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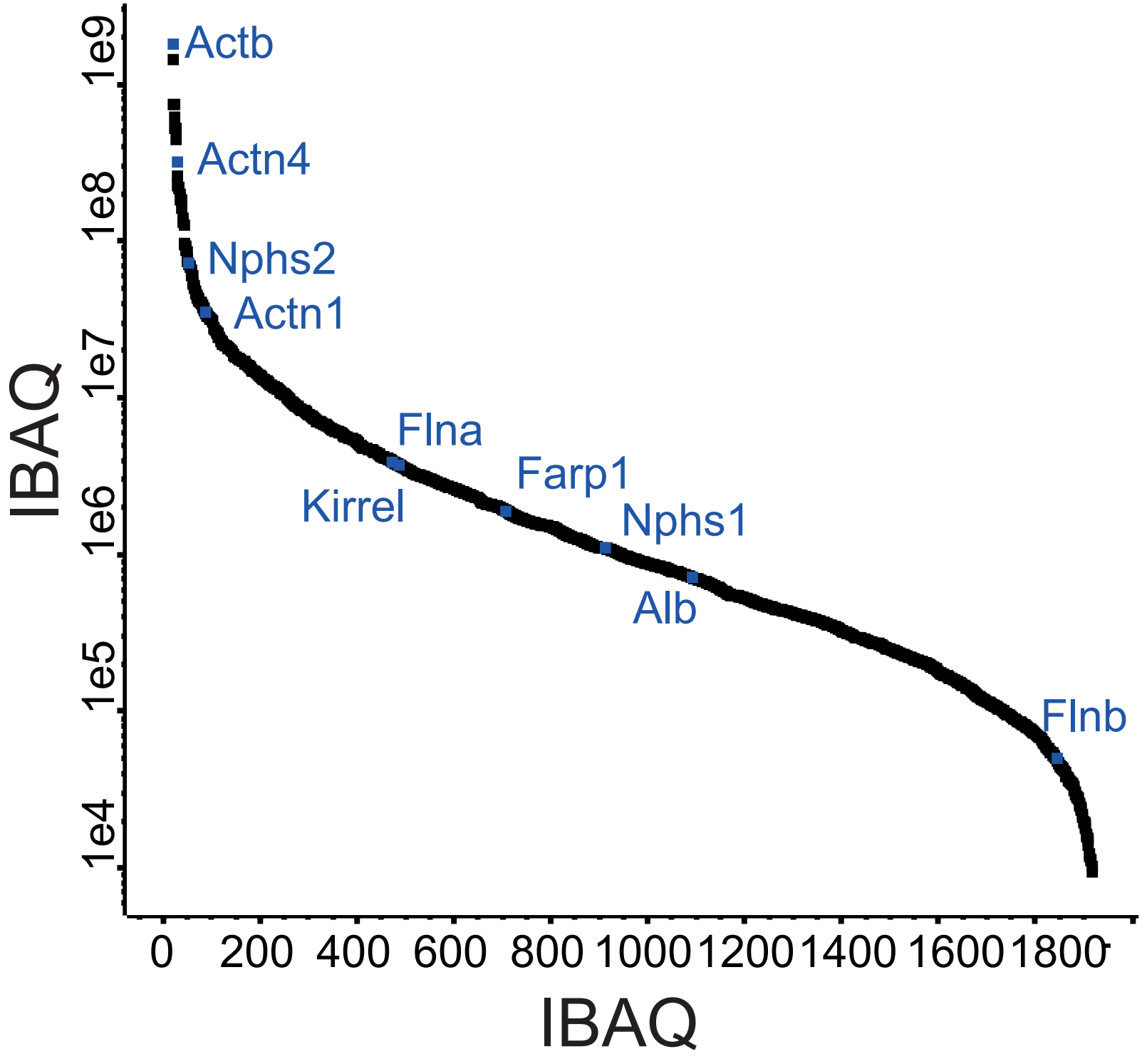
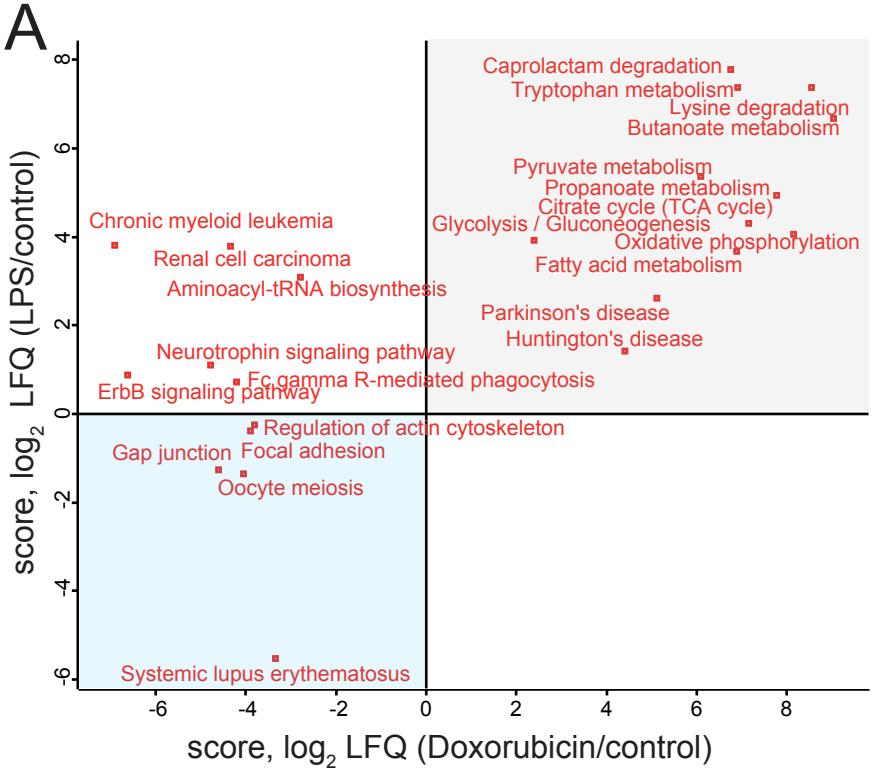
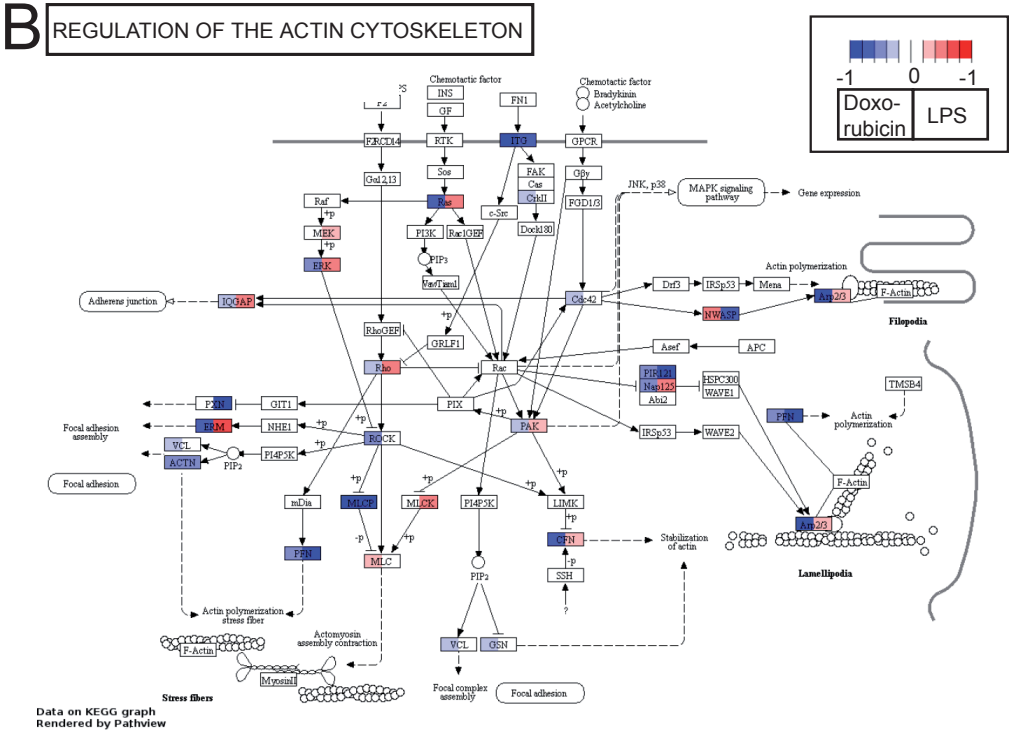
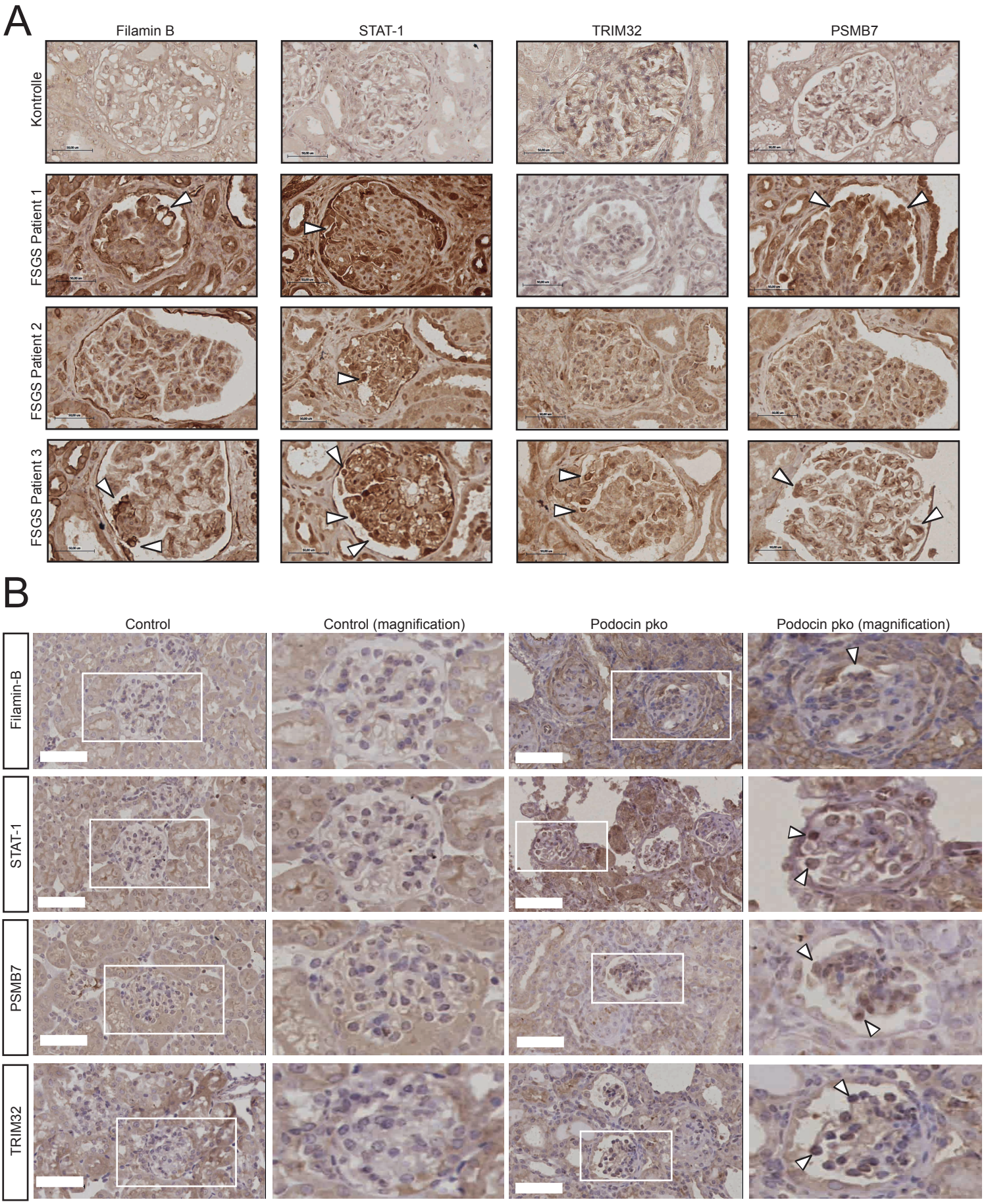


Supp Figure 1 Koehler et al.





Supp Figure 2 Koehler et al.



Supp Figure 3 Koehler et al.

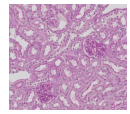
Proteomics of human tissue

Proteomics of podocyte injury

Priorization

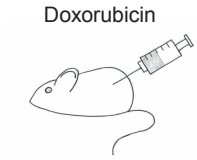
Functional analysis

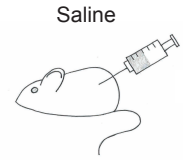
human FSGS patient glomerular proteomics



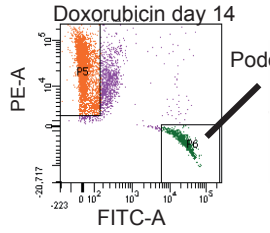
underrepresentation of podocyte-specific proteins

Ultrasensitive proteomics

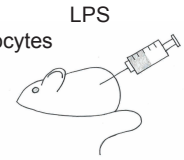




Isolation of native podocytes



FACS sorting



Isolation of native podocytes

225 changed proteins

120 changed proteins

Correlation of phenotype and protein expression

Increased Filamin-B

higher expression of Filamin-B in human FSGS and Podocin ko tissue

Filamin-Albumin-correlation in rat glomeruli treated with PAN

elevated filtration upon loss of Cher/dFilamin in *Drosophila* nephrocytes

increased Filamin levels in cells exposed to mechanical-stress

Disease mechanism

Supplementary Figure Legends

Supplemental Figure 1: Dynamic range. Protein copy numbers of podocytes isolated from a single mouse. The podocyte protein copy numbers are depicted as log10(iBAQ), a parameter corresponding to protein copy number.

Supplementary Figure 2: Different modes of injury cause an upregulation of metabolic signaling and a down-regulation of actin-cytoskeleton associated pathways. A Comparison of LFQ values of Doxorubicin and LPS treated animals using KEGG pathways revealed an up-regulation of metabolic signaling including citrate cycle, glycolysis and fatty acid metabolism. A common down-regulation was observed for actin-cytoskeleton associated pathways such as gap junctions, focal adhesions and regulation of the actin cytoskeleton. **B** KEGG pathway analysis revealed differential regulation of actin-cytoskeleton associated proteins in Doxorubicin and LPS treated mice.

Supplemental Figure 3: Immunhistochemistry on human FSGS and Podocin knockout tissue revealed an increased Filamin-B expression A Immunohistochemistry of FSGS patient samples revealed an increased expression of Filamin-B, STAT-1, Trim32 and PSMB7 upon podocyte injury. Scale bar = 50 um. Arrowheads mark positive podocytes. **B** Immunohistochemistry of kidney tissue from Podocin knockout mice also revealed an increased expression of Filamin-B, STAT-1, TRIM32 and PSMB7 upon podocyte injury. **C,D** Human and mouse tissue stained without a primary antibody served as negative control. Scale bar = 50 um.

Supplemental Figure 4: Overview of this study demonstrating the applicability of sensitive proteomics for phenotype-proteome correlations.