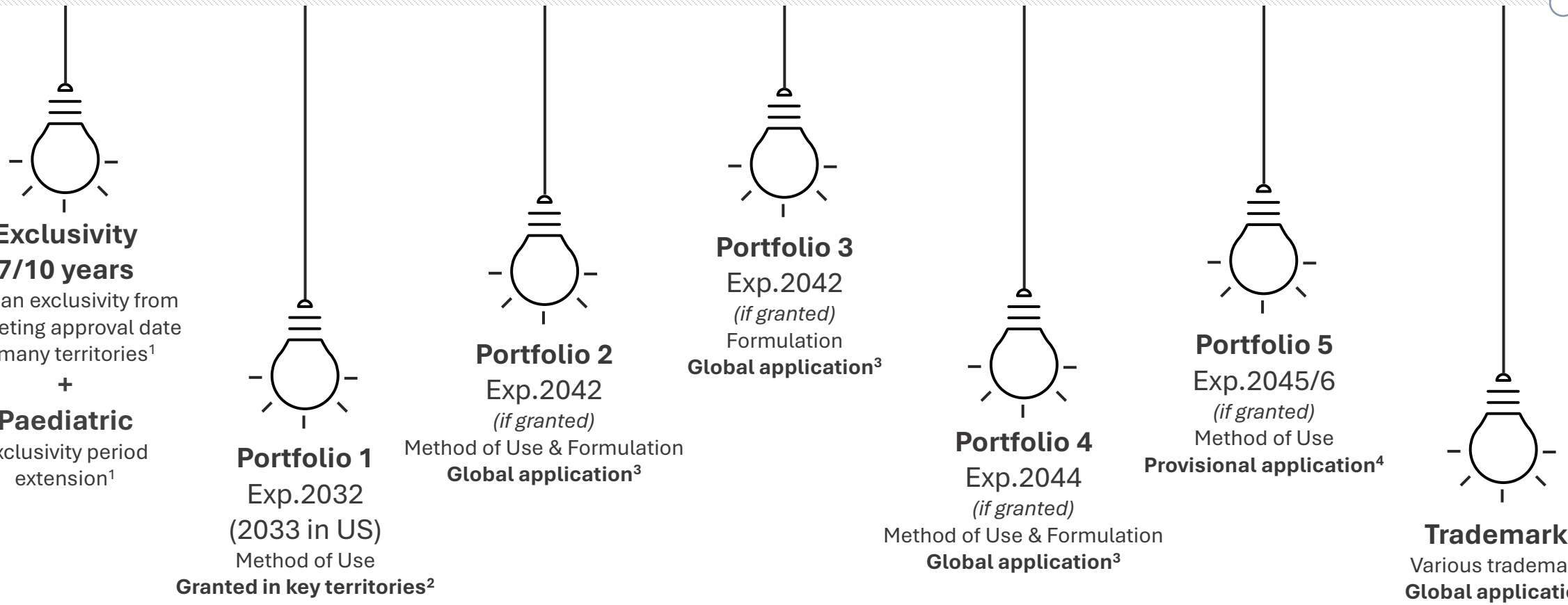
*uMCR = Urinary MCP-1 creatine ratio; PCR = protein creatinine ratio; *placebo adjusted difference*

1. ASX presentation 27 October 2020; 2. Liu Y. et al (2024) Role of MCP-1 as an inflammatory biomarker in nephropathy, Front. Immunol., Sec. Inflammation doi.org/10.3389/fimmu.2023.1303076

DMX-200 intellectual property portfolio

DMX-200



Exclusivity
7/10 years
Orphan exclusivity from marketing approval date in many territories[1]
+
Paediatric
exclusivity period extension[1]

Portfolio 1
Exp.2032
(2033 in US)
Method of Use
Granted in key territories[2]

Portfolio 2
Exp.2042
(if granted)
Method of Use & Formulation
Global application[3]

Portfolio 3
Exp.2042
(if granted)
Formulation
Global application[3]

Portfolio 4
Exp.2044
(if granted)
Method of Use & Formulation
Global application[3]

Portfolio 5
Exp.2045/6
(if granted)
Method of Use
Provisional application[4]

Trademarks
Various trademarks
Global applications

1. DMX-200 is a "New Chemical Entity: an active moiety not previously approved, which can attract exclusivity periods in various territories; 2. Granted patents US9,314,450, US10,058,555, US10,525,038, US11,382,896, US12,083,102, CN2012800046165, CA2,821,985, EP12734251.7, HK 1404477.8, IL227414, JP2013-547780, SA2013/5897, AU2012206945; 3. Patent applications: PCT/AU2022/050013, PCT/AU2022/050249 and PCT/AU2024/050416; 4. US Provisional Application 63/887,984

Dimerix

DMX-652 Phase 1 study met primary endpoint

Completed in healthy volunteers: strong safety & pharmacokinetic foundation to support Phase 2 clinical trial

85	0	Up to 200mg	100mg
Healthy volunteers received DMX-652; across 5 study sites	No serious adverse events (SAE) reported in any cohort	Single dose well tolerated; maximum tolerated dose not reached	Once daily x 14 days; multiple ascending dose; well tolerated

Safety

- No serious adverse events
- Almost all TEAEs of mild severity
- No ECG signal: no QT/QTc prolongation
- Well tolerated in elderly subjects
- Wide therapeutic margin in non-clinical tox studies

Pharmacokinetics & Drug/Drug Interactions (DDI)

- Rapid oral absorption; no effect of food or formulation
- Once-daily dosing profile supported
- Clean DDI profile: not a CYP450 inducer/inhibitor
- Modelled 100mg loading / 50mg daily achieves ~80% target occupancy
- Phase 2 dose selected using validated PK modelling

Source: Mission Therapeutics MTX-652 Phase 1 Clinical Study Report (MTX652_001), 2024; FDA Pre-IND meeting minutes

DMX-652: first-in-class, best-in-class potential

Unlike competing programs targeting specific inflammatory or fibrotic pathways, DMX-652 acts on a downstream, convergent driver of kidney injury — making it applicable across causes of AKI

1

Only USP30 inhibitor in clinical development for CS-AKI

No other mitophagy modulators are in clinical development for cardiac surgery-associated AKI

2

Damage-sensing mechanism

USP30 inhibition selectively amplifies mitophagy only where mitochondria are already damaged — avoiding the indiscriminate effects of broader pathway activators

3

Targets a ‘cause-neutral’ pathway

Mitochondrial dysfunction drives AKI regardless of cause — making DMX-652 mechanistically broad and complementary to existing care

4

Expansion potential beyond CS-AKI

Same mitophagy mechanism applicable to a large range of other diseases, including other AKI causes, kidney transplant, cardiac disease, and lung disease

Tang C et al, Nat Rev Nephrol 2021; Dimerix competitive landscape analysis (2026); clinicaltrials.gov searches; CS-AKI = cardiac surgery related acute kidney injury

DMX-652 - significant unmet need in AKI

With no approved disease-modifying therapies, severe consequences for patients, surgeons, and health systems

0	1 in 4	30-50%
No approved therapies for the prevention of cardiac surgery-associated AKI	Cardiac surgery patients develop AKI after on-pump surgery[1]	AKI rate in high-risk patients *(e.g. age, diabetes, low cardiac function, prior kidney disease)*[1]

Consequences of CS-AKI[2]:

- Prolonged intensive care and hospital length of stay
- Higher rates of renal replacement therapy (dialysis)
- Increased short- and long-term mortality
- Substantially elevated risk of progression to chronic kidney disease and end-stage renal disease

"CS-AKI is a predictable, frequent and devastating complication of cardiac surgery. Effective preventive therapies are a critical unmet need."
Consensus position, 2024 ADQI Conference

1. Alshaikh HN et al, Ann Thorac Surg 2018; Zarbock A et al, Intensive Care Med 2023; 2. Scurt FG et al, Kidney360 2024; Lau D et al, J Thorac Cardiovasc Surg 2021

Dimerix

DMX-652 Intellectual property

*The acquisition secures **owned composition-of-matter IP**, plus exclusive freedom-to-operate rights and regulatory exclusivity*

ACQUIRED · ASSIGNED TO DIMERIX

Composition-of-matter patent family

Directly covers DMX-652 (originally MTX-652) — a novel USP30-inhibitor compound; this is the core, 100% owned patent underpinning the asset

Granted in initial territories (incl. Japan) · US Notice of Allowance received

2041

Anticipated patent expiry

19

Territories filed worldwide

US · EU · Japan · China

LICENSED · EXCLUSIVE RIGHTS

Freedom-to-operate patents

An exclusive licence to specific USP30-inhibitor patent families secures DMX-652's freedom to operate — granted broadly across the US, Europe and Asia.

Licensed patents: PCT/GB2016/050851 · PCT/GB2019/050608

REGULATORY EXCLUSIVITY

New Chemical Entity exclusivity

≥5 yrs

US data exclusivity (NCE), from marketing approval

up to 7 / 10 yrs

US / EU, with orphan drug designation

Dimerix

Acquired (assigned to Dimerix): composition-of-matter family PCT/EP2021/064897, anticipated expiry 2041. Licensed (exclusive, freedom to operate): PCT/GB2016/050851 & PCT/GB2019/050608. Regulatory exclusivity anticipated on marketing approval; orphan drug designation not yet granted. DMX-652 was previously known as MTX-652 at Mission Therapeutics

Dimerix board

Mark Diamond
BSc, MBA
Non-Executive Chairman

Previous experience:

percheron THERAPEUTICS · Pfizer (Faulding)[1]

- Senior pharmaceutical executive with a demonstrated record of achievement and leadership over more than 30 years within the pharmaceutical and biotechnology industries
- Significant accomplishments in capital raising initiatives, pipeline development and licensing
  - ✓ BSc – Chemistry
  - ✓ MBA – Business

Nina Webster
PhD, MBA, M.IP.Law
CEO & Managing Director

Previous experience:

Wyeth[2] · ACRUX · immuron

- Experienced in product development, commercial strategy development & execution
- Successfully commercialized pharmaceutical products globally
  - ✓ BSc (Hons) – Pharmacology
  - ✓ PhD – Pharmaceutics
  - ✓ MBA – Business
  - ✓ M.IP.Law – Intellectual Property Law

Hugh Alsop
BSc (Hons), MBA
Non-Executive Director

Previous experience:

mayne pharma[1] · ACRUX · HatchTech · KINOXIS

- Extensive biotech drug development & commercial manufacturing experience
- Responsible for successful global commercialization programs & NDA registrations
  - ✓ BSc (Hons) – Chemistry
  - ✓ MBA – Business

Sonia Poli
PhD
Non-Executive Director

Previous experience:

Roche · addex · AC Immune · minoryx

- Experienced executive in pharmaceutical operations
- Background in small molecules development and analytical development
  - ✓ BSc (Hons) – Chemistry
  - ✓ PhD – Industrial Chemistry

Clinton Snow
BEng (Hons), BCom
Non-Executive Director

Previous experience:

Woodside Energy · PolyActiva · icetana[AI]

- Experienced technology and governance professional with a focus in operations, risk management, assurance, and AI
- Provides advisory services to a family office with multiple Australian biotech investments
  - ✓ BEng (Hons) – Chemical Engineering
  - ✓ BCom – Commerce

1. Acquired by Pfizer in 2015; 2. Acquired by Pfizer in 2009

Dimerix management

Nina Webster
PhD, MBA, M.IP.Law
CEO & Managing Director

Previous experience:

Wyeth[1] ACRUX immuron

- Experienced in product development, commercial strategy development & execution
- Successfully commercialised multiple pharmaceutical products
  - ✓ BSc (Hons) – Pharmacology
  - ✓ PhD – Pharmaceutics
  - ✓ MBA – Business
  - ✓ M.IP.Law – Intellectual Property Law

Mike Tonroe
BSc (Hons) FCA, MAICD
CFO & Company Secretary

Previous experience:

IMUGENE OPTHEA Genetic Technologies

- Experienced finance and governance executive with extensive experience of both ASX and NASDAQ-listed companies.
- Brings more than 30 years' international finance leadership experience across Australia, US, Canada, the UK and Hong Kong.
  - ✓ BSc (Hons) – Business Studies
  - ✓ MAICD
  - ✓ Chartered Accountant

David Fuller
B. Pharm (Hons), MBBS
CMO

Previous experience:

RACE ONCOLOGY Syneos Health genzyme

- 35 years international experience in drug development, commercialization and corporate leadership
- Planning, Financing, Pre-clinical, Clinical Development, Regulatory Approval, Product Launch, Pharmacovigilance, and Medical Affairs
  - ✓ B.Pharm (Hons) - Pharmacy
  - ✓ MBBS - Medicine and Surgery

Robert Shepherd
PhD, MBA,
COO

Previous experience:

MONASH University Medicines Development for Global Health

- Experienced pharmaceutical executive in project management, clinical development and research translation
- BD and strategic alliance leader
- Led multidisciplinary R&D&C teams for 13 years
  - ✓ BSc (Hons) – Genetics
  - ✓ PhD – Molecular Immunology
  - ✓ MBA – Business & Leadership

1. Acquired by Pfizer in 2009;

Medical Advisory Board

Professor
Hiddo Heerspink
PhD

Professor of Clinical Trials and Personalized Medicine: University Medical Center Groningen, the Netherlands. He specializes in the research of novel treatment approaches to slow the onset of diabetic cardiovascular and renal disease. Hiddo has been instrumental in interactions between industry, researchers and regulatory agencies in the validation of surrogate endpoints for renal trials.

Professor
Alessia Fornoni
MD, PhD, FASN

Professor of Medicine & Molecular & Cellular Pharmacology: University of Miami. Chief of the Katz Family Division of Nephrology and Hypertension. She has an extensive history of translational excellence for patients with renal disease and has uncovered novel pathogenetic mechanisms and therapeutic approaches for glomerular disorders.

Professor
Jonathan Barratt
MD, PhD, FRCP

Mayer Professor of Renal Medicine: Department of Cardiovascular Sciences; University of Leicester and Nephrologist. Jonathan is the IgA nephropathy Rare Disease Group lead for the UK National Registry of Rare Kidney Diseases (RaDaR) and a member of the steering committee for the International IgA Nephropathy Network.

Associate Professor
Lesley Inker
MD, MS, FRCPC

An attending physician and Director of the Kidney and Blood Pressure Center in the Division of Nephrology at Tufts Medical Center. Lesley's major research interest is in the estimation and measurement of glomerular filtration rate (GFR) and in defining alternative endpoints for CKD progression trials based on GFR decline and changes in albuminuria.

Dr Muh Geot Wong
MBBS, PhD, FRCP

Renal Physician and Head of the Renal Clinical trials at the Royal North Shore hospital, Sydney, Australia. Muh Geot's main areas of research are in understanding the mechanisms of kidney fibrosis, biomarkers research, and identifying strategies in delaying progressive kidney disease including glomerular diseases.

Professor
Howard Trachtman
MD, FASN

Graduated from Haverford College and the University of Pennsylvania School of Medicine. He has been a practicing pediatric nephrologist for 35 years. Has been the PI of NIDDK and industry sponsored clinical trials in glomerular disease and am a Co-Investigator in the NEPTUNE and CureGN observational cohort studies.

Associate Professor
Laura Mariani
MD, MSCE

Assistant Professor in the Division of Nephrology at the University of Michigan. Interest in observational studies in glomerular disease, including NEPTUNE and CureGN. Lead on PARASOL program to define FSGS endpoints with by applying statistical methods for clinical outcome definition and prediction of kidney disease progression.