BIOGRAPHICAL SKETCH

NAME: Balzer, Michael Sören

eRA COMMONS USER NAME (credential, e.g., agency login): mibalzer

POSITION TITLE: Group Leader, Assistant Professor

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
University of Music Karlsruhe, Karlsruhe (DE)	Junior undergrad	03/2001	Violoncello & Piano
University of Music Würzburg, Würzburg (DE)	Junior undergrad	03/2005	Violoncello & Piano
University of British Columbia, Vancouver (CA)		12/2010	Med School (Yr6), Nephrology
University College London, London (GB)		01/2011	Med School (Yr6), Pediatric Nephrology
University of Oxford, Oxford (GB)		03/2011	Med School (Yr6), Pediatrics
University of Berne, Berne (CH)		07/2011	Med School (Yr6), Surgery
Friedrich-Alexander University Erlangen-Nuremberg, Erlangen (DE)	MD	06/2012	Med School
Hannover Medical School, Hannover (DE)	Postdoctoral	10/2019	Residency, Internal Medicine; Fellowship, Nephrology
University of Pennsylvania, Philadelphia (US)	Postdoctoral	10/2022	Experimental Nephrology
Charité - Universitätsmedizin Berlin, Berlin (DE)	Postdoctoral		Nephrology

A. Personal Statement

I am a Group Leader and Attending Physician at the Department of Nephrology and Medical Intensive Care at Charité - Universitätsmedizin Berlin. My research focuses on the cellular and molecular mechanisms of adaptation, recovery, and repair in acute kidney injury (AKI), acute kidney disease (AKD), and the transition to chronic kidney disease (CKD). I have a background in medicine and nephrology, with specific training and expertise in experimental kidney research, single-cell transcriptomics, and translational approaches linking human disease to model systems. I studied Medicine at the Universities of Erlangen-Nuremberg, British Columbia, University College London, Oxford, and Berne, and obtained my MD through experimental laboratory work on systemic sclerosis. I completed residency and fellowship training in Internal Medicine and Nephrology at Hannover Medical School, where I also pursued postdoctoral training in experimental peritoneal dialysis. In 2019, I joined the laboratory of Professor Katalin Susztak at the University of Pennsylvania, where I studied single-cell transcriptomic responses in AKI and CKD. In 2022, I established my independent research group at Charité under the mentorship of Professor Kai-Uwe Eckardt. As principal investigator or co-investigator on multiple competitive fellowships and grants, including an ERC Starting Grant, I have laid the groundwork for the proposed research by developing and applying single-cell and systems-level approaches to identify adaptive versus maladaptive cellular programs in kidney injury and regeneration. My work has established mechanistic links between cellular differentiation trajectories, druggable pathways, and disease outcomes, as documented in the publications listed below. In parallel, I have successfully administered research projects, supervised trainees, collaborated across disciplines, and translated experimental findings toward clinically relevant questions.

Ongoing and recently completed projects that I would like to highlight include:

1. European Research Council (ERC) Starting Grant (SINGuLAR)
   Balzer (PI), 2026–2031, €1,500,000
   Mechanisms of kidney regeneration and adaptive repair

2. Clinician Scientist Program Fellowship, Berlin Institute of Health (BIH)
   Balzer (PI), 2023–2025, €60,000
   Cellular trajectories of repair and fibrosis following AKI

3. BIH Single Cell Approaches for Personalized Medicine Grant
   Balzer (Co-PI), Halbritter (Co-PI), Kaminski (Co-PI), 2025, €30,000
   Single-cell personalized medicine for ADPKD leveraging a founder mutation

4. Else Kröner Memorial Fellowship
   Balzer (PI), 2022–2024, €230,000
   Endophenotypes distinguishing adaptive and maladaptive regeneration in AKI

5. German Research Foundation (Deutsche Forschungsgemeinschaft, DFG) Grant Ba 6205/2-1
   Balzer (PI), 2019–2022, €173,000
   Single-cell mechanisms of renal regeneration and fibrosis in CKD

Selected Publications:

1. Miao Z.*, **Balzer, M.S.*** et al. (2021). Single cell regulatory landscape of the mouse kidney highlights cellular differentiation programs and disease targets. *Nat Commun*. doi:10.1038/s41467-021-22266-1
2. **Balzer, M.S.** et al. (2022). Single-cell analysis highlights differences in druggable pathways underlying adaptive or fibrotic kidney regeneration. *Nat Commun*. doi:10.1038/s41467-022-31772-9
3. **Balzer, M.S.**, Rohacs, T., Susztak, K. (2022). How many cell types are in the kidney and what do they do? *Annual Review of Physiology*. doi:10.1146/annurev-physiol-052521-121841
4. **Balzer, M.S.** et al. (2023). Treatment effects of soluble guanylate cyclase modulation on diabetic kidney disease at single-cell resolution. *Cell Rep Med*. doi:10.1016/j.xcrm.2023.100992
5. Abdank, K., et al., **Balzer, M.S.** (2024). A comparative scRNAseq data analysis to match mouse models with human kidney disease at the molecular level. *Nephrol Dial Transplant*. doi:10.1093/ndt/gfae030

B. Positions, Scientific Appointments, and Honors

Positions and Scientific Appointments

2025	Appointment to W2tt professorship for translational molecular nephrology and single-cell phenotyping (Kiel University)
2025	Habilitation and venia legendi (Privatdozent, Priv.-Doz.) for Internal Medicine and Nephrology, “Decoding phenotypes of kidney disease at the single-cell level”, Charité
2025–Present	Attending physician, Department of Nephrology and Medical Intensive Care, Charité
2024–Present	Fellow of the American Society of Nephrology (FASN)
2024–Present	Member, The New York Academy of Sciences (NYAS)
2024	Specialist in Internal Medicine and Nephrology
2023–Present	Fellow, Berlin Institute of Health (BIH), Charité Clinician Scientist Program
2023–Present	Board member, Diabetes and Metabolism commission (DGfN)
2022–Present	Group leader, Charité
2022–2025	Editorial fellow, *Journal of the American Society of Nephrology (JASN)*
2019–Present	Member, German Academic International Network (GAIN)
2019–2022	Member, Penn Program in Single Cell Biology (PPSCB)
2017–2019	Mentee, Nephrofuture mentoring program, class of 2017, DGfN
2015–Present	Member, American Society of Nephrology (ASN)
2015–Present	Member, Junge Niere (Young Kidney), DGfN

2015–Present	Member, European Renal Association-European Dialysis and Transplant Association (ERA-EDTA)
2015–Present	Member, ERA-EDTA Young Nephrologists' Platform
2014–Present	Member, DGfN
2014–Present	Member, International Society of Nephrology (ISN)
2014–Present	Member, International Society for Peritoneal Dialysis (ISPD)
2009–Present	Honorary member, Green Templeton College, Oxford

Honors

2023	Dr. Werner Jackstädt research prize for scientific excellence in AKI research, DGfN
2015–2025	Various (n=12) presentation & abstract awards (Keystone, DGfN, ERA/EDTA, Penn Genetics, Penn IDOM)
2010–2021	Various (n=11) travel grants & congress scholarships (Keystone, DGfN, ERA/EDTA, DAAD)

C. Contributions to Science

1. Redefinition of kidney cell states and maladaptive repair mechanisms in acute and chronic kidney disease
   My early and recent work has redefined cellular states in the kidney across multiple animal models of acute kidney injury (AKI) and chronic kidney disease (CKD) using single-cell transcriptomic approaches. These studies identified stromal cells and proximal tubule cells as key drivers of disease development and progression. In particular, I demonstrated that subsets of proximal tubule cells undergoing necrotic cell death programs, including pyroptosis and ferroptosis, play a central role in maladaptive repair processes following AKI and contribute to fibrotic remodeling. This work challenged traditional bulk-tissue paradigms of kidney injury and provided a mechanistic framework to distinguish adaptive regeneration from fibrotic outcomes at cellular resolution. These contributions have been recognized by scientific awards and have influenced the field's understanding of kidney injury biology.
   a. Balzer, M. S. et al. (2023). Treatment effects of soluble guanylate cyclase modulation on diabetic kidney disease at single-cell resolution. *Cell Rep Med*, doi:10.1016/j.xcrm.2023.100992
   b. Doke, T. et al. (2022). Single-cell analysis identifies the interaction of altered renal tubules with basophils orchestrating kidney fibrosis. *Nat Immunol*, doi:10.1038/s41590-022-01200-7
   c. Balzer, M. S., Rohacs, T. & Susztak, K. (2022). How Many Cell Types Are in the Kidney and What Do They Do? *Annu Rev Physiol*, doi:10.1146/annurev-physiol-052521-121841
   d. Balzer, M. S. et al. (2022). Single-cell analysis highlights differences in druggable pathways underlying adaptive or fibrotic kidney regeneration. *Nat Commun*, doi:10.1038/s41467-022-31772-9
   e. Miao, Z.* Balzer, M. S.* et al. (2021). Single cell regulatory landscape of the mouse kidney highlights cellular differentiation programs and disease targets. *Nat Commun*, doi:10.1038/s41467-021-22266-1
   f. Dhillon, P. et al. (2021). The Nuclear Receptor ESRRA Protects from Kidney Disease by Coupling Metabolism and Differentiation. *Cell Metab*, doi:10.1016/j.cmet.2020.11.011
   g. Balzer, M. S., Ma, Z., Zhou, J., Abedini, A. & Susztak, K. (2021). How to Get Started with Single Cell RNA Sequencing Data Analysis. *J Am Soc Nephrol*, doi:10.1681/ASN.2020121742
   h. Abedini, A. et al. (2021). Urinary Single-Cell Profiling Captures the Cellular Diversity of the Kidney. *J Am Soc Nephrol*, doi:10.1681/ASN.2020050757

2. Creation of widely used single-cell atlases and community-accessible kidney disease resources
   To accelerate discovery and reproducibility in renal research, I contributed to the development of interactive single-cell atlases of kidney disease across species and disease contexts. These publicly accessible resources enable researchers to explore gene expression patterns across cell types and disease states and have been accessed more than 10,000 times within the past year. By integrating single-cell transcriptomic data across mouse, rat, and human kidney disease models, this work provides a translational bridge between experimental systems and human pathology and supports hypothesis generation for therapeutic target discovery.
   a. Developing mouse kidney scRNA-seq & snATAC-seq atlas: URL
   b. Mouse kidney ischemia-reperfusion injury scRNA-seq atlas: URL
   c. Mouse kidney fibrosis (UUO) scRNA-seq atlas: URL

d. Rat diabetic kidney disease snRNA-seq atlas: URL
e. Human kidney spatial atlas: URL
f. Mouse cross-model kidney disease single-cell atlas: URL
g. Multispecies-integrated single-cell kidney atlas: URL

3. Pioneering single-cell and multi-omic approaches to study kidney disease progression and fibrosis
In addition to transcriptomic profiling, I was instrumental in applying state-of-the-art single-cell, spatial transcriptomic, and epigenomic approaches to define regulatory programs underlying kidney development, injury, and fibrosis. This work identified rare immune and stromal cell populations, including basophils, as key contributors to fibrotic microenvironments and demonstrated how altered epithelial-immune interactions drive disease progression. I also contributed to the first studies showing that urine-derived kidney cells recapitulate physiological and pathological kidney cell states, enabling non-invasive longitudinal monitoring of kidney disease. Collectively, this body of work established foundational methods and concepts that are now widely used in the field.
    a. Klötzer, K. A. et al. (2025). Analysis of individual patient pathway coordination in a cross-species single-cell kidney atlas. *Nat Genet*, doi:10.1038/s41588-025-02285-0
    b. Abedini, A. et al. (2024). Single-cell transcriptomics and chromatin accessibility profiling elucidate the kidney-protective mechanism of mineralocorticoid receptor antagonists. *J Clin Invest*, doi:10.1172/JCI157165
    c. Abedini, A. et al. (2024). Single-cell multi-omic and spatial profiling of human kidneys implicates the fibrotic microenvironment in kidney disease progression. *Nat Genet*, doi:10.1038/s41588-024-01802-x
    d. Abdank, K. et al. (2024). A comparative scRNAseq data analysis to match mouse models with human kidney disease at the molecular level. *Nephrol Dial Transplant*, doi:10.1093/ndt/gfae030
    e. Zhou, J. et al. (2023). Unified Mouse and Human Kidney Single-Cell Expression Atlas Reveal Commonalities and Differences in Disease States. *J Am Soc Nephrol*, doi:10.1681/ASN.0000000000000217

Complete List of Published Work
https://pubmed.ncbi.nlm.nih.gov/?term=%28Balzer+MS%29OR%28S+Balzer+M%29&sort=date