interdisziplinäres Zentrum für Klinische Forschung

Clinician Scientist Programme - Biosketch Module Step 2

Title/ First Name/ Last Name

Dr.med. Tilman Jobst-Schwan

Clinic

Nephrology and Hypertension

CV (Current position, academic career, achievements and extracurricular activities, languages)

Clinical experience
10/2018 – present Resident and Clinician scientist at the Department of Nephrology and Hypertension, Friedrich-Alexander-University Erlangen-Nuremberg, Germany
03/2013 – 04/2015 Resident at the Department of Nephrology and Hypertension, Friedrich-Alexander-University Erlangen-Nuremberg, Germany

Scientific experience
01/2021 – present Principal Investigator, Department of Nephrology and Hypertension, Friedrich-Alexander-University Erlangen-Nuremberg, Germany
01/2021 – present RECORD – Research Center On Rare Kidney Diseases fellowship
10/2018 – 09/2021 Project leader Junior project J70 "Gene discovery in kidney disease" (funded by the Interdisciplinary Center for Clinical Research Erlangen)
04/2015 – 07/2018 Postdoctoral Research Fellow in the laboratory of Prof. Friedhelm Hildebrandt, MD, Division of Nephrology, Boston Children´s Hospital, Harvard Medical School, Boston, MA, USA
07/2014 Doctoral Degree under supervision of Prof. Michael S. Wiesener, MD, Department of Nephrology and Hypertension, Friedrich-Alexander-University Erlangen-Nuremberg, Germany; Grade summa cum laude

Honors and awards
08/2015 – 07/2018 Research fellowship of the German Research Foundation (DFG)
2018 Podocyte Conference 2018 Poster Award
2016 and 2019 ASN Kidney Week ARC Travel Award
2016 Marine Biology Laboratory scholarship "Zebrafish genetics and development"
2015 Doctoral thesis award of the Research Foundation (University Hospital Erlangen)

Additional training
11/12/2020 Good Scientific Practice (GSP) course (Interdisciplinary Center for Clinical Research, Erlangen, Germany)
22/01/2020 Good Clinical Practice (GCP) basic course (Winicker Norimed, Nuremberg, Germany)
10/2019 8th Annual International Zebrafish husbandry Course, Buguggiate, Italy
08/2016 Course "Zebrafish genetics and development" at the Marine Biology Laboratory, Woodshole, MA, USA

Languages
German, native language

Research Area

Renal and Vascular Research

Nephrology

CSP Project Outline

Proteinuria describes the state of plasma protein loss via the urine. Proteinuric kidney diseases is divided into glomerular or non-glomerular forms, depending on whether protein loss occurs across the glomerular filtration barrier or results from impaired reabsorption of filtered protein by the proximal tubule.

Glomerular forms are caused by impairment of the glomerular epithelial cell, the podocytes, either as a primary podocytopathy or secondary to external factors. Primary podocytopathies often have a genetic background with mutations affecting the function of structural proteins of the filtration barrier or proteins involved in regulation of the actin cytoskeleton. Secondary causes (that may have a genetic background as well) include metabolic conditions like diabetes mellitus or adipositas, and dysfunction of the immune system.

In an internal cooperation with the group of Prof. Michael Wiesener and the Institute of Human Genetics we are employing Whole Exome Sequencing to identify underlying genetic causes in our local patients with proteinuric kidney disease, and employ patient derived primary cells to analyze the detrimental effects of the candidate variants identified.

In this context, we are focusing primarily on the Wnt/β-catenin pathway, a signaling pathway that is important in development, but which is reactivated in podocytes and tubular cells upon cellular stress (such as proteinuria) as a protective mechanism. However, maintained Wnt/β-catenin activation is detrimental to kidney function, and drives cellular dedifferentiation with progression to chronic kidney disease. We aim to understand the underlying mechanisms and how we can modify these to alleviate disease progression. For this purpose, we are employing cell culture models, as well as zebrafish and mouse animal models.

Methods, Skills and Know-How

Nephrogenetics, Whole Exome Sequencing analysis, zebrafish animal model

Current Funding

DFG, Else Kröner-Fresenius Memorial Stipendium 2020, RECORD Program

Intended Funding Applications

SFB, IZKF

Most important publications (3)

Jobst-Schwan, T, Klambt, V, Tarsio, M, Heneghan, J F, Majmundar, A J, Shril, S, Buerger, F, Ottlewski, I, Shmukler, B E, Topaloglu, R, Hashmi, S, Hafeez, F, Emma, F, Greco, M, Laube, G F, Fathy, H M, Pohl, M, Gellermann, J, Milosevic, D, Baum, M A, Mane, S, Lifton, R P, Kane, P M, Alper, S L, and Hildebrandt, F: Whole exome sequencing identified ATP6V1C2 as a novel candidate gene for recessive distal renal tubular acidosis. Kidney international 97, 567-579, 2020.
Jobst-Schwan, T, Hoogstraten, CA, Kolvenbach, CM, Schmidt, JM, Kolb, A, Eddy, K, Schneider, R, Ashraf, S, Widmeier, E, Majmundar, AJ, Hildebrandt, F: Corticosteroid treatment exacerbates nephrotic syndrome in a zebrafish model of magi2a knockout. Kidney international, 95: 1079-1090, 2019.
Jobst-Schwan, T, Knaup, KX, Nielsen, R, Hackenbeck, T, Buettner-Herold, M, Lechler, P, Kroening, S, Goppelt-Struebe, M, Schloetzer-Schrehardt, U, Furnrohr, BG, Voll, RE, Amann, K, Eckardt, KU, Christensen, EI, Wiesener, MS: Renal uptake of the antiapoptotic protein survivin is mediated by megalin at the apical membrane of the proximal tubule. American journal of physiology Renal physiology, 305: F734-744, 2013.