



Pioneering a World Free From Complement Disease

Stifel Healthcare Conference

November 16, 2022



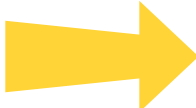
Our Name Reflects Our Mission

Advancing transformative
therapies for patients with
complement-mediated diseases

Complement-mediated Disease: A Dysregulation of the Complement System that Activates Immune Response

Broad Impact

- Nephrology
- Neurology
- Hematology
- Dermatology
- Ophthalmology
- Immuno-oncology



Goal of Therapy

- ✓ Achieve better, targeted and longer-acting immune modulation
- ✓ Preserve body's ability to fight new pathogens

Kira is Unique in the Complement Space

Clinical-Stage Company

Bi-functional Lead Asset

Proprietary LOGIC Platform

Robust Pipeline: 9 Drugs

Quick and Cost-Effective

- Lead Asset: Successful Phase 1, Entering Phase 2
 - Multiple high value indications
- Potent Inhibitor of proximal and terminal pathways provides potential for improved efficacy across many disease areas
- Unparalleled ability to address complex biology
- Potential for first-in-class, best-in-class compounds
 - Unique potency, duration and PKPD properties
- Potential for 2 more assets to enter clinic within 18-24 months
- Unrivaled speed and cost-effectiveness through discovery, manufacturing, and clinical development via Chinese offices

World-Class Science Led by a Global Team of Successful Executives and Entrepreneurs



Frederick Beddingfield, MD, PhD
CEO
 Board Certified Dermatology, Mohs Micrographic Surgery, and Cutaneous Onc & Emergency Medicine
 Associate Clinical Professor at UCLA



Wenchao Song, PhD
Scientific Founder & Chair of SAB
 Professor of Systems Pharmacology & Translational Therapeutics
 U Penn Perelman School of Medicine



Wenru Song, MD, PhD
President & Head of R & D Co-Founder



Teri Loxam
Chief Operating Officer & Chief Financial Officer



Richard Lee, MD
Chief Medical Officer



Martin Rabe, MSc
SVP, Head of Global Regulatory Affairs



Gonghua Pan, MBA, PhD
Head of External Alliances & Special Projects, Co-Founder



Angela (Hui) Yan
President of China and Asia Development and Operations

formerly

Allergan
 CMO (Allergan Medical)

KYTHERA
 biopharmaceuticals
 CMO

sienna
 biopharmaceuticals
 CEO

AstraZeneca
 Global VP I/O

Pfizer
 Global Clin Lead

sqzBIOTECH
 CFO

MERCK
 SVP IR and Global Communications

Bristol Myers Squibb

ALEXION
 Head of Hematology

Bristol Myers Squibb
 Group Medical Director

Eisai

Pfizer

MERCK

WuXi AppTec
 VP Operations

Pfizer

dMed
 Chief Operation Officer

Takeda

Scientific Advisors Include 3 Globally Known Complement Experts



Wenchao Song, PhD

Scientific Founder & Chair of SAB
Professor of Systems Pharmacology &
Translational Therapeutics
UPenn Perelman School of Medicine



Paul Morgan, MB, PhD

Professor of Immunology, Director of
Systems Immunity Research Institute
Cardiff University



Jörg Köhl, MD

Professor and Founding Director, Institute for Systemic
Inflammation Research
University of Lübeck (UzL), Germany



Garret A. Fitzgerald, MD, FRS

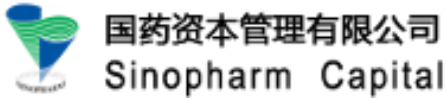
Professor of Medicine and
Systems Pharmacology & Translational Therapeutics
Perelman School of Medicine, University of Pennsylvania



Ronald Levy, MD

Robert K. Summy & Helen K. Summy
Professor of Medicine
Stanford University

Strong, Committed Investors Across World's Largest Capital Markets



Increased Interest in Complement Space: The “Complement Revolution”

Major Partnerships (Mergers and Acquisitions)

\$3.7B

AMGEN



\$1.5B

NOVARTIS

GYROSCOPE
VISION FOR LIFE

\$39B

AstraZeneca

ALEXION

\$1.2B

Apellis

sobi

\$930M

ALEXION



\$2.1B



Ra Pharma

\$825M

Bioverativ

TRUE NORTH
THERAPEUTICS

Encouraging Clinical Evidence Validates Complement Across Many Diseases

Nephrology

IgAN

IgA Nephropathy

C3G

Complement 3 glomerulopathy

Neurology

gMG

Generalized myasthenia gravis

NMO

Neuromyelitis optica

GBS

Guillain-Barre syndrome

Hematology

PNH

Paroxysmal Nocturnal Hemoglobinuria

aHUS

Atypical hemolytic-uremic syndrome

ANCA-AAV

antineutrophil cytoplasmic antibody-associated vasculitides

CAD

Coronary artery disease

ITP

Idiopathic thrombocytopenic purpura

Dermatology

HS

Hidradenitis suppurativa

BP

Bullous pemphigoid

PG




Pyoderma gangrenosum

Ophthalmology

dAMD

Dry age-related macular degeneration

Kira LOGIC* Platform Overcomes Three Key Challenges of Developing Complement Therapeutics

Challenge	Kira's Solution	Benefit
1 Complex biology	Unparalleled animal models for target validation & indication selection	 Selecting best target and indication
2 High load and turnover of targeted complement system proteins	State-of-the-art protein design allowing superior potency, antibody recycling, and longer lasting inhibition	 Best-in-class efficacy and dosing
3 Unreliable translation from clinic	Unique complement protein humanized transgenic mice allowing for unrivaled PK/PD predictability	 High probability of success in clinic

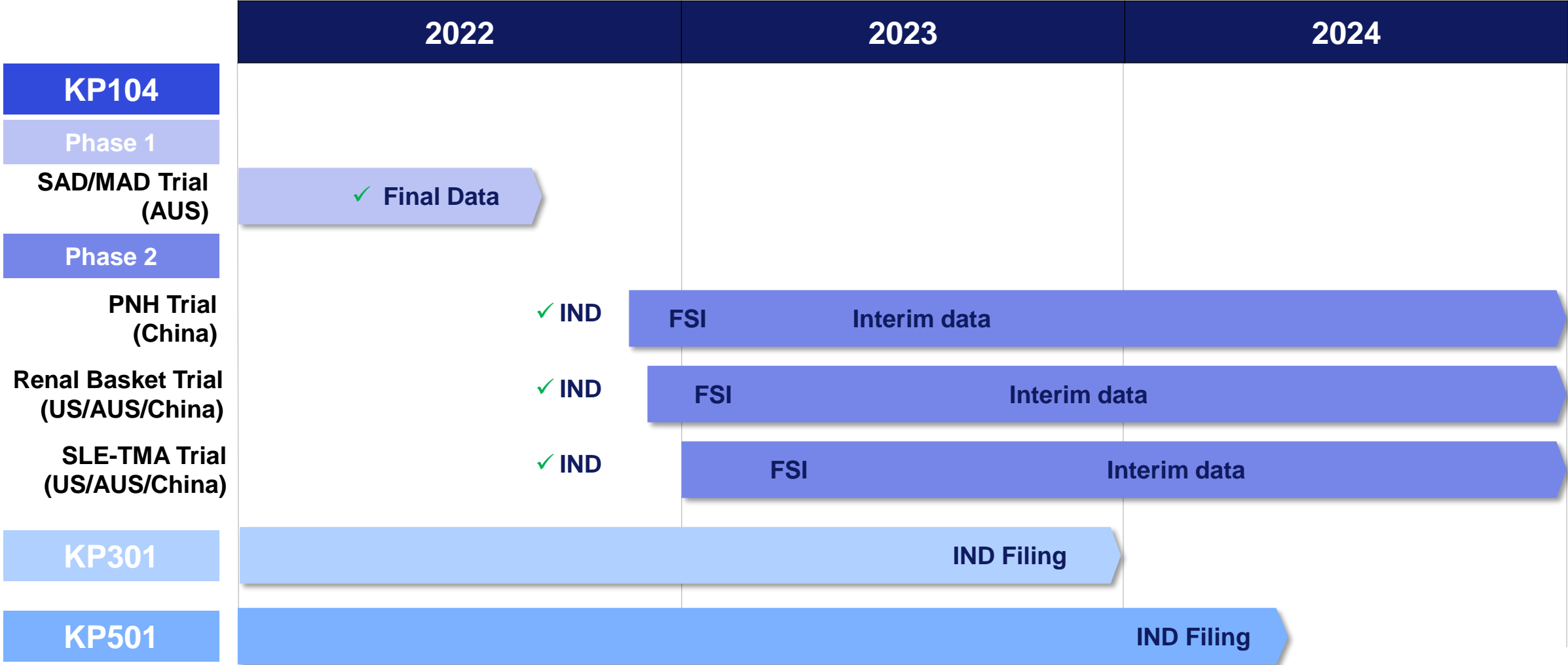
* Kira "LOGIC" Platform: Lead identification, Optimization and attributes Generation, In vivo Confirmation

Broad Pipeline with Differentiated Assets Spanning Complement Pathways

Product Candidate	MOA	Lead Indications	Development Stage					Next Anticipated Milestone
			Discovery	Preclinical	Phase 1	Phase 2	Phase 3	
KP104	Anti-C5 mAb +Factor H bifunctional biologic	Renal basket study (IgAN, C3G)						Phase 2 Interim Data H2-2023
		Thrombotic Microangiopathies Secondary to Systemic Lupus Erythematosus (SLE-TMA)						Phase 2 Interim Data Late 2023/Early 2024
		Paroxysmal Nocturnal Hemoglobinuria (PNH)						Phase 2 Interim Data mid-2023
KP301	Long-acting mAb against clinically validated target	Neutrophilic and other autoimmune inflammatory disorders						2H2023 FIH IND
KP501	Long-acting Classical and Lectin Pathway biologic	Autoantibody-mediated disorders						2024 FIH IND
KP1020/2050	Bi-specific biologics	dAMD						2022 Lead Generation

- 5 additional assets in the pipeline
- Precisely engineered biologics for novel MOA and potentially better efficacy

Multiple Value-Add Catalysts Over Next 12-18 Months



Lead Asset – KP104

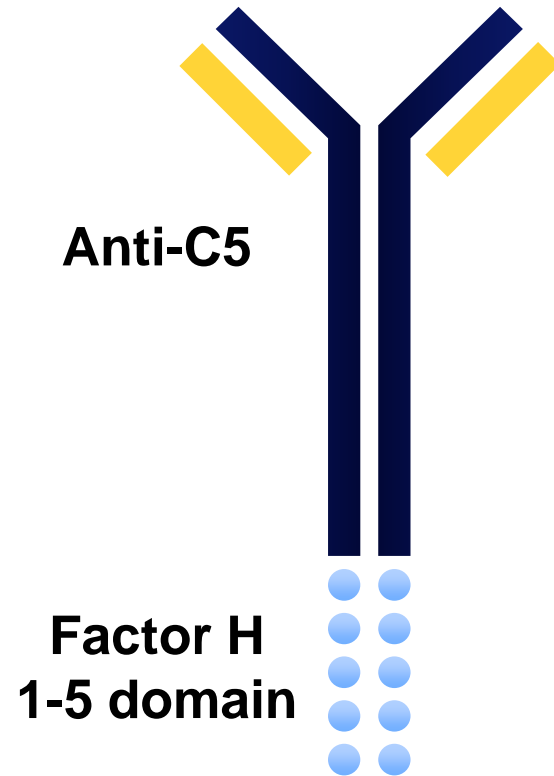
First-in-class, bi-functional biologic

Treats diseases where C5 or C3 inhibition alone is insufficient



Lead Asset KP104: Breakthrough in Complement

First-in-Class, Bifunctional Biologic, Overcomes Challenges of C5 or C3 Inhibition Alone



KP104 Differentiation

- Inhibits Alternative (Factor H) and Terminal (C5) Pathways
 - FH is natural AP regulator
 - C5 engineered to have long half-life
 - Unique C5 binding epitope
 - C5 serves as tissue targeting for FH, creating synergistic effect
- Superior efficacy and potency
- High manufacturing yield & high purity

Advantageous for diseases with AP contribution and where C5 inhibition alone is inadequate

Phase 1 Demonstrated Proof of Mechanism for KP104 Dual Inhibition and Provides High Confidence for Phase 2 Success

- Potent and sustained Terminal Pathway (TP) and Alternative Pathway (AP) Inhibition
 - Fully reduced free C5 levels ($<0.5\mu\text{g/mL}$) (biomarker for TP inhibition)
 - Continuously inhibited C3b deposition (biomarker for AP inhibition)
 - Continuously inhibited rabbit RBC hemolysis (Mixed model for AP & TP inhibition)
- Single IV loading dose achieved quick inhibition and SubQ dosing achieves steady state thereafter
 - MAD PK half-life ~ 16 days with IV loading followed by SQ weekly
- KP104 is only biologic in clinical development that simultaneously inhibits C5 and Alternative Pathway
- Potential for significant differentiation with ability for dual inhibition and SubQ dosing

Phase 1 Demonstrated KP104 was Safe and Well Tolerated

- No major safety findings in SAD and MAD
 - No deaths
 - No Serious Adverse Events
 - No drug-related treatment discontinuations due to AEs
 - No severe TEAEs (Grade 3) - majority of TEAEs were mild (Grade 1)
 - No Dose Limiting Toxicities
- Low incidence of ADAs and presence of ADAs did not impact safety, PK or PD
- Safety Review Committee deemed KP104 to be safe and well tolerated

Need for Dual AP/TP Inhibition in PNH Called Out in Recent NEJM Article



The NEW ENGLAND
JOURNAL of MEDICINE

REVIEW ARTICLE

Dan L. Longo, M.D., *Editor*

Breakthrough Hemolysis in PNH with Proximal or Terminal Complement Inhibition

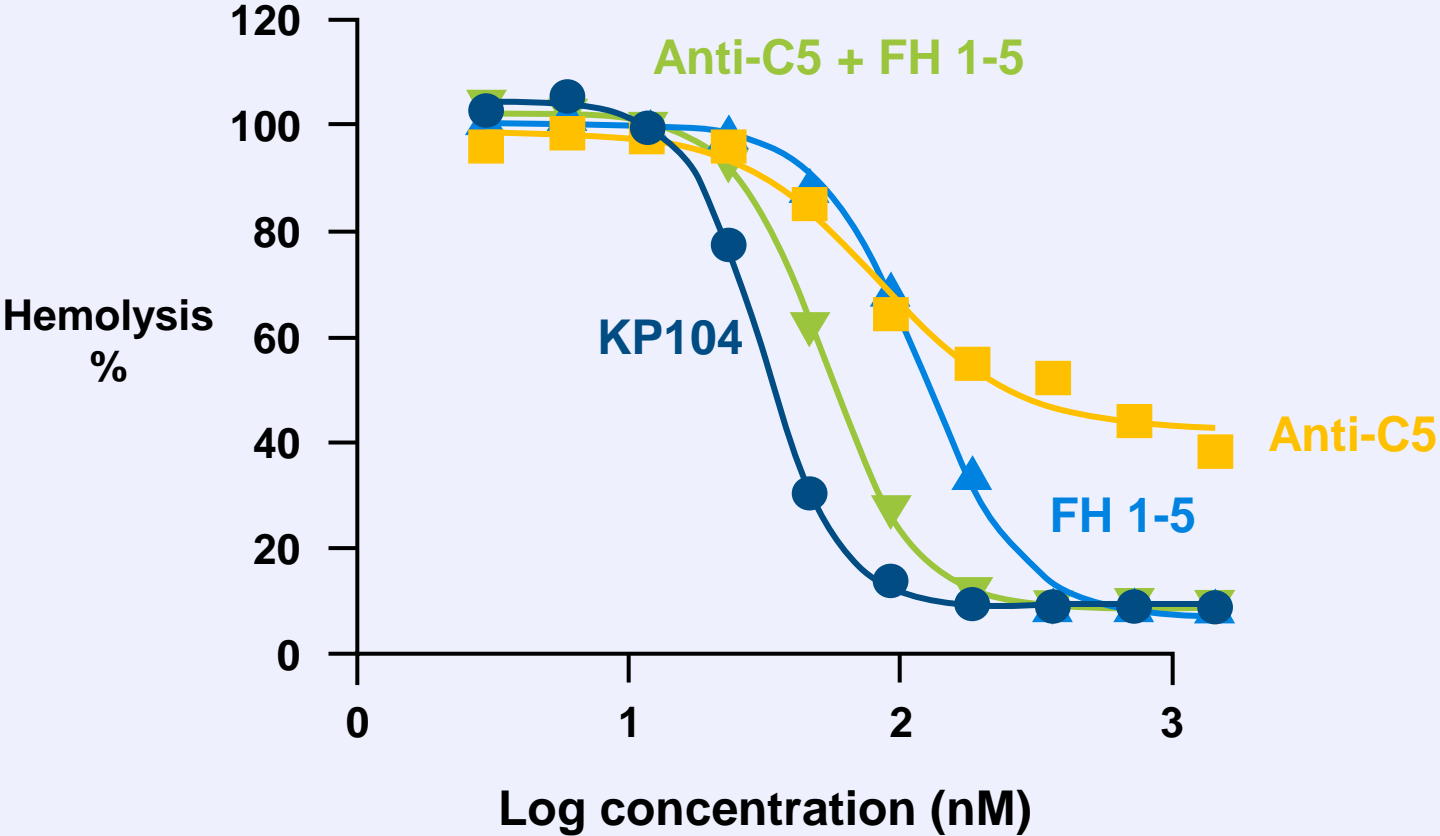
Rosario Notaro, M.D., and Lucio Luzzatto, M.D.

DOI: 10.1056/NEJMra2201664

- “ We now understand more clearly how even partial inhibition of the terminal complement pathway may be synergistic with proximal complement inhibition. ”***
- “ The combined approach might prevent C3 binding to PNH red cells and thus extravascular hemolysis, in addition to preventing massive breakthrough hemolysis. ”***
- “ Such an approach would improve clinical benefits with respect to both efficacy and safety in controlling hemolysis in patients with PNH. ”***

KP104 as Single Agent TP/AP Dual-Inhibitor Could Resolve Affordability Issue of Combining Single Agents While Also Potentially Providing Synergistic Efficacy

Rabbit RBC Lysis



- KP104 more potent than
 - Anti-C5 mAb alone
 - AP regulator (FH 1-5) alone
 - Anti-C5 mAb and AP regulator (FH 1-5) together
- Conclusion: there is synergy in potency when the anti-C5 mAb and AP regulator are combined in a single molecule of KP104

KP104 Targeting Novel Indications with High Unmet Need and First-in-Class or Best-in-Class Opportunities Where C5 or C3 Inhibition Alone is Insufficient

 **C5 Inhibition is Effective but Inadequate**

 **Involvement of Alternative Pathway**

 **Factor H Deficiency as a Potential Driver**

 **Aberrant AP Activity Manifests in Clinical Outcomes**



Lead Asset (KP104) Opportunities

Nephrology

- IgA Nephropathy (IgAN)
- C3 Glomerulopathy (C3G)
- Lupus Nephritis (LN)

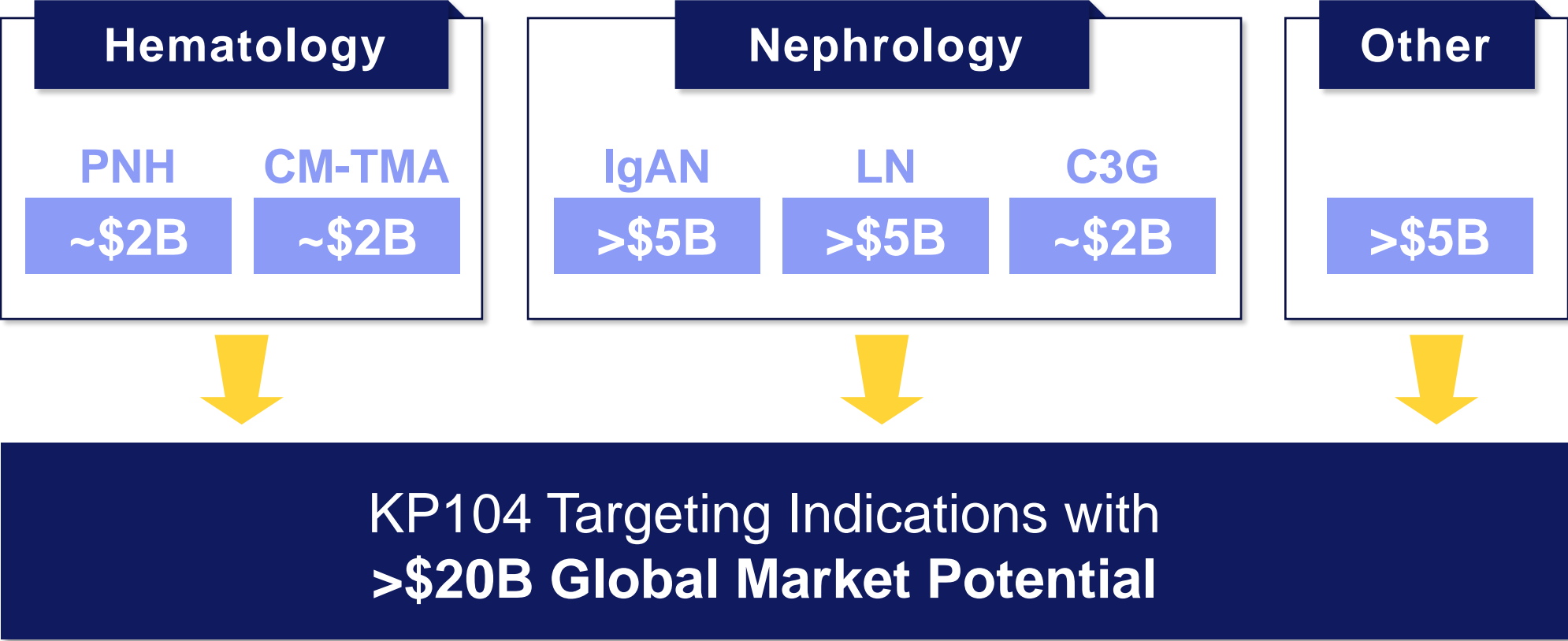
Hematology

- Complement-Mediated Thrombotic Microangiopathy (CM-TMA)
- Paroxysmal Nocturnal Hemoglobinuria (PNH)

Other

- Neurology

KP104 Indications Have Significant Global Market Potential



Summary of Strategy



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