Novel therapies targeting kidney disease

Non-confidential summary

May 2025

GUARD THERAPEUTICS

PIONEERING TRANSFORMATIVE MEDICINES FOR KIDNEY DISEASE

RMC-035 for **kidney protection** in open-heart surgery

- **Phase 2b *POINTER*** study ongoing – **> enrolment to be completed in June, results expected year-end 2025**
- **Granted FDA Fast Track Designation**; eligible for Breakthrough Therapy Designation
- **Clinical proof-of-concept established in Phase 2a AKITA study with 177 patients**
  - **59% reduction vs placebo (MAKE, regulatory endpoint)**
- **First-to-market potential**; >USD 1 billion market – no approved therapies

Additional **opportunities** with RMC-035 & GTX peptides

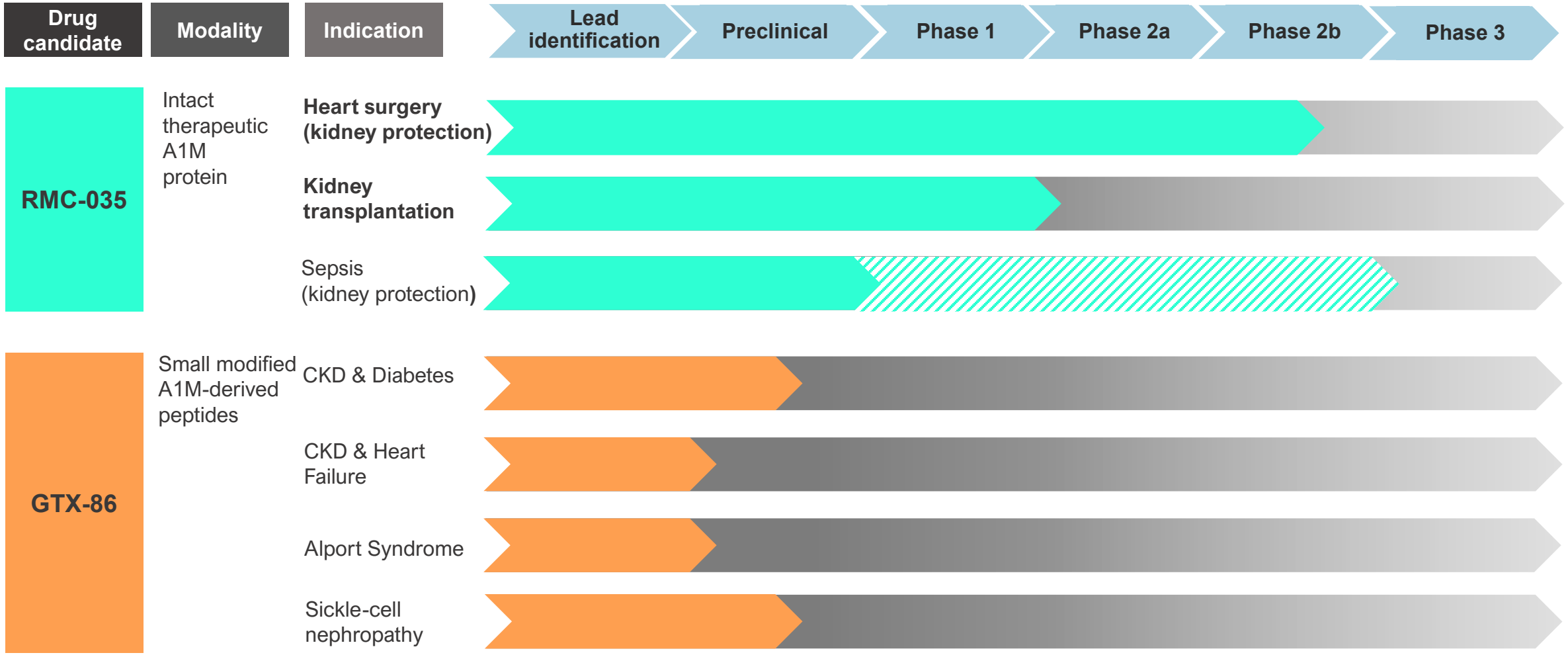
- **Phase 3 ready sepsis program** and **Phase 2a/b ready kidney transplantation program** for >USD 5.6 billion market
- **Unique positioning of preclinical GTX peptides** in chronic kidney disease for >USD 8 billion market
- Listed in Stockholm with top shareholders including Industrifonden and Swedbank Robur [Nasdaq FN Growth Market: GUARD]

GUARD THERAPEUTICS

EXPERIENCED MANAGEMENT TEAM WITH STRONG & PROVEN TRACK RECORD IN DRUG DEVELOPMENT

NAME / POSITION	EXPERIENCE		NAME / POSITION	EXPERIENCE	
 **TOBIAS L. AGERVALD** MD, PhD, CEO	**+10** years in industry	astellas	 **KARIN BOTHA** MSc, CFO	**+20** years in industry	NOVARTIS, SmithKline Beecham, GlaxoSmithKline
 **MICHAEL REUSCH** MD, CMO	**+30** years in industry	Fujisawa, astellas	 **PETER GILMOUR** MSc, PhD, CSO/Head of Preclinical	**+20** years in industry	AstraZeneca, astellas
 **TORBJÖRN LARSSON** BSc, Head of CMC	**+30** years in industry	Medivir, aprea therapeutics, Pharmacia &Upjohn	 **SARA THURESSON** MSc, Head of Clinical Operations	**+15** years in industry	oncopeptides, IQVIA, WILSON THERAPEUTICS, Medivir

DIFFERENTIATED PIPELINE BASED ON A1M MECHANISM



* Opportunity to initiate pivotal Phase 3 study in sepsis following results in ongoing Phase 2b study (POINTER) in open-heart surgery.

** Multiple GTX peptides fulfill criteria for candidate drug nomination. GTX-86 at nomination stage.

A1M, alpha-1-microglobulin.

CHRONIC KIDNEY DISEASE & END-STAGE RENAL DISEASE – A GLOBAL HEALTH CONCERN

Acute Kidney Injury (AKI):

- Multiple causes, often resulting from in-hospital complications like severe infections & sepsis and major surgeries (e.g., open-heart surgery, kidney transplantation)
- 50% or more of high-risk open-heart surgery patients develop AKI; addressable patient population ~100,000-120,000 cases per year in the US alone (~30,000 patients with pre-operative CKD)

Progression to Chronic Kidney Disease (CKD):

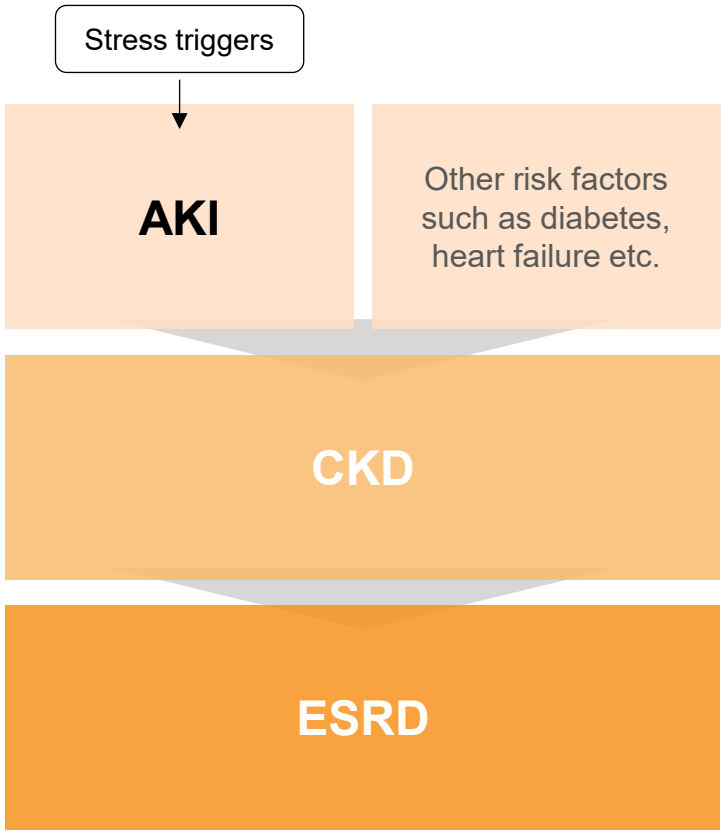
- AKI raises the risk of CKD; 15-20% progress to advanced CKD within 24 months
- CKD leads to severe complications, e.g., cardiovascular disease and kidney failure
- Years of life lost (YLL) from CKD are expected to surpass diabetes by 2040

AKI in patients with pre-existing CKD:

- CKD is a strong risk factor for AKI
- AKI in CKD accelerates progression to ESRD – high unmet need

CKD to End-Stage Renal Disease (ESRD):

- 10-15% of CKD patients advance to ESRD; requires dialysis or kidney transplant
- High mortality rate (15-20%), worse than many cancers
- Represents 7% of Medicare costs but affects 1% of the population



Francis et al, Nature Reviews Nephrology 2024, P Stenvinkel and TE Larsson, Am J Kidney Dis, 2013 Aug;62(2):339-51, 2021 USRDS Annual Data Report
July 2024 Data Book: Health Care Spending and the Medicare Program – MedPAC , Chawla, L. S., & Kimmel, P. L. (2012). Acute kidney injury and chronic kidney disease: an integrated clinical syndrome. Kidney International, 82(5), 516-524

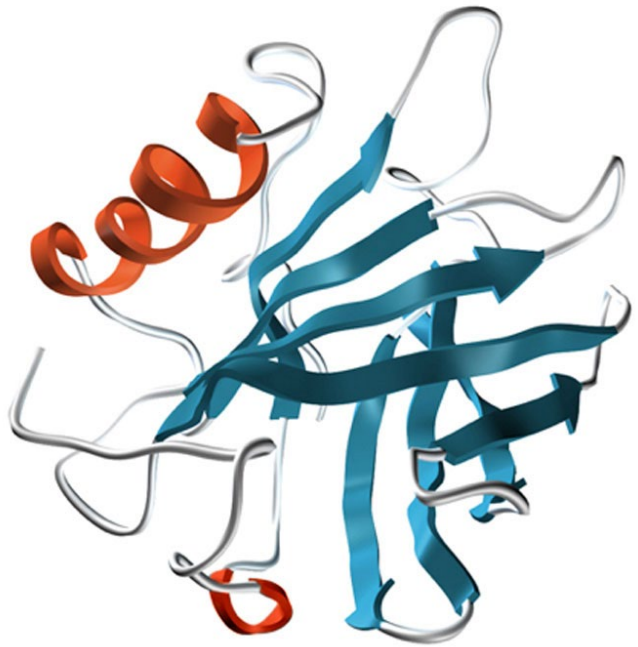
THERAPEUTIC A1M DELIVERY – A NOVEL PARADIGM WITH CLINICALLY VALIDATED MECHANISM

Harnessing natural properties of alpha-1-microglobulin (A1M) to protect the kidneys

- Endogenous ~22 kDa circulating glycoprotein
  - Liver main source of expression
- Ubiquitous distribution & cellular uptake
- Renal clearance
- Evolutionary conserved house-keeping mechanism:

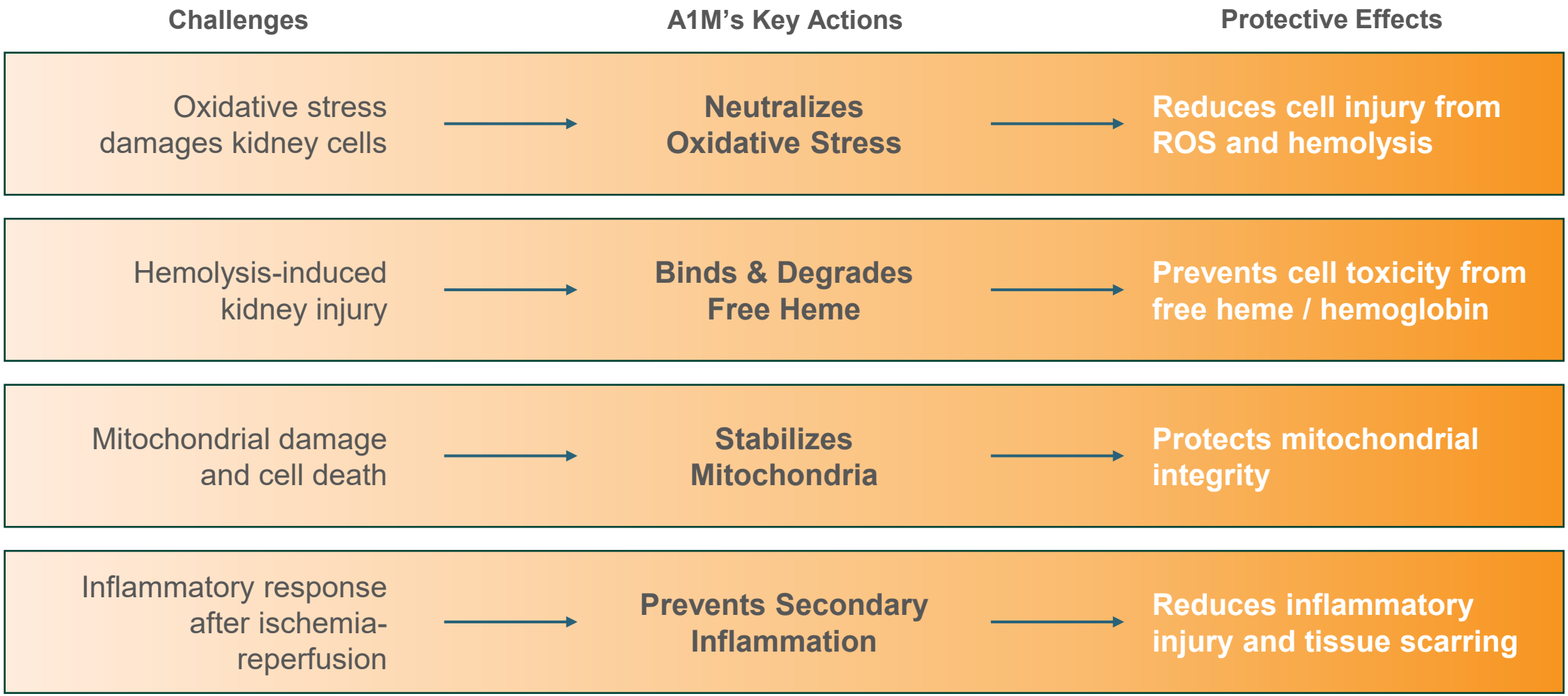
➤ ***Potent dual-action inhibitor of oxidative stress*** (reductase, radical scavenging)

➤ **Heme binding**



3-D structure of A1M protein

A1M PROTECTS KIDNEY FUNCTION BY TARGETING CRITICAL DISEASE PATHWAYS



Bergwik et al., Front Physiol 2021

DIFFERENTIATING ATTRIBUTES OF RMC-035 (VS OTHER DRUGS TARGETING ACUTE RENAL PROTECTION)

- **Distributes to & activity at primary site of cellular injury**
  - Kidney proximal tubules
- **Several protective & evolutionary conserved mechanisms**
  - Oxidative stress / ischemia & reperfusion injury
  - Heme injury
  - Mitochondrial dysfunction
- **Targets cellular events upstream of initial injury – *a paradigm not previously tested in clinical settings***
- **Robust & consistent kidney protection** in a **diverse set of in vivo models & species**

RMC-035 – therapeutic intact A1M

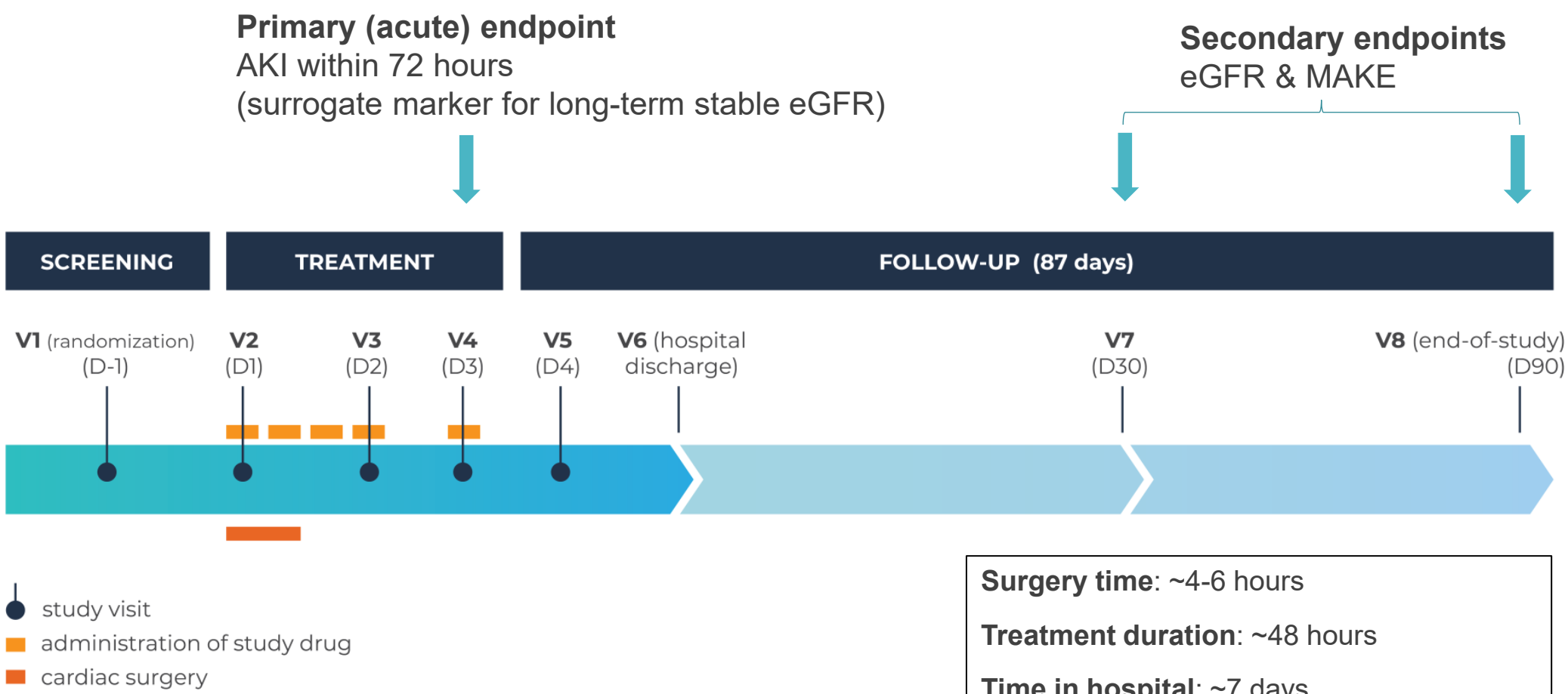
Kidney protection in open-heart surgery

COMPLETED PHASE 2a *AKITA* STUDY – OUTLINE & OBJECTIVES

- Recruitment in the **US, Canada & Europe**
- Double-blind, placebo-controlled (1:1 RMC-035:placebo) in patients undergoing **open-heart surgery at increased risk for kidney injury**
- **177 patients** randomized and dosed
- Main objective: **proving efficacy** & safety with **the maximum possible dose**
  - *Primary endpoint: acute SCr change meeting AKI criteria, not accepted for regulatory approval*
  - *Secondary endpoints: Long-term eGFR, accepted for regulatory approval*
- Start dose 1.3 mg/kg; reduced to 0.65 mg/kg for patients with low pre-operative renal function
  - *Overexposure risks linked to acute SCr increase (tubular overload)*

SCr, serum creatinine; AKI, acute kidney injury; eGFR, estimated glomerular filtration rate

COMPLETED PHASE 2a *AKITA* STUDY – FLOWCHART



Surgery time: ~4-6 hours

Treatment duration: ~48 hours

Time in hospital: ~7 days

Follow-up time: up to 90 days after surgery

AKI, acute kidney injury; eGFR, estimated glomerular filtration rate; MAKE, major adverse kidney events.

PHASE 2a RESULTS SUPPORT ROBUST EFFICACY ON HARD KIDNEY ENDPOINTS

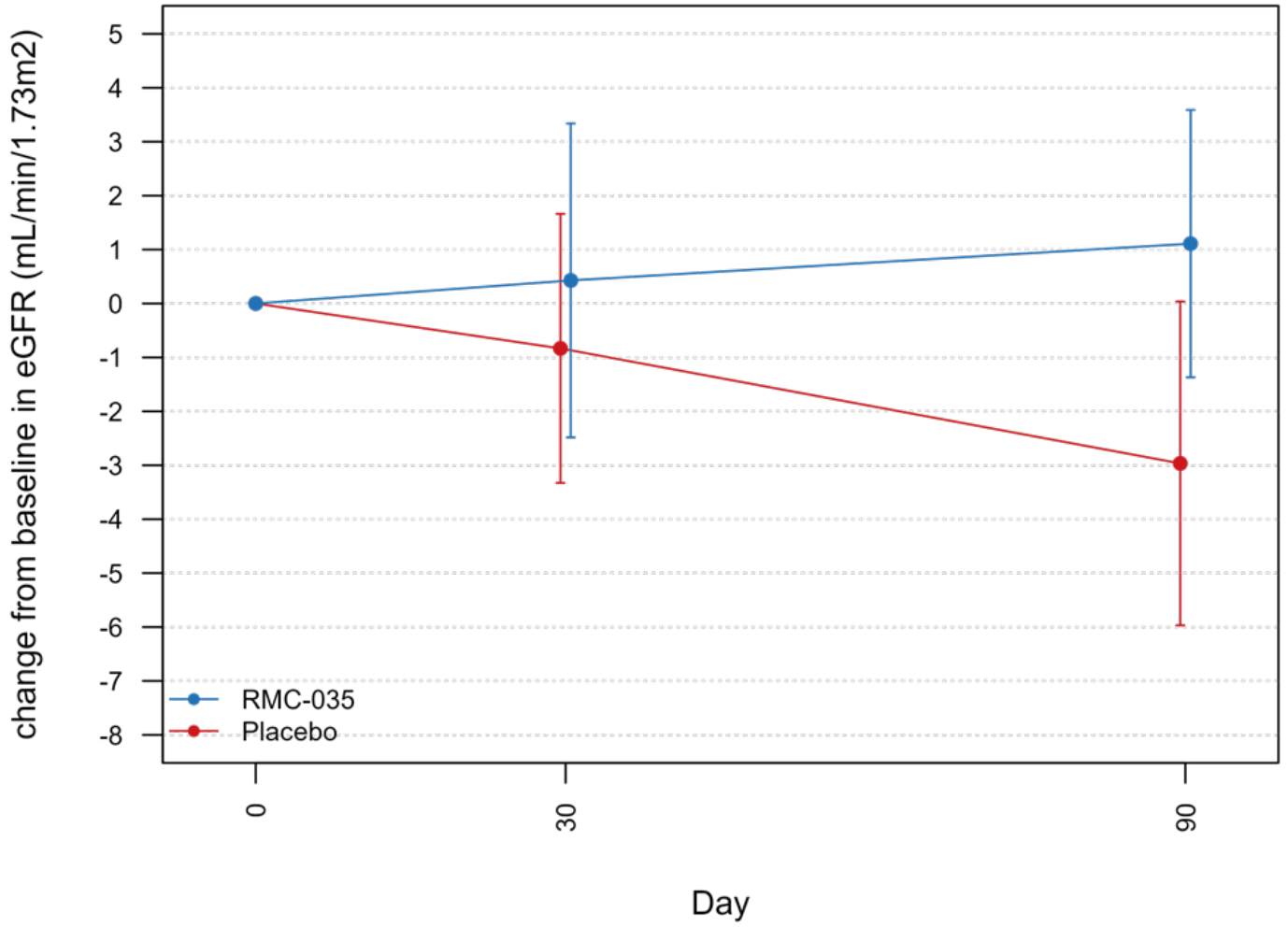
Efficacy stronger than required for regulatory approval based on renal function (eGFR) & Major Adverse Kidney Events (MAKE) on Day 90

- **Statistically significant & clinically relevant improvement of renal function (eGFR) vs placebo**
  - Improved eGFR vs placebo 4.3 mL/min (full population)
  - Improved eGFR vs placebo 7.9 mL/min (pre-defined subgroup of patients with chronic kidney disease [CKD])
- **Reduced proportion of patients with MAKE (i.e., severe loss of kidney function)**
  - 59% risk reduction vs placebo for composite endpoint MAKE (death, dialysis or ≥ 25% eGFR loss)
  - FDA recommends **MAKE as primary endpoint in Phase 3 – 20% risk reduction sufficient for approval**

Results support progression to Phase 2b

Study ROS-05 Clinical Study Report: Zarbock et al., eClinical Medicine 2024. eGFR, estimated glomerular filtration rate.

RMC-035 PREVENTS LOSS OF RENAL FUNCTION AFTER OPEN-HEART SURGERY



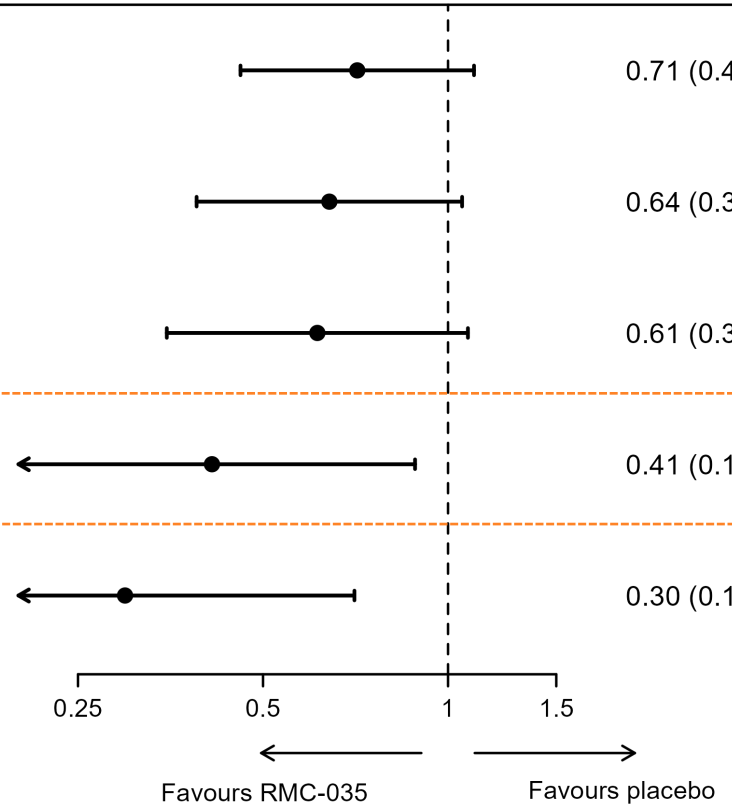
eGFR difference
4.3 mL/min **p=0.06***

Pre-defined alpha level was 0.1.
P-values < 0.1 are statistically significant.

Study report 21-ROS-05; eGFR, estimated glomerular filtration rate

RMC-035 CONSISTENTLY REDUCES MAKE90 USING VARIOUS THRESHOLDS OF eGFR LOSS

	RMC-035 Evs/Pts	Rate (90% CI)	Placebo Evs/Pts	Rate (90% CI)	Risk ratio (90 % CI)	p-value
MAKE 90 days (10 % eGFR cutoff)	18/89	20.2 (13.2-27.2)	25/88	28.4 (20.5-36.3)	0.71 (0.46-1.10)	0.200
MAKE 90 days (15 % eGFR cutoff)	14/89	15.7 (9.4-22.1)	22/88	25.0 (17.4-32.6)	0.64 (0.39-1.05)	0.138
MAKE 90 days (20 % eGFR cutoff)	11/89	12.4 (6.6-18.1)	18/88	20.5 (13.4-27.5)	0.61 (0.35-1.08)	0.150
MAKE 90 days (25 % eGFR cutoff)	6/89	6.7 (2.4-11.1)	14/88	15.9 (9.5-22.3)	0.41 (0.19-0.88)	0.047
MAKE 90 days (30 % eGFR cutoff)	4/89	4.5 (0.9-8.1)	14/88	15.9 (9.5-22.3)	0.30 (0.13-0.70)	0.010



Study report 21-ROS-05 & post-hoc analysis of 21-ROS-05. MAKE, major adverse kidney events; eGFR, estimated glomerular filtration rate

PHASE 2b *POINTER* STUDY – STATUS UPDATE

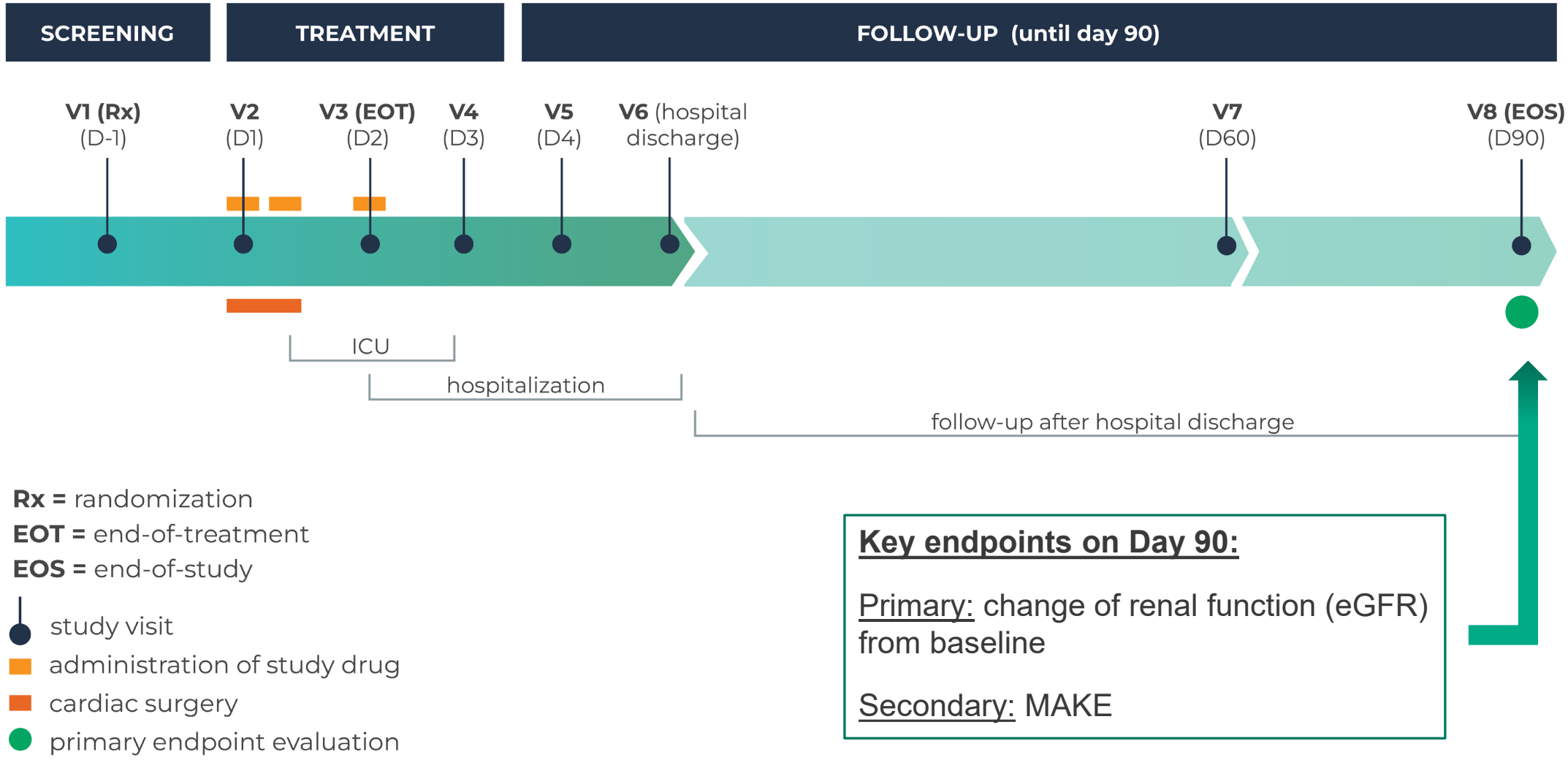
- **Final optimization step before Phase 3**
- ~160 patients (Europe & North America)
- Two RMC-035 dose arms (30 & 60 mg) and Placebo (2:2:3 randomization)
  - 3 infusions during the first 24 h from surgery
- **Primary endpoint:** change in eGFR from before surgery to Day 90

Important study milestones

- ✓ **>80% of patients enrolled** (April 2025)
- ✓ **Positive outcome of first DSMC interim safety review** (Q1 2025); 2nd review in Q2
- Recruitment to be completed this summer; results year-end 2025

eGFR, estimated glomerular filtration rate; DSMC, data safety monitoring committee

ONGOING PHASE 2b *POINTER* STUDY – FLOWCHART



Rx = randomization
EOT = end-of-treatment
EOS = end-of-study

- study visit
- administration of study drug
- cardiac surgery
- primary endpoint evaluation

Key endpoints on Day 90:

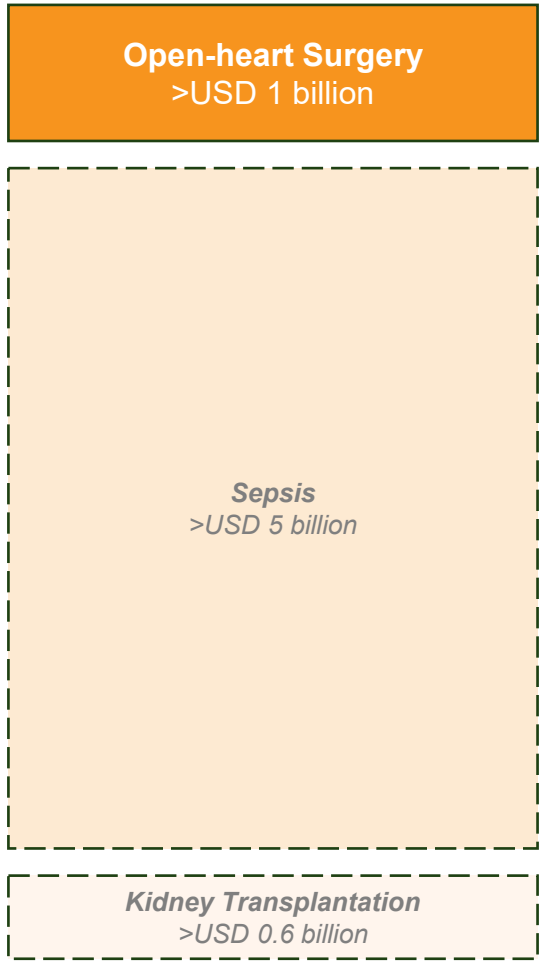
Primary: change of renal function (eGFR) from baseline

Secondary: MAKE

ICU, intensive care unit; eGFR, estimated glomerular filtration rate; MAKE, major adverse kidney outcomes

CLEAR PATH TOWARDS MARKET APPROVAL FOR RMC-035

- **Fast Track Designation** granted by the US FDA
  - *Reducing risk for death, dialysis or irreversible loss of kidney function in patients undergoing open-chest cardiac surgery at high risk for acute kidney injury*
- Indication eligible for **Breakthrough Therapy Designation**
- **Single pivotal Phase 3 study** sufficient to support market approval
  - *Primary endpoint MAKE at Day 90 after surgery (~600 patients)*
  - *Potential for accelerated approval based on interim analysis of eGFR (~300 patients)*
  - *First-to-market potential*
- **Phase 3 Expansion Opportunities**
  - ***Sepsis*** *Phase 3 ready and* ***Kidney Transplant*** *Phase 2a/b ready, following successful Phase 2b POINTER results*



External analysis (September 2022) & interviews with Health Care Professionals & Hospital & Therapeutics Committee members, Data from US Renal Data System (USRDS) & Organ Procurement and Transplantation Network (OPTN), HCUP 2019, J Thorac Dis. 2018 Mar; 10(3): 1960–1967, Pickering JW et al. Am J Kidney Dis. 2015. eGFR, estimated glomerular filtration rate; MAKE, major adverse kidney events

FIRST-TO-MARKET POTENTIAL WITH NO APPROVED THERAPIES – COMPETITOR LANDSCAPE

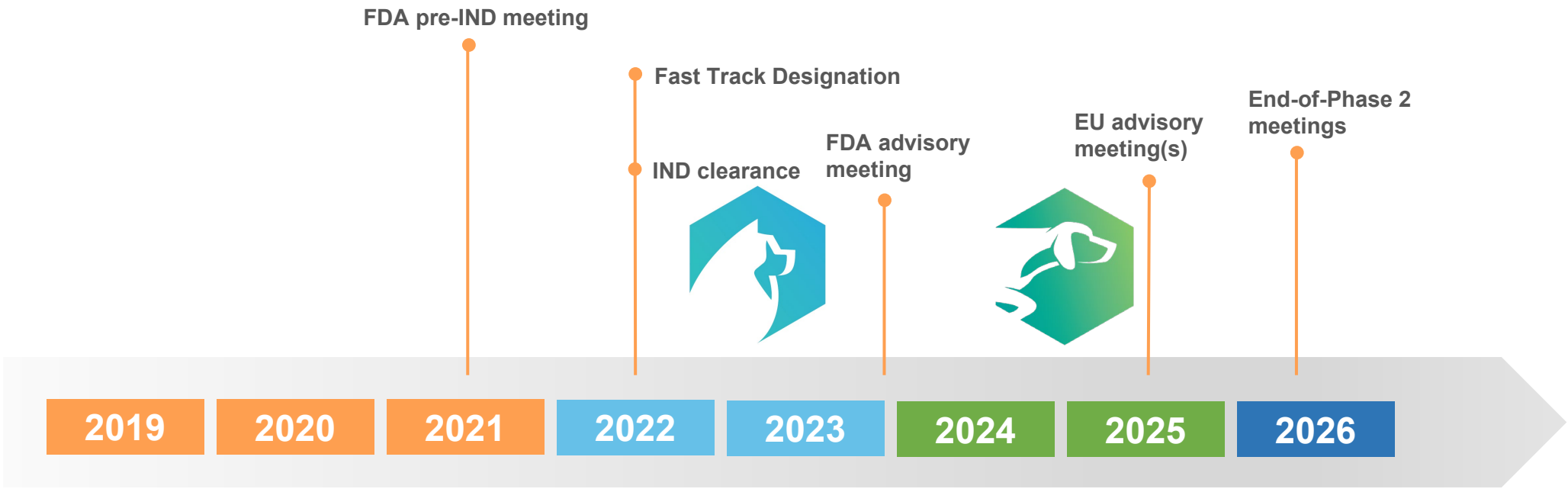
COMPANY (DRUG)	PHASE	MODE OF ACTION	POC DATA HEART SURGERY	COMMENT
Guard Therapeutics (RMC-035)	**2b**	**A1M analog**	**Yes**	Ongoing – expected results year-end 2025
Novartis (TIN-816)	2a	Human CD39 enzyme	-	Ongoing – expected results Q3 2025 N=120, acute primary endpoint No efficacy data available
AstraZeneca / Alexion (Ultomiris)	3	Complement 5 inhibitor	-	Ongoing – expected results Q1 2027 N=736, MAKE is primary endpoint No efficacy data available in open-heart surgery
Genentech (GDC-8264)	2	RIP-1 inhibitor	-	Ongoing – expected results Q4 2027 N=404, MAKE is primary endpoint No efficacy data available
AM Pharma (Ilofotase alfa)	2a	ALP analog	-	Ongoing – results expected Q4 2025 N=250, acute primary endpoint No efficacy data available in open-heart surgery
Renibus Therapeutics (RBT-1)	3	Iron sucrose + stannus protoporhyrin	-	Targets acute endpoints like length of hospital stay & hospital re-admission rate. Did not show efficacy on renal endpoints in Phase 2a study.

RECENT PHARMA DEALS IN NEPHROLOGY
– TOTAL DEAL VALUE OVER $11BN 2023-24 YTD

TARGET	ACQUIRER	YEAR	DEAL VALUE	STAGE	LEAD ASSET	INDICATION
Alpine Immune Sciences	Vertex Pharma	2024	**$4.9bn**	Phase 2	Povetacicept	IgAN
Human Immunology Biosciences	Biogen	2024	**$1.15bn + milestones**	Phase 2	Feltzartamab	IgAN, Primary membranous nephropathy & antibody-mediated rejection
Jnana Tx	Otsuka	2024	**$800m**	Preclinical	Panel of solute carrier inhibitors	Ion transporter kidney disease
Calliditas	Asahi Kasei	2024	**$1.1bn**	Marketed	Tarpeyo (Budesonide)	IgAN
Chinook Tx	Novartis	2023	**$3.5bn**	Phase 3	Atrasentan & Zigakibart	IgAN
CinCor Pharma	AstraZeneca	2022	$1.8bn	Phase 2	Baxdrostat	Treatment-resistant hypertension, primary aldosteronism and CKD
Vifor Pharma	CSL	2021	$12.3bn	-	Product portfolio in nephrology	-
Sanifit Tx	Vifor Pharma	2021	$205m + milestones	Phase 3	SNF472	Treatment for calciphylaxis ESRD patients
Corvidia Tx	Novo Nordisk	2020	$2.1bn	Phase 2	Zilitivekimab	Therapies within CKD segments

IgAN, IgA nephropathy; CKD, chronic kidney disease; ESRD, end-stage renal disease; NDA, new drug application

KEY MILESTONES & DELIVERY ACCORDING TO PLAN



Clinical Phase 1 program

- ✓ ROS-01 (single dose, healthy subjects)
- ✓ ROS-02 (multiple doses, healthy subjects)
- ✓ ROS-03 (renal impairment study)
- ✓ ROS-04 (safety/PK study in heart surgery)

Phase 2a *AKITA* study

- ✓ ROS-05 (proof-of-concept, heart surgery)

Phase 1b study

- ✓ ROS-06, kidney transplant

Phase 2b *POINTER* study

ROS-07 (dose-finding)

Phase 3 study heart surgery

Optional Phase 3 study in sepsis

Optional Phase 2a/b study in kidney transplantation

GTX platform (A1M-derived peptides)

Treatment of chronic kidney disease (CKD)

GTX PEPTIDES – 2nd GENERATION A1M DRUGS

Delivered SC

Intended as chronic therapy with intermittent dosing (e.g., for CKD)

- Panel of novel **A1M-derived peptides with preserved functionality** vs native A1M
  - ~15-35 aa, chemical synthesis
- Enables **clinically validated A1M mechanism** in **non-acute settings**
- **Robust preclinical efficacy** across diverse acute and chronic kidney models
- **Strong IP**, composition of matter until 2044
- Broad clinical development opportunity with **unique positioning in CKD**
  - ***High degree of optionality – strategy yet to be refined***
- Candidate drug nomination pending; transfer into slow-release formulation
- ~2 years to IND

GTX PEPTIDES (A1M-DERIVED) – MASSIVE OPPORTUNITY IN LATE-STAGE CKD

- **High potency and efficacy, comparable to RMC-035**
- **Broad impact across CKD etiologies, including orphan diseases**
  - *Demonstrated robust efficacy in a wide range of preclinical disease models*
- **Unique opportunity in late-stage CKD patients:**
  - *Highest risk for progression to ESRD*
  - *Often excluded from clinical trials*
  - *Current CKD therapies ineffective or contraindicated*

CKD, chronic kidney disease; ESRD, end-stage renal disease.
CKD classification based on Kidney Disease Improving Global Outcomes (KDIGO)

PIONEERING TRANSFORMATIVE MEDICINES FOR KIDNEY DISEASE

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- **Clinical proof-of-concept established in Phase 2a AKITA study with 177 patients**
  - **59% reduction vs placebo (MAKE, regulatory endpoint)**
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GUARD THERAPEUTICS

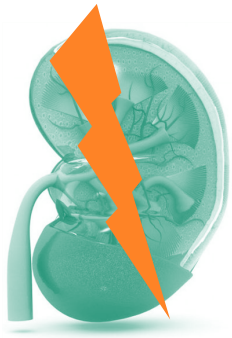
Appendix

DIFFERENTIATING ATTRIBUTES OF GTX PEPTIDES VS SoC & LATE-STAGE COMPETITORS IN CKD

- **Preferential distribution & activity at kidney proximal tubules**
  - Enables specific tubular protection
- **Evolutionary conserved protective mechanisms common to all CKD patients**
  - Oxidative stress / IRI, heme injury, mitochondrial dysfunction
- **Does not rely on renal hemodynamic mechanisms for kidney protection**
  - **Complementary to SoC**
  - **Potential efficacy & safety benefits**
- **Offers MoA-driven enrichment** of high-risk patients
  - **E.g., hematuria & tubulo-interstitial engagement**
    (acute tubular injury is a common feature of progressive CKD)

THERAPEUTIC POTENTIAL ACROSS THE SPECTRUM OF CKD

	Glomerular dysfunction, podocyte & mesangial activation[1]	Loss of filtration barrier	Inflammatory mediator release & tubular cell injury	Metabolic dysfunction
Patho-physiology	**Podocyte effacement Mesangial expansion ROS & NOX4 Inflammation**	**Proteinuria Hematuria**	**Tubular inflammation Fibrosis ROS & NOX4**	**Metabolic dysfunction ROS & NOX4**
GTX intervention	Protection of podocytes / mesangial cells[2,3]	Maintenance of glomerular structure[2,3]	Antioxidant activity & tubular cell activation[4,5,6]	Stabilization of mitochondrial structure & function, reduced ROS[7,8]



Targeting critical & common disease pathways in late-stage CKD – independent of CKD etiology

ROS, reactive oxygen species; NOX4, nicotinamide adenine dinucleotide phosphate oxidase 4

1 Wickman et al 2016. PLoS One. 11(5): e0155255.
2 Nääv et al 2015. PLoS One. 0125499
3 Wester-Rosenlöf et al 2014. 9(1): e86353.
4 Åkerström et al 2019, Antioxidants & Redox Signal. 30(4): 489
5 Burmakin et al. 2024 Am J Physiol Renal Physiol. doi: 10.1152/ajprenal.00067.2024
6 Study reports with in vitro and in vivo GTX activity on file
7 Kristiansson et al. 2020. Int J Mol Sci. 21(6): 5825
8 Olsson et al 2013. Antiox & Redox Signal. 18(6): 2017

GENERIC CKD THERAPIES LARGELY RELY ON HEMODYNAMIC (GLOMERULAR) MECHANISMS

DRUG CLASS	MODE OF ACTION	PRIMARY SITE OF ACTION & KEY MECHANISM FOR SLOWING CKD PROGRESSION
ACE inhibitors	*Inhibition of Angiotensin-Converting Enzyme*	Systemic; reduction of blood pressure & intra-glomerular pressure via vasodilation
ARBs	*Blocking of Angiotensin II Type 1 receptors*	Systemic; reduction of blood pressure & intra-glomerular pressure via vasodilation
ERAs	*Blocking of Endothelin Type A receptors*	Systemic; reduction of blood pressure & intra-glomerular pressure via vasodilation; reduction of inflammation & fibrosis
SGLT2 inhibitors	*Inhibition of Sodium-Glucose Co-Transporter 2*	Kidney proximal tubules; reduction of intra-glomerular pressure via tubulo-glomerular feedback
AS inhibitors	*Inhibition of Aldosterone Synthase*	Systemic; reduction of blood pressure & intra-glomerular pressure via vasodilation; reduction of inflammation & fibrosis
ISTs (eg corticosteroids)	*Anti-inflammatory, immuno-suppressant based on pleiotropic effects*	Systemic; reduction of inflammation & fibrosis via multiple mechanisms

Untapped opportunity for specifically targeting tubular kidney compartment

ACE: angiotensin converting enzyme
ARB: angiotensin II type 1 receptor blockers
ERA: Endothelin receptor antagonists
SGLT2: sodium-glucose co-transporter 2
AS: aldosterone synthase (not yet marketed)
IST: immunosuppressive therapies

ADDITIONAL OPPORTUNITY FOR RMC-035 – SEPSIS

- Sepsis is **leading cause of acute kidney injury (AKI)**. Multifactorial etiology, kidney cell stress due to reduced perfusion, oxidative stress, endotoxins and inflammation
- **RMC-035 efficacious in preclinical sepsis models**
- In the US, **~1.7 million** patients develop sepsis each year**; ~800,000 patients** with sepsis develop AKI; and **~250,000 patients** develop CKD
- Dosing regimen: once daily up to 5 days; First dose given at sepsis diagnosis (ICU admission)
- Clear regulatory path to market approval – **Major Adverse Kidney Events (MAKE) at 90 days**
- **Single confirmatory Phase 3 study** sufficient for approval
  - *Sample size ~400-600 patients depending on eligibility criteria*
  - *Recruitment time ~2 years*
- **Pivotal Phase 3 study in sepsis enabled by Phase 2b POINTER study (heart surgery)**
  - *Interim analysis with sample size re-estimation to be built in in the absence of preceding efficacy study*
  - *Should be preceded by a Phase 1b study of approximately 15-20 patients to evaluate exposure & safety*

ADDITIONAL OPPORTUNITY FOR RMC-035 – KIDNEY TRANSPLANTATION

- **Phase 1b study of RMC-035 completed**
- Acute kidney graft dysfunction & impaired long-term dysfunction in deceased donor transplantation due to **graft ischemia & ischemia-reperfusion injury,** and **inflammatory / fibrotic response**
- Approximately **20,000 deceased donor kidney transplantation** performed annually in US – eligible for **orphan drug designation**
- Treatment goal: **protect long-term graft function in recipient**, avoiding need for re-transplantation
- Dosing regimen: once daily up to 5 days; First dose given intra-operatively to graft recipient
- **Clear regulatory path** to market approval – **eGFR at 1 year** after transplantation
- **Single confirmatory Phase 3 study** sufficient for approval
  - *Sample size 300-600 patients depending on eligibility criteria*
  - *Recruitment time 2-3 years*
- New formulation considered, may enable higher price point than in open-heart surgery

eGFR, estimated glomerular filtration rate.

GLOBAL MARKET OPPORTUNITY FOR RMC-035 SUPPORTS BLOCKBUSTER POTENTIAL

- **Open-heart surgery: >USD 1 billion (global)**
  - *100,000 patients annually in the US (~30,000 with CKD). Total US population ~250,000 patients.*
  - *Price estimate: USD 5,000 – 10,000 per patient*
  - *Total US market potential: USD 0.5 – 1 billion*
- **Sepsis: >USD 5 billion (global)**
  - *~1.7 million adults in the US develop sepsis each year*
  - *~500,000 patients in scope for treatment*
  - *Total US market potential: USD 2.5 – 5 billion*
- **Kidney transplantation: >USD 0.6 billion (global)**
  - *~20,000 patients in the US undergoing deceased donor kidney transplantation each year*
  - *Total US market potential: USD 0.3 – 0.6 billion (estimated price USD 15,000 – 30,000 per patient)*

External analysis (September 2022) & interviews with Health Care Professionals & Hospital & Therapeutics Committee members.
Data from US Renal Data System (USRDS) & Organ Procurement and Transplantation Network (OPTN)
HCUP 2019, J Thorac Dis. 2018 Mar; 10(3): 1960–1967
Pickering JW et al. Am J Kidney Dis. 2015

STRONG VALUE PROPOSITION – EFFICACY & COST-BENEFIT PROFILE

Robust kidney protection in AKITA study

- *~5 x greater eGFR effect than one year of standard-of-care CKD treatment*
- *3 x greater risk reduction of MAKE than required for FDA approval*

Strong evidence for eGFR in Cost Effectiveness Models

- *Value dossier supported by available health economic data in patients with CKD*

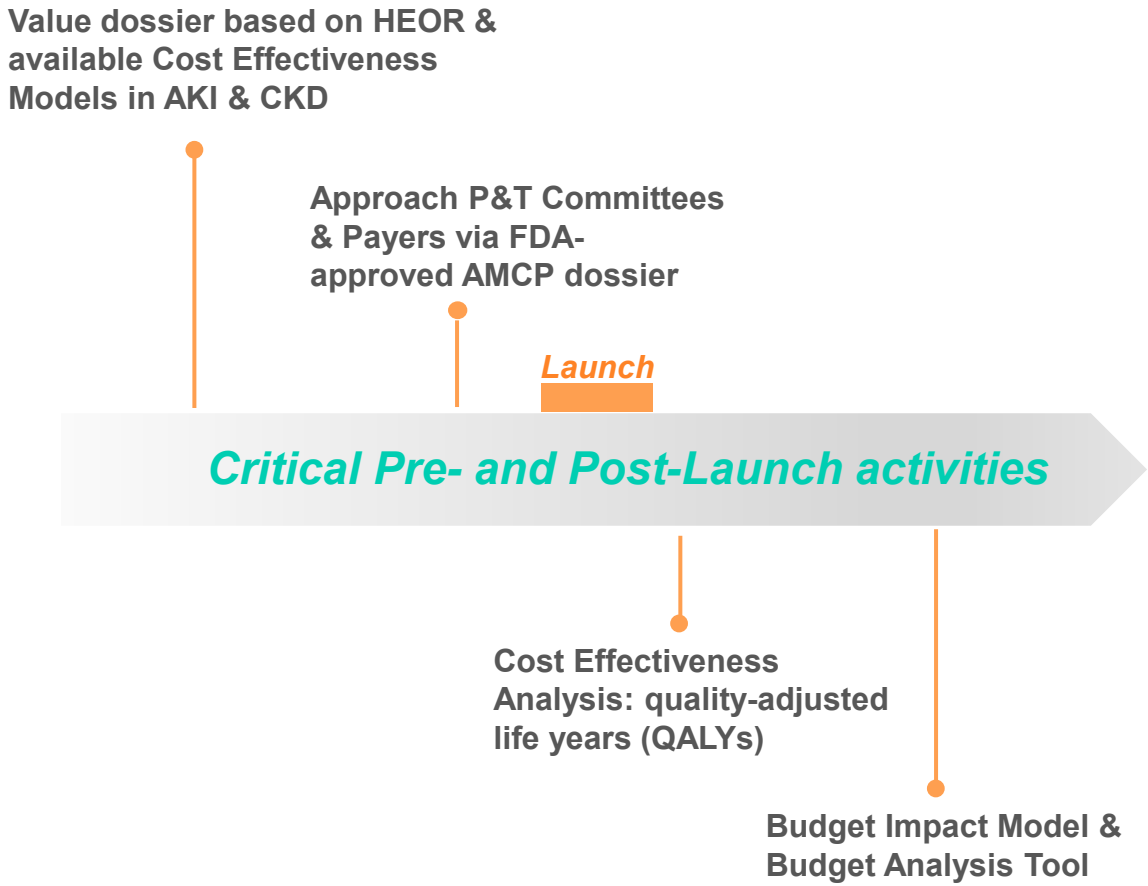
Short-term therapeutic benefit

(AKI, dialysis, length of hospital stay & re-admission)

- *Acute benefits offer direct & indirect hospital savings*

Attractive cost-benefit profile

- *Anticipated formulary inclusion with marginal impact on hospital cost*
- *Fulfils NTAP criteria*



eGFR, estimated glomerular filtration rate; CKD, chronic kidney disease; MAKE, major adverse kidney events; AKI, acute kidney injury; ICU, intensive care unit; AMCP; academy of managed care pharmacy; NTAP, new technology add-on payment