New Insights into Podocyte Biology in Glomerular Health and Disease

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ABSTRACT

Podocyte and glomerular research is center stage for the development of improved preventive and therapeutic strategies for chronic progressive kidney diseases. Held April 3–6, 2016, the 11th International Podocyte Conference took place in Haifa and Jerusalem, Israel, where participants from all over the world presented their work on new developments in podocyte research. In this review, we briefly highlight the advances made in characterizing the mechanisms involved in podocyte development, metabolism, acquired injury, and repair, including progress in determining the roles of genetic variants and microRNA in particular, as well as the advances made in diagnostic techniques and therapeutics.

The meeting began with the newest work on the origin of podocytes and glomerular development (Figure 1). Nephron and subsequently, podocyte numbers have been shown to influence long-term kidney health.1,2 Therefore, the ability of podocyte precursors to maintain the progenitor cell pool throughout kidney development is paramount for nephron endowment. During development, nephron progenitor cells decide whether to self-renew or exit the progenitor population to form new nephrons until a final burst of differentiation results in the complete depletion of progenitor cells.3 Kopan and colleagues4 showed that nephron progenitor cells undergo transient intrinsic changes during aging, resulting in an increasing propensity of cells to exit the progenitor pool at later stages of development, whereas cell-cell contacts with younger cells rejuvenated older progenitors, indicating an important role for the stem cell niche during the course of development. On differentiation requiring Wnt signals, the nephron precursors are polarized early on, resulting in segmentation and patterning of the nephron.

In the chick embryo, Schultheiss and colleagues5 showed that Wnt signals are also necessary to determine the proximal distal nephron axis, because ectopic Wnt signals could repress podocyte transcription factors Wt1 and Tcf21/Pod1, leading to reorientation of the nephron.

Next to influencing nephron numbers, faulty differentiation functions as a cause for transformation and development of Wilms tumor. Dekel and colleagues6 could trace this to the markers NCAM1 and CD133 in the human fetal kidney, which are expressed in opposing gradients in multipotent renal cells and differentiated tubular structures, respectively, with the NCAM1+/CD133−/FDZ7+/ALDH1+ population harboring Wilms tumor cancer stem cells. Using anti-NCAM1 immunocjugates in combination with the newly identified markers could potentially lead to more effective targeted treatment strategies for patients with Wilms tumor.

GLOMERULAR STRUCTURAL AND FUNCTIONAL INTEGRITY

On differentiation, podocytes develop regularly spaced foot processes, which control glomerular filtration via the slit diaphragm. The assembly of the slit diaphragm was originally described as a zipper-like structure.7 However, new technologies indicate that the condensed midline seen in electron tomography might stem from fixation artifacts. Using cryoelectron microscopy techniques, Huber et al.8 showed that the two main components of the slit, nephrin and

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Neph1, form two distinct compartments with different widths (45 and 23 nm, respectively) on top of each other, representing a multilayered sieving structure.

Not only the podocyte, but also the other components of the glomerular filtration barrier, the fenestrated endothelium and the glomerular basement membrane (GBM), are known to influence filtration, and changes in this part of the filtration barrier can lead to albuminuria. Foster et al. focused on the endothelial glycocalyx, an intravascular compartment that is frequently sulfated and negatively charged, which protects the vessel wall against pathogenic insults. When disrupted, such as in early diabetes, the endothelial glycocalyx promotes an inflammatory environment and becomes leaky. Foster and colleagues showed in vitro vascular permeability measurements using perfusion with fluorescent-labeled albumin as readout of the effects of diabetes or various vascular endothelial growth factor isoforms and angiopoietins on endothelial glycocalyx and permselectivity.

The extracellular matrix (ECM) plays a crucial role in glomerular function, which has been shown in genetic diseases caused by mutations of COL4A5 (Alport syndrome) or LAMB2 (Pierson syndrome) among others. Using serial block face-scanning electron microscopy, Lennon and colleagues elegantly showed in three-dimensional (3D) glomerular reconstruction models that podocyte projections invade the GBM with disease progression in an Alport Col4a3−/− mouse model but not in Col4a3+/− or wild-type controls. Furthermore, they deciphered the ECM of human glomeruli with mass spectrometry, confirming many published components and adding novel ECM structural and regulating proteins to the human “matrisome,” the matrix signature of the glomerulus. Using C57BL/6 (glomerular disease resistant) and FVB (glomerular disease susceptible) mouse strains, they also revealed a shift in the matrisome, which is dependent on both mouse background and sex. Analysis of in vitro-cultured human podocytes on different ECM and glomerular endothelial cells suggested a pivotal role of cell-cell crosstalk in the production of the ECM.

**PODOCYTE CELL SIGNALING AND METABOLISM IN HEALTH AND DISEASE**

Podocyte metabolism has been shown to be controlled by the mammalian target of rapamycin (mTOR) pathway, which is upregulated in several glomerular disease states. Nevertheless, podocyte and glomerular injury could be triggered by direct inhibition of mTOR complex components, making mTOR inhibition problematic as a therapeutic means. New data from Inoki and colleagues showed, both in vivo and in vitro, that an increase in nutrients leads to recruiting of mTORC1 to lysosomal membranes by the Ragulator complex. Interestingly, knockout of Ragulator component p18 maintained basal TORC1 activity in podocytes but attenuated its robust activation and subsequent cell injury under diabetic conditions. This points to mTOR-lysosome recruitment as a potential therapeutic target for the treatment of glomerular disease.

In recent years, mitochondria have emerged as crucial contributors to glomerular health and disease. In early diabetic nephropathy and other glomerular diseases, mitochondrial dysfunction, altered number, and dynamics were observed and suggested to be involved in glomerular cell crosstalk. He and colleagues showed that, in adriamycin-induced nephropathy, Krüppel-like factor 6, a transcription factor and essential regulator of mitochondrial function, is upregulated, thereby protecting against mitochondrial injury and podocyte apoptosis. Conversely, susceptibility to developing adriamycin-induced nephropathy and focal segmental glomerulosclerosis (FSGS) was increased in the absence of Krüppel-like factor 6 in podocytes of C57BL/6 mice.

Additional metabolic function is performed by the mitochondrial protein...
Prohibitin-2 (PHB2), which was shown by Brinkkoetter and colleagues\(^{28}\) to also localize to the slit diaphragm and interact with Podocin. Podocyte-specific knockout of PHB2 presented with mitochondrial dysfunctions, whereas knockout of PHB2 orthologs in Caenorhabditis elegans showed impaired touch sensitivity in mechanosensory neurons, indicating an additional function at the plasma membrane and slit diaphragm.\(^{28}\)

Podocytes are highly dependent on their actin cytoskeleton to stabilize their complex architecture. FERM-domain protein EPB4.1L5, which links the actin cytoskeleton to cell membrane proteins, is highly enriched in podocytes and conserved in evolution. Schell showed that depletion of EPB4.1L5 from podocytes leads to proteinuria, foot process effacement, and early lethality from kidney disease (C. Schell, unpublished data). In vitro studies of knockout cells show impaired spreading and contractility behavior, indicating a role for EPB4.1L5 in generating focal adhesions (C. Schell, unpublished data).

Gain of function mutations in the transient receptor potential channel C6 (TRPC6) have been shown to cause familial FSGS.\(^{29}\) Because GPCR/Gq signaling can activate TRPC6, Spurney and colleagues\(^{30}\) overexpressed Gq\(^{Q209L}\) in the mouse podocyte. This led to calcineurin-dependent upregulation of TRPC6 and aggravation of puromycin aminonucleoside nephropathy nephrosis with albuminuria and 50% FSGS.\(^{30}\) Conversely, knockout of TRPC6 in these mice was able to prevent FSGS and proteinuria in puromycin aminonucleoside nephropathy nephrosis, indicating a potential therapeutic benefit from targeting Gq/TRPC6 signaling.

**IMAGING AND INTRACELLULAR TRAFFICKING**

Rac and Rho are in mutual balance in podocytes under normal conditions.\(^{31}\) However, on podocyte injury, Rac is upregulated, leading to changes in podocyte morphology, detachment, and migration.\(^{32}\) Shaw and colleagues\(^{33,34}\) used intravitral (in vivo) and kidney slice two-photon (in vitro) imaging to monitor the motility of podocytes. On constitutive activation of Rac1, podocytes showed increased membrane dynamics, foot process retraction, and podocyte motility, occasionally even crawling through tubular structures.\(^{33,34}\) Increased membrane ruffling was also seen in nephrotic serum nephritis, indicating Rac and Rho as potential future therapeutic targets.

In the zebrafish pronephros, the fused glomerulus is an easy target for live imaging.\(^{35}\) Using different-sized fluorescent dyes, Endlich and colleagues\(^{36}\) were able to monitor podocyte behavior, filtration, and barrier function of the GBM. In larvae, two-photon imaging of podocyte foot processes, up to 26 hours, showed stationary behavior of podocytes, with the Nitroreductase/Metrodinazole–induced injury model leading to podocyte apoptosis, foot process effacement, and edema but not leading to migration on the denuded GBM.\(^{36}\) In zApol1 or Myh9-morpholino–injected larvae, intravascular injection of dextran showed compromised filtration with loss of the 78-kD vitamin D binding protein DBP-EGFP.\(^{37,38}\)

Using imaging of the intact kidney via an abdominal window, Peti-Peterdi and colleagues\(^{39}\) could show with in vivo multiphoton microscopy that laser injury/excitation of one podocyte can lead to spreading of Ca\(^{2+}\) across neighboring cells. Using different inducible Cre lines labeling cells from the macula densa and mesenchyme, cells could be imaged over time migrating into the glomerulus in disease conditions. This phenomenon was increased with low-salt diet and angiotensin-converting enzyme inhibition and may point to a contribution of macula densa cells in glomerular disease (J. Peti-Peterdi et al., unpublished data).

Actin is known to play a crucial role in podocyte architecture. Sever showed that actin also plays multiple roles in endocytosis (S. Sever, unpublished data); 40% of clathrin-coated pits are associated with actin. Using rhodamine-conjugated transferrin, internalization was visualized by structured illumination microscopy–total internal reflection fluorescence microscopy. Using this approach, the initiation density of clathrin-coated pits could be shown to be sensitive to actin drugs and alter membrane tension. Using atomic force microscopy, the actin cytoskeleton was also shown to regulate membrane stiffness (S. Sever, unpublished data).

**ORGANISMSAL AND STEM CELL EXPERIMENTAL PLATFORMS FOR GLOMERULAR AND PODOCYTE FUNCTION IN HEALTH AND DISEASE**

New progress has been made in the generation of kidney organoids from induced pluripotent stem cells.\(^{40,41}\) Here, Freedman\(^{42}\) presented his work on two-dimensional and 3D cultures using sequential induction of stem cells by the GSK3\(^{b}\) inhibitor CHIR, activin, and FGF9. By replicating developmental pathways and tissue induction, 3D cultures formed spheroids, which differentiated into tubular organoids on CHIR treatment. In these, podocyte as well as tubular markers could be identified. Freedman et al.\(^{40,42}\) also showed that these organoids can be used as a model for nephrotoxicity and genetically modified using CRISPR/Cas9 to study knockout phenotypes, such as polycystic kidney disease 1 or polycystic kidney disease 2, which correspondingly formed cysts in induced pluripotent stem cell–derived organoids.

High plasma levels of soluble urokinase-type plasminogen activator receptor were proposed to be from an unknown nonkidney origin, and they were shown to be associated with FSGS and predictive for chronic kidney disease (CKD).\(^{43,44}\) Using a series of bone marrow chimera, cell ablation, and transfer studies as well as a humanized mouse models from patients with FSGS, Gr-\(^{lo}\) immature myeloid cells turned out to be a main source for elevated soluble urokinase-type plasminogen activator receptor in renal disease.\(^{45}\)

The zebrafish (Danio rerio) has served as an informative vertebrate model readily
amenable to genetic interrogation. Endlich and colleagues\textsuperscript{37,46} provided informative examples, including a phenotype resulting from the interaction of the endogenous zebrafish sole homolog of the \textit{APOL1} gene family. This led to an interesting consideration of the gain versus loss of function modes of injury in the evolutionary history of the gene family.\textsuperscript{37,46}

A recently emerging model organism for podocyte biology is the Drosophila nephrocyte with its Garland cells and pericardial nephrocytes resembling podocytes. Drosophila provides a model that is readily amenable to genetic manipulation, and therefore, it is used for disease gene validation by various groups.\textsuperscript{47} Han and colleagues\textsuperscript{48} investigated uptake of Red Fluorescent Protein in the pericardial nephrocytes by screening 3800 genes with 268 hits, such as RabGTPases Rab5, -7, and -11, leading to changes in nephrocyte morphology and endocytosis. They furthermore showed that knockdown of dKANK, the Drosophila homolog of kidney Ankyrin repeat-containing proteins KANK1, -2, and -4 (newly identified as disease-causing genes in patients with steroid-resistant nephrotic syndrome [SRNS]), has evolutionary conserved functions in the nephrocyte.\textsuperscript{48}

### Mechanistic Insights from the Genetics of Glomerular Disease

Point mutations in single genes that cause disruption of structure and function of encoded proteins, leading to diseases or syndromes, such as FSGS, are one of the strongest cause-effect relationships observed in clinical medicine. To date, mutations in \textgreek{>}40 podocyte genes, \textgreek{>}32 with recessive mode of inheritance and eight with dominant mode of inheritance, have been identified in patients with SRNS that also exemplify genetic locus heterogeneity. Recently, in a worldwide cohort of 1782 families, Hildebrandt and colleagues\textsuperscript{49} found single-gene mutations in about 30% of young patients with SRNS, of which 10% and 7% can be attributed to mutations in the \textit{NPHS2} and \textit{NPHS1} genes, respectively. Interestingly, many products of these genes interact in protein-protein complexes, mostly involved in the slit diaphragm, Actin binding and regulation, Integrin/Laminin signaling, calcium signaling, lysosome, CoQ10 biosynthesis, nuclear transcription factors, and nucleopores.\textsuperscript{50} Hildebrandt also identified six novel monogenic disease genes causing partial treatment sensitivity to corticosteroids manifesting as steroid-dependent or frequently relapsing nephrotic syndrome (F. Hildebrandt, unpublished data). The genes are involved in the regulation of Rho/Rac/Cdc42 signaling, underscoring the importance of balanced Rac/Rho signaling for podocyte function, migration, foot process formation, and slit diaphragm signaling and function (F. Hildebrandt, unpublished data).

\textit{APOL1} gene variants, G1 and G2, are highly associated with risk for FSGS and other progressive nephropathies in patients of West African ancestry.\textsuperscript{51,52} The genetics indicate a recessive mode of inheritance, whereas the biology points to a gain of function/injury rather than a loss of function. \textit{APOL1} is expressed in podocytes; however, not all patients carrying the risk alleles develop renal disease. The exact mechanisms of toxicity of risk variants are not fully understood, even under meticulous investigation.\textsuperscript{53–55} Kopp reported on a novel \textit{APOL1} splice variant, B3 (J. Kopp, unpublished data). This splice variant does not express exon 4 and is intracellular. B3 enhances the inflammasome and associates with NLRP12 and NLRP3. Expression of \textit{APOL1}-B3 in the podocyte enhances podocyte damage after LPS treatment. Protein kinase R (PKR), which is a central player in the innate immune system and antiviral response, is increased by \textit{APOL1} and leads to increased proteinuria. A mechanism whereby \textit{APOL1} variant mRNA transcripts might engage PKR in cell injury was presented. PKR could also be shown to be increased in human kidney biopsies.

Susztak and colleagues\textsuperscript{54} developed a mouse model with inducible podocyte-specific expression of G1 and G2 \textit{APOL1} alleles. The model resembles human disease at the functional, structural, and molecular levels. Mechanistically, Apol1 G1 and G2 expression likely results in a gain of function injury, with evidence for interference in intracellular vesicle trafficking.

Type IV collagens (encoded by \textit{COL4A1}–\textit{COL4A6} genes) are crucial components of basement membranes; \textalpha1 \textalpha2(IV) is the most abundant collagen trimmer in the embryonic kidney and other organs, partly switching to \textalpha3 \textalpha4 \textalpha5(IV) and \textalpha5 \textalpha5 \textalpha6(IV) in the adult kidney.\textsuperscript{56} Although Alport syndrome affects \textit{COL4A3}, \textit{COL4A4}, and \textit{COL4A5} genes, mutations in \textit{COL4A1} lead to \textit{COL4A1}-related brain small vessel disease, familial porencephaly, and hereditary angiopathy with nephropathy, aneurysms, and muscle cramps syndrome. The G498V mutation causing the latter was analyzed by Plaisier and colleagues\textsuperscript{57} in a mouse model. The mice presented with mild proteinuria, glomerular defects, foot process effacement, and glomerular basement duplications. Newborn mice showed delayed glomerulogenesis, glomerular cysts, and reduced nephrin expression, indicating the developmental importance of \textit{COL4A1}. At the molecular level, the G498V mutation led to activation of CD44, pointing toward activation of parietal epithelial cells, collagen receptor DDR1 and ILK activation-regulating ECM-induced signaling pathways, and matrix metalloproteinase induction.\textsuperscript{57}

### The Role of MicroRNAs in the Podocytes

Progress was made in the analysis of microRNA (miRNA) expression and function in podocytes. These approximately 22-nucleotide-long noncoding RNAs are crucial for the post-transcriptional regulation of gene expression. They have come into the focus of podocyte research in the past decade in both health and disease, emerging as biomarkers, regulators, and potential targets of future therapies.\textsuperscript{58–60} Schiffer tested patient urine to gain cell- and disease-specific
miRNA signatures in various glomerulonephritides (M. Schiffer, unpublished data). In membranous nephropathy (MN), seven podocyte-specific miRNAs were detected, such as miR-378a, which is specific for Nephronectin (NPNT). NPNT localizes to the ECM and is inversely correlated with the expression of miR-378a. TGFβ induces expression of miR-378a, leading to loss of NPNT expression. In a zebrafish model, knockdown of NPNT or expression of miR-378a leads to edema, podocyte foot process effacement, and altered GBM (M. Schiffer, unpublished data).

In an attempt to explain why diverse courses of disease occur among patients with diabetic kidney disease (DKD), Gupta and colleagues found differential expression of miR-146a, which is known to be involved in immune system regulation. In humans, reduction of miR-146a expression was correlated with disease progression and high-grade proteinuria. At the molecular level, target genes of miR-146a, ErbB4 (a member of the epidermal growth factor receptor [EGFR]) and Notch-1, were upregulated in DKD. In miR-146a knockout mice, streptozotocin-induced hyperglycemia significantly accelerated development of albuminuria and glomerular injury compared with in the wild-type mice. Blocking ErbB4 with a known antagonist, erlotinib, protected animals from the development of albuminuria and significantly reduced glomerular damage, suggesting a role for EGFR cell signaling in DKD.

Recently, the beneficial effect of erlotinib and the role of EGFR signaling in glomerular crescent formation have been also shown by Tharaux and colleagues. To further unravel the consequences of activated EGFR signaling, Tharaux took a closer look at Stat3, which increases in many glomerular diseases, with explicit attention to miRNA-92a, a known downstream target of Stat3 (P.L. Tharaux, unpublished data). He could show that upregulation of miR-92a controls the expression of cell cycle regulator p57 specifically in podocytes, therefore presenting as a potential key regulator of podocyte quiescence (P.L. Tharaux, unpublished data). This effect could be reversed by an antagonomir, which delayed or prevented crescent formation (P.L. Tharaux, unpublished data).

**ADVANCES IN GLOMERULAR AND PODOCYTE DIAGNOSTICS AND THERAPEUTICS**

The concept of “fatty kidney” was introduced recently and describes the accumulation of lipid droplets in glomerular and tubular cells. Herman-Edelstein et al. suggested that disturbed lipid metabolism in DKD and obesity-related glomerulopathy has a role in disease progression. Lipid deposition is commonly seen in histopathologic samples of human DKD, causing dysregulation of fatty acid metabolism and lipotoxicity that correlates with renal damage. The public health effect is noteworthy as a component of what has been termed as “diabesity.”

Fornoni and colleagues analyzed the mechanism of injury related to lipids accumulating in sera of patients with types 1 and 2 diabetes mellitus. They identified tumor necrosis factor-α (TNF-α) and its receptors as biomarkers for DKD progression, causing downregulation of ATP binding cassette transporter A1-mediated cholesterol efflux.
and reduced cholesterol esterification by sterol-O-acyltransferase 1. Subsequent cholesterol accumulation in podocytes causes cell apoptosis via nuclear factor of activated T cells (NFAT). Of note, local TNF expression is sufficient to cause podocyte injury and likely independent of its receptors or circulating serum levels of TNF. The decreased cholesterol efflux in mouse models was counteracted using overexpression of ATP binding cassette transporter A1 or the drug cyclodextrin, which induces intracellular cholesterol depletion. This cholesterol efflux pathway needs to be further explored as a potential target for alleviating other proteinuric glomerular diseases.

In the kidney, the chemokine SDF-1/CXCL12 is exclusively expressed by podocytes. Anders and colleagues could show that an SDF-1 Spiegelmer counteracts the inflammatory reaction, leading to less proteinuria and an increase in the number of podocytes. Using undifferentiated human progenitor cells, it was shown that blocking SDF-1 activated progenitor cells, indicating that SDF-1 promotes parietal epithelial cell quiescence. Recently, an integrative biology approach on the basis of large datasets of prospective European and North American patient cohorts with glomerular diseases, seeks to reclassify patients on the basis of mechanistic rather than histopathologic patterns of injury. Recognizing the diversity in presentation, progression, and response to therapies among patients, this paradigm shift will enable clinicians to tailor future precise therapies. Kretzler and colleagues showed a correlation of urinary EGF with intrarenal EGF expression, interstitial fibrosis, and tubular atrophy. This makes urinary EGF a potential noninvasive biomarker to predict CKD progression in patients with chronic glomerular diseases. They also showed that shared transcriptional signatures divide minimal change disease and FSGS data into distinct subgroups. One FSGS cluster revealed no remission of proteinuria when followed over time. In this group, differentially regulated gene clusters were shown to be downstream of the TNF activation pathway. Therefore, the challenge was again to identify a noninvasive biomarker from proteomic data comparing kidney biopsy tissue and urine. A set of molecules (e.g., Monocyte chemoattractant protein-1) predicted upregulation of TNF and showed correlation with urinary biomarkers.

Another example of precision medicine is the discovery of M-type phospholipase A2 receptor (PLA2R) as an autoantigen in MN, which transformed our understanding of MN to an autoimmune disease. About 70% of patients with MN develop autoantibodies, mostly IgG4, reacting with PLA2R. The immunodominant epitope was identified, and high anti-PLA2R activity and epitope spreading beyond the cysteine-rich region were correlated with poor renal outcome. The seminal study of Salant and colleagues (Salant was the recipient of the 2016 Marilyn Farquhar Award at the conference) set the stage for later identification of Thrombospondin type 1 domain–containing 7A as a second autoantigen in about 5% of anti–PLA2R-negative patients with MN.

Recent scientific exchange and integrative approaches in the field of glomerular diseases, especially podocytopathies, have vastly enhanced the depth of our understanding and accelerated pathophysiologic insights. Correspondingly, the emerging knowledge and technologies are already proving useful in the reclassification of diseases on the basis of mechanism, risk stratification of patients, and identification of diagnostic and progression biomarkers as well as target identification for the development of novel therapeutics, some of which have already reached the stages of clinical implementation of investigation and validation in clinical trials (Figure 2).

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DISCLOSURES

None.

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