SULFOGENIS

A New Era in Kidney Medicine

MBK2910 for the Treatment of Acute Kidney Injury (AKI)

20%
of patients develop AKI during hospital stay.

20%
is the AKI-associated 40-days mortality.

$24B
hospitalization cost in the EU and the US per year.

$240B
follow-up annual costs (dialysis and transplantation).

The Growing Crisis in Kidney Medicine

Acute kidney injury (AKI) is a frequent threat for hospitalized patients feared for its substantial morbidity and mortality. Despite its huge burden and socio-economic impact, effective therapeutic approaches in the clinic are lacking.

Sulfogenis' Solution

Sulfur metabolism as a key mediator of kidney protection is conserved in humans. MBK2910 renders kidneys resistant against reactive oxygen species due to its unique mode of action in sulfur metabolism. Our treatment protects the kidneys from ferroptosis, the key driver of AKI.

Translational Research

Built on translational research at the Cluster of Excellence for Aging Research (CECAD), the Cologne Center for Molecular Medicine (CMMC), and the University Hospital Cologne. Access to the Center of Excellence in Nephrology - European key hub for kidney innovation at the University Hospitals Aachen, Cologne, and Düsseldorf.

Known Mode of Action

MBK2910 increases intracelullar glutathione biosynthesis and improves recovery of oxidized glutathione . MBK2910 enters tubular epithelial cells via a known transporter and limits cell death in kidneys. This unique, and broad mode of action enables MBK2910 for the treatment of a wide range of diseases.

Scalable Access in Kidney Medicine

MBK2910 allows a broad clinical use and equitable access due to a very favourable side-effect profilie. MBK2910 will reduce AKI-associated mortality, prevent loss of kidney function, avoid dialysis, improve quality of live, and reduce total care costs. Further indications in kidney medicine are chronic kidney disease and transplantation.

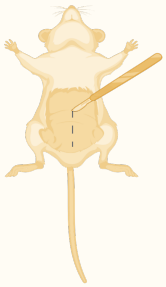
Expansion into Other Territories

MBK2910 protects organs from ferroptosis, a universal mode of cell death. The expansion of MBK2910 for the treatment of ferroptosis-caused diseases beyond kidney medicine includes stroke, myocardial infarction, non-alcoholic fatty liver disease, hemochromatosis, autoimmune diseases, and neurodegenerative disorders.

MBK2910 demonstrates Consistent Protective Effects.

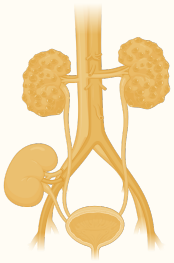
Known pharmacokinetics (*in-vitro, in-vivo*)

- Biochemical modifications directly influence bioavailability and pharmacokinetics with stable pharmacodynamics.
- Universal mode of action and modifiable pharmacokinetics enable broad usage of MBK2910 beyond kidney medicine.



Uninephrectomy + renal IRI (mouse, *in-vivo*)

- Consistent and robust induction of AKI by removal of the right kidney and clamping of the left vessels for 40 minutes.
- Improved survival and protected kidney function.
- Prevented histopathological damage and lowered tubular injury markers.
- Demonstrated potent anti-ferroptoptic activity.



Clinical trial during living kidney donation (human, *ex-vivo*)

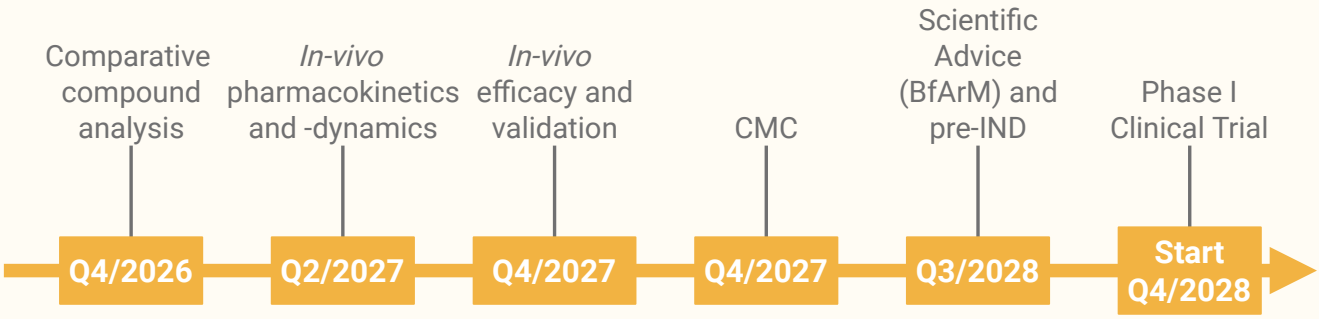
- Set-up of an accompanying clinical trial (NCT05709600) with specific, dietary interventions that mimic MBK2910's mode of action in sulfur metabolism.
- Collection of human kidney material: Conservation of MBK2910's mechanism of kidney protection in human tissue.

World-Class Advisory Board

Prof. Ton Rabelink

Prof. Hanns-Christian Mahler

Prof. Marcus Conrad



Raising CHF 3M to complete IND-enabling (FDA, EMA) studies and GMP manufacturing towards First-in-Human

Leadership Team

Prof. Günter Schwarz (CEO, Co-founder)
Dr. Felix Köhler (CSO, Co-founder)
Marc Kessemeier (CFO, Co-founder)
Prof. Thomas Benzing (Co-founder)
Prof. Roman-Ulrich Müller (Co-founder)

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