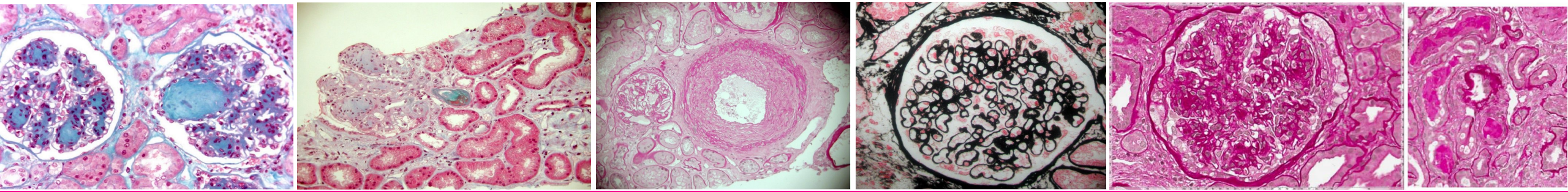
Dal fenotipo all'endotipo nella malattia renale cronica e DMT2: ex uno, plures

Loreto Gesualdo, MD, FERA

UNIVERSITÀ
DEGLI STUDI DI BARI
ALDO MORO



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Disclosures

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UNIONE EUROPEA
Fondo Sociale Europeo
AIM: Attraction and International Mobility
PON Ricerca e Innovazione 2014-2020

Codice progetto
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Ministero dell'Istruzione, dell'Università e della Ricerca

PON RICERCA E INNOVAZIONE 2014 - 2020

BEAT DKD

Spin-off e Start-up:

Persongene
MedPath

Speaker activity:

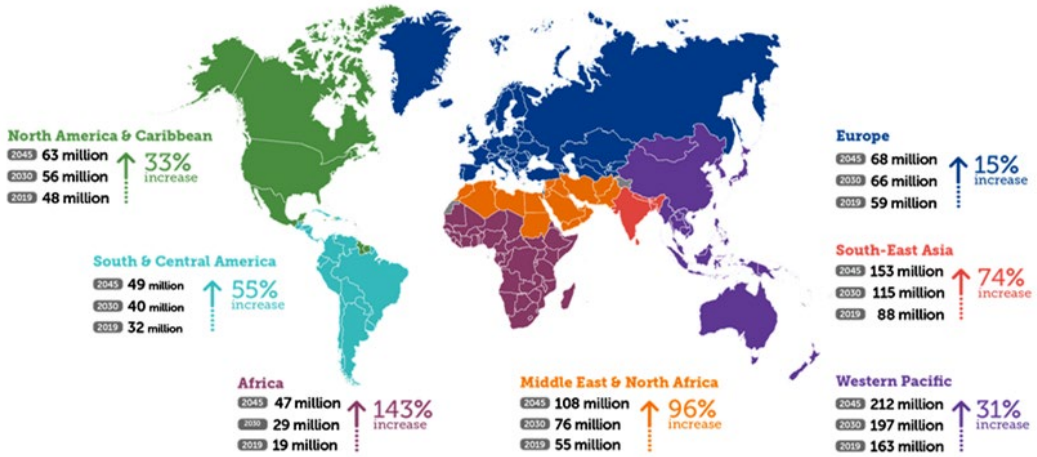
Fresenius
Estor
Werfen
Astellas
AstraZeneca
Travere
Bayer
Dr. Schaer

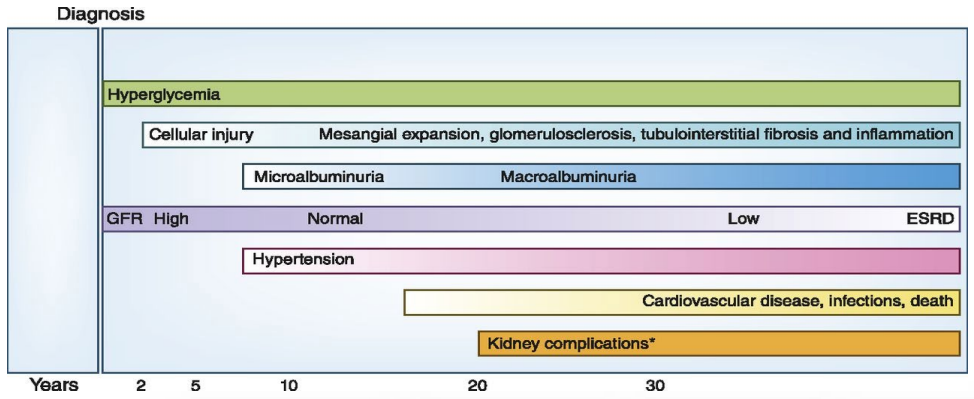
Advisory Board and Consulting Activity:

Sandoz
Sanofi
Baxter
Mundipharma
Estor
Pharmadoc
Travere
AstraZeneca
GS Pharma
Novartis
Chinook
CSL Vifor
Bayer

CKD in Type 2 Diabetes

2019-2030-2045 (around 700 million)





Develops in approximately 40% of patients who are diabetic and is the leading cause of CKD worldwide. ESRD lead to kidney replacement therapy or kidney transplantation.

CLASSIFICATION		HISTOLOGY	PREVALENCE
DN	• Class 1 →	Diabetic glomerulosclerosis	(39,6%)
NDRD	• Class 2 →	Vascular (arterioarteriolosclerotic) and ischemic glomerular changes	(15,2%)
DN+ NDRD	• Class 3a →	Glomerular diseases superimposed on diabetic glomerulosclerosis	(45,03%)
NDRD	• Class 3b →	Other glomerulonephritis without the presence of diabetic lesions	

Mazzucco G et al, *Am J Kidney Dis* 2002; 39: 713-716

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Rence

REAL WORLD EVIDENCE

OUTPATIENT SERVICE

Journal of Hypertension

ORIGINAL PAPERS: KIDNEY

Clinical significance of nonalbuminuric renal impairment in type 2 diabetes

Penno, Giuseppe[a]; Solini, Anna[b]; Bonora, Enzo[c]; Fondelli, Cecilia[d]; Orsi, Emanuela[e]; Zerbini, Gianpaolo[f]; Trevisan, Roberto[g]; Vedovato, Monica[h]; Gruden, Gabriella[i]; Cavalot, Franco[j]; Cignarelli, Mauro[k]; Laviola, Luigi[l]; Morano, Susanna[m]; Nicolucci, Antonio[n]; Pugliese, Giuseppe[o] for the Renal Insufficiency And Cardiovascular Events (RIACE) Study Group

Author Information

Journal of Hypertension 29(9):p 1802-1809, September 2011. | ***DOI:*** 10.1097/HJH.0b013e3283495cd6

Patients stratification

		Cases			
N		A1	A2	A3	Total
G1	>90	62% 9900	22% 3,497	5% 738	4,662 (29.6)
G2	60-90				8,152 (51.7)
G3a	45-60	11% 1638			1,951 (12.4)
G3b	30-45				750 (4.8)
G4	15-30				229 (1.5)
G5	<15				29 (0.2)
Total		11,538 (73.2)	3,497 (22.2)	738 (4.7)	15,773 (100.0)

eGFR ml/min

<30 mg/g >30 <300 mg/g >300 mg/g

ALBUMINURIA

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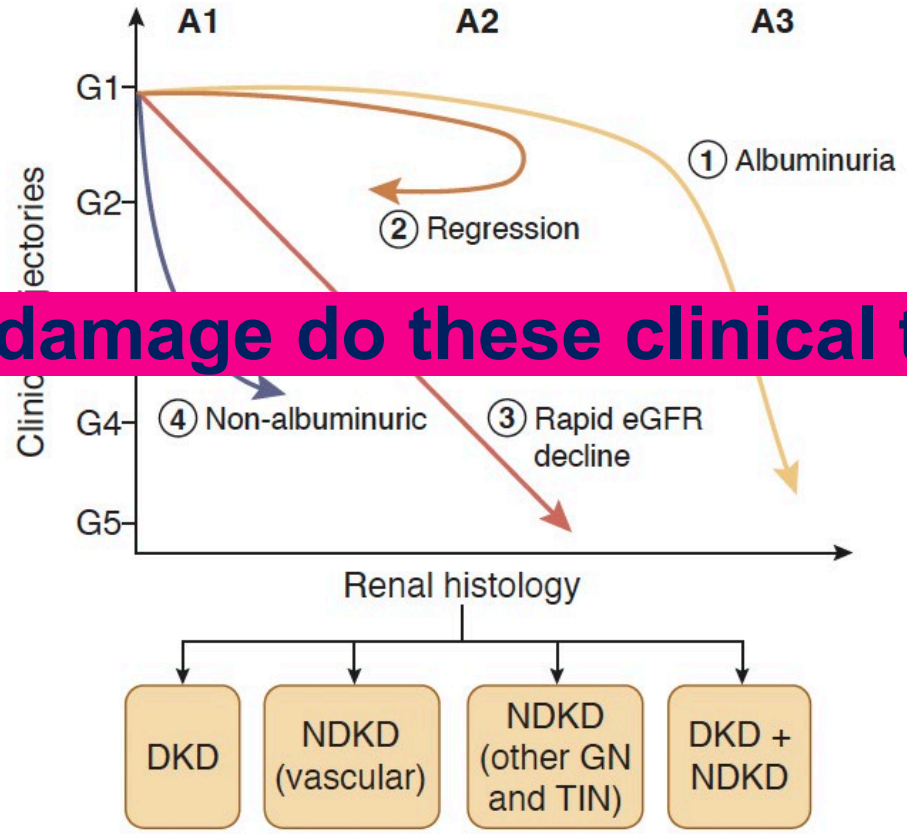
THE CHALLENGE



What type of kidney damage do these clinical trajectories have?

Gesualdo, Kreztler and Pontrelli, Kidney Int. 2026 Feb 27

THE CHALLENGE

Phenotype

CKD in diabetes

HERD OF ZEBRAS

the coat looks the same among them, but each of them has its own coat

Subtype

- DKD
- NDKD
- DKD+NDKD

CLINICAL DIAGNOSIS

Main unmet medical needs: Cardiovascular death and CKD progression

THE CHALLENGE

OPEN ACCESS Freely available online

PLOS ONE

Identifying Parameters to Distinguish Non-Diabetic Renal Diseases from Diabetic Nephropathy in Patients with Type 2 Diabetes Mellitus: A Meta-Analysis

Shuang Liang, Xue-Guang Zhang, Guang-Yan Cai*, Han-Yu Zhu, Jian-Hui Zhou, Jie Wu, Pu Chen, Shu-peng Lin, Qiang Qiu, Xiang-Mei Chen*

Department of Nephrology, State Key Laboratory of Kidney Diseases, Chinese PLA General Hospital, Beijing, China

Abstract

Background: Renal injuries in patients with diabetes include diabetic nephropathy (DN) and non-diabetic renal diseases (NDRD). The value of a clinical diagnosis of DN and NDRD remains inconclusive. We conducted a meta-analysis of the literature to identify predictive factors of NDRD and to compare the clinical characteristics of DN and NDRD for differential diagnosis.

Methods: We searched PubMed (1990 to January 2012), Embase (1990 to February 2009), and CNKI (1990 to January 2012) to identify studies that enrolled patients with DN and NDRD. Then, the quality of the studies was assessed, and data were extracted. The results were summarized as odds ratios (ORs) for dichotomous outcomes and weighted mean differences (WMDs) for continuous outcomes.

Results: Twenty-six relevant studies with 2,322 patients were included. The meta-analysis showed that the absence of diabetic retinopathy (DR) predicts NDRD (OR, 0.15; 95% confidence interval [CI], 0.09–0.26, $p<0.00001$). A shorter duration of diabetes mellitus (DM) also predicted NDRD (weighted mean difference, −34.67; 95% CI, −45.23–−24.11, $p<0.00001$). The levels of glycosylated hemoglobin (HbA1C%), blood pressure (BP), and total cholesterol were lower in patients with NDRD, whereas triglycerides and body mass index were higher. Other clinical parameters, including age, 24-h urinary protein excretion, serum creatinine, creatinine clearance, blood urea nitrogen, and glomerular filtration rate were not different between patients with NDRD and DN.

Conclusions: We identified that the absence of DR, shorter duration of DM, lower HbA1C, and lower BP may help to distinguish NDRD from DN in patients with diabetes. This could assist clinicians in making a safe and sound diagnosis and lead to more effective treatments.

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NDT Advance Access published May 4, 2016

Nephrol Dial Transplant (2016) 0: 1–14
doi: 10.1093/ndt/gfw070

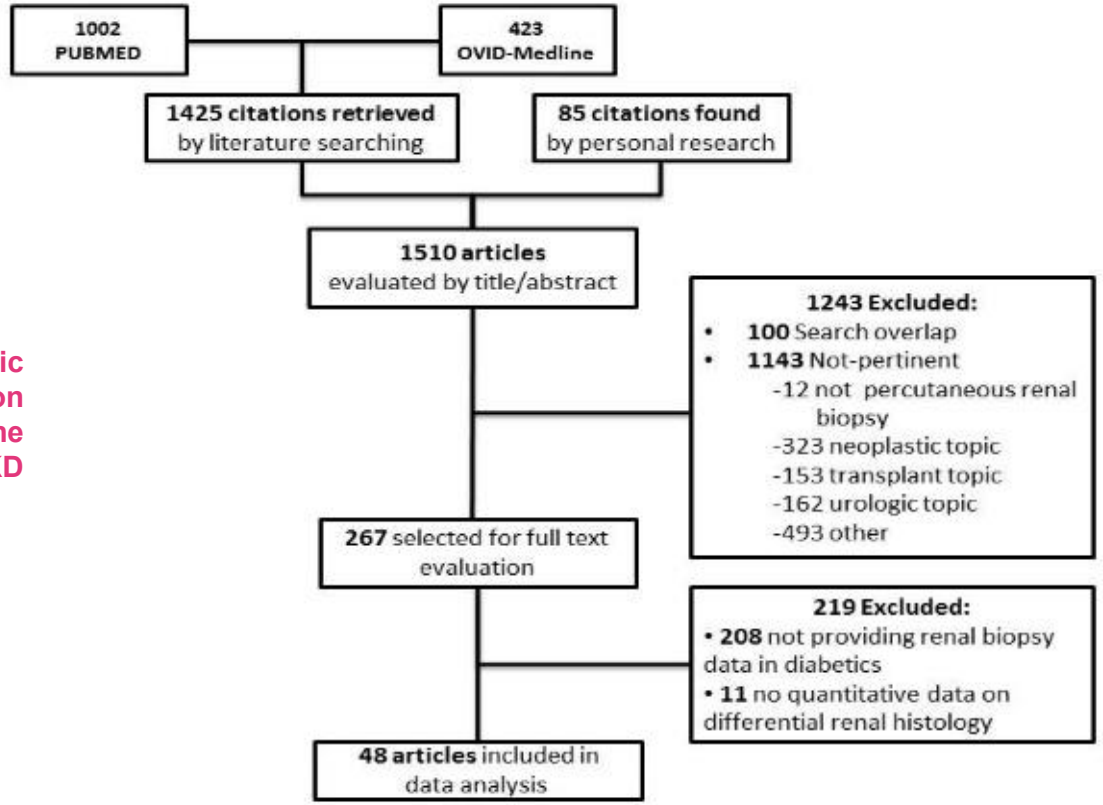
ndt Nephrology Dialysis Transplantation

Original Article

Renal biopsy in patients with diabetes: a pooled meta-analysis of 48 studies

Marco Fiorentino[1], Davide Bolignano[2,3], Vladimir Tesar[4], Anna Pisano[2], Wim Van Biesen[3], Giovanni Tripepi[3], Graziella D'Arrigo[2] and Loreto Gesualdo[1]on behalf of the ERA-EDTA Immunonephrology Working Group

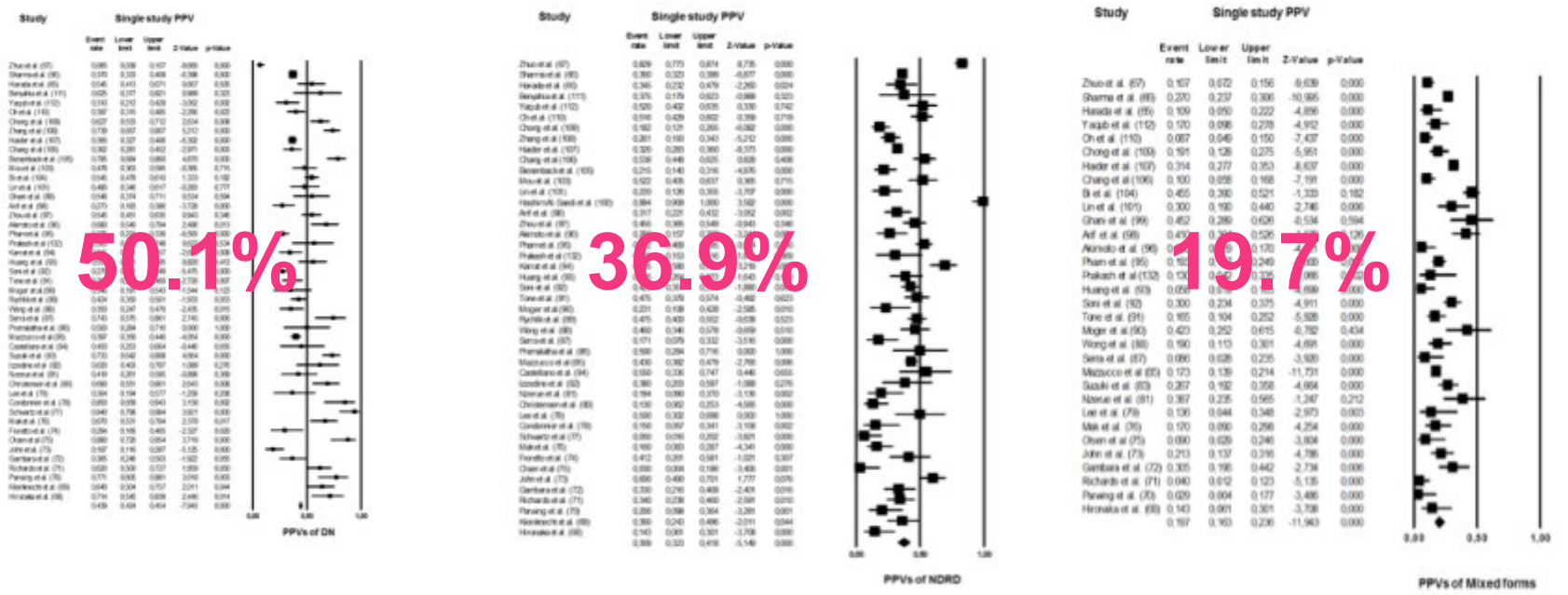
We performed a systematic search for studies on diabetics with data on the frequency of DKD, NDKD and mixed forms



THE CHALLENGE

Positive predictive values of clinical judgment for the diagnosis of DKD (2a), NDKD (2b) and mixed forms (2c) from pooled meta-analysis

Figure 2. Positive predictive values of clinical judgment for the diagnosis of DN (2a), NDRD (2b) and mixed forms (2c) from pooled meta-analysis



Positive predictive values (PPVs) (pooled data) of clinical judgment for identifying DKD, NDKD and mixed forms (as assessed by renal biopsy) were 50.1% (95% CI: 44.9 - 55.3), 36.9% (95% CI: 32.3-41.8) and 19.7% (95% CI: 16.3-23.6), respectively

Clinical Kidney Journal, 2024, vol. 17, no. 1, 1–4

https:/doi.org/10.1093/ckj/sfad266
Advance Access Publication Date: 26 October 2023
Pro/Con Debate

CLINICAL KIDNEY JOURNAL

PRO/CON DEBATE

Should we enlarge the indication for kidney biopsy in patients with diabetes? The pro part

Loreto Gesualdo, Marco Fiorentino, Francesca Conserva and Paola Pontrelli

Renal, Dialysis and Transplantation Unit, Department of Precision and Regenerative Medicine and Ionian Area (DIMEPRE-J), University of Bari, Bari, Italy

Correspondence to: Loreto Gesualdo; E-mail: loreto.gesualdo@uniba.it

Holistic T2D-CKD Board

- Nephrologist
- Cardiologist
- Diabetologist
- Ophthalmologist
- Radiologist

- Renal Pathologist
- Molecular Biologist
- Dietician
- Psychologist
- Music Therapist

Multidisciplinary Team

REAL WORLD EVIDENCE

DIABETOLOGY/NEPHROLOGY OUTPATIENT SERVICE

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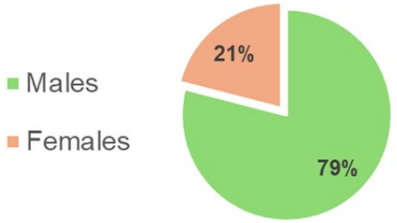
Figure 1: Cohort Characteristics and K-DIGO staging

DIABETIC PTS WITH CKD

SEX Distribution iBEAt DKD Cohort Bari



Patients Stratification in (260/113/100 pts)

eGFR ml/min	N	A1	A2	A3	Total
		Cases			
G1	>90	58% 150/3/2			65
G2	60-90				116
G3a	45-60		15% 39/39/34	21% 54/54/49	21
G3b	30-45	6% 17/17/15			46
G4	15-30				9
G5	<15				3
Total		167	39	54	260
ACR		<30 mg/g	>30 <300 mg/g	>300 mg/g	

DIABETOLOGY/NEPHROLOGY OUTPATIENT SERVICE

260 Patients screened

113 Patients Recruited

147 Pts G1-2/A1 refused kidney biopsy

100 pts underwent **KIDNEY BIOSPY**

13 pts do **NOT** have biopsy

Reasons for no biopsy

- Cysts
- High Blood Pressure
- Bleeding risk

96 kidney biopsies cores were collected in RNAlater for snRNA Seq

83 pts: Kidney, Eye, Heart and Clinical Variables

11 pts: Kidney, Heart and Clinical Variables

4 pts: Kidney, Eye and Clinical Variables

2 pts: Kidney and Clinical Variables

INCLUSION CRITERIA

- Type 2 Diabetes
- Albuminuria categories: A1, A2; A3
- Age: 18 to 80

EXCLUSION CRITERIA

- Renal Transplantation/ Dialysis
- Significant comorbidities with life expectancy of < 1 year
- Use of investigational drug within 1 month prior to screening
- Cirrhotic liver disease
- Current pregnancy
- History of Hepatitis B or Hepatitis C +
- Use of antiretroviral medication
- Concurrent other renal disease
- Current malignancy

CKD in Type 2 Diabetes: BEAT-DKD study (100 Renal Biopsies)

Phenotype

Subtype

Endotype

DIABETIC PTS WITH CKD

Normal

1% (1)

DKD

37% (37)

NDKD

56% (56)

DKD+NDKD

6% (6)

PURE METABOLIC

8%

VASCULAR METABOLIC

29%

VASCULAR (HYPERTENSIVE)

35%

VASCULAR GNs, TIN

17%

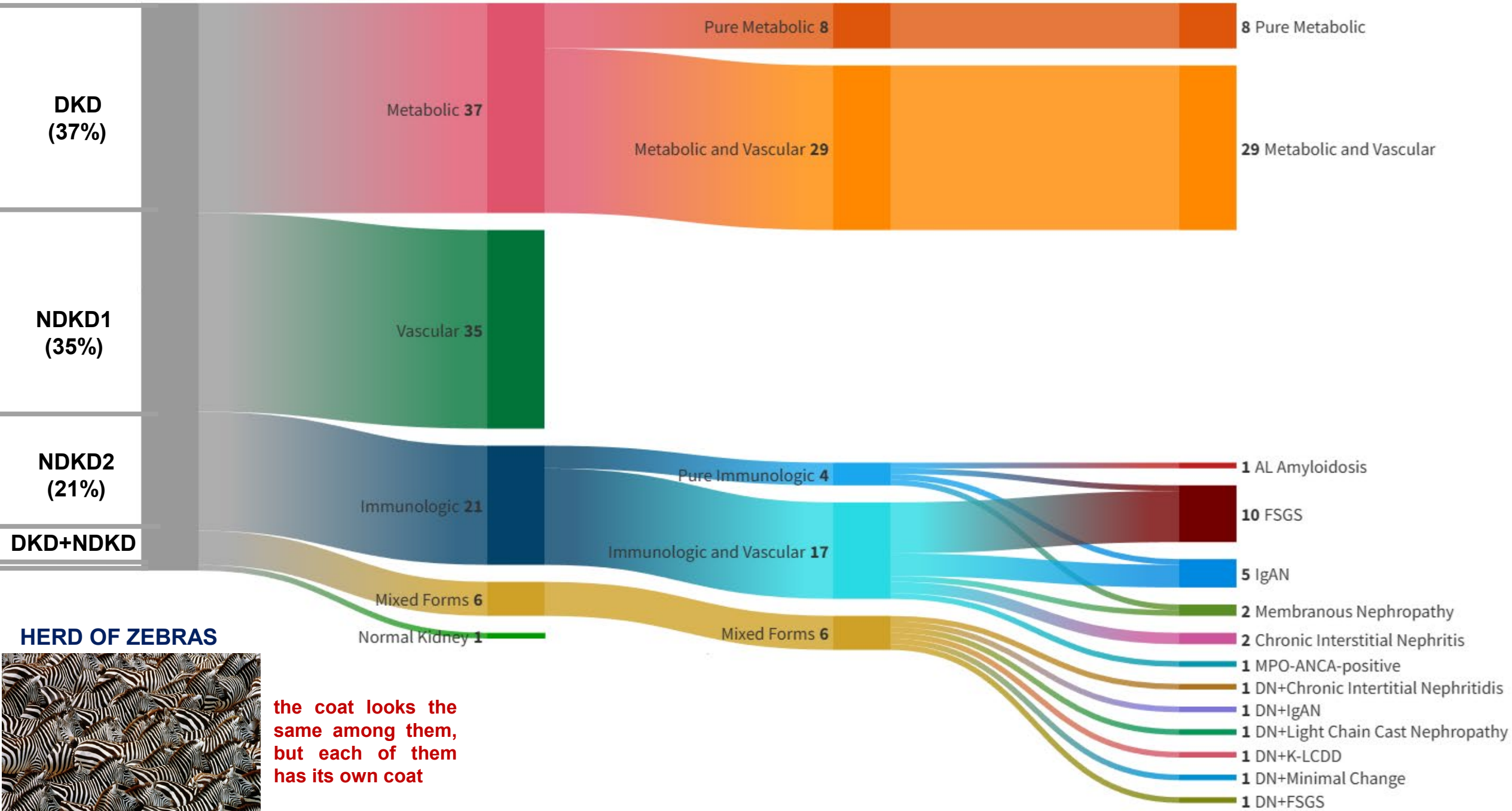
PURE GNs,TIN

4%

MIXED FORM

6%

CKD in T2D 100 Pts



HERD OF ZEBRAS

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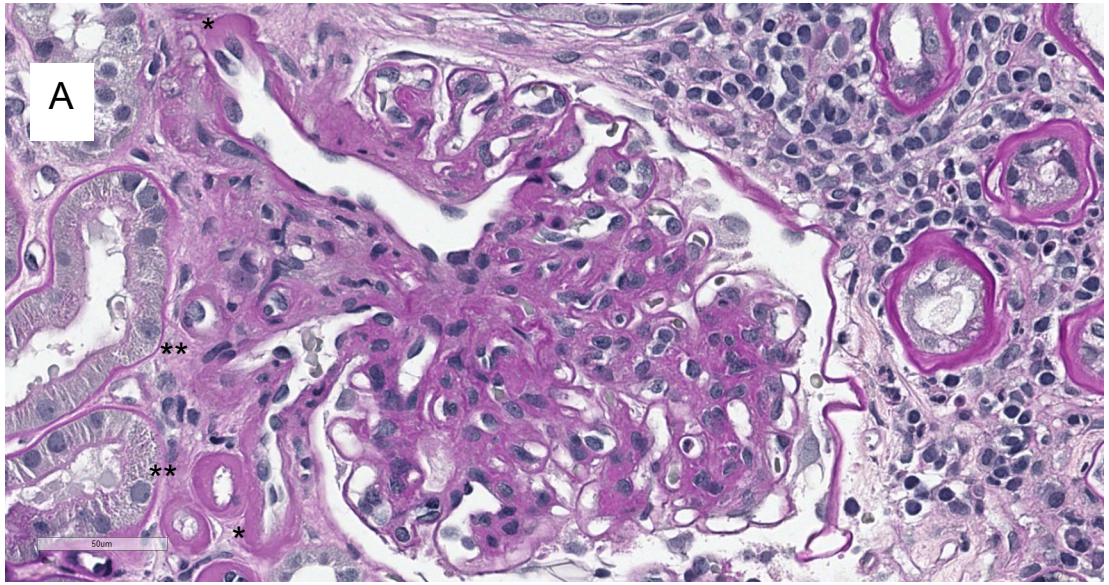
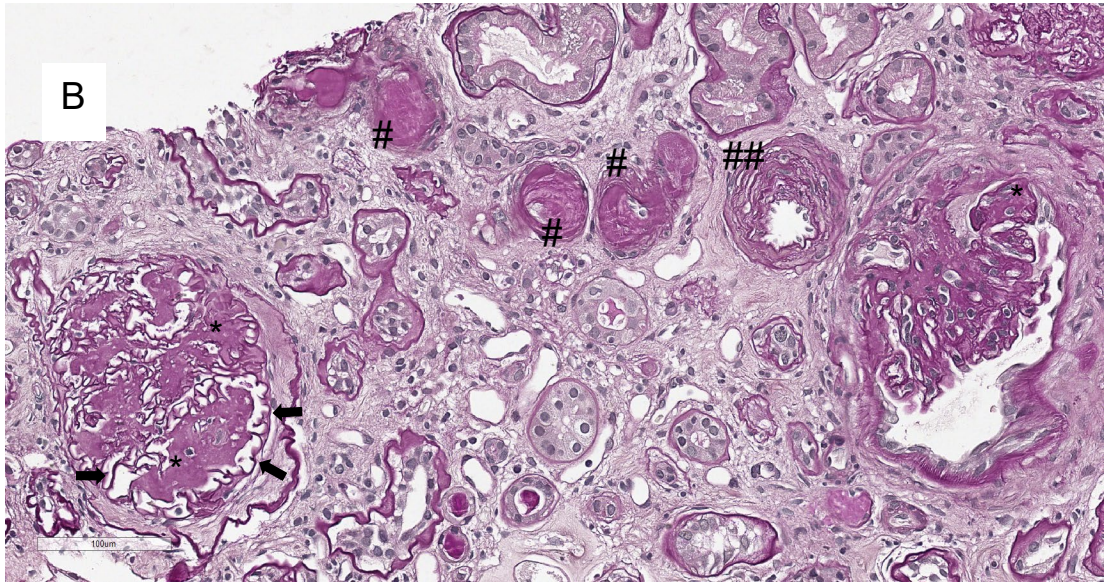
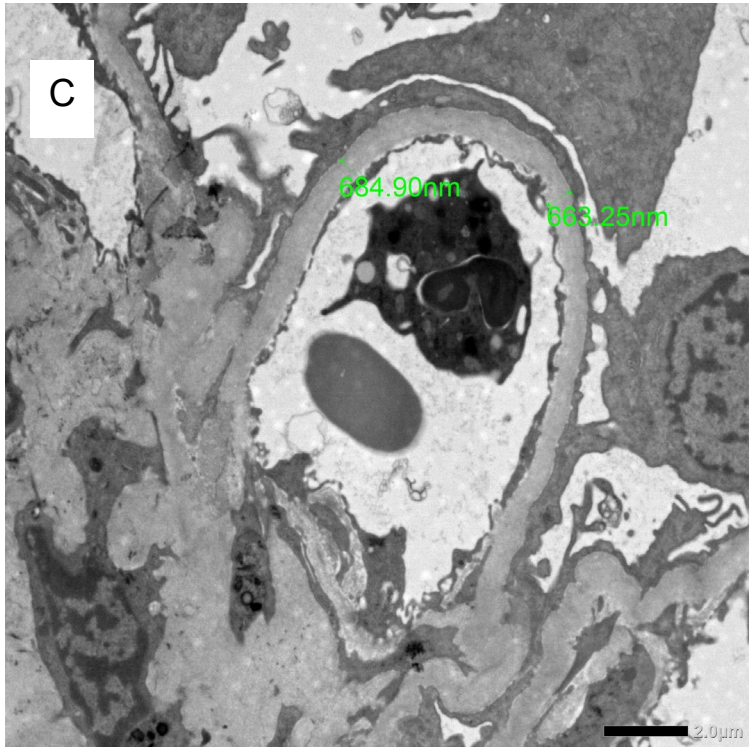
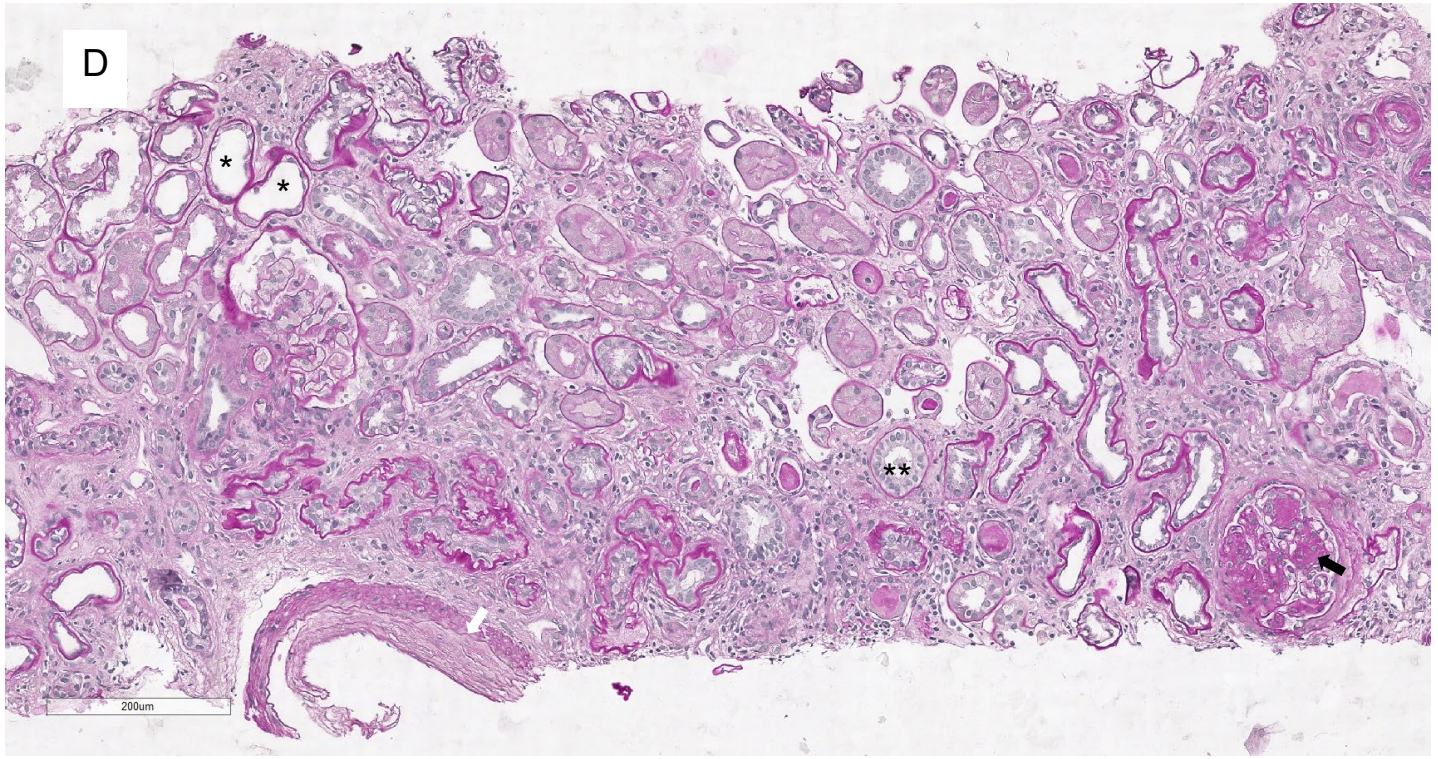
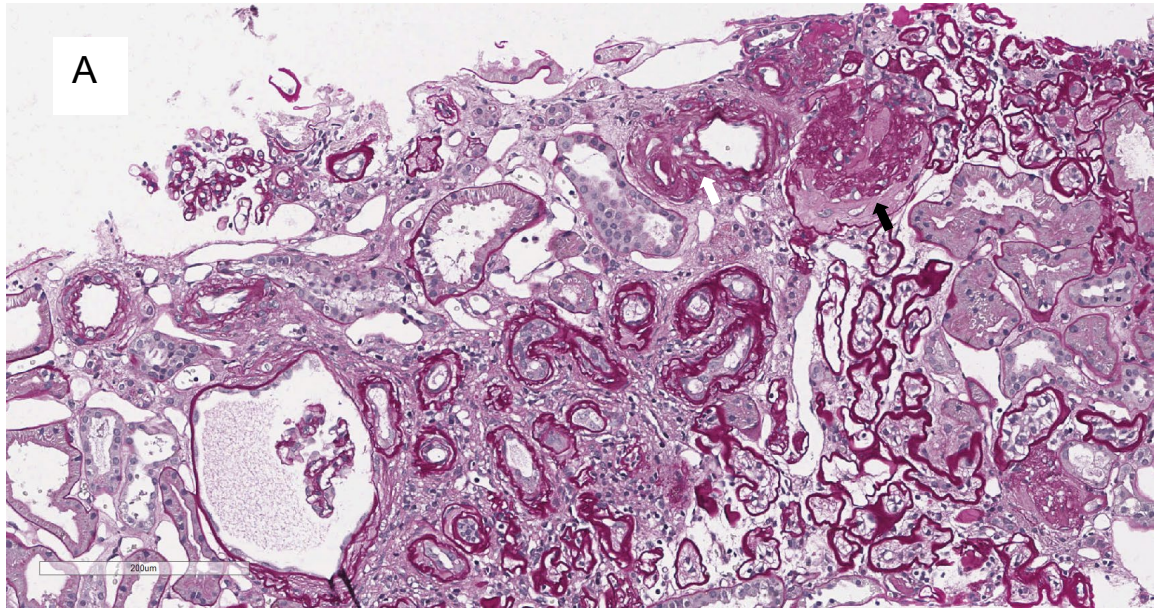


DKD

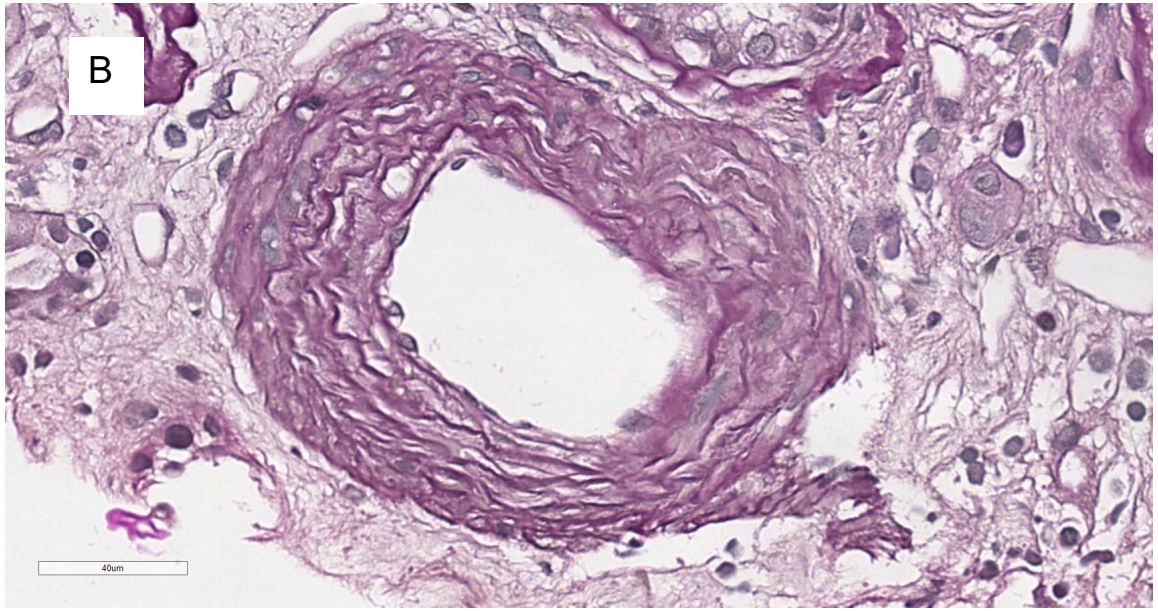


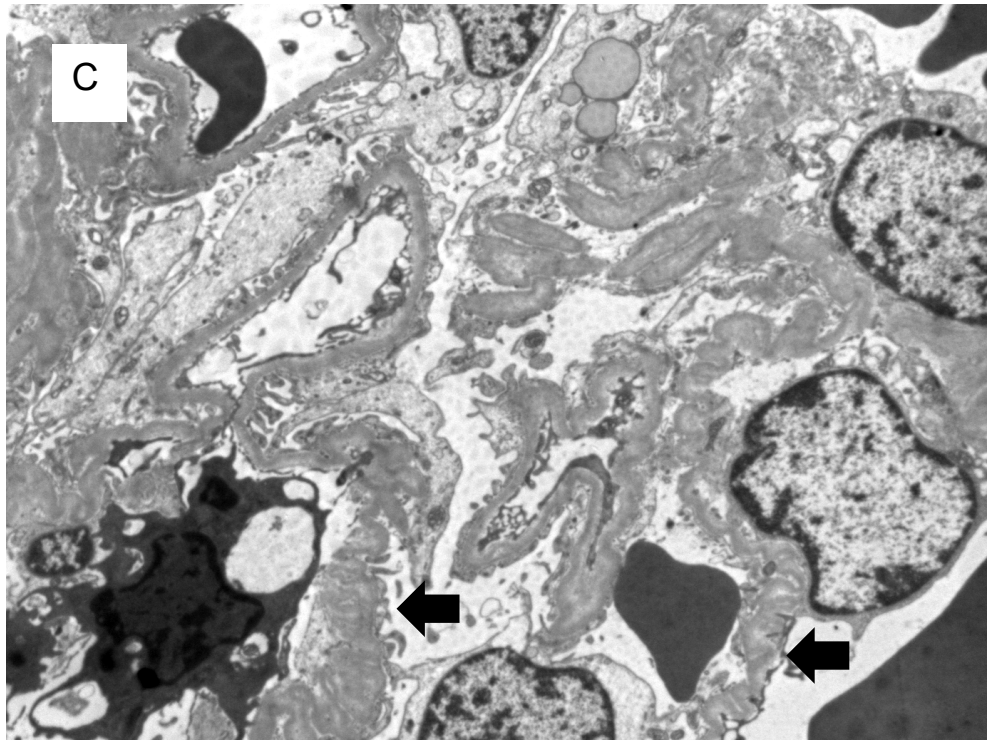


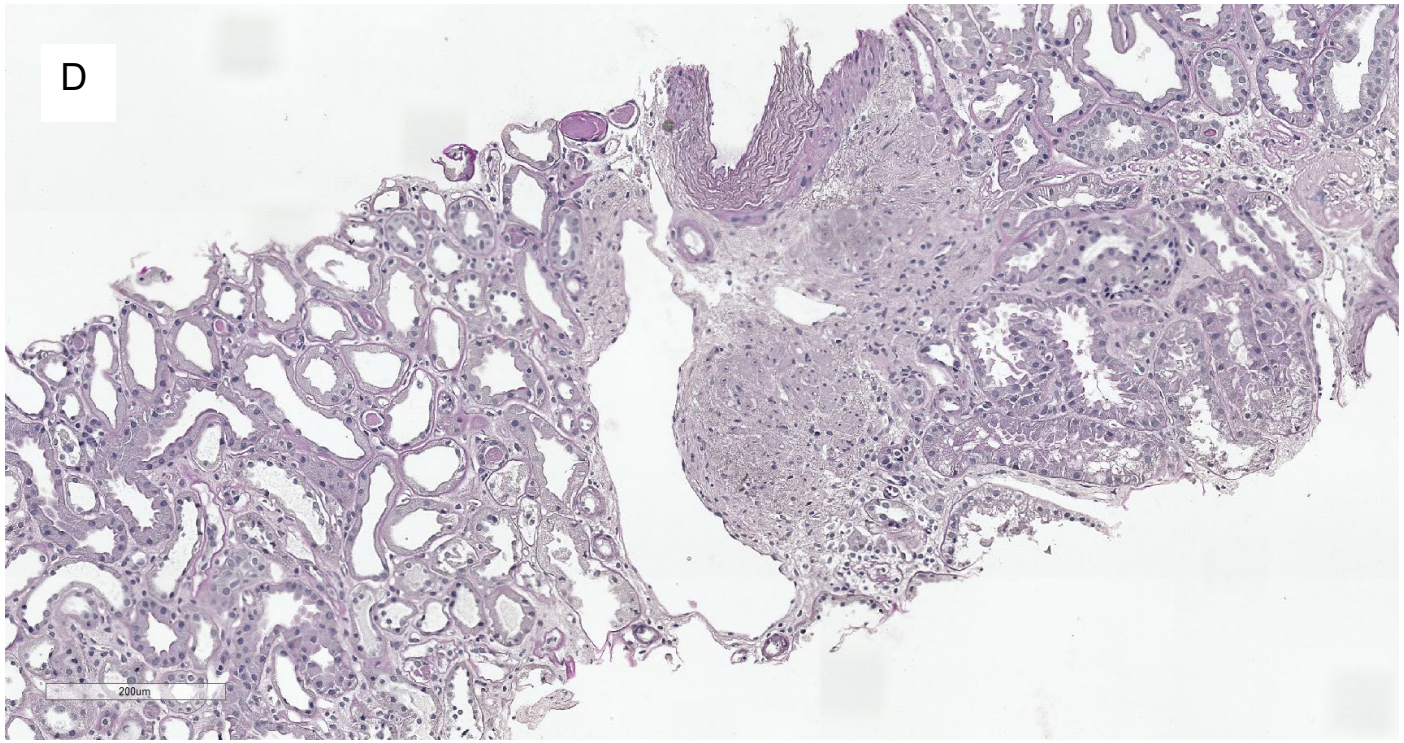




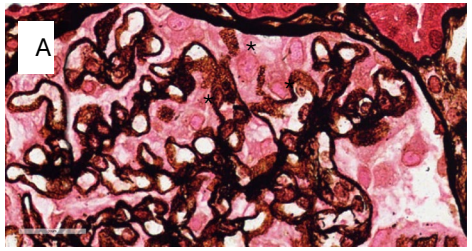
NDKD1

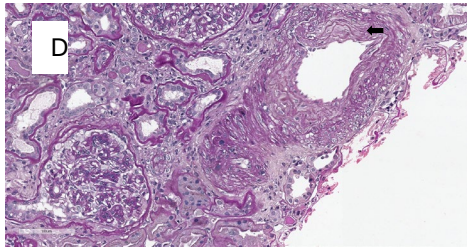


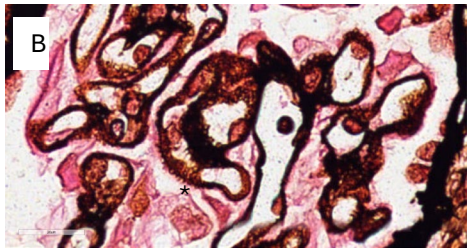


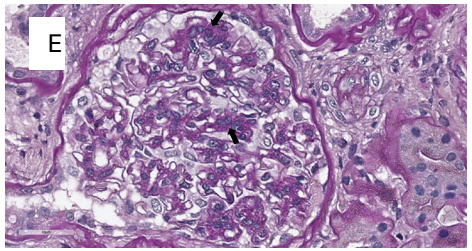


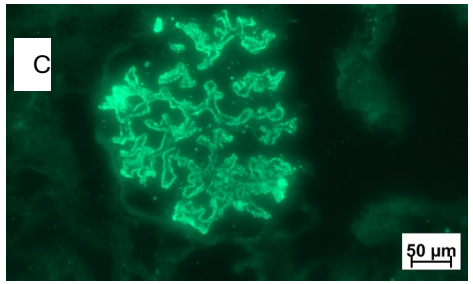
NDKD2

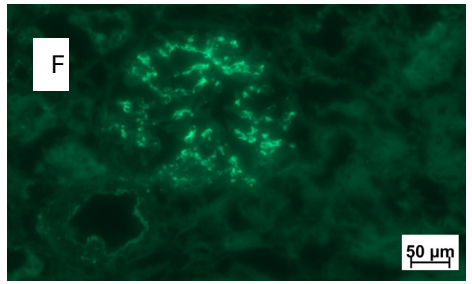




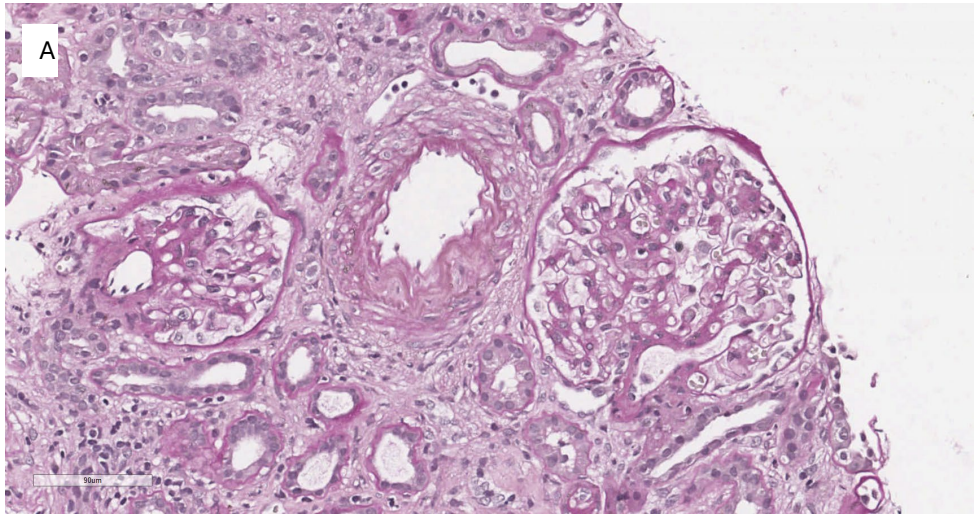


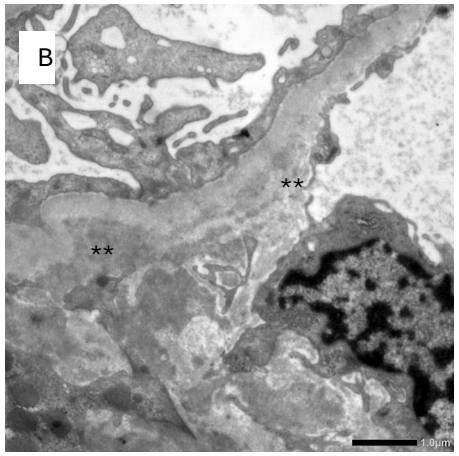


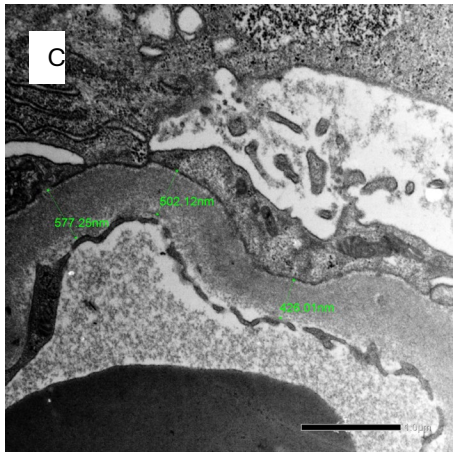




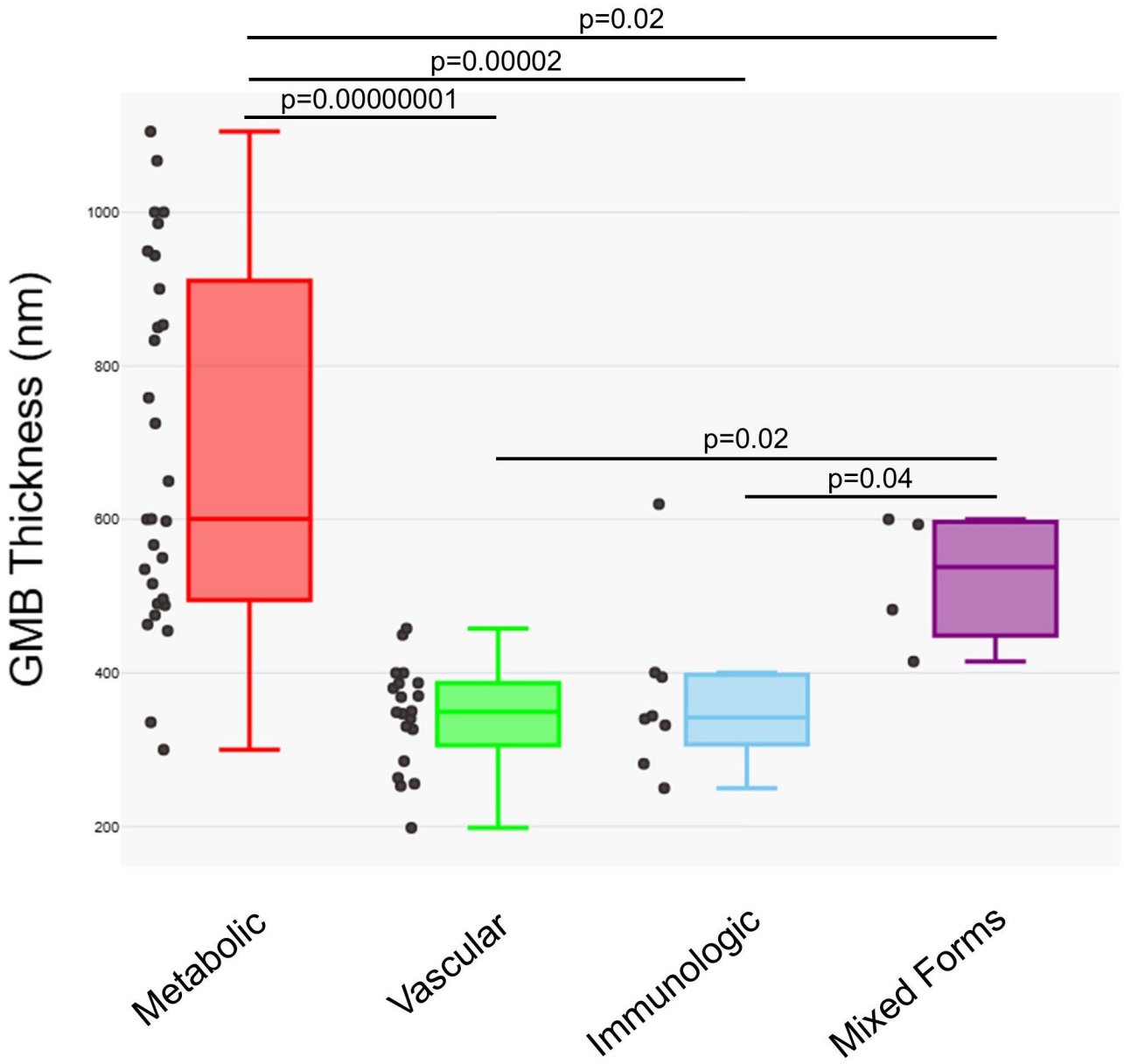
DKD+NDKD

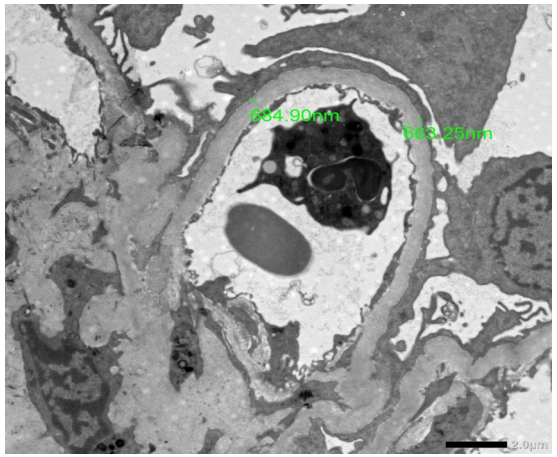


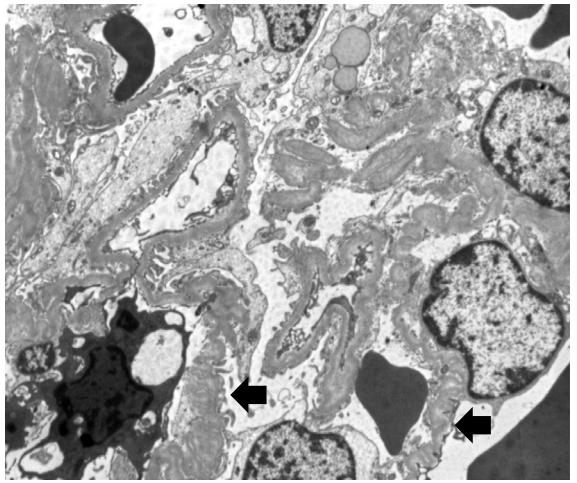




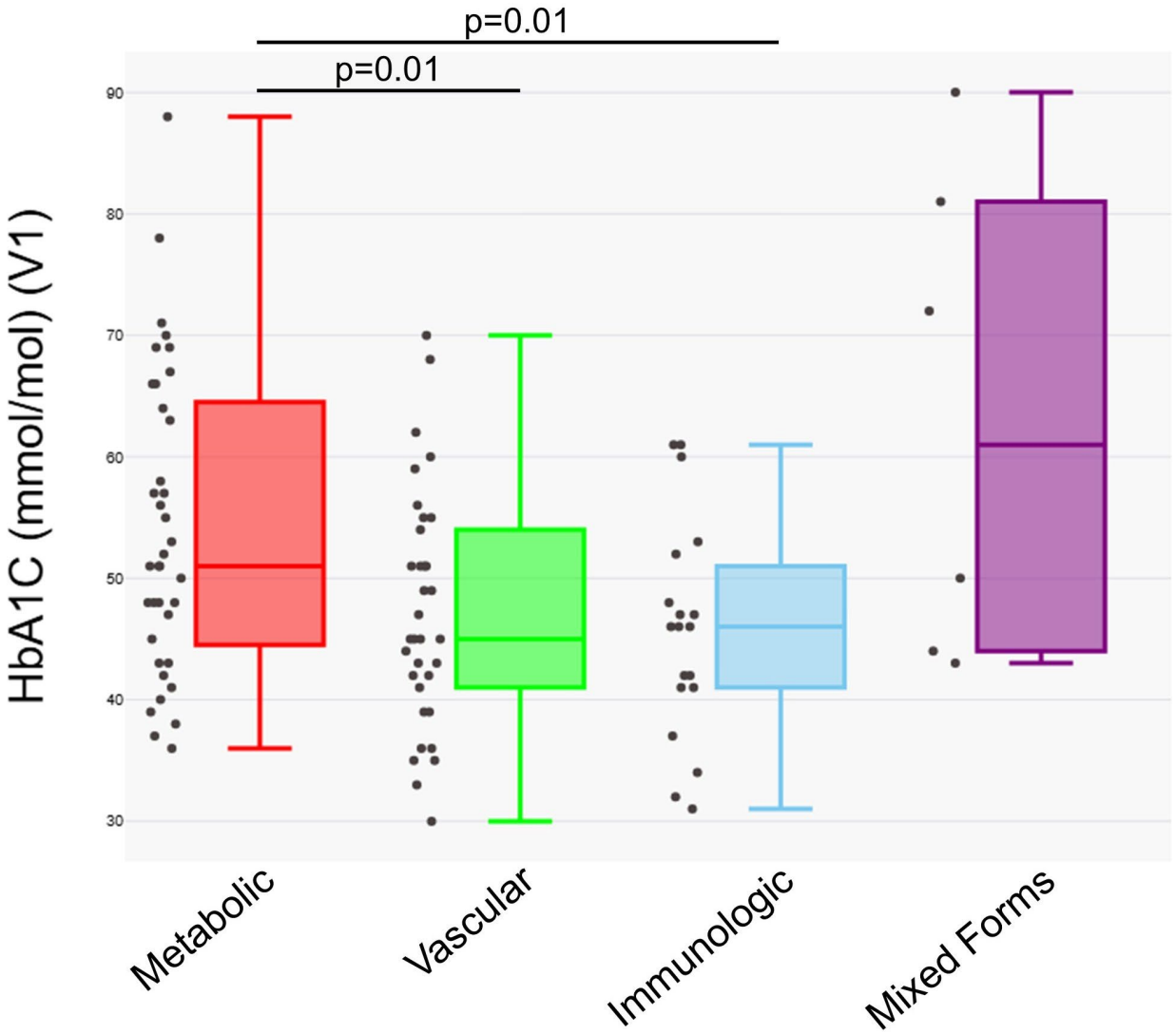
GBM Thickness

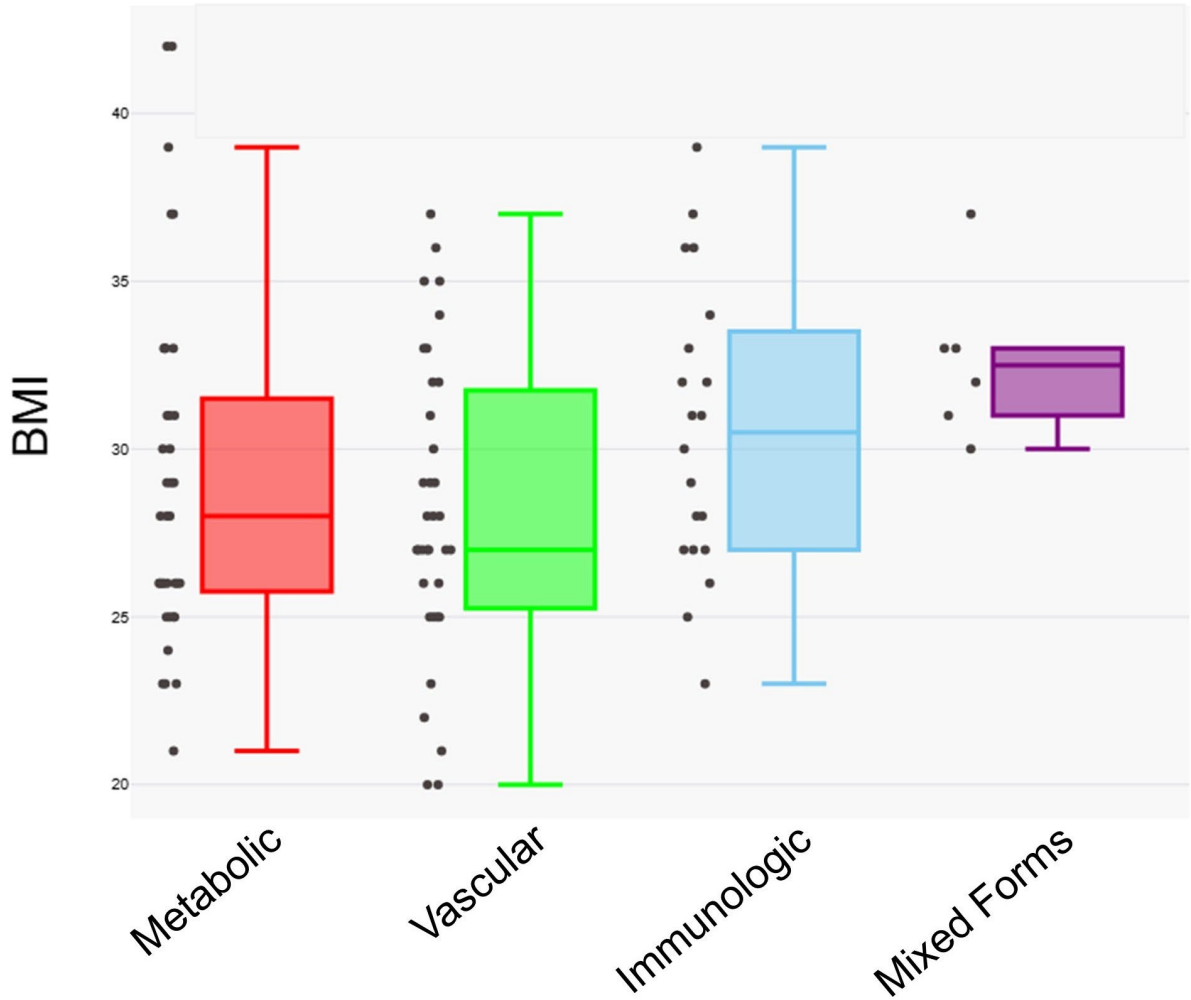




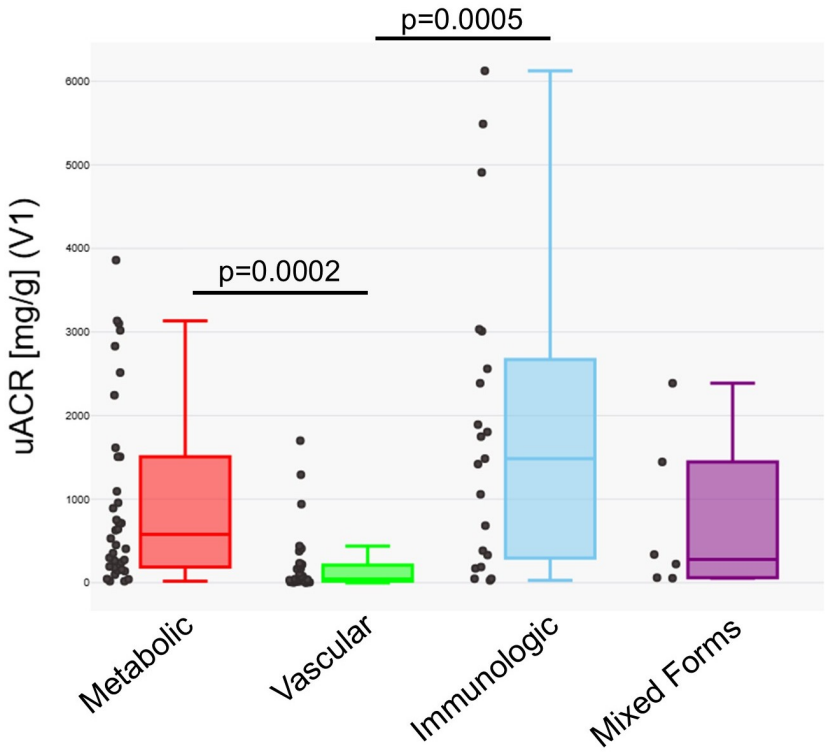


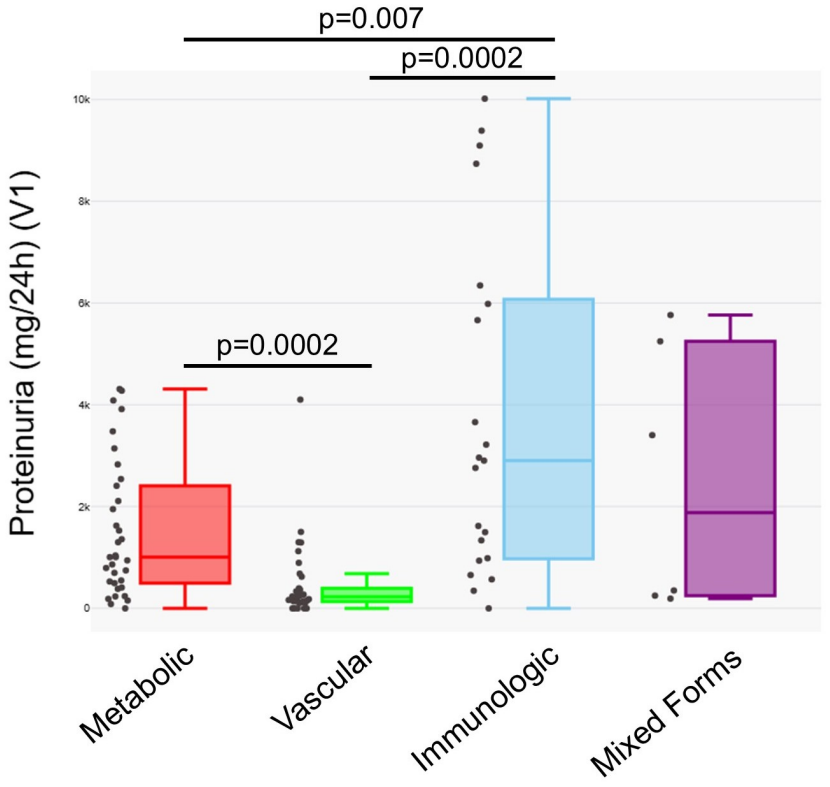
HbA1C and BMI

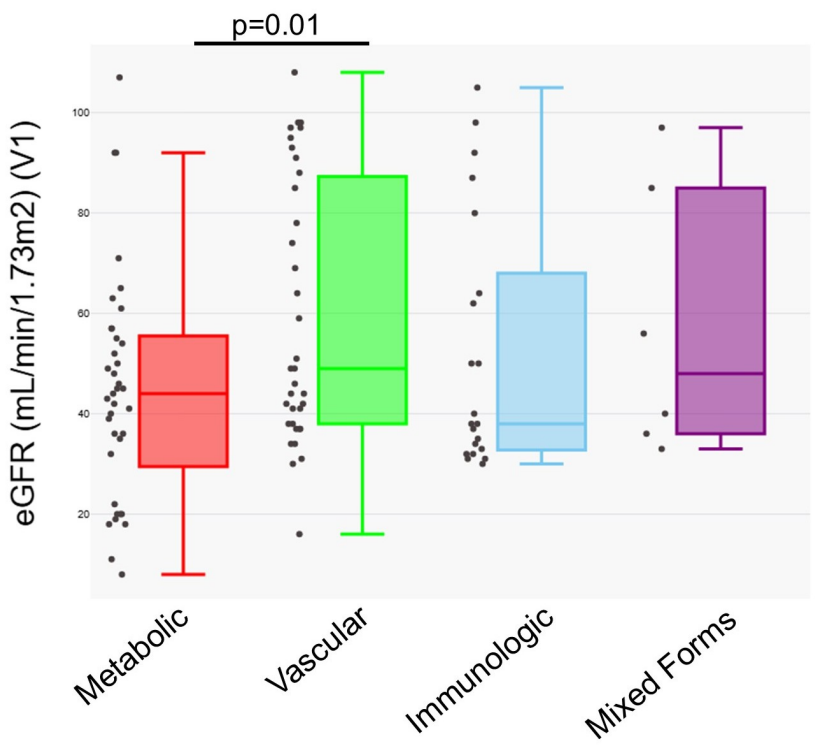




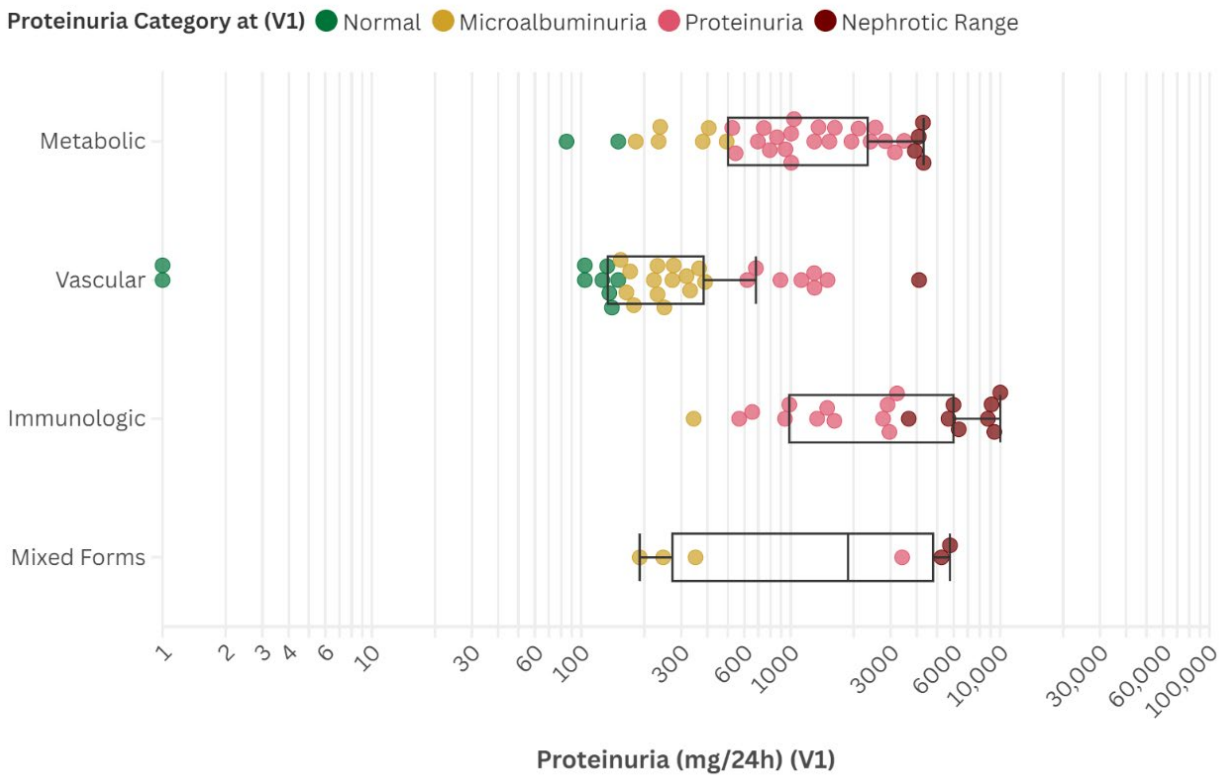
uACR, 24 hours Proteinuria, eGFR

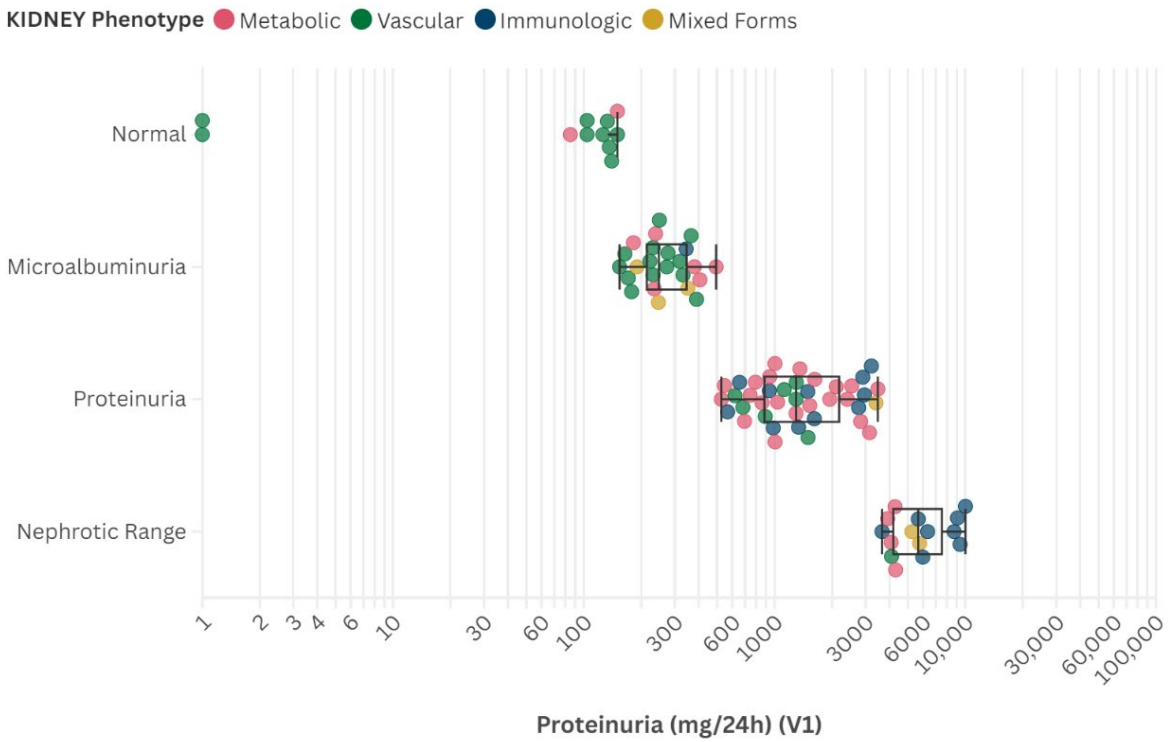




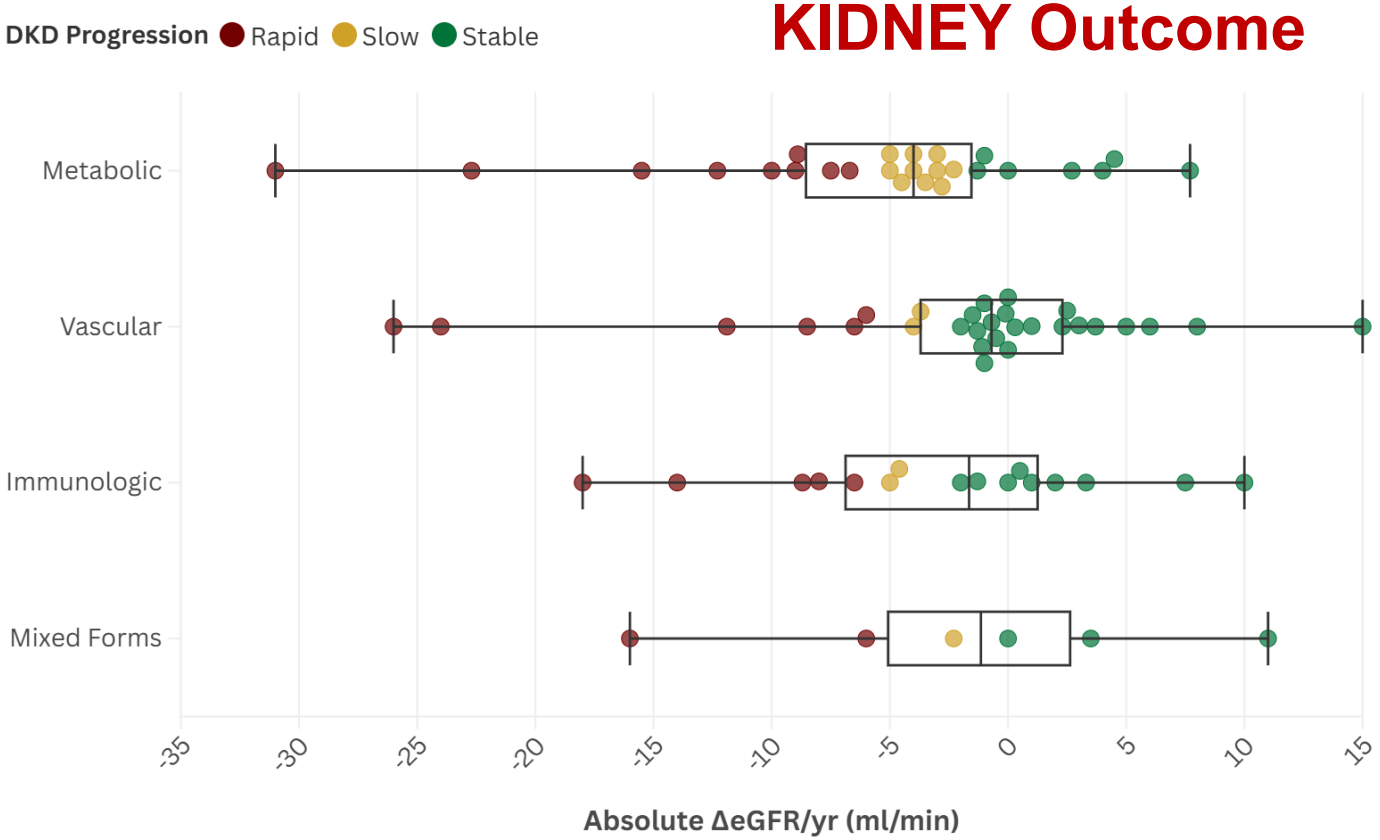


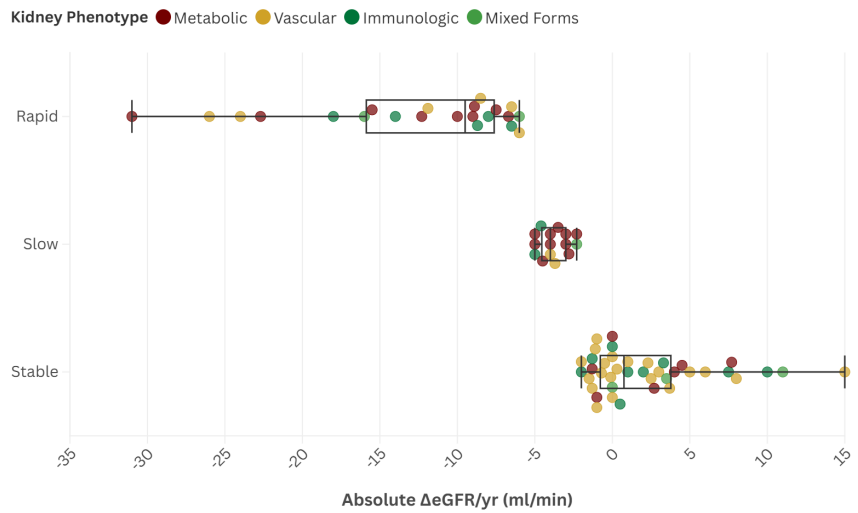
Type of Proteinuria





KIDNEY Outcome





absolute delta eGFR(ml/min)

(median 3.2 years [range 1–5 years])

	Rapid	Slow	Stable	Total Cases
Metabolic	9	10	7	26
Vascular	6	2	21	29
Immunologic	5	2	9	16
Mixed Forms	2	1	3	6

	Rapid	Slow	Stable	Total Cases
Metabolic	35%	38%	27%	34%
Vascular	21%	7%	72%	38%
Immunologic	31%	13%	56%	21%
Mixed Forms	33%	17%	50%	8%

HEART SUBTYPES

Echocardiographic variables	Meaning
SIV: Interventricular septum thickness	
PP: Back wall thickness VS	
DTD: Left ventricular end-diastolic diameter	
DTS: Left ventricular end-systolic diameter	
LVM: Left ventricular mass	
LVMi: left ventricular mass indexed by body surface area	
RWT: relative wall thickness	Assesses the type of ventricular remodeling, whether concentric or eccentric
LAVi: Left atrium volume indexed by body surface area	
E wave	Left Left Ventricular Rapid Filling Wave
A wave	Left ventricular filling wave due to atrial contraction
LVEF: left ventricular ejection fraction	
TAPSE: Trycuspidala anular plane systolic exursion	Right ventricular contractility index (the lower the value, the lower the contractility of the right ventricle)
Left ventricular geometry	Defines the type of left ventricular remodeling

Table 2

LV geometry	LVMi (g/m^2)	RWT
Normal	≤115 for men or ≤ 95 for women	<0.42
Concentric hypertrophy	>115 (men) or > 95 (woman	>0.42
Eccentric hypertrophy	>115 (men) or < 95 (women)	<0.42
Concentric remodelling	≤115 (men) or ≤ 95 (women)	>0.42

Table 3

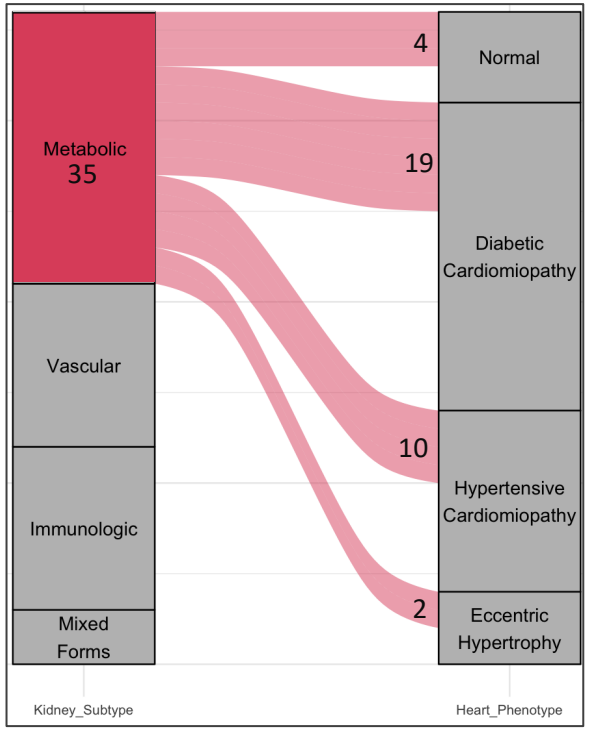
The variable "**left ventricular geometry**" defines the type of left ventricular remodeling:
-'*concentric remodeling*' identifies diabetic cardiomyopathy
- '*concentric hypertrophy*' is expression of hypertension damage.

Kidney to Heart trajectory

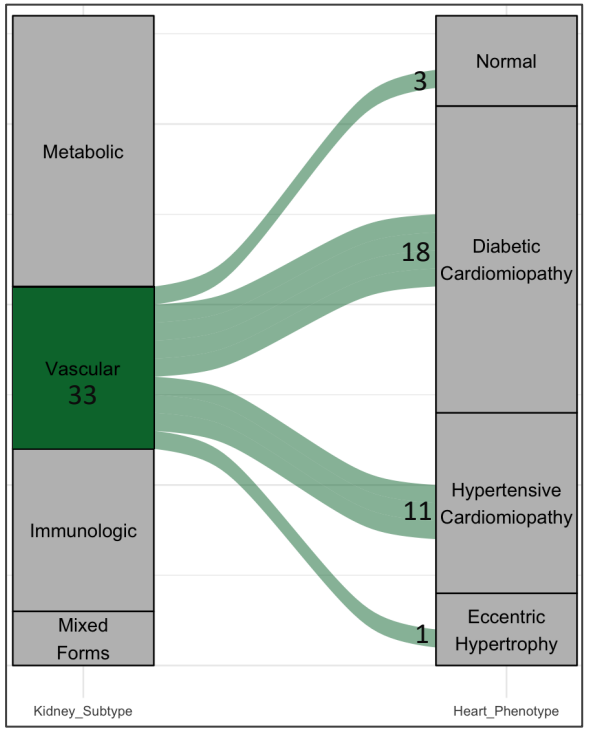
DC (%)	54	55	63	66
HC (%)	29	33	26	33

p= 0.89

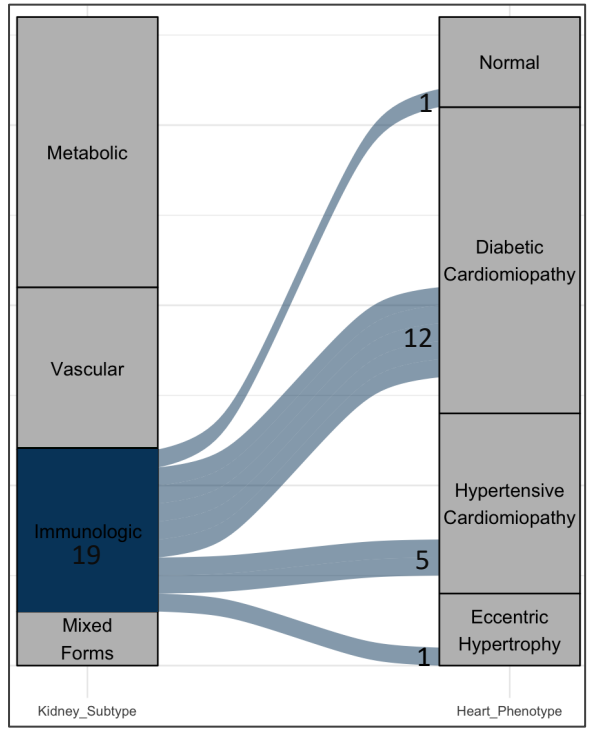
Metabolic Kidney(DKD)



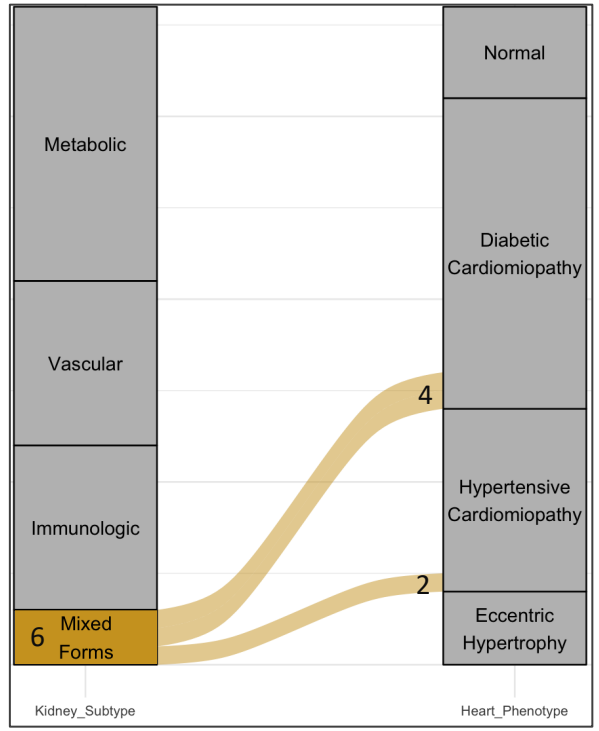
Vascular Kidney (NDKD1)



Immunologic/Inflamm Kidney (NDKD2)

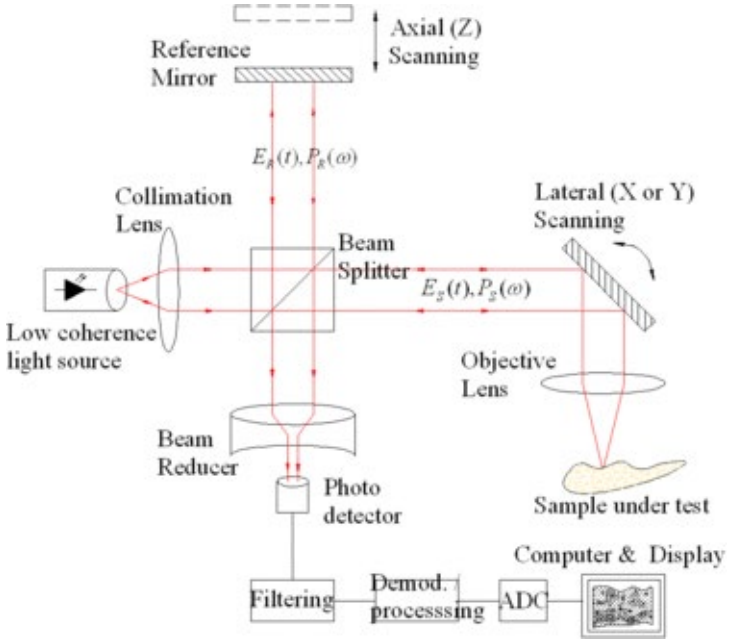


Mixed Forms Kidney (DKD+NDKD)



RETINA SUBTYPES

Optical Phase Coherence Tomography



Data included in the clinical classification:

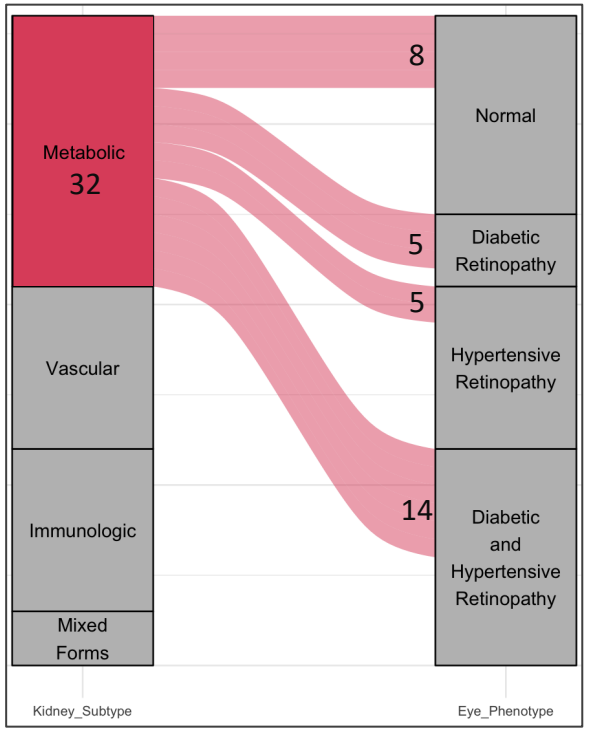
Diabetic Retinopathy V1 (Yes=1; no=0)	Hypertensive Retinopathy V1 (Yes=1; no=0)	Other Retinopathy V1 (yes=1; no=0)	Other Ocular lesions (0=no; 1= si)

Kidney to Retina Trajectory

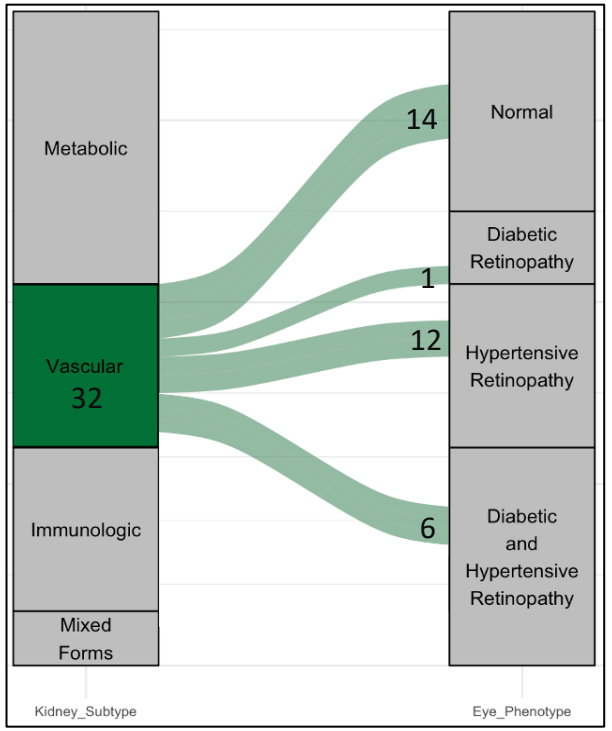
DR (%)	59	22	13	33
HR (%)	59	56	56	33

p= 0.03

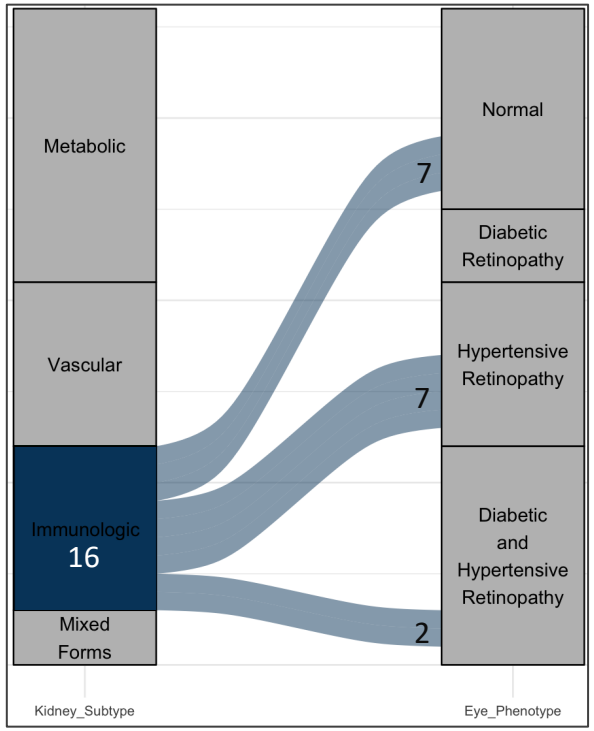
Metabolic Kidney(DKD)



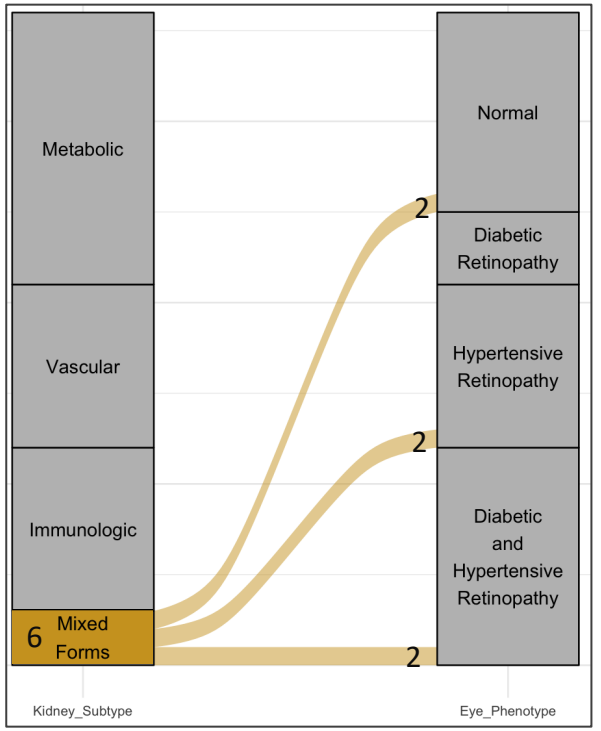
Vascular Kidney (NDKD1)



Immunologic/Inflamm Kidney (NDKD2)



Mixed Forms Kidney (DKD+NDKD)



Xgboost Variable importance plot for each subtype (4 models)

Metabolic Kidney(DKD)

Mixed Forms Kidney (DKD+NDKD)

Immunologic/Inflamm Kidney (NDKD2)

Vascular Kidney (NDKD1)

Shap Analysis

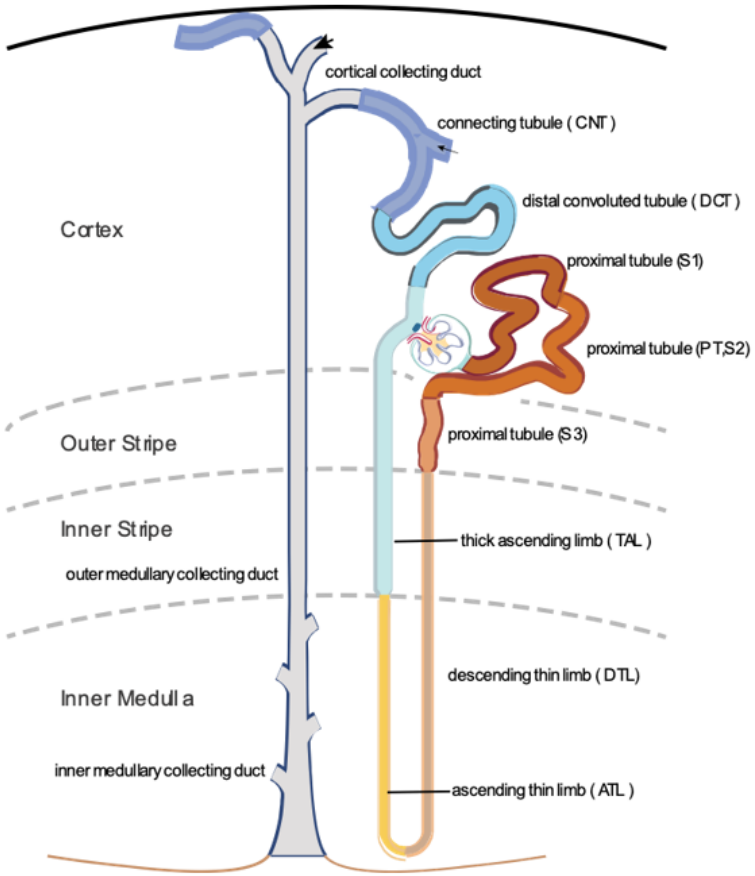
Metabolic Kidney(DKD)

Vascular Kidney (NDKD1)

Immunologic/Inflamm Kidney (NDKD2)

Mixed Forms Kidney (DKD+NDKD)

DKD protocol Biopsies snRNAseq



2-4 mm segments of 56 Protocol Research Biopsies (DKD) undergoing split-pool single nuclear RNA seq analysis. 61362 nuclei passing QC across all major cell types

Brad Godfrey

Edgar Otto
U Michigan

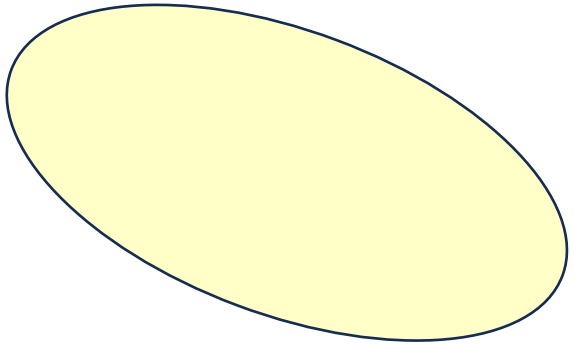
Phil McCown

Gene Classifier for each group in Tubuli

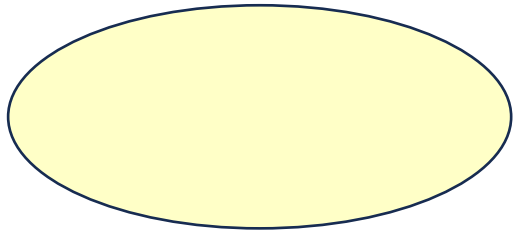
a) PCA using all genes

b)

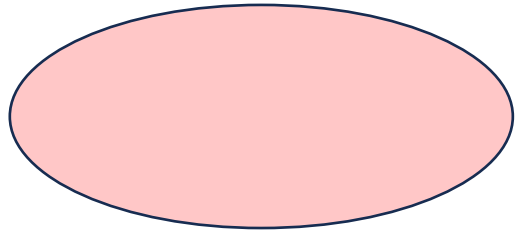
c)



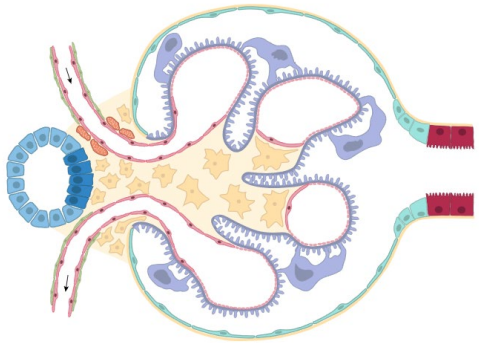
d)



e)

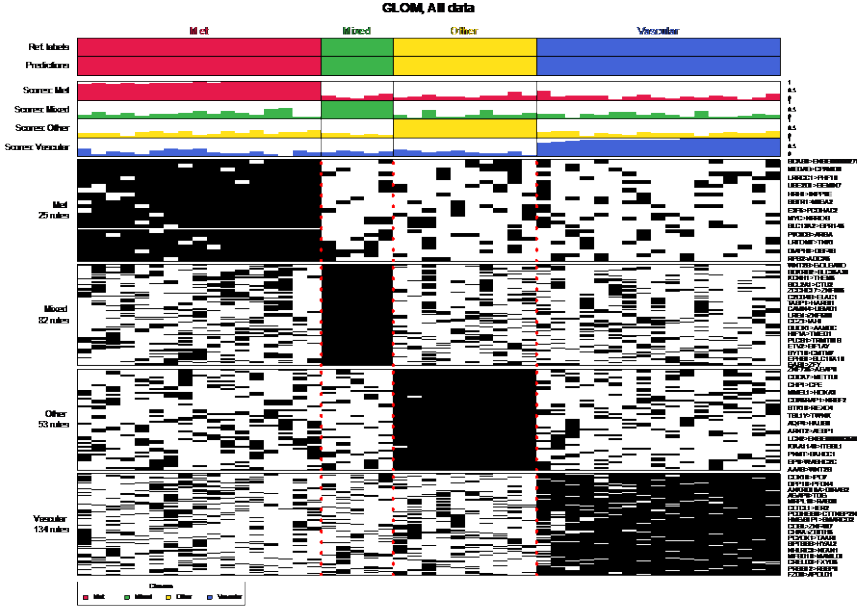


Gene Classifier for each group in Glomeruli

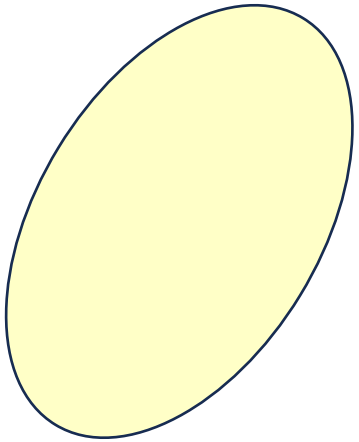


a) PCA using all genes

b)



c)



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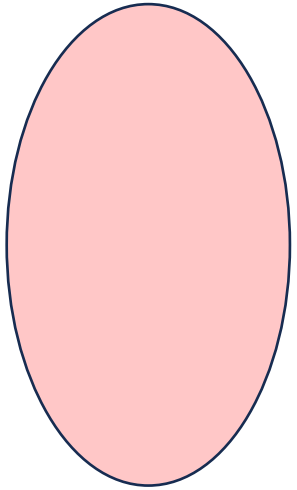

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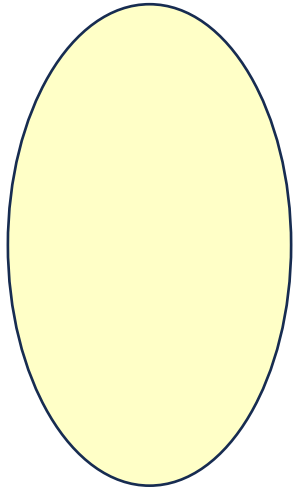
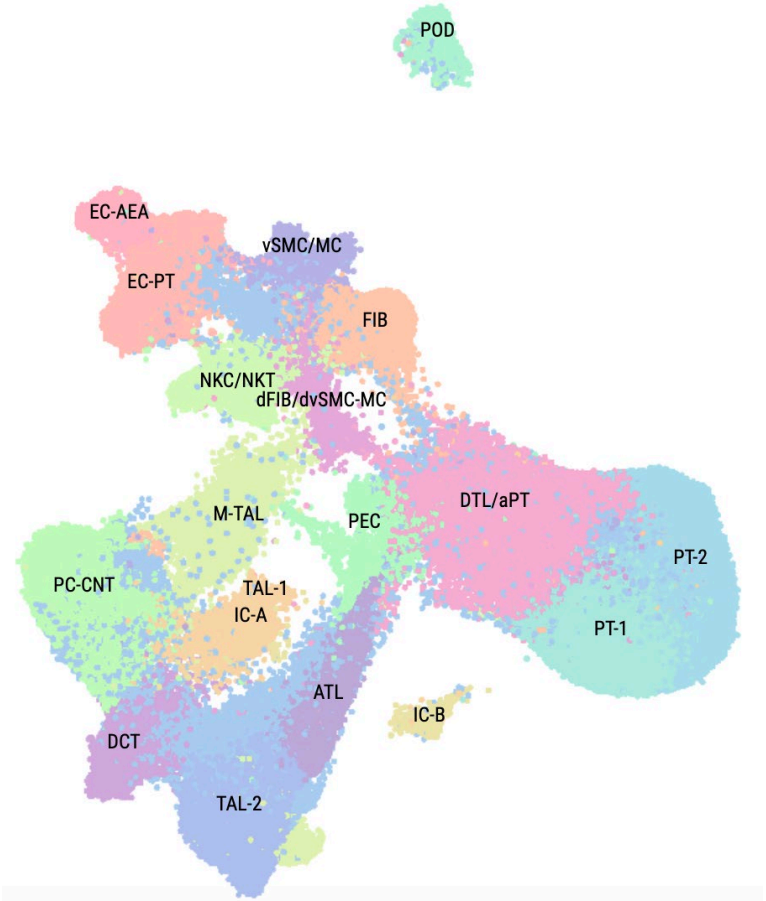
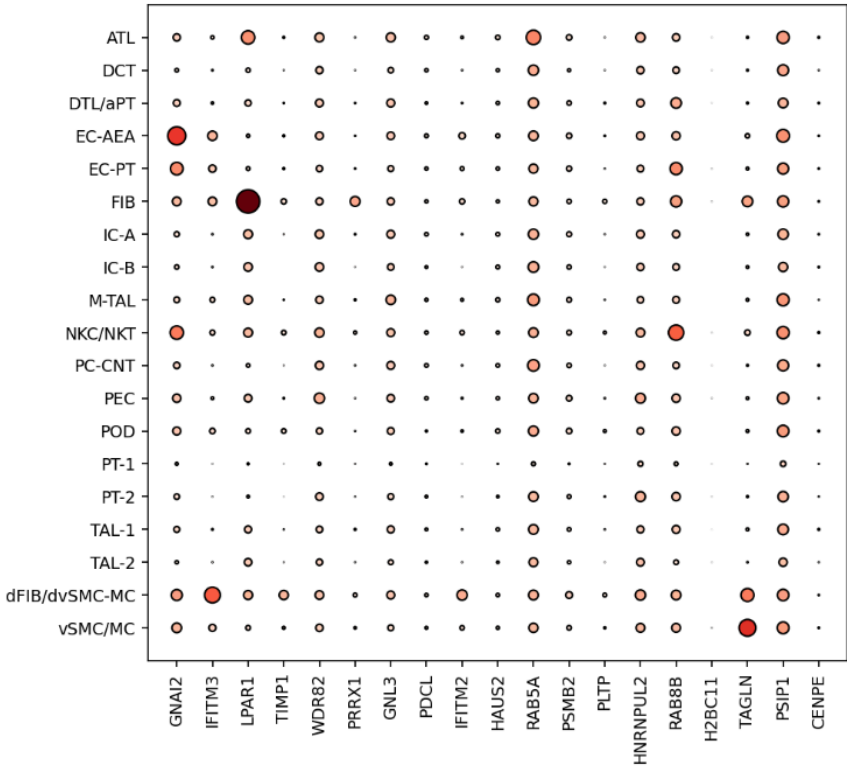


Figure 6

Ongoing analysis

2) Projecting the Classifier to enrich of cell types





3) Correlate the classifier with the clinical phenotype

gene	pearson	pval	fdr
GNAI2	-0.656	7.5E-07	4.05E-05
IFITM3	-0.586	1.88E-05	0.000338
LPAR1	-0.588	1.7E-05	0.000338
TIMP1	-0.574	3.05E-05	0.000376
WDR82	-0.569	3.72E-05	0.000376
PRRX1	-0.566	4.18E-05	0.000376
GNL3	-0.555	6.2E-05	0.000478
PDCL	-0.541	0.000105	0.000711
IFITM2	-0.528	0.000164	0.000957
HAUS2	-0.525	0.000177	0.000957
RAB5A	-0.485	0.000638	0.003091
PSMB2	-0.482	0.000687	0.003091
PLTP	-0.466	0.001086	0.004511
HNRNPUL2	-0.451	0.001649	0.005935
RAB8B	-0.454	0.001542	0.005935
H2BC11	-0.437	0.002373	0.008008
TAGLN	-0.426	0.003175	0.010087
PSIP1	-0.419	0.003759	0.011277
CENPE	-0.411	0.004582	0.013024
CEP85L	-0.39	0.007394	0.019964
MFSD6L	-0.38	0.009153	0.023537
CFAP47	-0.376	0.010029	0.024008
EXT2	-0.375	0.010226	0.024008
ZNF625	-0.371	0.011192	0.025181
INSYN2A	-0.369	0.011669	0.025205
IRF2	-0.36	0.013878	0.028824
CCR4	-0.343	0.019486	0.038563
UBQLN3	-0.342	0.019996	0.038563

Figure 1: TNNT2 expressed in Podocyte and PEC cells across all samples

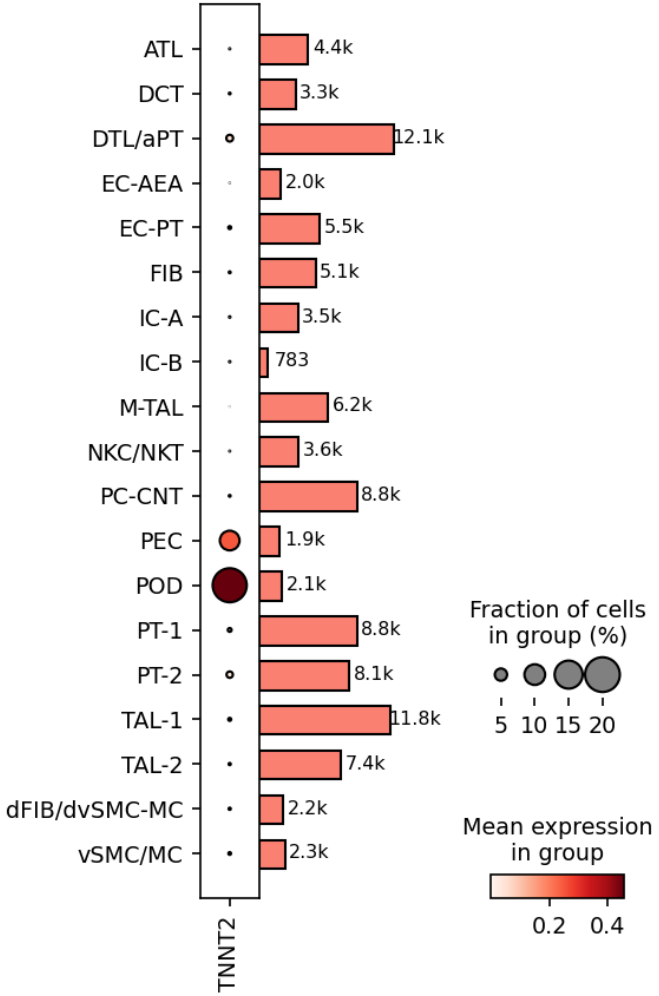
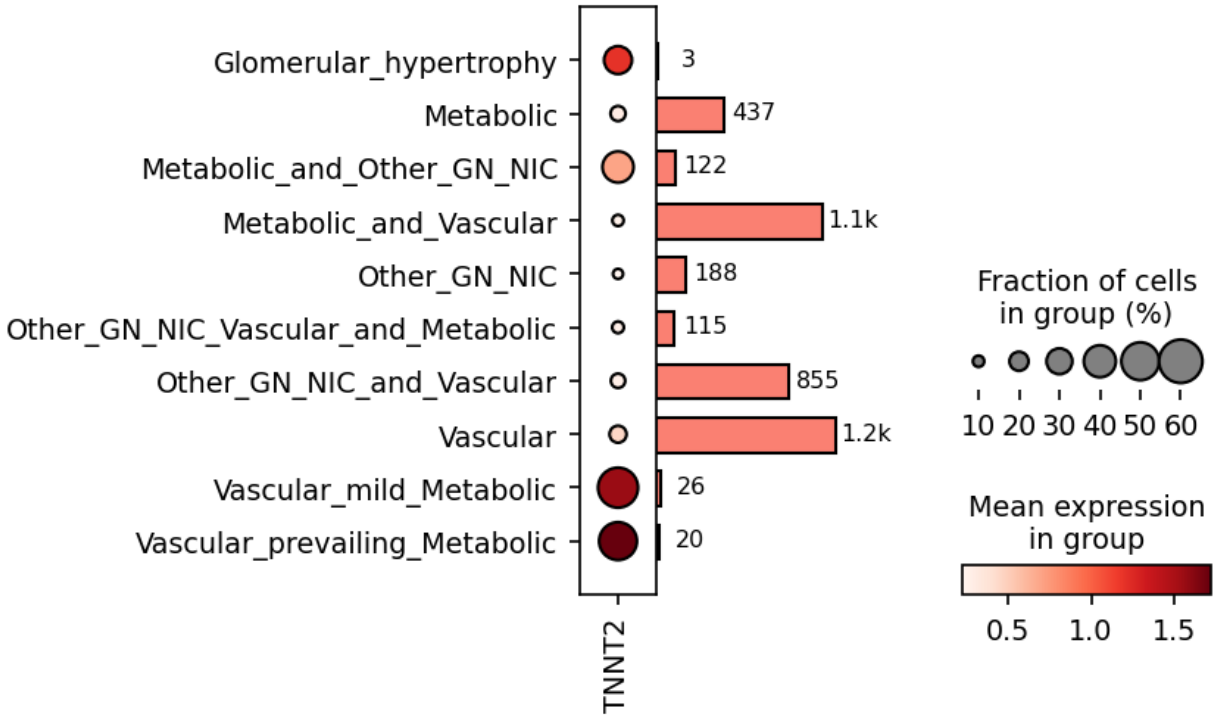
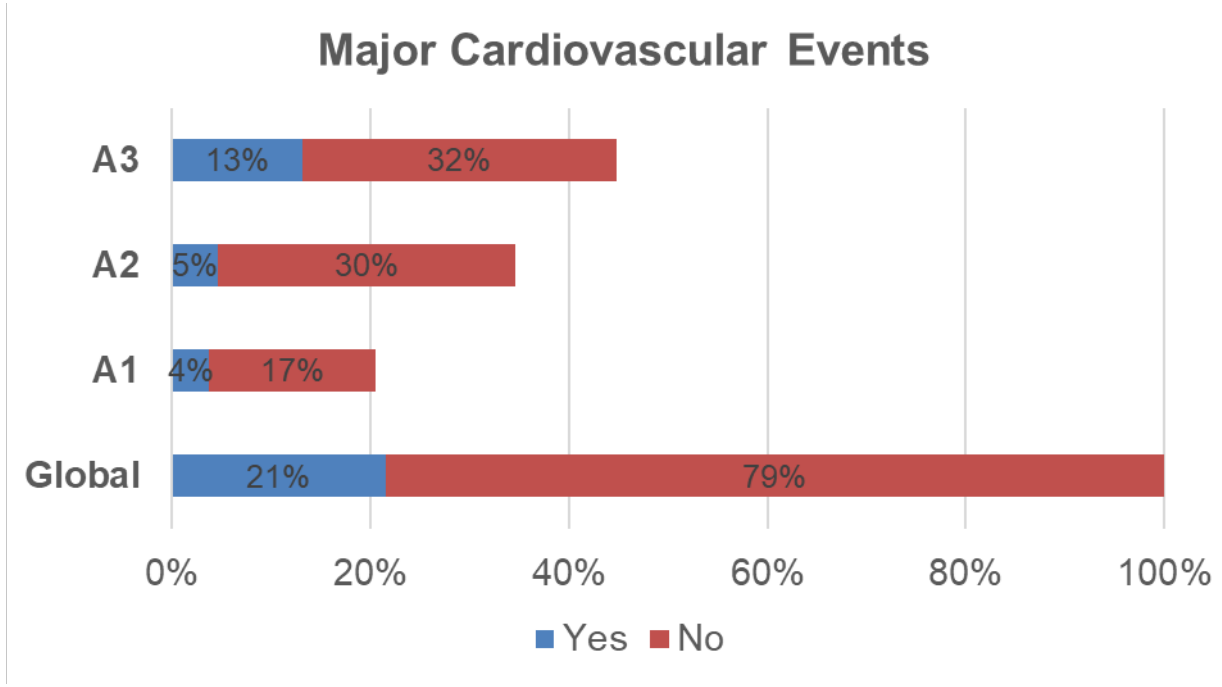
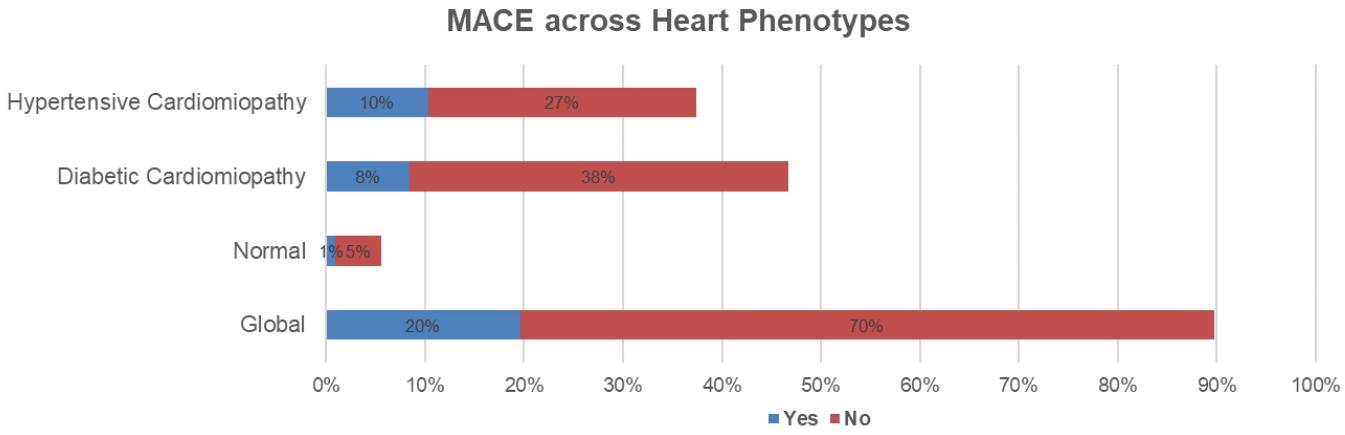


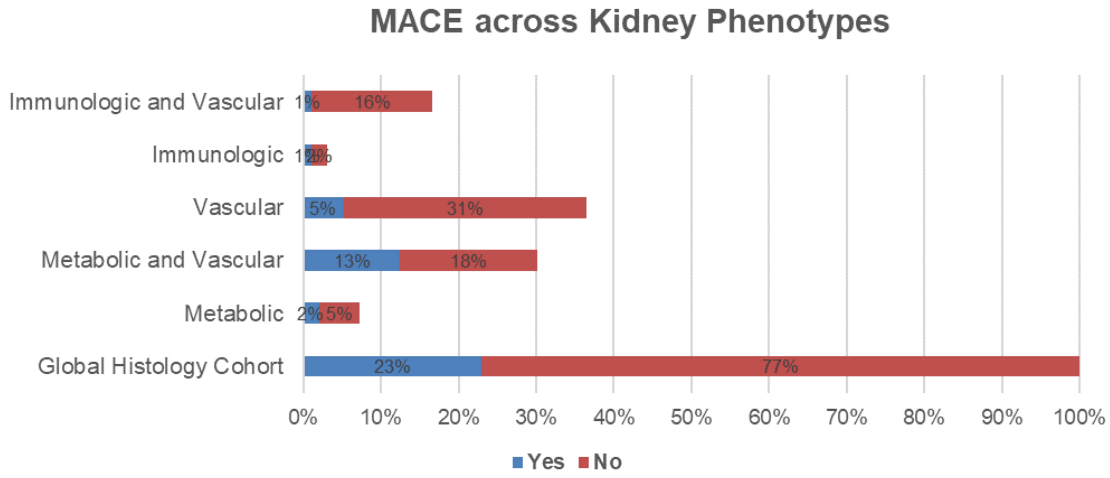
Figure 2: TNNT2 POD and PEC cells across the Bari histology label higher expression in the "metabolic/vascular endotype-DKD"



Frequency of Major Cardiovascular Events

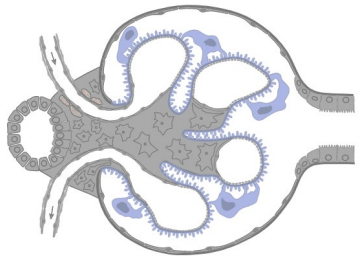






Preliminary omics results

Podocytes



TNNT2 (Cardiac Muscle Troponin T) expression in podocytes was associated with metabolic/vascular damage

KNOW-CKD, 2017 patients with CKD1-5 (pre-dialysis)

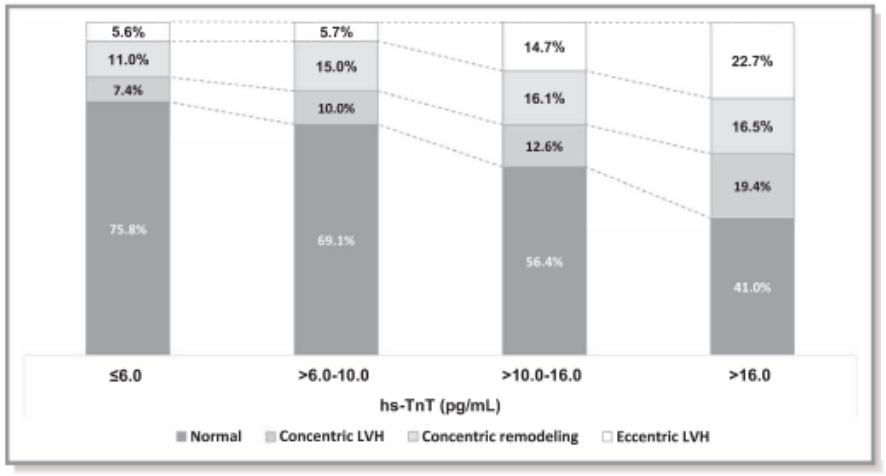


Figure 2. Elevated hs-TnT levels are associated with LVH in patients with chronic kidney disease. With increasing quartiles of hs-TnT, the prevalence of concentric and eccentric LVH and concentric remodeling increases. hs-TnT indicates high-sensitivity cardiac troponin T; LVH, left ventricular hypertrophy;

hs-TnT and Echocardiographic Parameters in CKD *Kang et al*

Clinical Perspective

What Is New?

- In patients with chronic kidney disease, high-sensitivity troponin T (TnT) is strongly associated with alterations of left ventricular structure and diastolic dysfunction, regardless of estimated glomerular filtration rate strata.
- Baseline levels of high-sensitivity TnT are predictive of new left ventricular hypertrophy on follow-up.

What Are the Clinical Implications?

- This research suggests that a high-sensitivity TnT assay could be a useful targeted strategy for risk stratification of patients who may need further cardiac evaluation, regardless of renal function.
- Further studies are needed to confirm the ideal cutoff values for high-sensitivity TnT in chronic kidney disease.

Kang E, et al. J Am Heart Assoc. 2019

RNA expression profiles from DN datasets

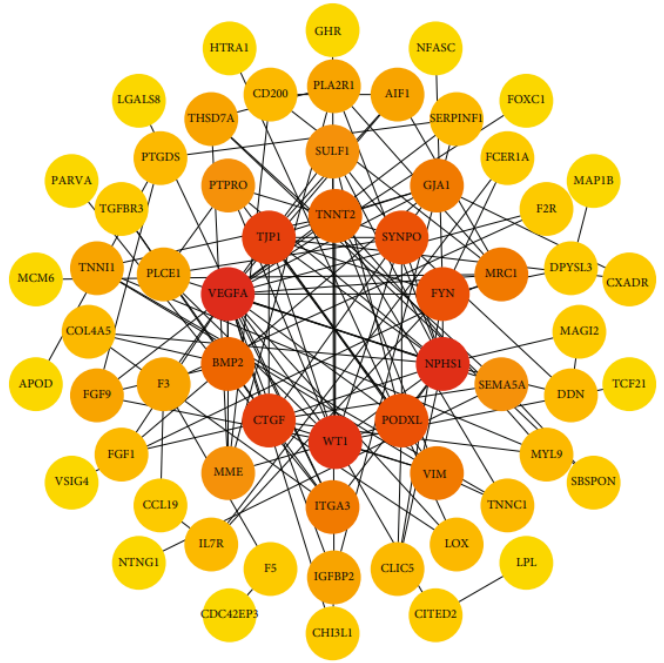
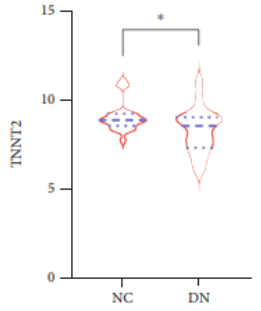


FIGURE 6: The PPI network of common DEGs and top 10 hub genes of DN. The top 10 hub genes were represented in the central of the network. The color of the nodes reflects the degree of connectivity (red color represents a higher degree, and yellow color represents a lower degree).



Chen S, et al. Dis Markers. 2022

kidney INTERNATIONAL ISN

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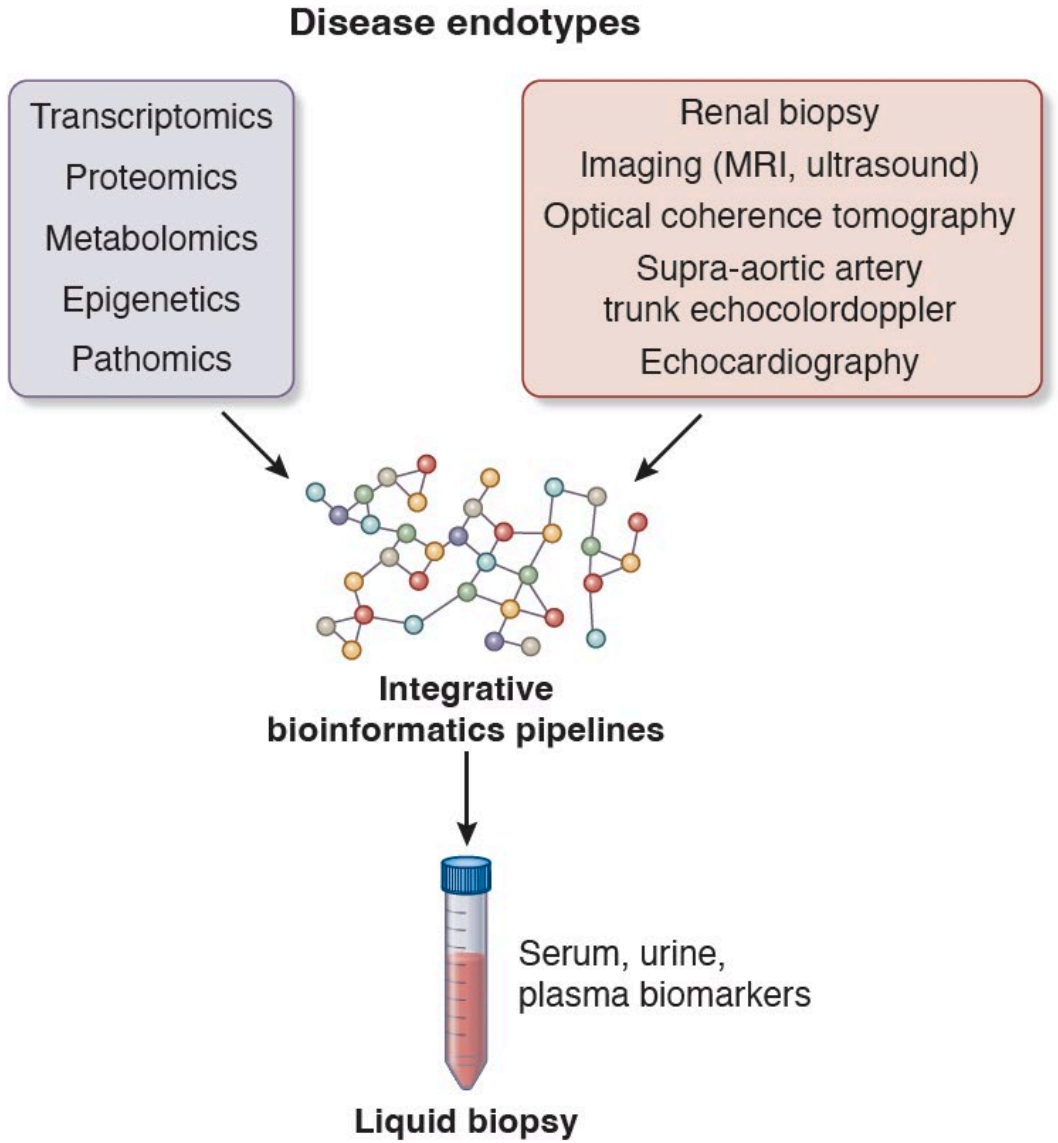
Deep endotyping of renal damage in type 2 diabetes: ex uno, plures

Loreto Gesualdo [1] · Matthias Kretzler [2] · Paola Pontrelli [1]



Gesualdo et al. Kidney Int 2026 Feb 27

DEEP PHENOTYPING

The details of disease

Precision medicine demands precise matching of deep genomic and phenotypic models — and the deeper you go, the more you know.

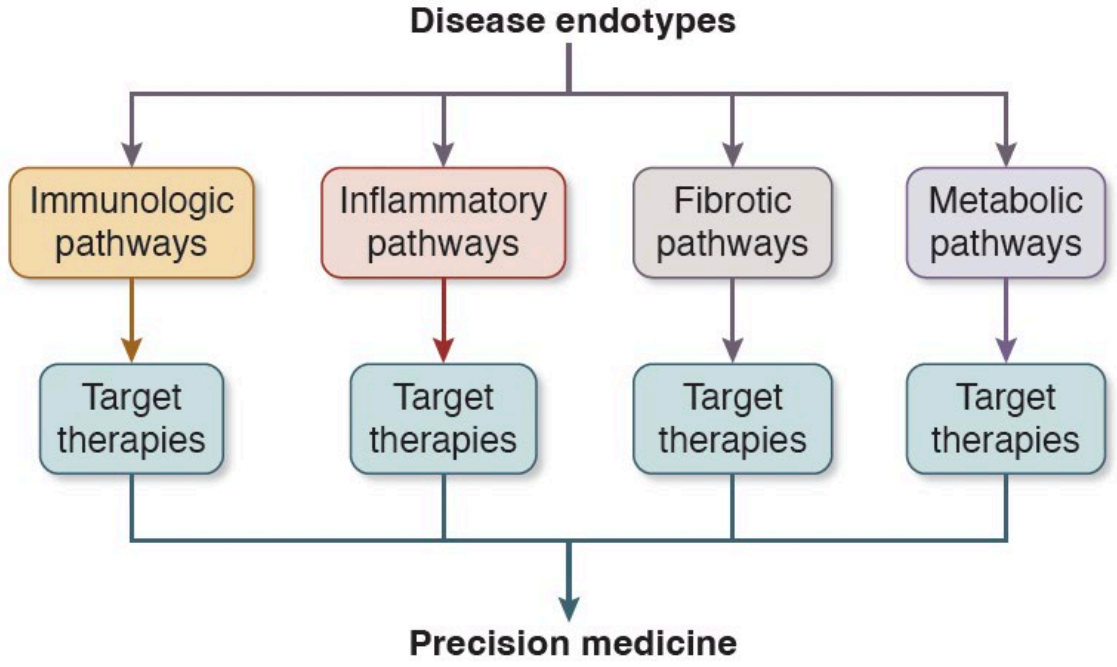
DIVIDING DIABETES

Diabetes exemplifies the problem of imprecise phenotypes. "***There are a hundred ways to be diabetic,*** *involving different processes in the pancreas, liver, muscle, brain and fat*" says Gary Churchill, a mouse geneticist at the Jackson Laboratory in Bar Harbor, Maine.

Different genes are responsible for particular subtypes of diabetes, so mixing them together obscures the reasons why people with the same genetic mutation respond differently to the same treatment.

Cathryn M. Delude, Nature 2015

BREAKTROUGH CHALLENGES



clinical decisions treat y/n

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We are trying to refine each individual subtype through endotyping to more accurately stratify renal and cardiovascular risk and deliver a precision medicine approach.

CONCLUSIONS

- The BEAT-DKD Cohort represents an opportunity to better define the true prevalence of NDKD in diabetes and refine patient's stratification to guide treatment, given the possibility that a diagnosis of NDKD may offer additional treatment options. Other protocol biopsy studies are needed!

- The search for new biomarkers in DKD is still an open challenge and is constantly evolving also thanks to the continuous evolution of technologies and should take into account the different patient's endotype.

- The use of biopsy should be considered, at this time, in the enrollment of more homogeneous patient populations in clinical trials and might help to address the unmet medical needs of these patients and improve the overall effectiveness of these studies.

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BEAT DKD

ABIONYX

Aferetica Purification Therapies

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Prof. J. Kellum

Prof. M. Gomez

Prof. S. Sourbron

Prof. Ali G Gharavi

Prof. R. Monteiro

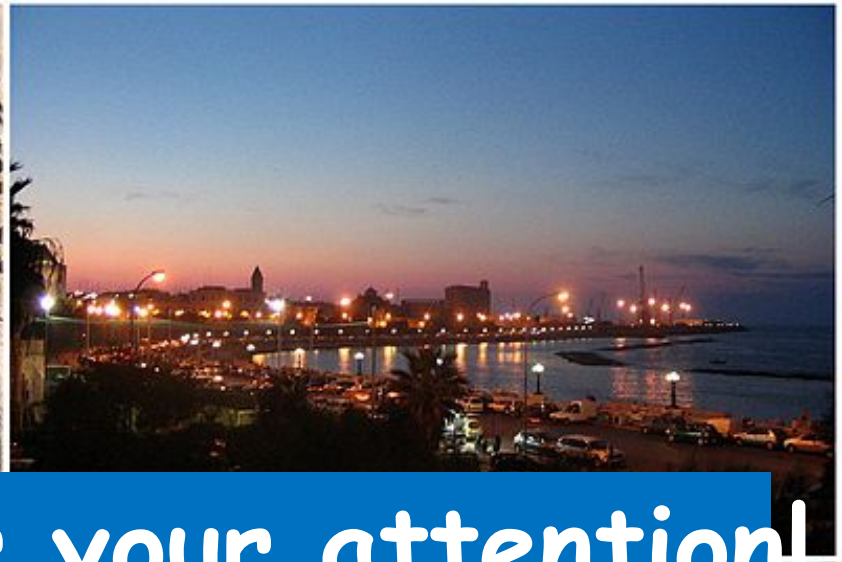
Prof. Simon C Satchell
Prof. Richard J M Coward

Prof. Hiddo Heerspink

Prof. Alessia Fornoni

Prof. Barisoni Laura

To all patients participating in the study!

Thank you for your attention!