ERKNet
The European Rare Kidney Disease Reference Network

european society for paediatric nephrology

IPNA International Pediatric Nephrology Association
GREAT CARE FOR LITTLE KIDNEYS. EVERYWHERE

WELCOME TO

ESPN/ERKNet
Educational Webinars on Pediatric Nephrology & Rare Kidney Diseases

Date: 08 Sept 2020

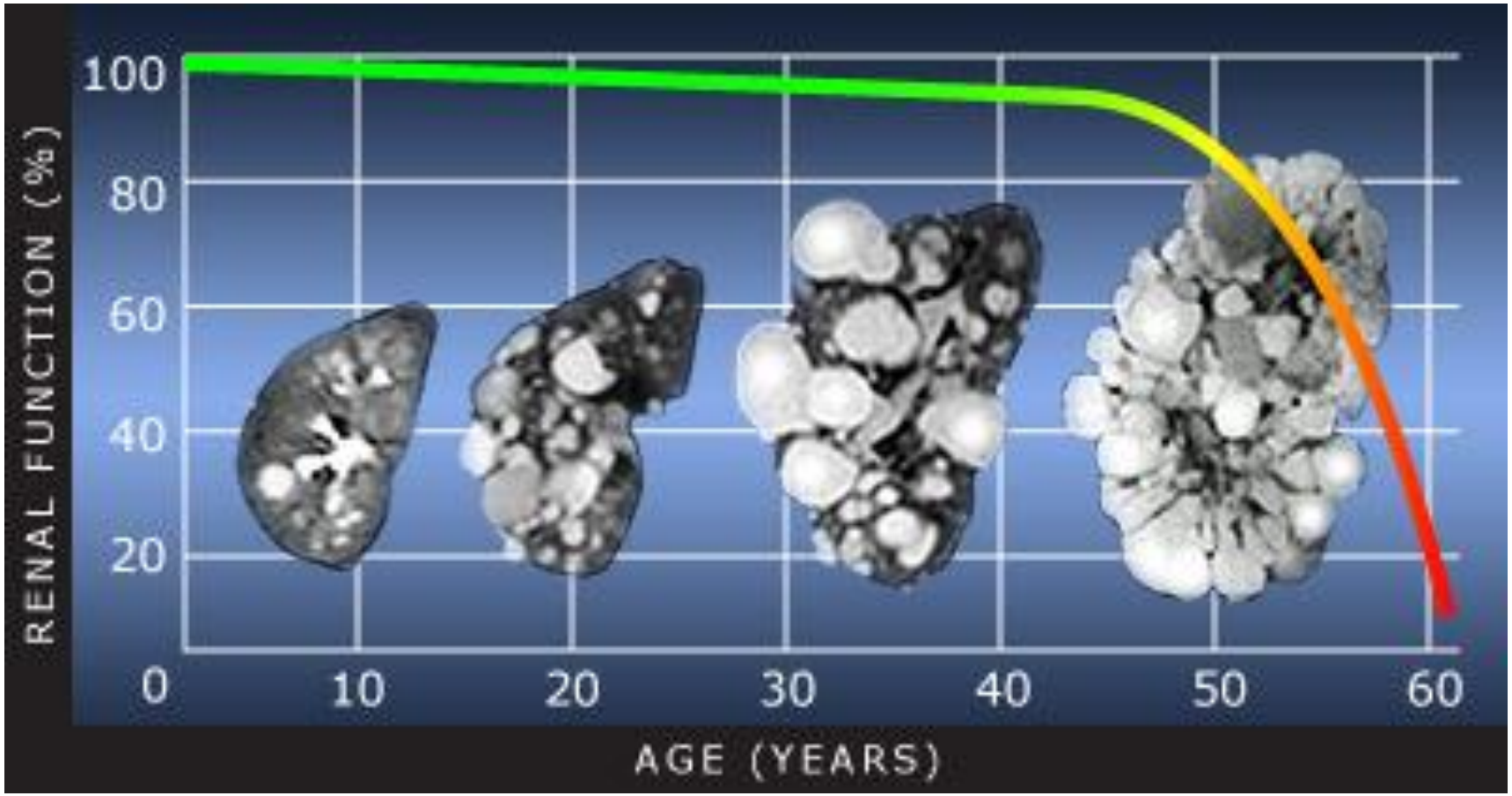
Topic: ADPKD

Speaker: Bert Bammens

Moderator: Max Liebau

Autosomal Dominant Polycystic Kidney Disease

1/400 to 1/1000 live births – progressive cystic deformation and growth of kidneys



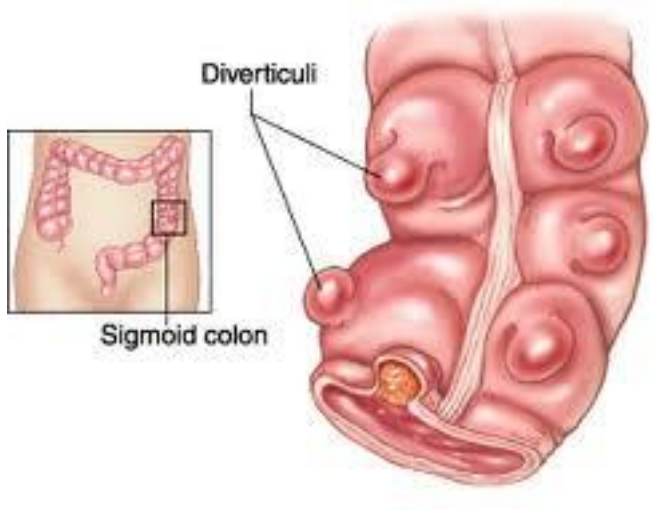
CKD stage 5D (median age): 58y PKD1, 79y PKD2 - 5-10% of ESRD incidence

ADPKD – extrarenal manifestations

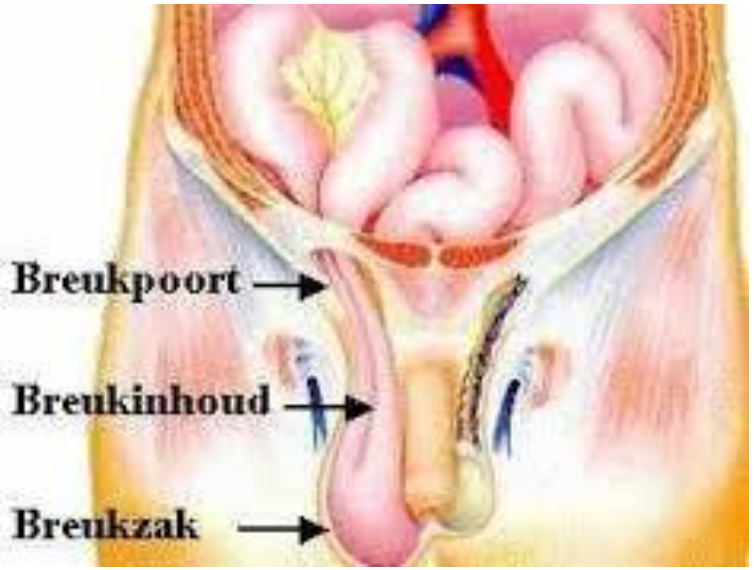
- **Hepatic and pancreatic cysts**
  - asymptomatic in many patients, but can expand and cause pain and infection; rarely massive PLD
- **Cardiac valvular abnormalities**
  - Mitral valve prolapse, tricuspid and aortic regurgitation
- **Intracranial aneurysms**
  - Found in approximately 5% of patients with no family history and about 22% of patients with family history of ICA or SAH
- **Seminal vesicle cysts**
  - Found in ~39-60% of men; undefined risk of infertility

ADPKD – extrarenal manifestations

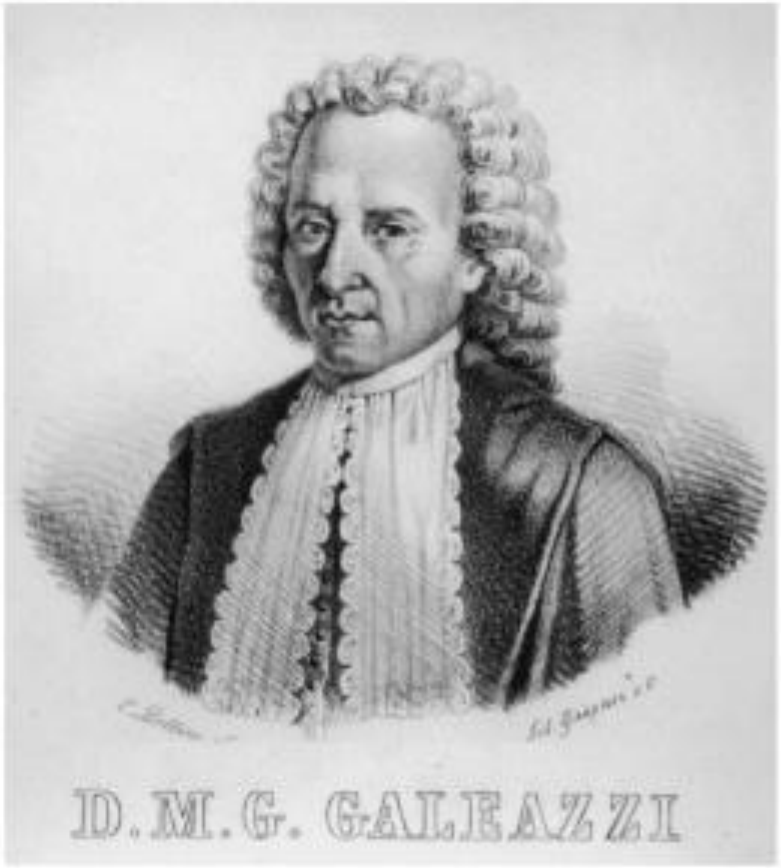
Colonic diverticulosis



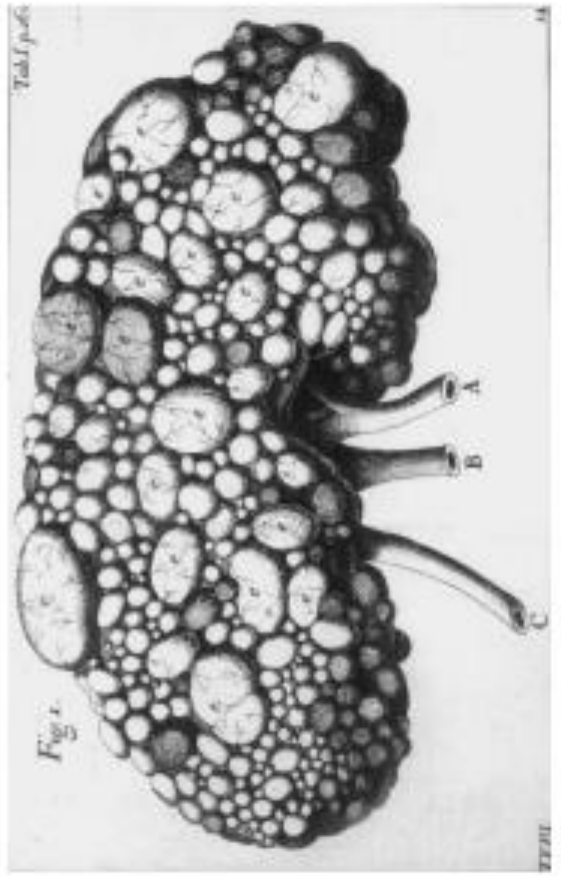
Abdominal wall hernia



First reports: descriptive



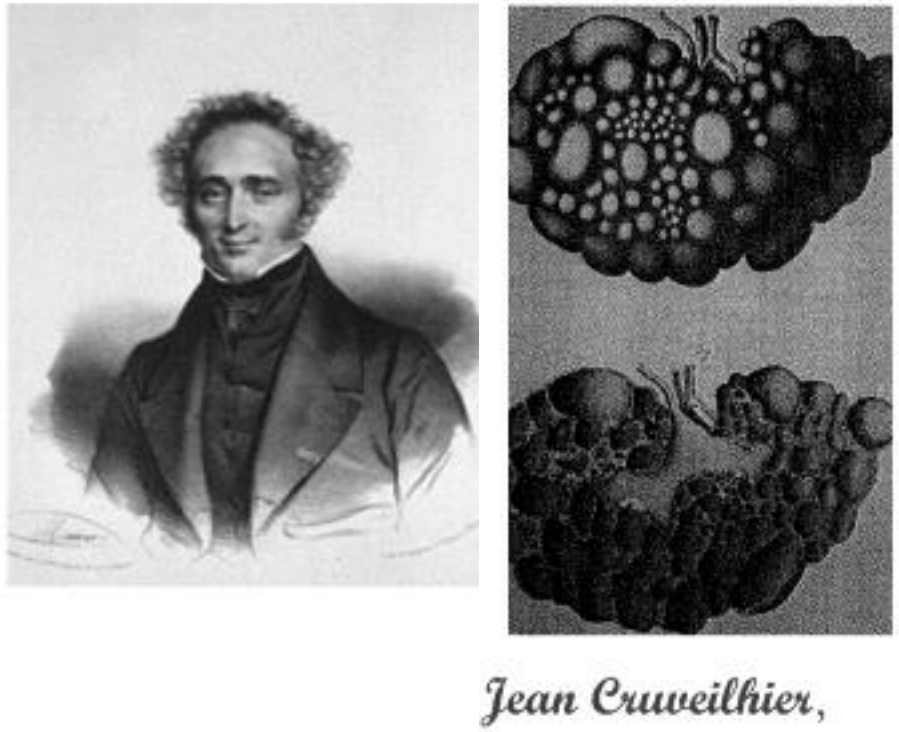
Domenico Galeazzi (1686-1775)



De renum morbis 1757

Fogazzi et al. Nephrol Dial Transplant 13: 1039-1040, 1998

First reports: descriptive



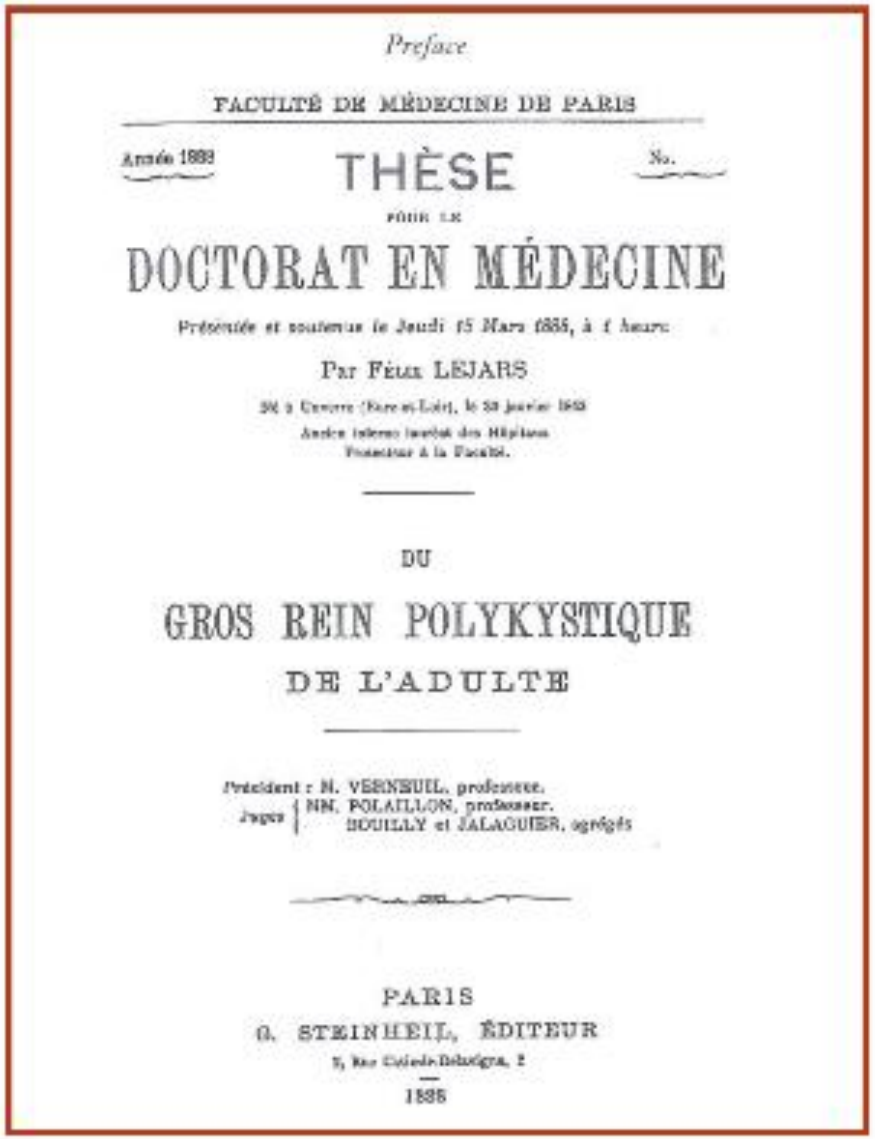
Jean Cruveilhier,

Anatomie pathologique du corps humain, 1829

Pierre Rayer,

Traité des maladies des reins, 1841

First reports: descriptive



Preface

FACULTÉ DE MÉDECINE DE PARIS

Année 1888 — THÈSE — No.

POUR LE

DOCTORAT EN MÉDECINE

Présentée et soutenue le Jeudi 15 Mars 1888, à 1 heure

Par FÉLIX LEJARS

Né à Cuncy (Nièvre-et-Loire), le 30 janvier 1860

Ancien interne lauréat des Hôpitaux

Prosecteur à la Faculté.

DU

GROS REIN POLYKYSTIQUE

DE L'ADULTE

Président : M. VERNEUIL, professeur.

Juges : MM. POLAILLON, professeur.
BOUILLY et JALAGUIER, agrégés

PARIS

G. STEINHEIL, ÉDITEUR

2, Rue Casimir-Delavigne, 2

1888

"La polykystose rénale sera peut-être un jour traitable"

F. Lejars, 1888

Till second decade of 21st century (and still): treatments focus on symptoms & complications

Standard care	Lifestyle approaches (general – like in many other diseases)
Blood pressure control	**Maintenance of healthy BW**
Pain control	Frequent H_2O intake
Antibiotics for UTIs	Avoidance of caffeine and smoking
Antidepressants	**Salt restriction (sodium chloride <6 g/d)**
Dialysis	**Low protein intake (<1 g/kg BW/d)**
Renal transplantation	Bed rest
	Regular exercise

BW, body weight; UTIs, urinary tract infections

Halvorson CR et al. Int J Nephrol Renovasc Dis 3: 69-83, 2010

The identification of the genetic background of ADPKD…

…has helped to better predict the (renal) prognosis of the disease.

…has paved the way for research into the many mechanisms of the disease.

…has been pivotal in moving treatment from “only symptomatic” to “disease-modifying”.

…is possible in every individual patient.

Which statement is NOT correct?

Genetics: a giant step forward!

Polycystic Kidney Disease: The Complete Structure of the *PKD1* Gene and Its Protein

The International Polycystic Kidney Disease Consortium*

Cell, Vol. 81, 289–298, April 21, 1995

PKD 1 (± 78%)

Cytogenetic location 16p13.3

Genetics: a giant step forward!

Chromosome 4 localization of a second gene for autosomal dominant polycystic kidney disease

D.J.M. Peters[1], L. Spruit[1], J.J. Saris[1], D. Ravine[2], L.A. Sandkuijl[3], R. Fossdal[3], J. Boersma[1], R. van Eijk[1], S. Nørby[4], C.D. Constantinou-Deltas[5], A. Pierides[5], J.E. Brissenden[6], R.R. Frants[3], G.-J.B. van Ommen[1] & M.H. Breuning[1]

Nature Genetics **5**, 359 - 362 (1993)

PKD 2 (± 15%)

Autosomal Dominant Polycystic Kidney Disease: Localization of the Second Gene to Chromosome 4q13–q23

WILLIAM J. KIMBERLING,*,[1] SHRAWAN KUMAR,* PATRICIA A. GABOW,† JUDITH B. KENYON,* CHRISTOPHER J. CONNOLLY,* AND STEFAN SOMLO‡

GENOMICS 18, 467–472 (1993)

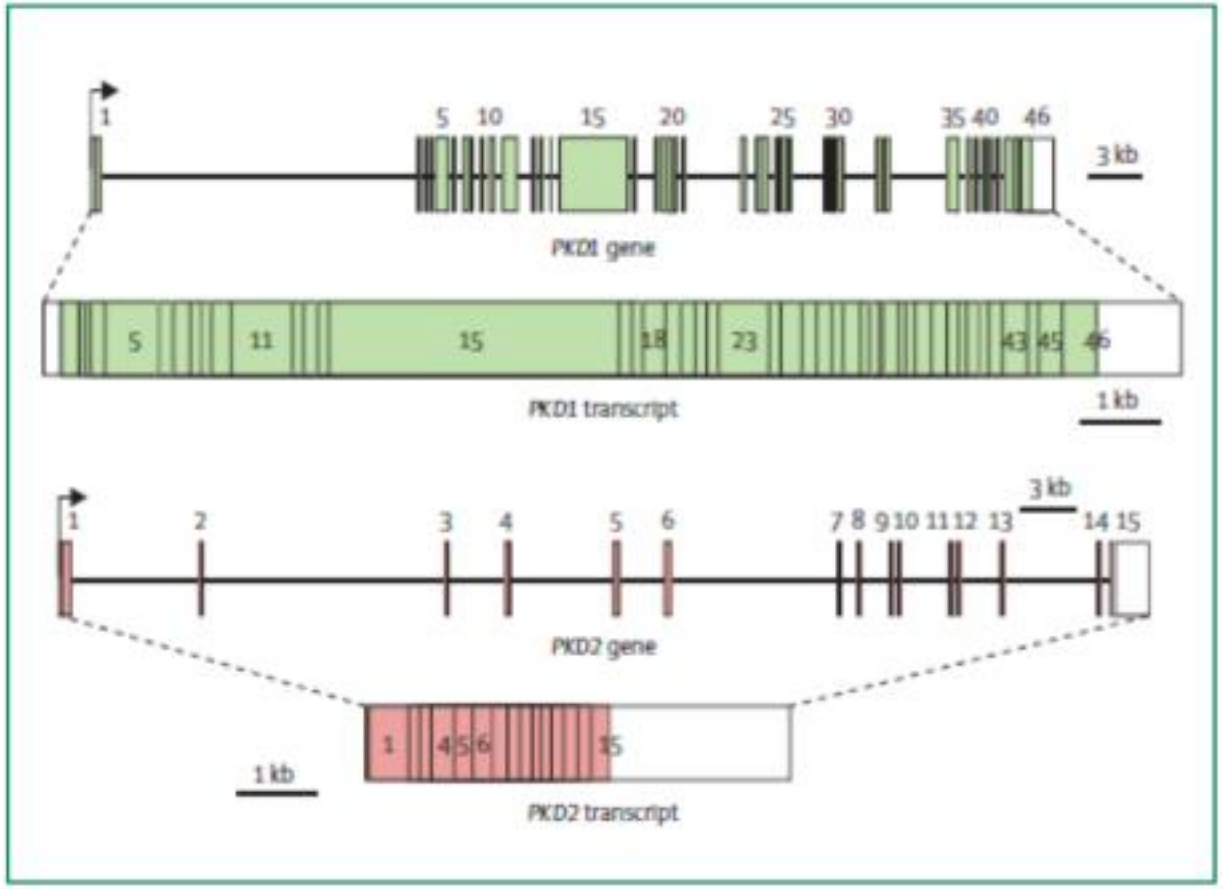
cytogenetische locatie 4q22.1

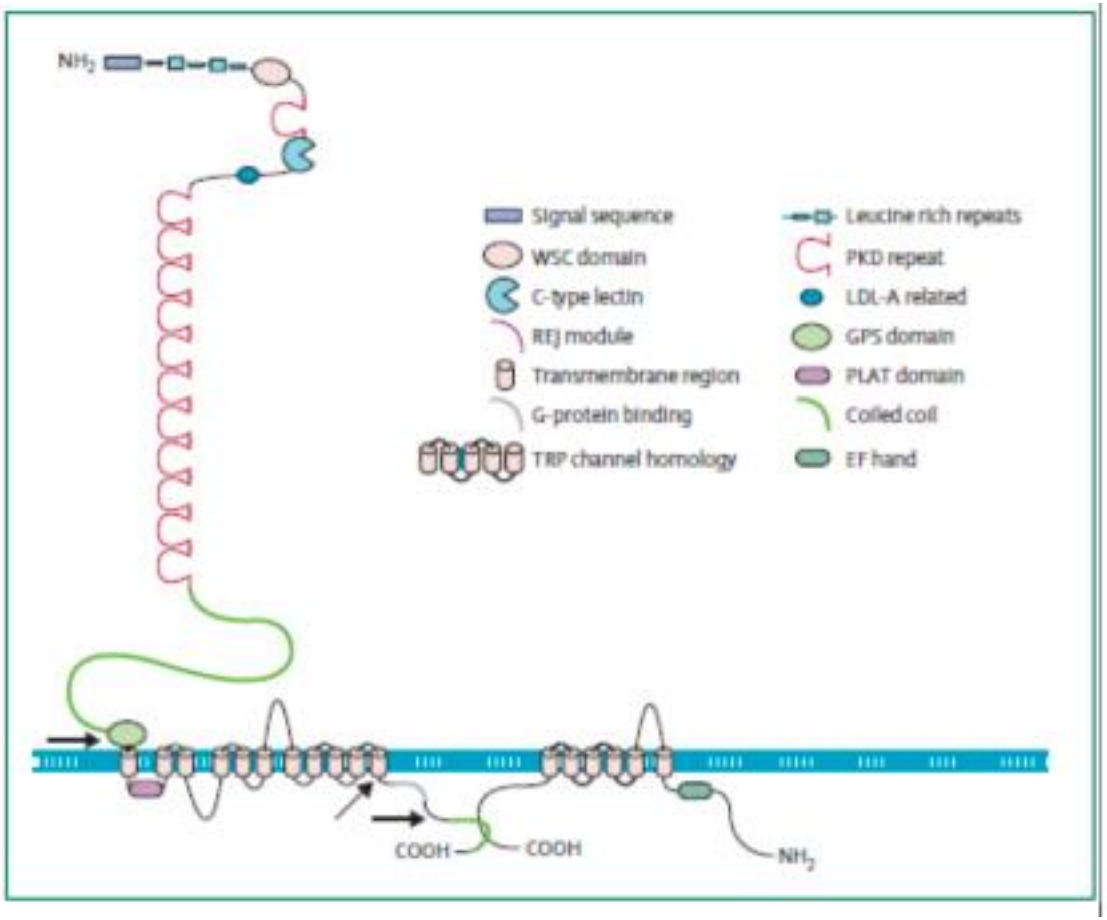
***PKD2*, a Gene for Polycystic Kidney Disease That Encodes an Integral Membrane Protein**

Toshio Mochizuki, Guanqing Wu*, Tomohito Hayashi*, Stavroulla L. Xenophontos, Barbera Veldhuisen, Jasper J. Saris, David M. Reynolds, Yiqiang Cai, Patricia A. Gabow, Alkis Pierides, William J. Kimberling, Martijn H. Breuning, C. Constantinou Deltas, Dorien J. M. Peters, Stefan Somlo†

Science. 1996 May 31;272(5266):1339-42.

Genetics: a giant step forward!





PKD1 > polycystin 1, a receptor-like, large integral membrane glycoprotein

PKD2 > polycystin 2, a transmembrane calcium channel

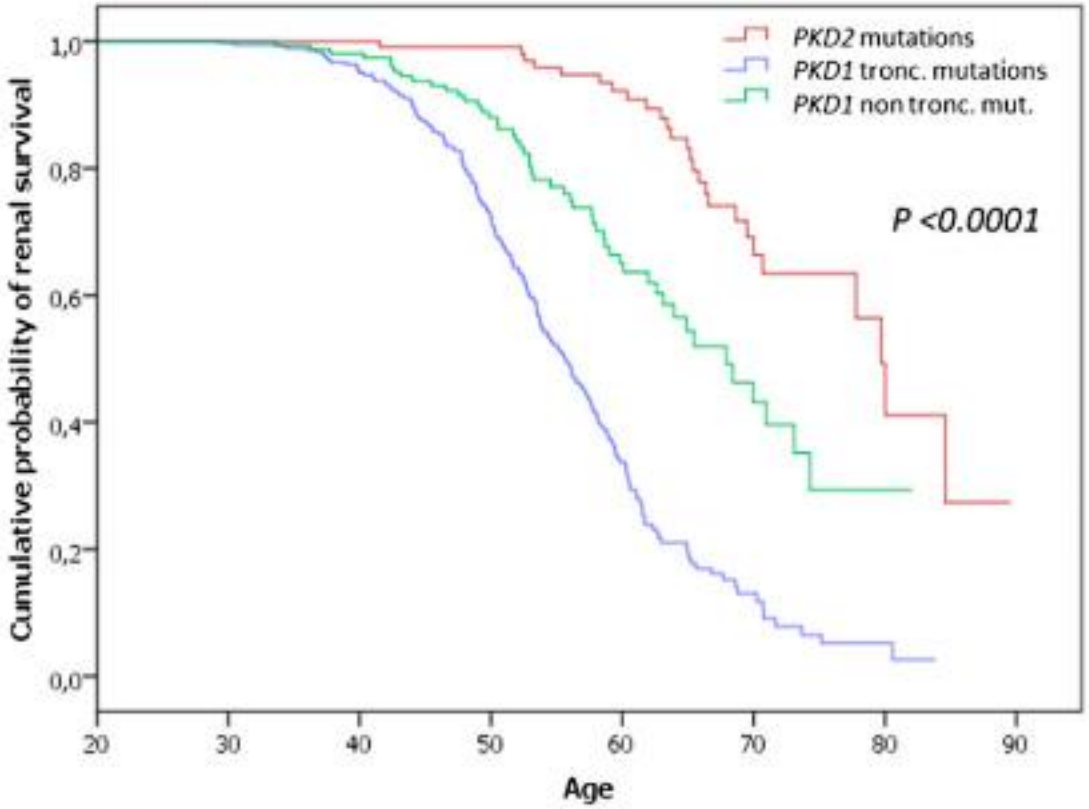
Genetics: a giant step forward!

Type of *PKD1* Mutation Influences Renal Outcome in ADPKD

Emilie Cornec-Le Gall,*† Marie-Pierre Audrézet,†‡ Jian-Min Chen,†‡ Maryvonne Hourmant,§ Marie-Pascale Morin,‖ Régine Perrichot,¶ Christophe Charasse,** Bassem Whebe,†† Eric Renaudineau,‡‡ Philippe Jousset,§§ Marie-Paule Guillodo,‖‖ Anne Grall-Jezequel,*† Philippe Saliou,†‡ Claude Férec,†‡ and Yannick Le Meur*†

PKD1 truncating vs. PKD1 non-truncating vs. PKD2

Cornec-Le Gall et al. J Am Soc Nephrol 24: 1006-1013, 2013



Patients at risk :

PKD1 truncating mutations (n=387)	356	296	175	53	11	2
PKD1 non truncating mutations (n=184)	172	144	134	48	15	1
PKD2 Mutations (n=133)	127	116	99	63	23	5

median age ESRD

55y PKD1 truncating

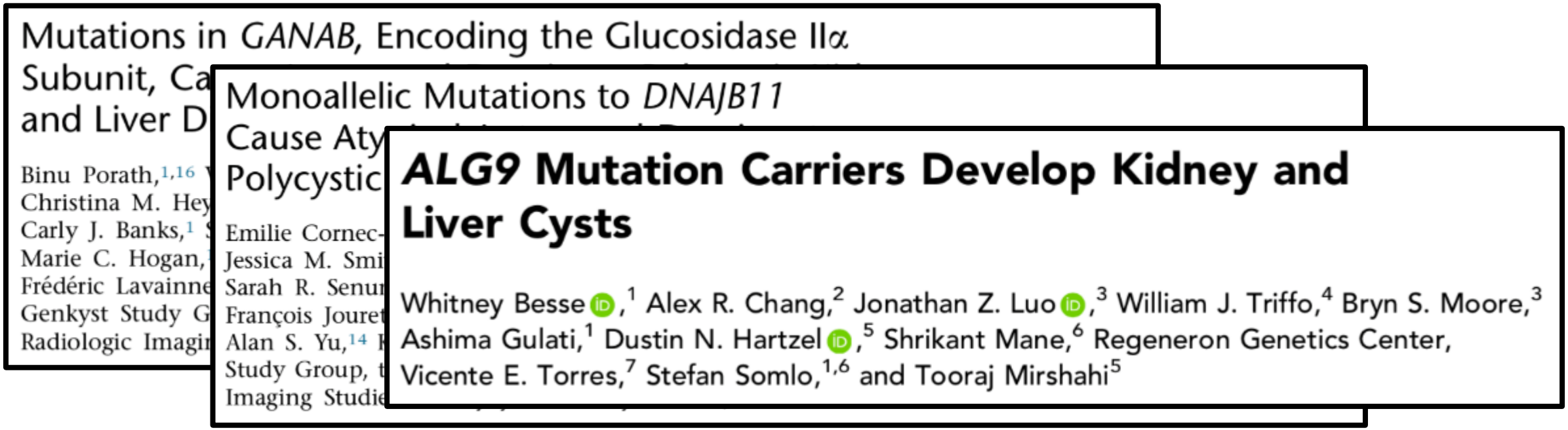
67y PKD1 non-truncating

79y PKD2

Cornec-Le Gall et al. J Am Soc Nephrol 24: 1006-1013, 2013

By the way, the identification of the genetic background of ADPKD…

…is not (yet) possible in every individual patient.
…and some have other than PKD1 or PKD2 mutations.

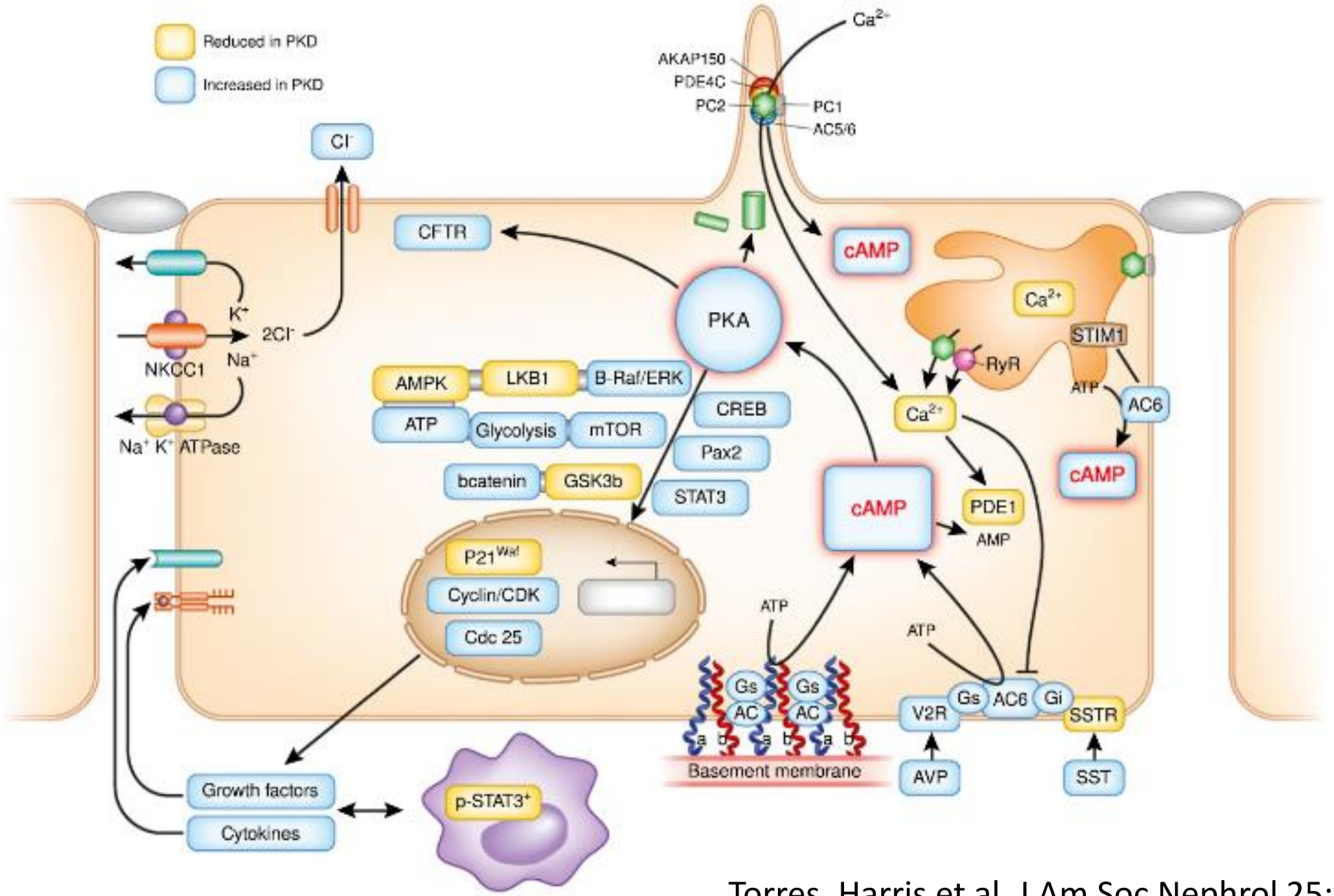


Porath et al. Am J Hum Gen 98: 1193-1207, 2016
Cornec-Le Gall et al. Am J Hum Gen 102: 832-844, 2018
Besse et al. J Am Soc Nephrol, 2019 https://doi.org/10.1681/ASN.2019030298

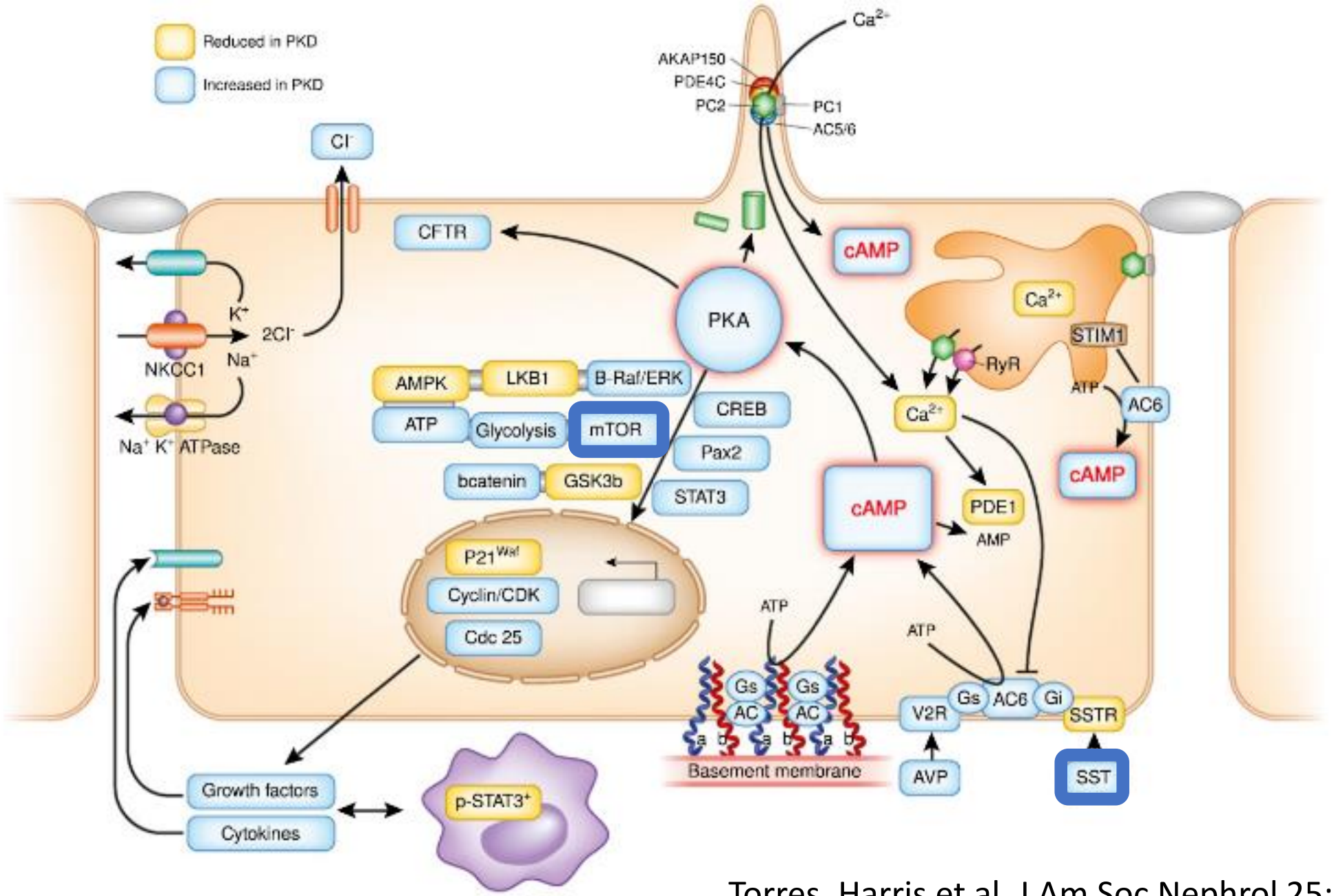
Genetics

Table 1
Viable ADPKD mouse models suitable for preclinical trials

Strain	Induction	
	Cre promoter	Target renal tubules/time
***Pkd1* floxed deletion (constitutive)**		
fl/–	*Ksp*-Cre	CD, DT
fl/fl	*Pkhd1*-Cre	CD
fl/fl	*Nestin*-Cre	Multiple mosaic
fl/–	*γGT*-Cre	PT, CD
***Pkd2* floxed deletion (constitutive)**		
fl/–	*γGT*-Cre	PT, CD
fl/fl	*Pkhd1*-Cre	CD
***Pkd1* floxed deletion (induced)**		
fl/fl	*CAG*-cre/Esr1 +OHT	All<P12
fl/fl	*CAG*-cre/Esr1 +OHT	All>P14
fl/fl	*Ksp*-Cre/ER +tam	CD, DT 4 d
fl/fl	*Ksp*-Cre/ER +tam	CD, DT >3 mo
fl/fl	*Mx1*-Cre/IFN +pI:pC	All P7
fl/–	*Mx1*-Cre/IFN +pI:pC	All P7
fl/fl	*Mx1*-Cre/IFN +pI:pC	All 5 wk
fl/fl	*Pax8*^{rtTA};TetC-Cre +dox	All 4 wk
***Pkd2* floxed deletion (induced)**		
fl/–	*Mx1*-Cre/IFN +pI:pC	All 6 wk
fl/fl	*Pax8*^{rtTA};TetC-Cre +dox	All 4 wk
***Pkd1* hypomorphic**		
nl/nl		
L3/L3		
T3041V/T3041V		
R3277C/R3277C		
R3277C/–		
***Pkd2* hypermutable**		
WS25/–		



Torres, Harris et al. J Am Soc Nephrol 25: 18-32, 2014
Harris, Torres et al. J Clin Invest 124: 2315-2324, 2014



Torres, Harris et al. J Am Soc Nephrol 25: 18-32, 2014
Harris, Torres et al. J Clin Invest 124: 2315-2324, 2014

RESULTS

Somatostatin analogues

vs. placebo: **5 studies, 123 participants**

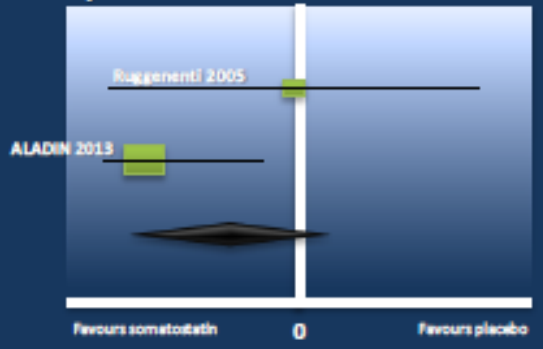
vs. mTOR-inhibitors: 1 study, 15 participants

Creatinine

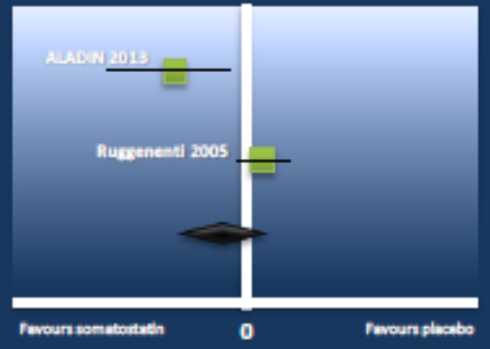
GFR

Total kidney volume

Cyst volume



Parenchimal volume



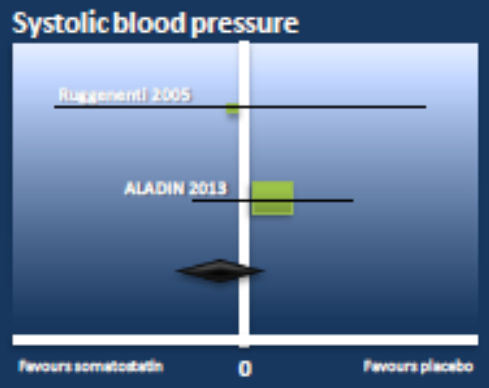
Albuminuria

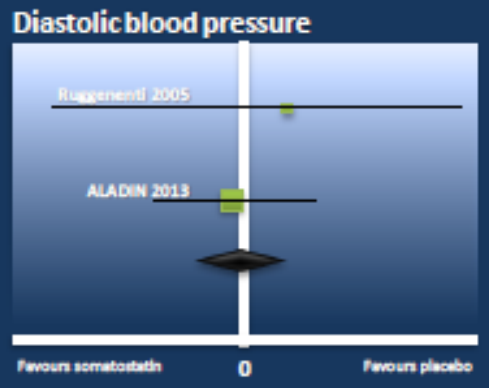
RESULTS

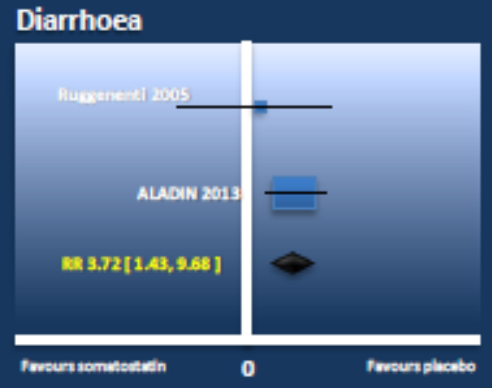
Somatostatin analogues

vs. placebo: **5 studies, 123 participants**

vs. mTOR-inhibitors: **1 study, 15 participants**







Rationale and Design of the DIPAK 1 Study: A Randomized Controlled Clinical Trial Assessing the Efficacy of Lanreotide to Halt Disease Progression in Autosomal Dominant Polycystic Kidney Disease

Meijer et al. Am J Kidney Dis 63: 446-455, 2014

JAMA | Original Investigation

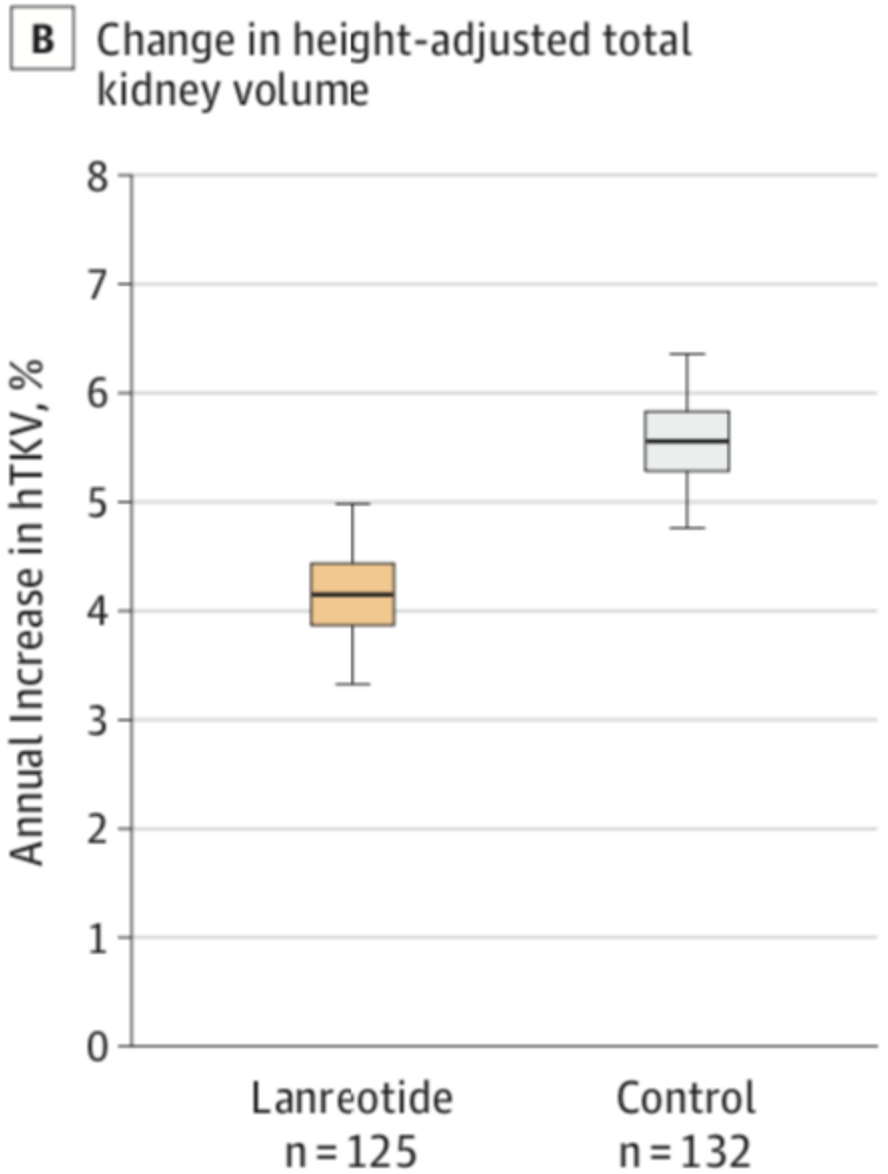
Effect of Lanreotide on Kidney Function in Patients With Autosomal Dominant Polycystic Kidney Disease

The DIPAK 1 Randomized Clinical Trial

DESIGN, SETTING, AND PARTICIPANTS An open-label randomized clinical trial with blinded end point assessment that included 309 patients with ADPKD from July 2012 to March 2015 at 4 nephrology outpatient clinics in the Netherlands. Eligible patients were 18 to 60 years of age and had an estimated glomerular filtration rate (eGFR) of 30 to 60 mL/min/1.73 m^2. Follow-up of the 2.5-year trial ended in August 2017.

INTERVENTIONS Patients were randomized to receive either lanreotide (120 mg subcutaneously once every 4 weeks) in addition to standard care (n = 153) or standard care only (target blood pressure <140/90 mm Hg; n = 152).

Meijer et al. JAMA 19: 2010-2019, 2018



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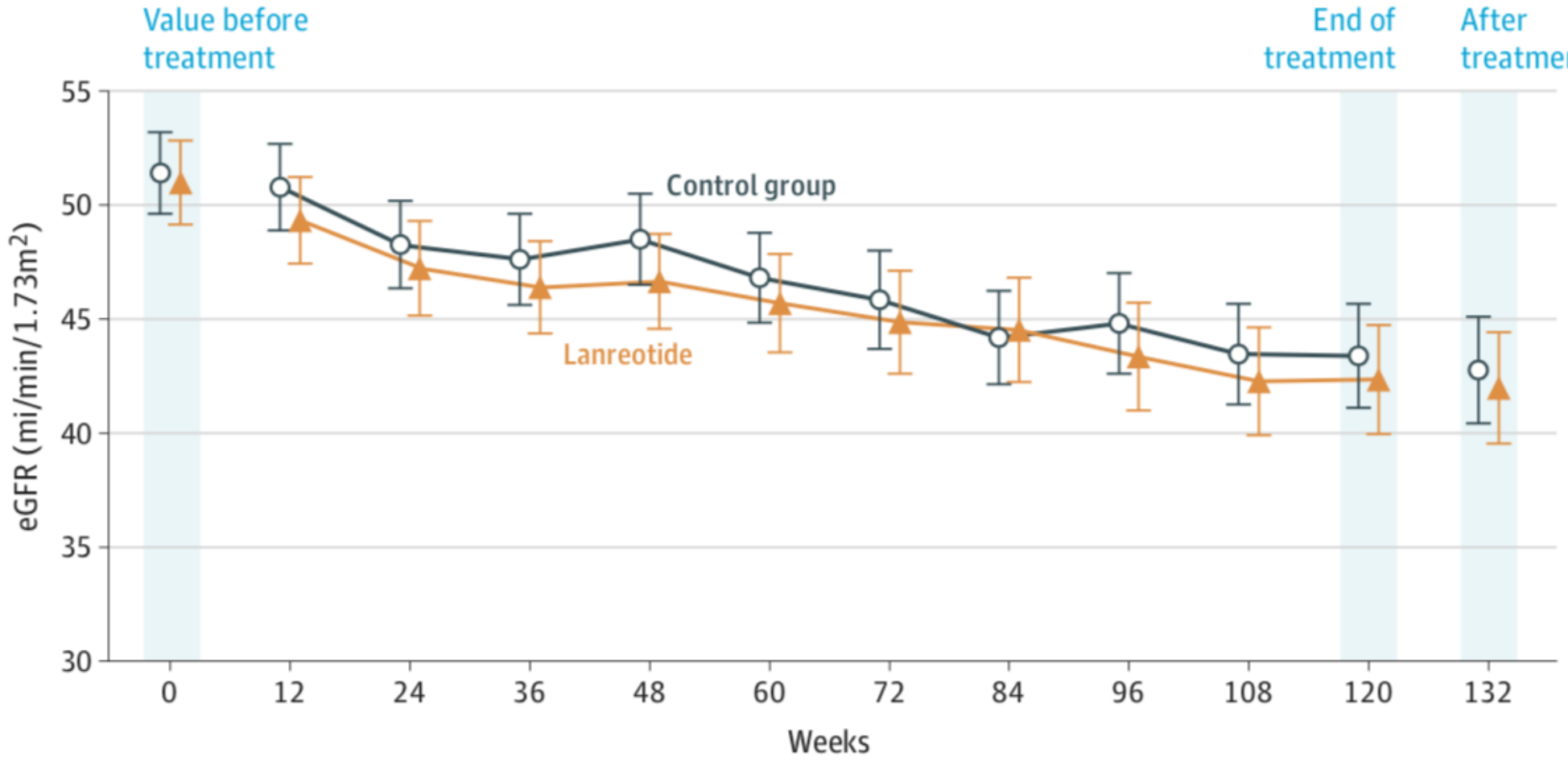
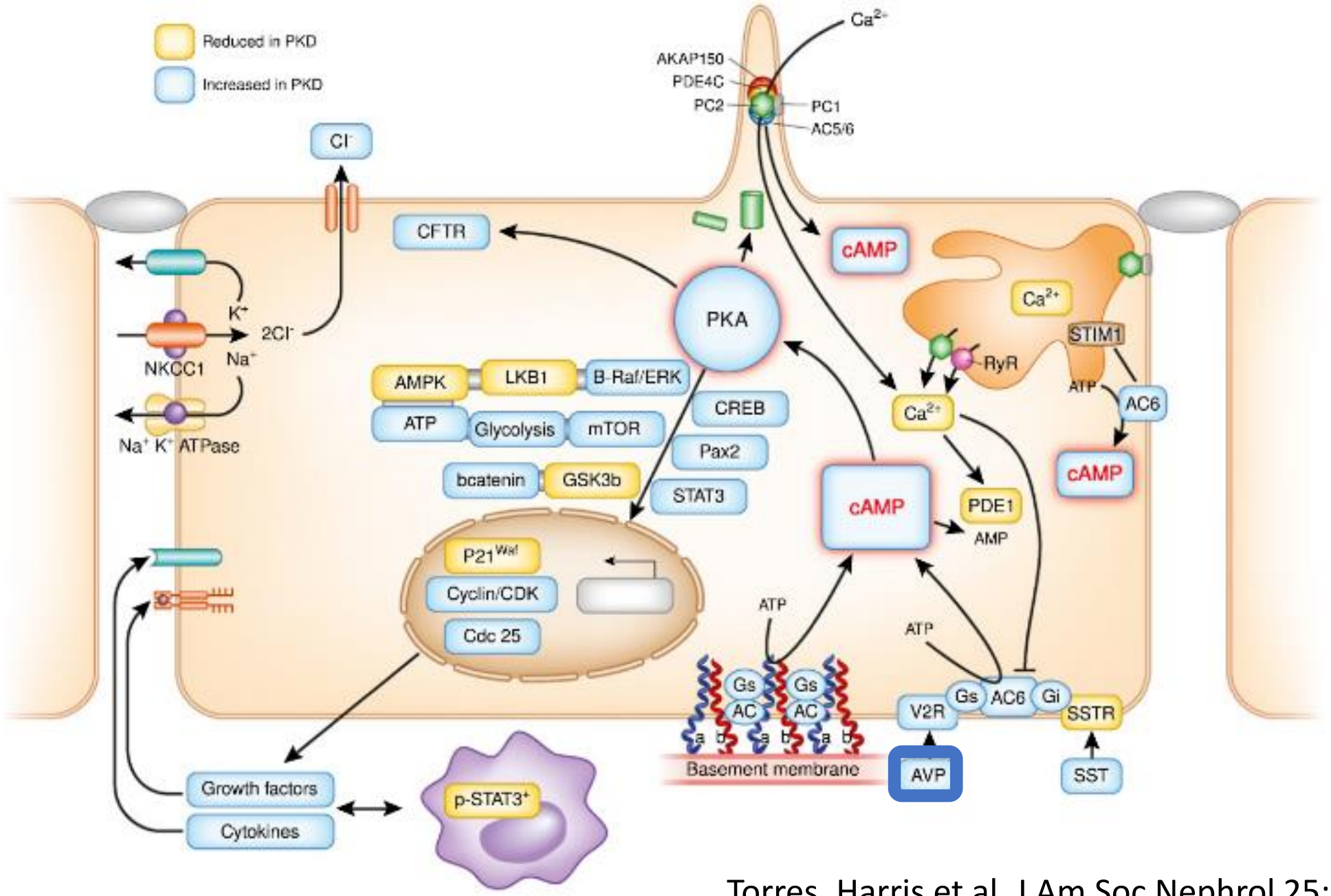


No. of patients												
Lanreotide	153	145	143	142	145	144	138	132	140	136	135	114
Control group	152	148	148	143	144	147	141	140	144	137	142	141

Meijer et al. JAMA 19: 2010-2019, 2018



Torres, Harris et al. J Am Soc Nephrol 25: 18-32, 2014
Harris, Torres et al. J Clin Invest 124: 2315-2324, 2014

What about high water intake?

Animal models: high water intake promotes diuresis by decreasing plasma concentrations of arginine vasopressin (AVP) and renal cAMP concentrations, which slows cyst progression.[1,2]

Human models:
Wang et al 2011[3]: The variation in the urinary cAMP rate is related to the osmolality according to a small study of 8 cases

Barash et al 2010[4]: 13 patients ADPKD and 10 healthy subjects / 7 days

3.14 +/- 0.32 l/day water intake day decreased Uosm in most ADPKD subjects below 270 mOsm/L (46%, p=0,04). Non-significant decrease in 24-hour urine cAMP excretion.

Higashihara E et al 2014[5]: high (H-, n = 18) and free (F-,n = 16) water intake / 1year

Plasma AVP and copeptin were lower in H- group (p=0.02).

Non-significant trends toward faster eGFR decline and TKV growth in H- group.

1. Nagao et al J Am Soc Nephrol 17: 2220-2227,2006
2. Hopp K, et al AM J Physiol Renal Physiol 308: F261-F266, 2015
3. Wang CJ et al Clin J Am Soc Nephrol 6: 192-197, 2011
4. Barash et alnClin J Am Soc Nephrol 5: 693-697, 2010
5. Higashihara E et al Nephrol Dial Transplant 29: 1710-1719, 2014

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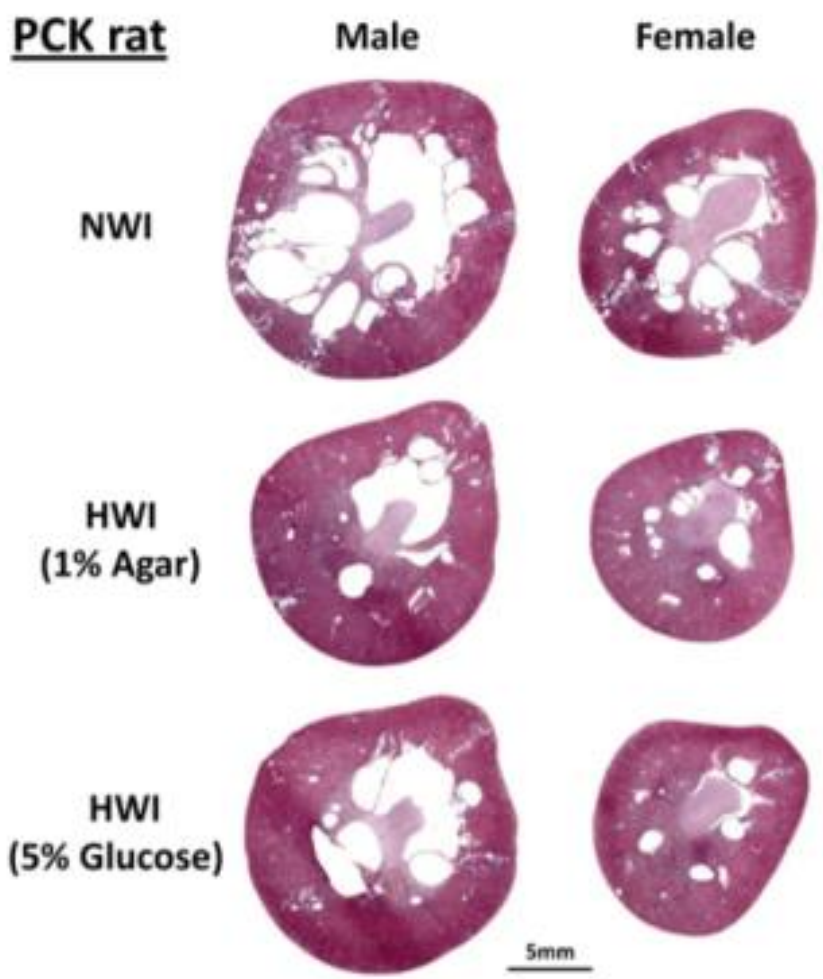
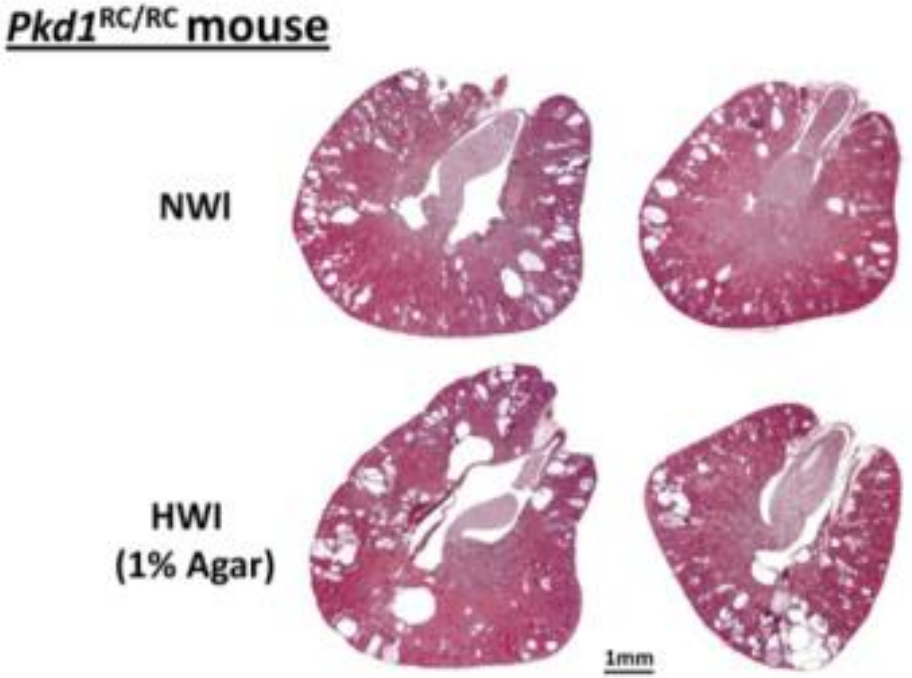




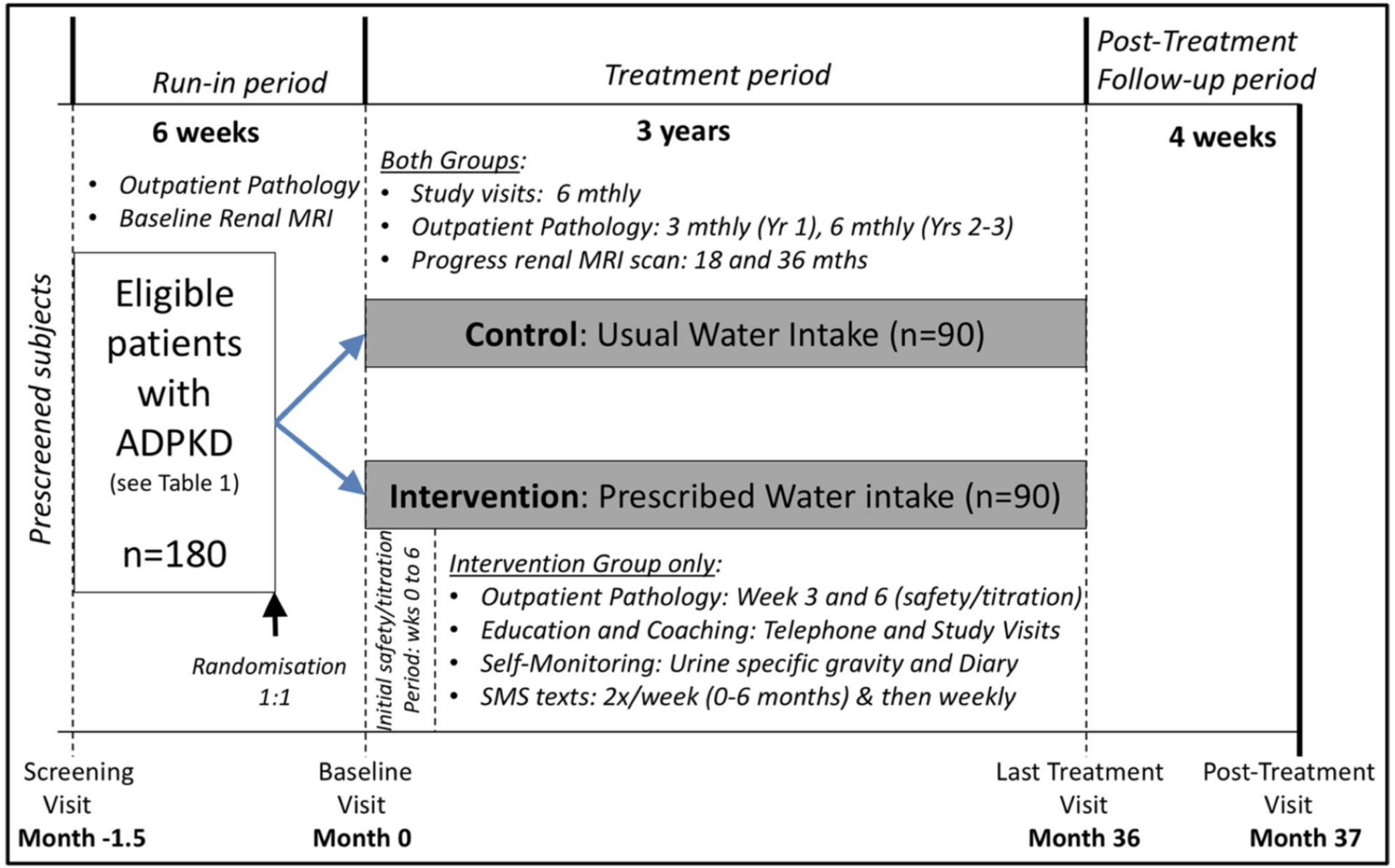
Rat: YES! Mouse: small effect

Hopp K, et al AM J Physiol Renal Physiol 308: F261-F266, 2015

Randomised controlled trial to determine the efficacy and safety of prescribed water intake to prevent kidney failure due to autosomal dominant polycystic kidney disease (PREVENT-ADPKD)

In humans?

Wong et al. BMJ Open 2018 Jan 21;8(1):e018794. doi: 10.1136/bmjopen-2017-018794.



Wong et al. BMJ Open 2018 Jan 21;8(1):e018794. doi: 10.1136/bmjopen-2017-018794.

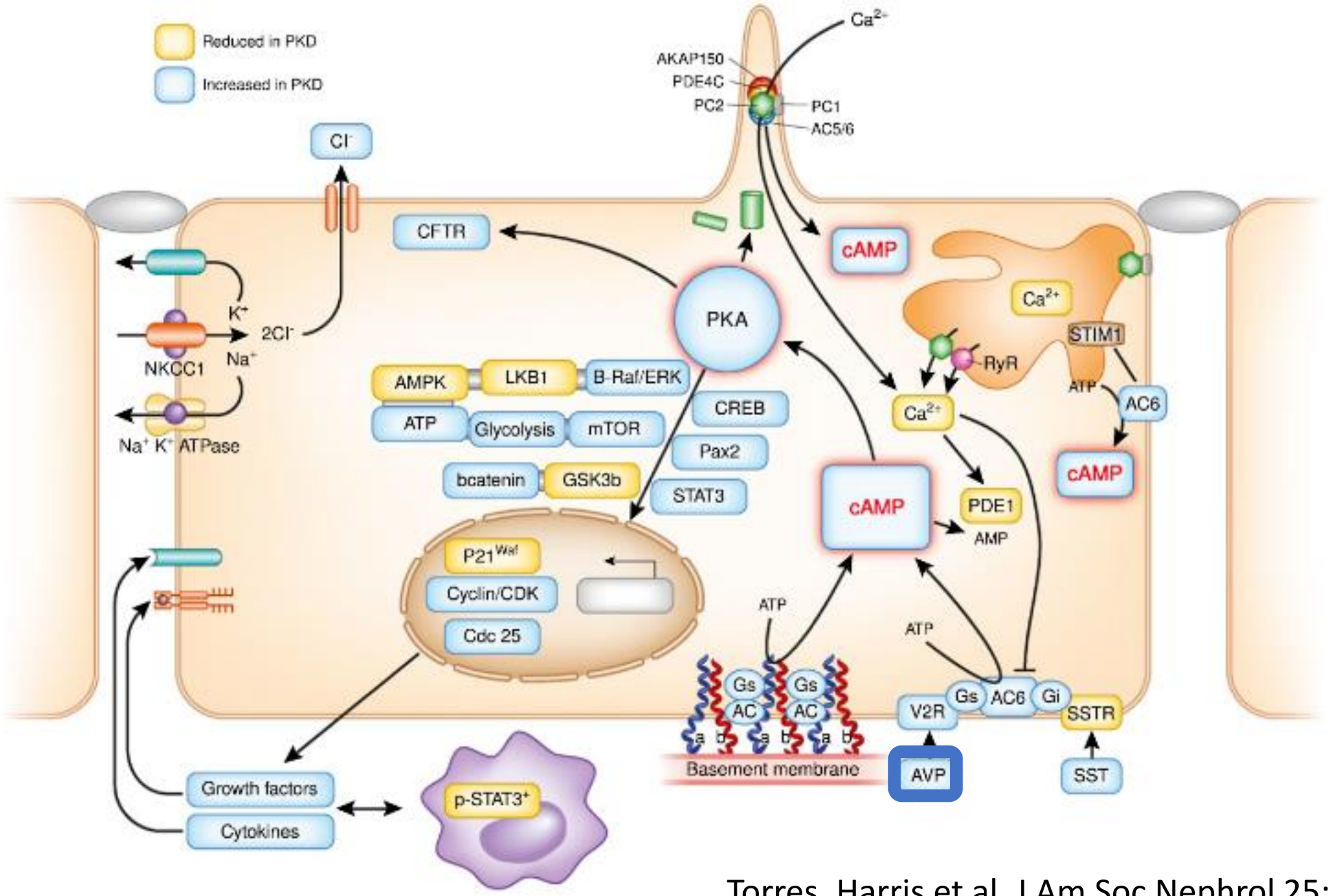
Till second decade of 21st century (and still): treatments focus on symptoms & complications

Standard care	Lifestyle approaches (general – like in many other diseases)
Blood pressure control	**Maintenance of healthy BW**
Pain control	Frequent H_2O intake
Antibiotics for UTIs	Avoidance of caffeine and smoking
Antidepressants	**Salt restriction (sodium chlorid**
Dialysis	**Low protein intake**
Renal transplantation	Bed rest
	Regular exercise

Disease modifying?

BW, body weight; UTIs, urinary tract infections

Halvorson CR et al. Int J Nephrol Renovasc Dis 3: 69-83, 2010



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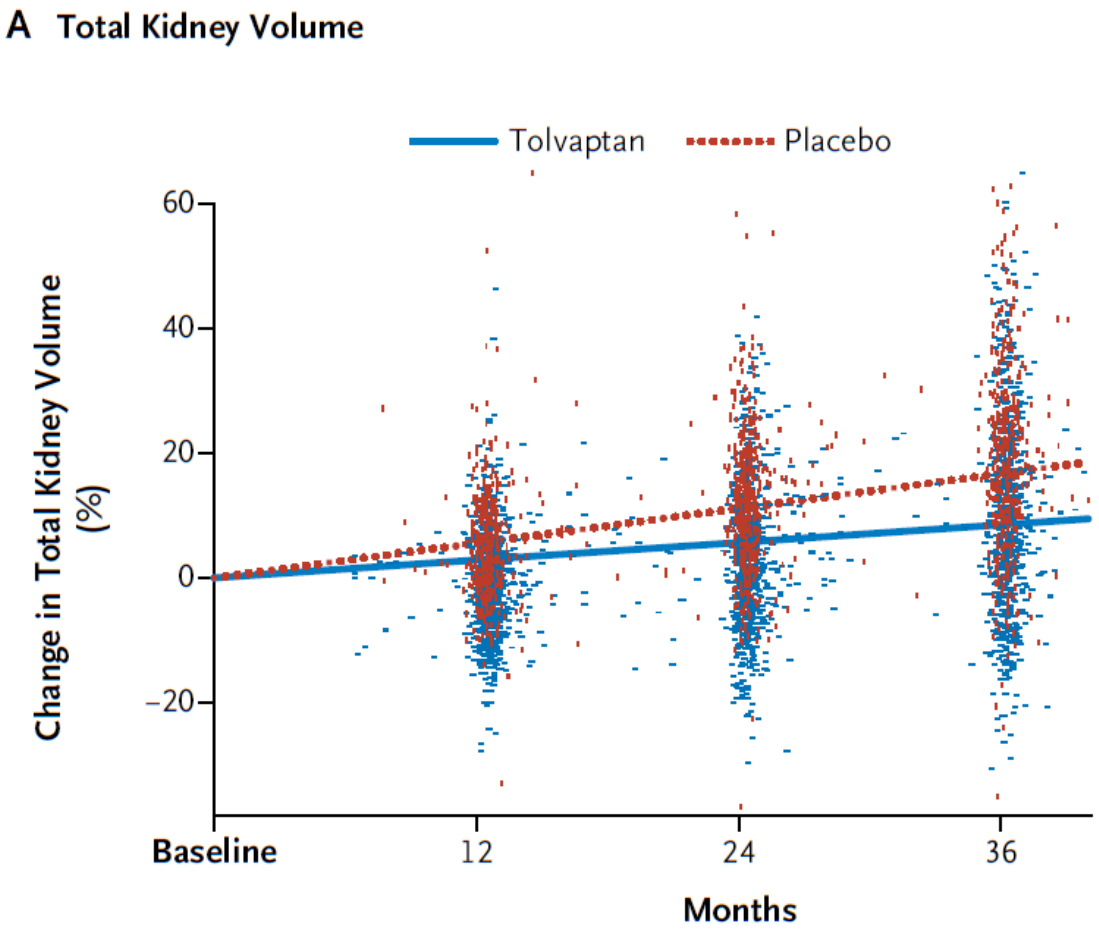
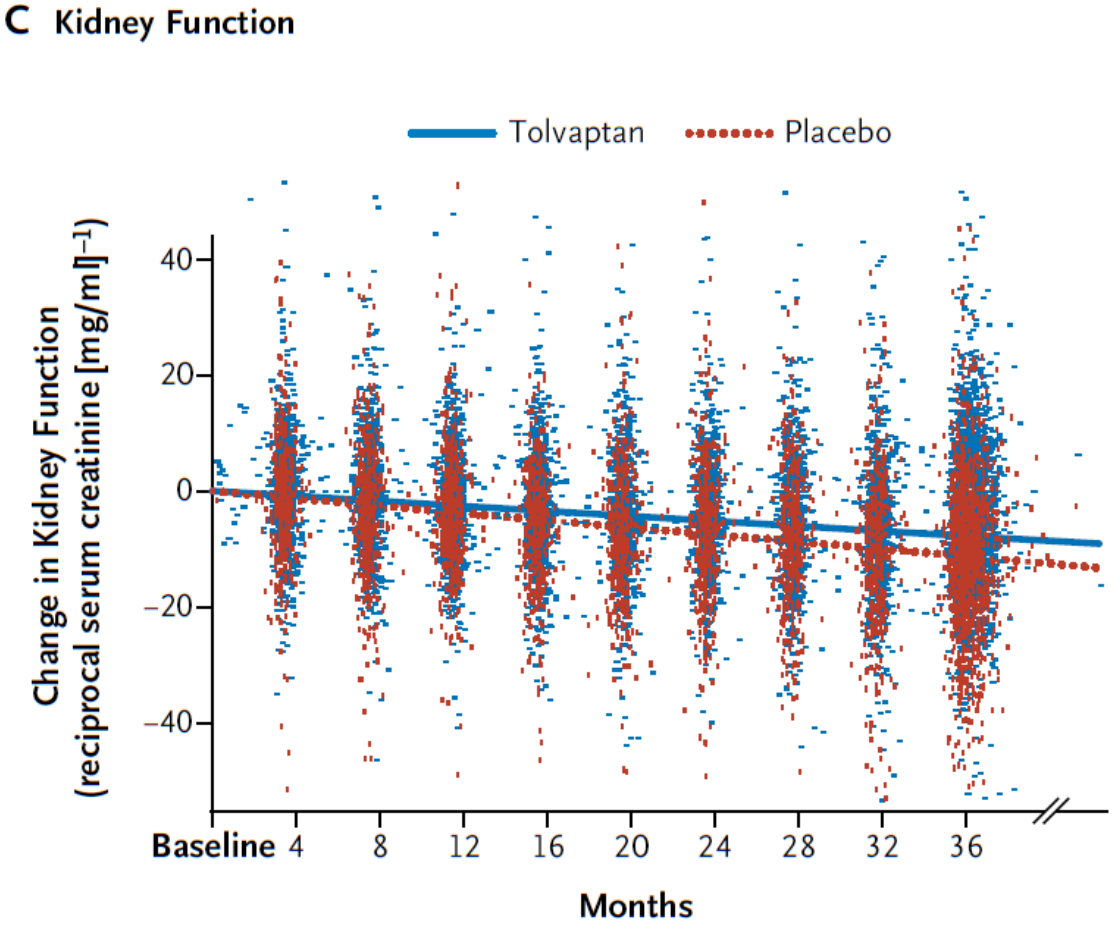
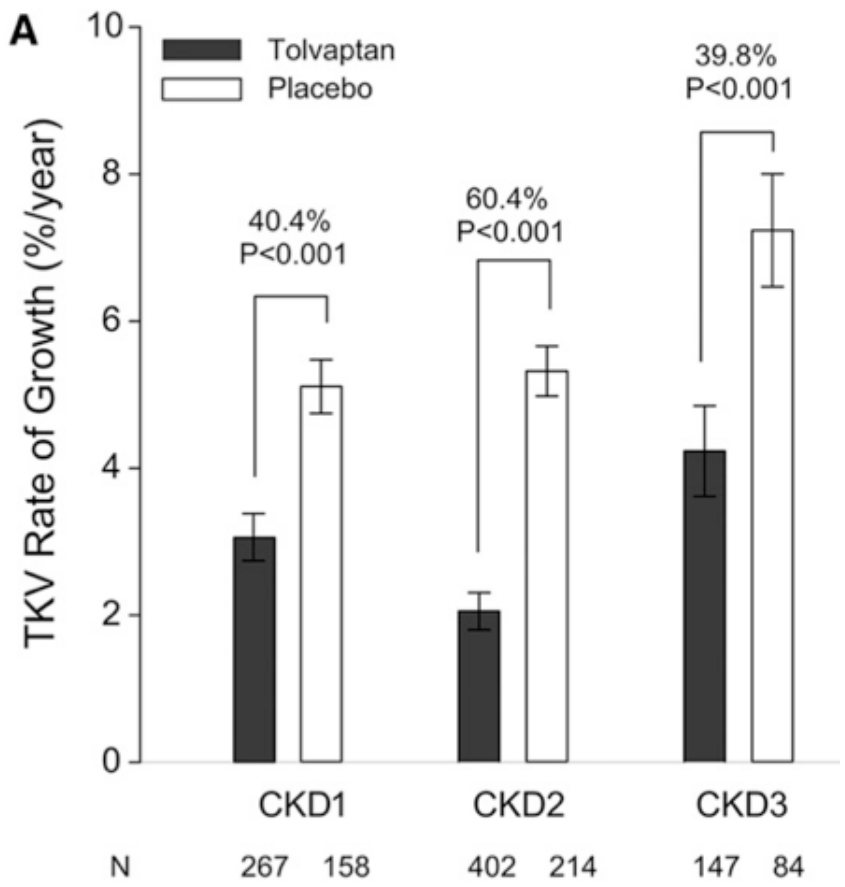
Tolvaptan in Patients with Autosomal Dominant Polycystic Kidney Disease

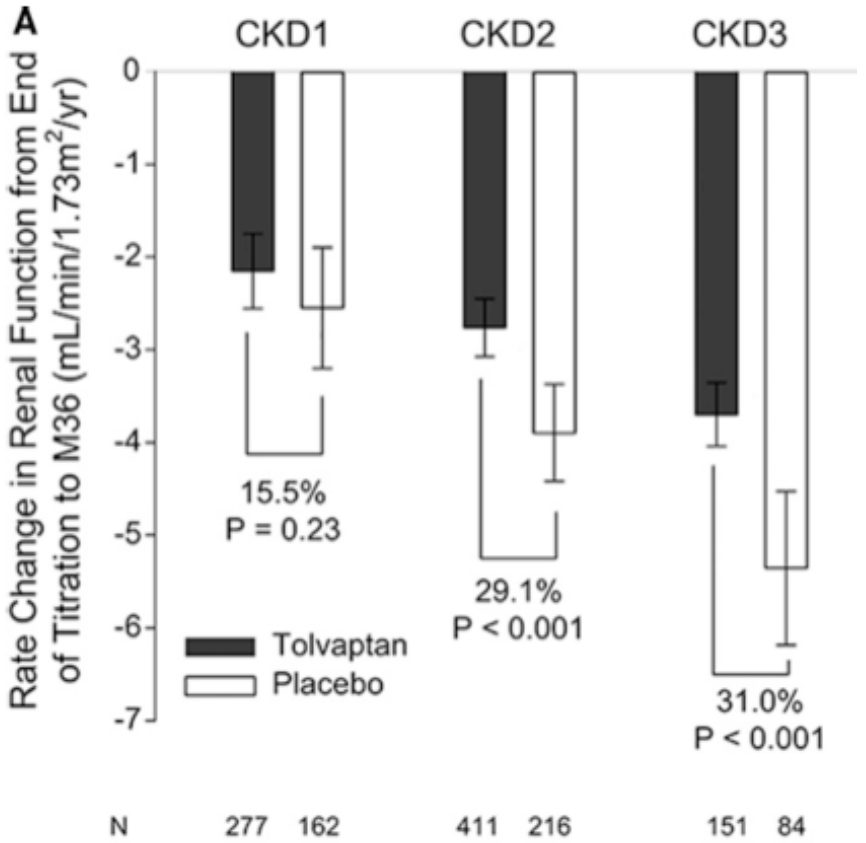




Torres et al. N Engl J Med 367: 2407-2418, 2012

Effect of Tolvaptan in Autosomal Dominant Polycystic Kidney Disease by CKD Stage: Results from the TEMPO 3:4 Trial

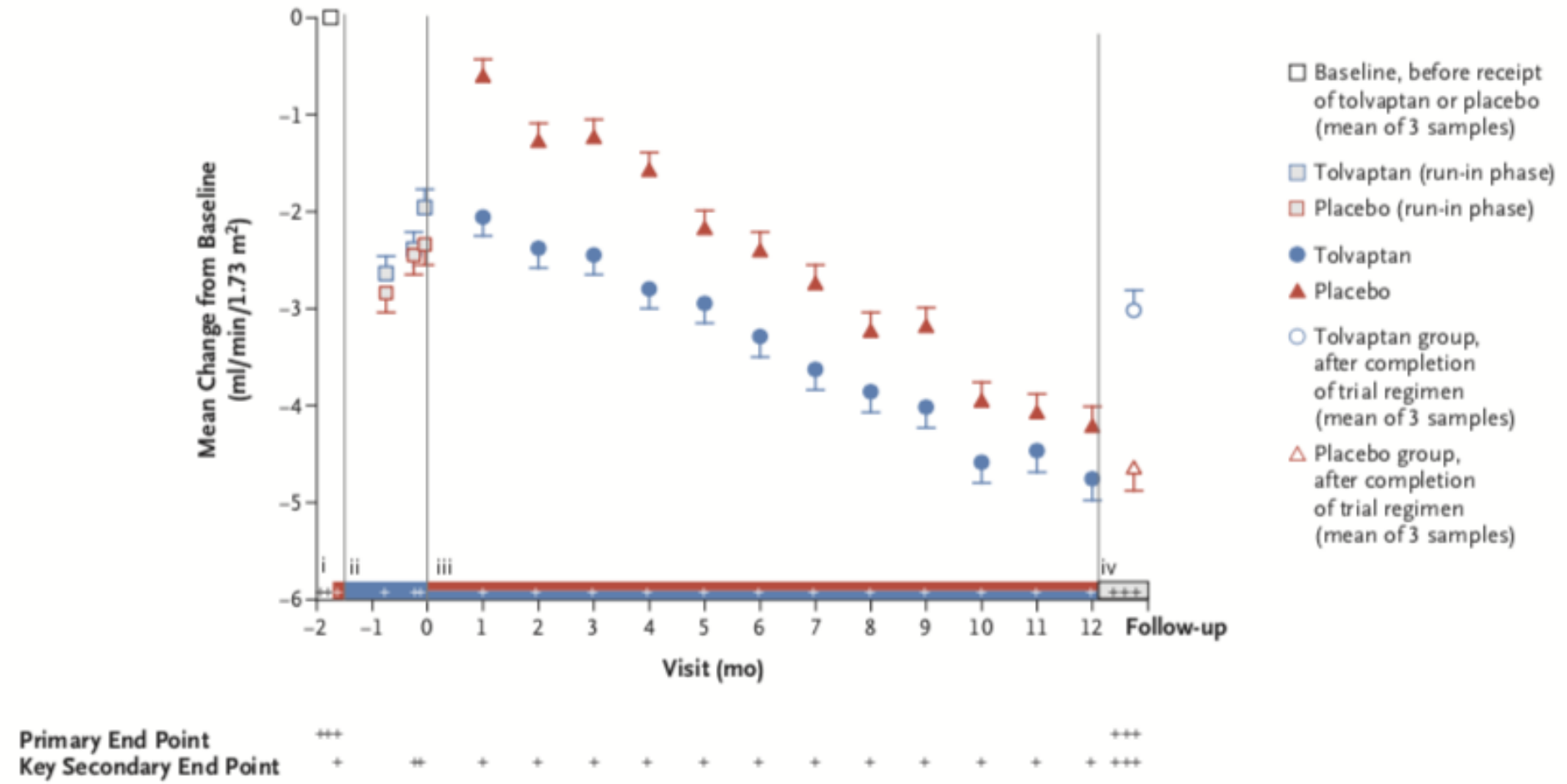




Torres et al. Clin J Am Soc Nephrol 11: 803-811, 2016

Tolvaptan in Later-Stage Autosomal Dominant Polycystic Kidney Disease

B Change in Estimated GFR over Course of the Trial



Torres et al. N Engl J Med 377: 1930-1942, 2017

thirst, polyuria

cost

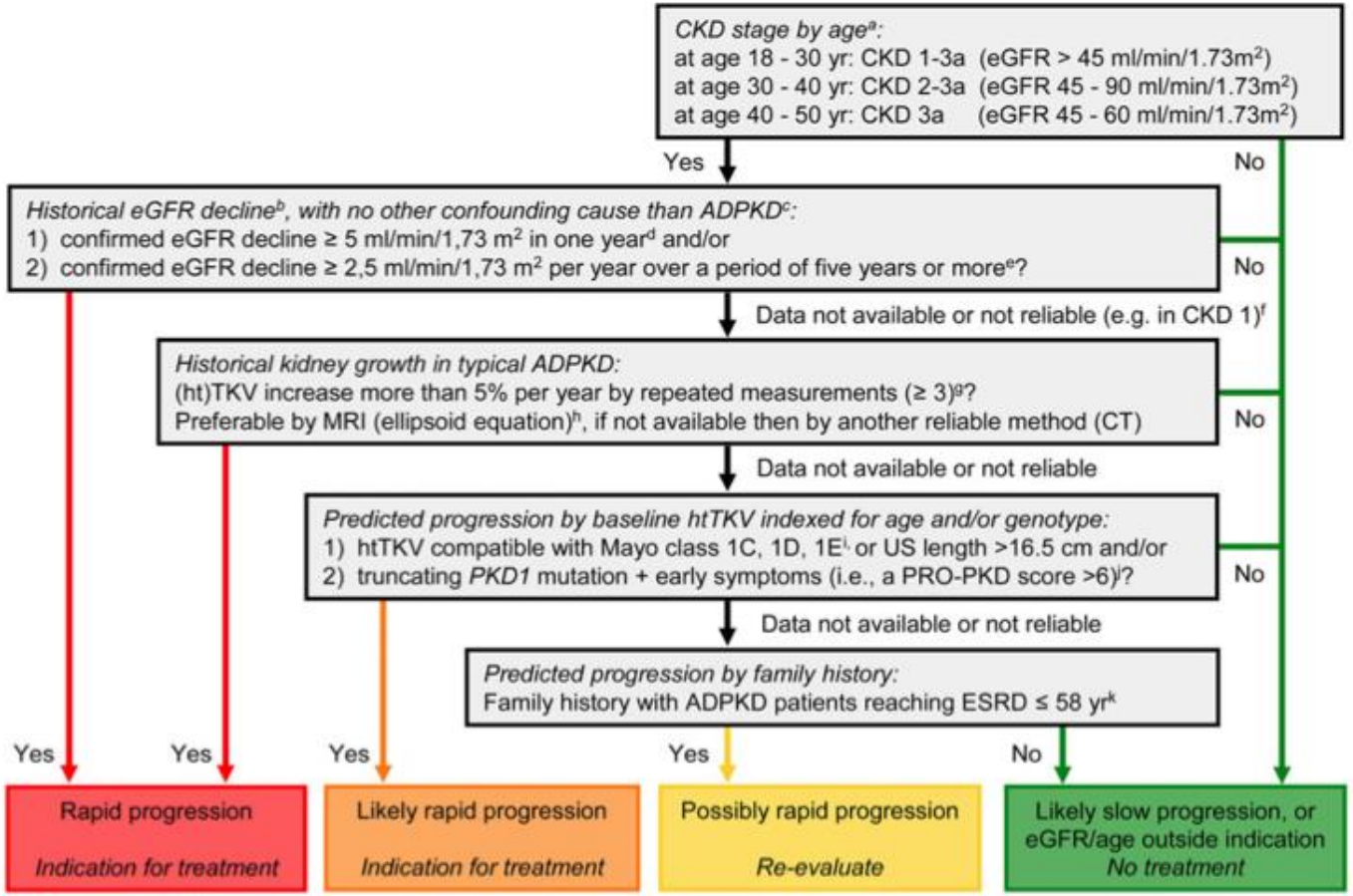
Event	Tolvaptan (N=961)	Placebo (N=483)
	no. of patients with event (%)	
Adverse events more common in tolvaptan group		
Thirst	531 (55.3)†	99 (20.5)
Polyuria	368 (38.3)†	83 (17.2)
Nocturia	280 (29.1)†	63 (13.0)
Headache	240 (25.0)	120 (24.8)
Pollakiuria‡	223 (23.2)†	26 (5.4)
Dry mouth	154 (16.0)	59 (12.2)
Diarrhea	128 (13.3)	53 (11.0)
Fatigue	131 (13.6)	47 (9.7)
Dizziness	109 (11.3)	42 (8.7)
Polydipsia	100 (10.4)†	17 (3.5)

Serious adverse events more common in tolvaptan group		
Alanine aminotransferase elevation	9 (0.9)	2 (0.4)
Aspartate aminotransferase elevation	9 (0.9)	2 (0.4)
Chest pain	8 (0.8)	2 (0.4)
Headache	5 (0.5)	0

liver dysfunction

Torres et al. N Engl J Med 367: 2407-2418, 2012

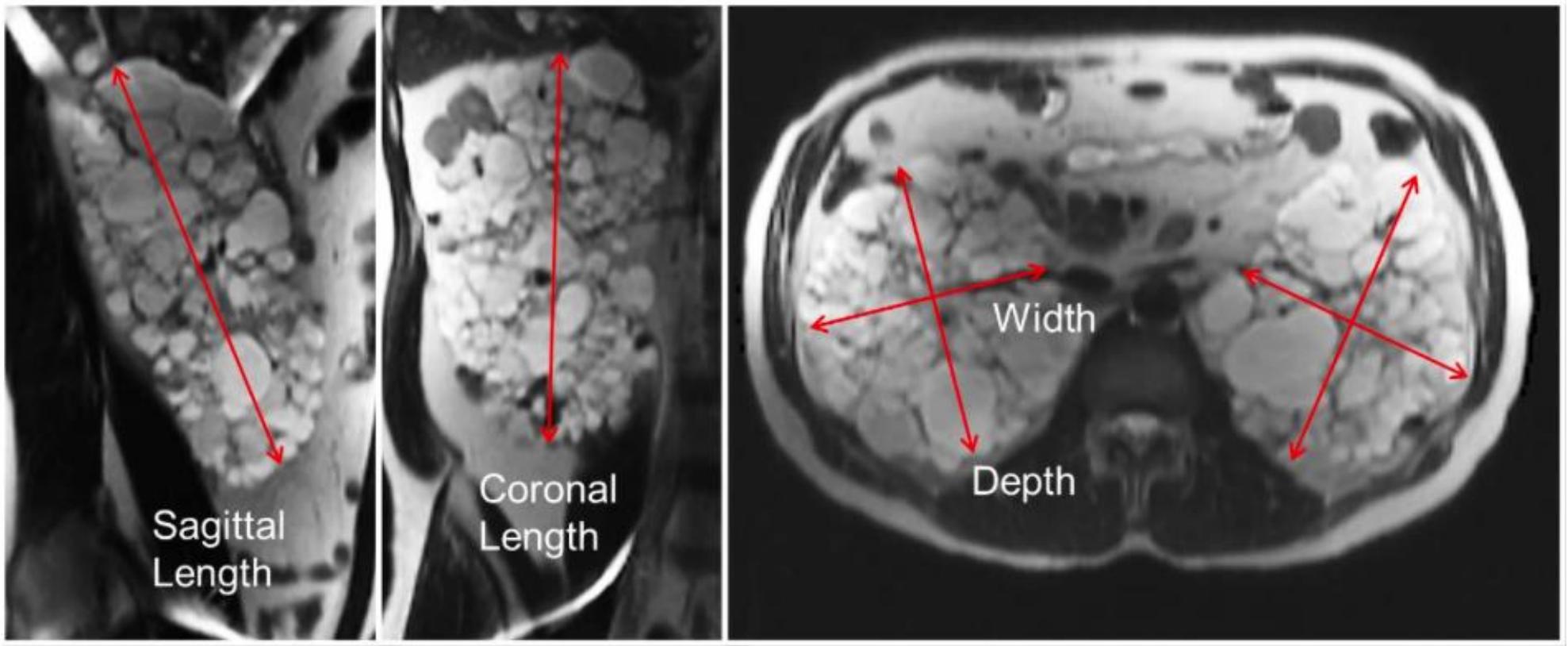
Recommendations for the use of tolvaptan in autosomal dominant polycystic kidney disease: a position statement on behalf of the ERA-EDTA Working Groups on Inherited Kidney Disorders and European Renal Best Practice



Gansevoort et al. Nephrol Dial Transplant 31: 337-348, 2016

Patient selection for treatment

(height-adjusted) Total Kidney Volume (TKV)



Irazabal et al. J Am Soc Nephrol 26: 160-172, 2015

Patient selection for treatment

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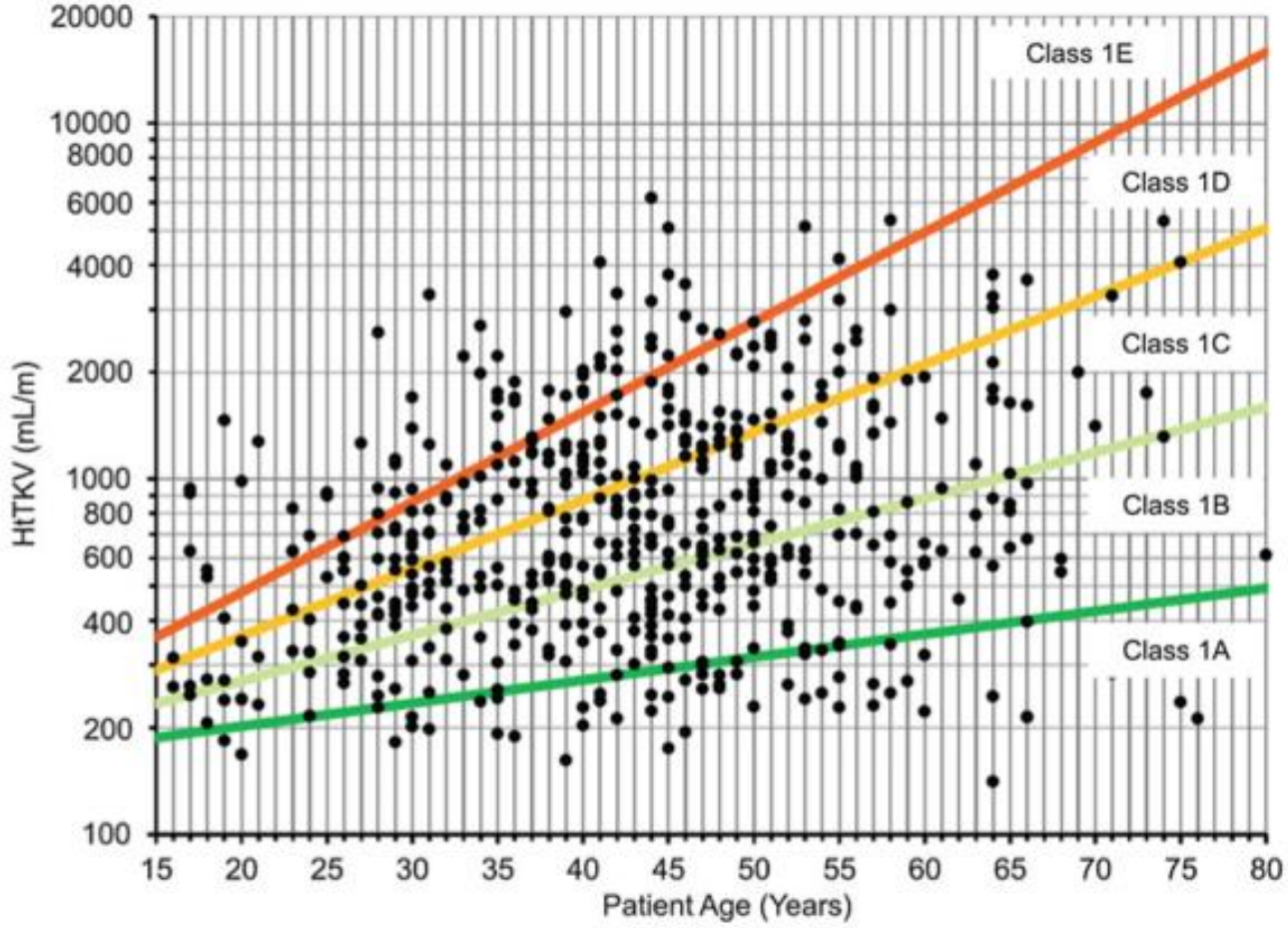


Irazabal et al. J Am Soc Nephrol 26: 160-172, 2015

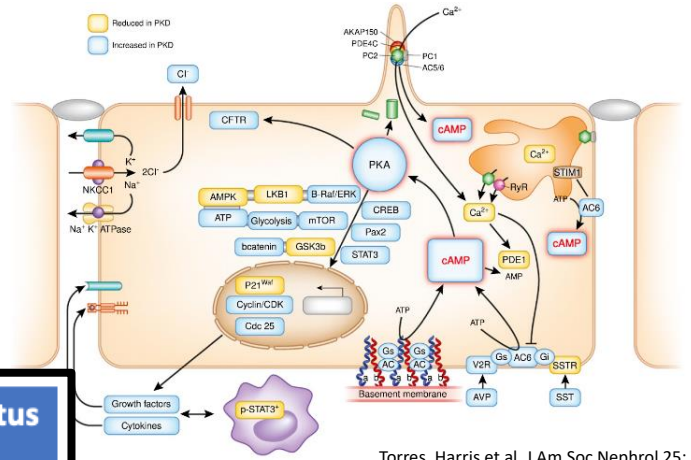
Till second decade of 21st century (and still): treatments focus on symptoms & complications

From second half of second decade of 21st century:

Disease modifying treatments

are slowly becoming available!

Disease modifying treatments



Torres, Harris et al. J Am Soc Nephrol 25: 18-32, 2014
Harris, Torres et al. J Clin Invest 124: 2315-2324, 2014

Trial name	Investigational product	Phase Design	Duration	Inclusion criteria	End-points	Current status
STAGED-PKD Stage 1 (Sanofi)	Venglustat (glucocerebrosidase inhibitor)	Phase 2/3 RCT, placebo-controlled 2 doses of IP	24 months	18-50y ADPKD Mayo 1C, D or E eGFR 45-90	1° TKV 2° eGFR safety	recruitment target reached
STAGED-PKD Stage 2 (Sanofi)	Venglustat (glucocerebrosidase inhibitor)	Phase 2/3 RCT, placebo-controlled 1 dose of IP	24 months	18-50y ADPKD Mayo 1C, D or E eGFR 45-90	1° eGFR 2° TKV safety	recruiting
FALCON (Reata)	Bardoxolone methyl (Nrf2 activator, anti-inflammatory, anti-oxidative)	Phase 3 RCT, placebo-controlled 1 dose of IP	24 months	18-70y ADPKD eGFR 30-90 (for 18-55y) eGFR 30-44 (for 56-70y) "progressive" eGFR (for eGFR 60-90 and 56-70y)	1° eGFR 52w 2° eGFR 104w	recruiting
GLPG2737 (Galapagos)	GLPG2737 (CFTR inhibitor)	Phase 2a RCT, placebo-controlled 1 dose of IP	24 weeks	18-50y ADPKD TKV > 750mL Mayo 1C, D or E eGFR 30-90 (for 18-40y) eGFR 30-60 (for 40-50y)	1° TKV safety 2° eGFR PK	initiating

Till second decade of 21st century (and still): treatments focus on symptoms & complications

Standard care	Lifestyle approaches (general – like in many other diseases)
Blood pressure control	**Maintenance of healthy BW**
Pain control	Frequent H_2O intake
Antibiotics for UTIs	Avoidance of caffeine and smoking
Antidepressants	**Salt restriction (sodium chlorid**
Dialysis	**Low protein intake**
Renal transplantation	Bed rest
	Regular exercise

Disease modifying?

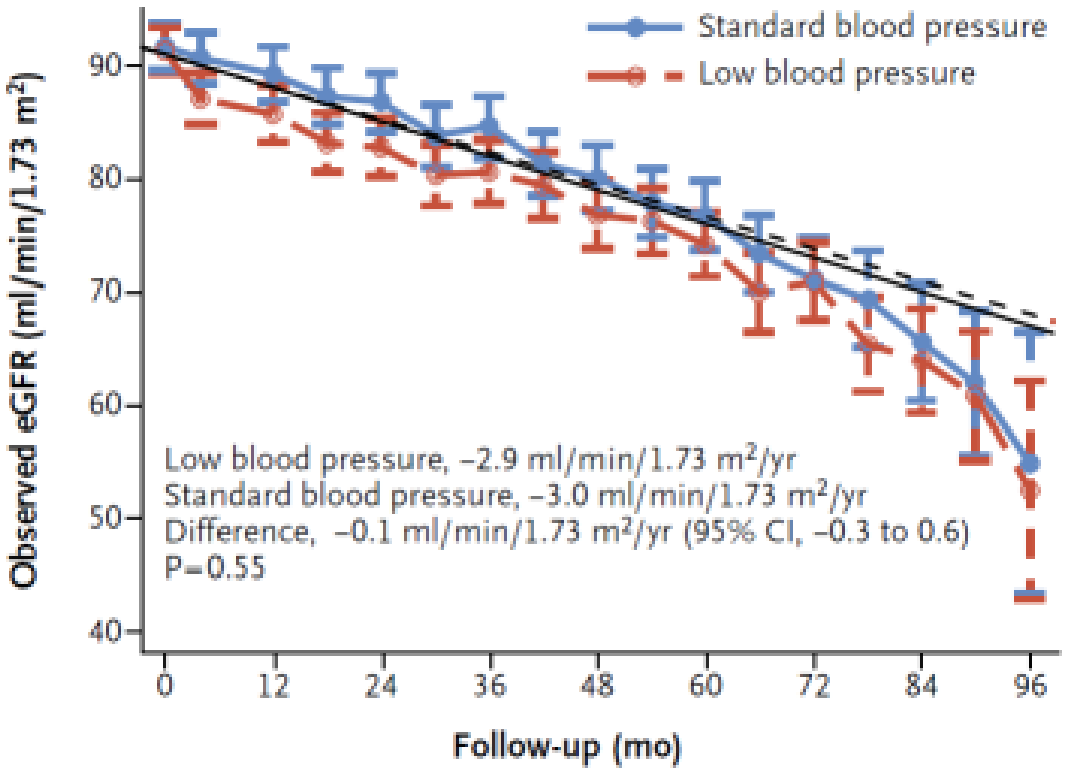
BW, body weight; UTIs, urinary tract infections

Halvorson CR et al. Int J Nephrol Renovasc Dis 3: 69-83, 2010

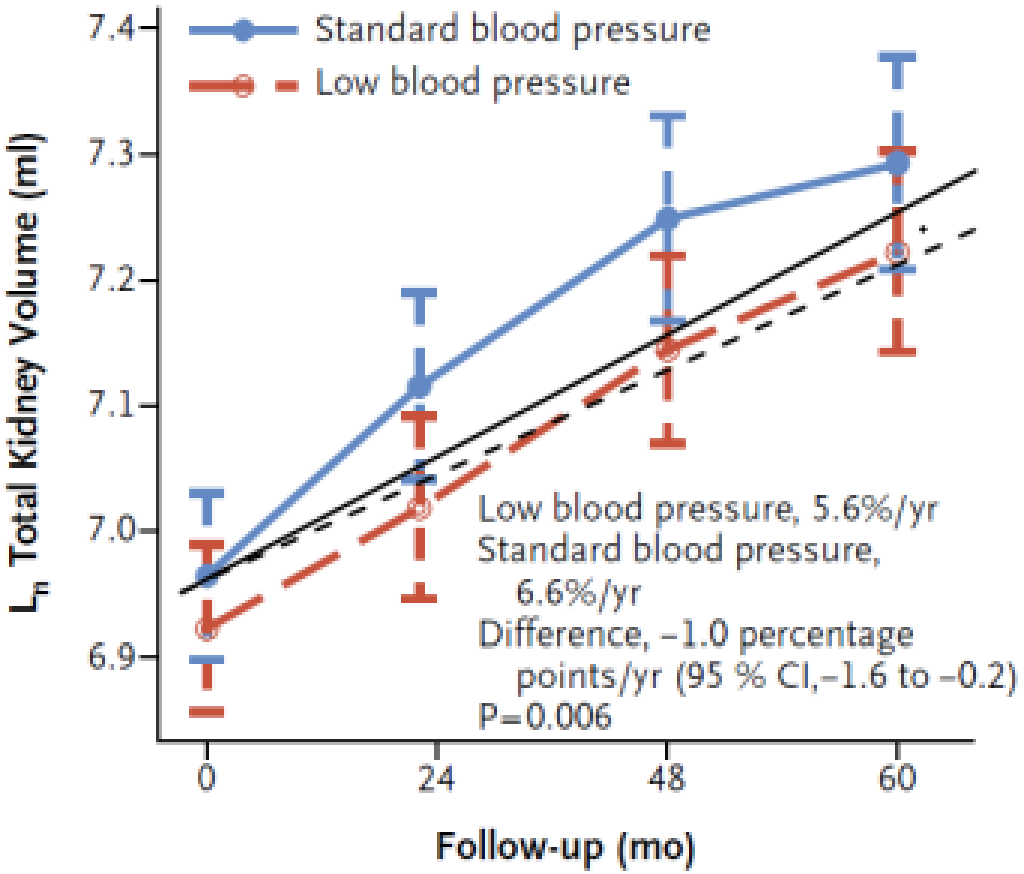
Blood Pressure in Early Autosomal Dominant Polycystic Kidney Disease

HALT-PKD study

Changes in eGFR over Time



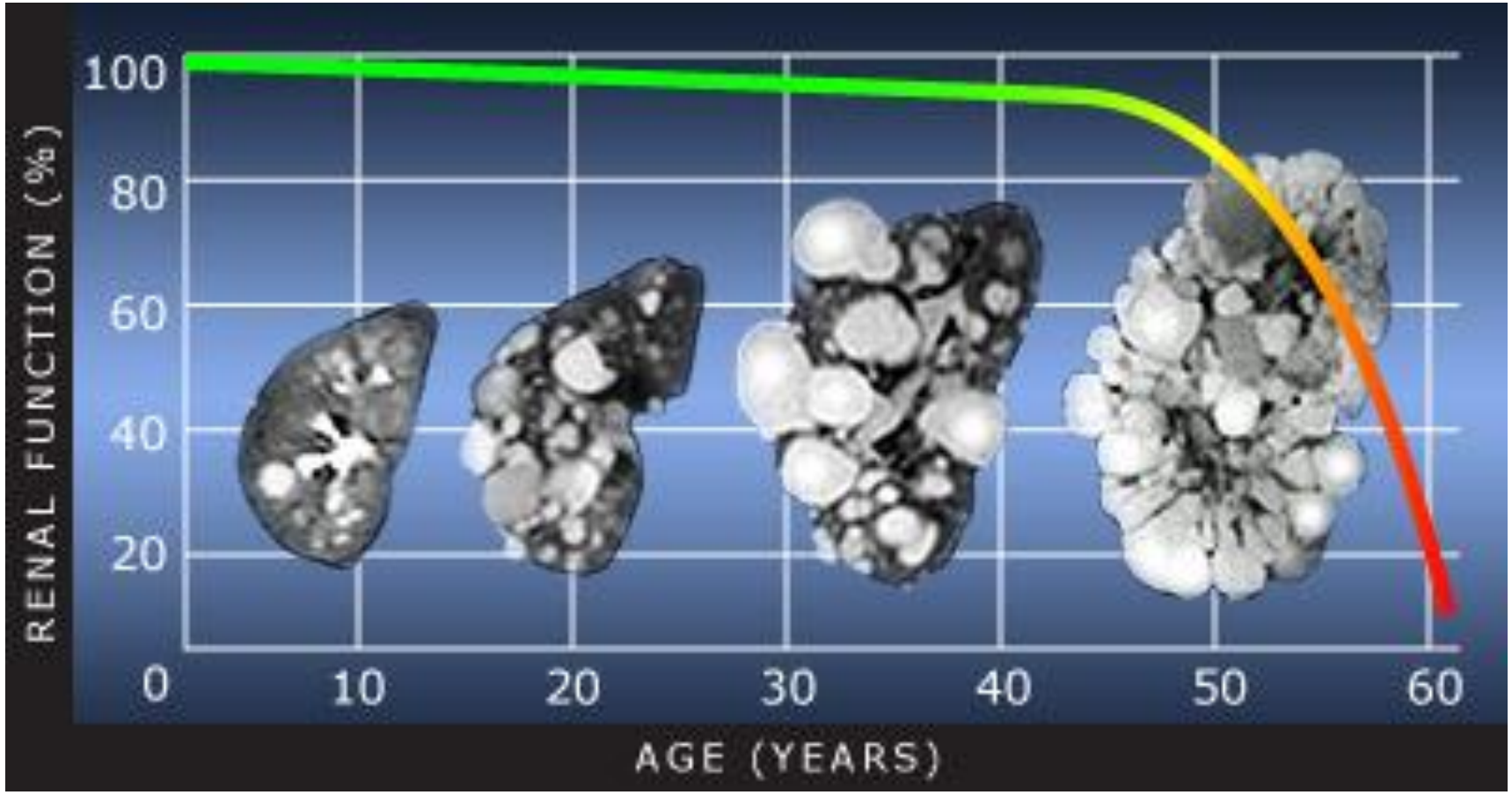
Changes in Total Kidney Volume over Time



Schrier et al. N Engl J Med 371: 2255-2266, 2014

Autosomal Dominant Polycystic Kidney Disease

1/400 tot 1/1000 live births – progressive cystic deformation and growth of kidneys

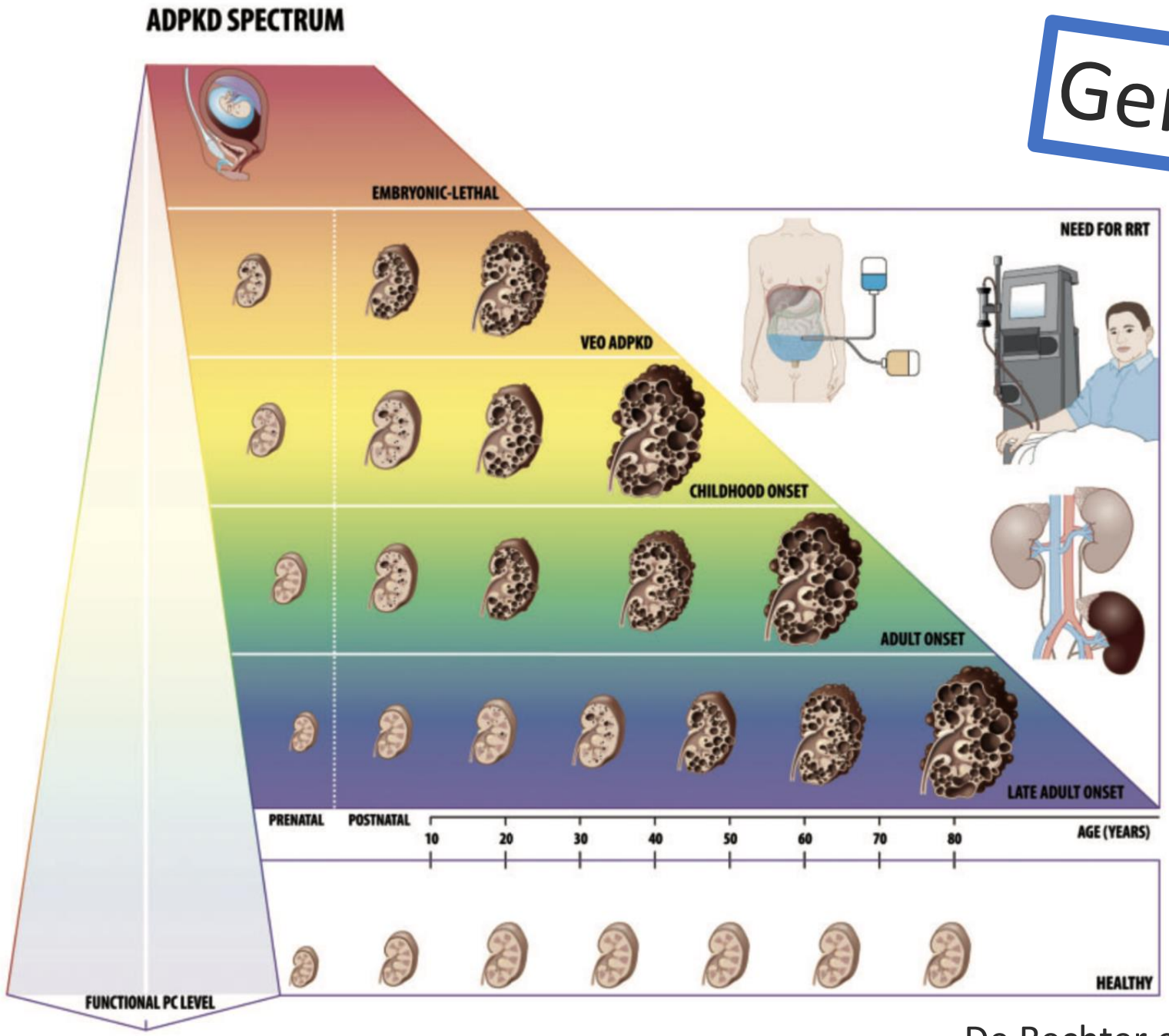


CKD stage 5D (median age): 58y PKD1, 79y PKD2 - 5-7% of ESRD incidence in Belgium

Unmet needs and challenges for follow-up and treatment of autosomal dominant polycystic kidney disease: the paediatric perspective

Adult onset ADPKD is part of a spectrum of the disease,
with symptomatic disease presentations spanning over the entire age range.

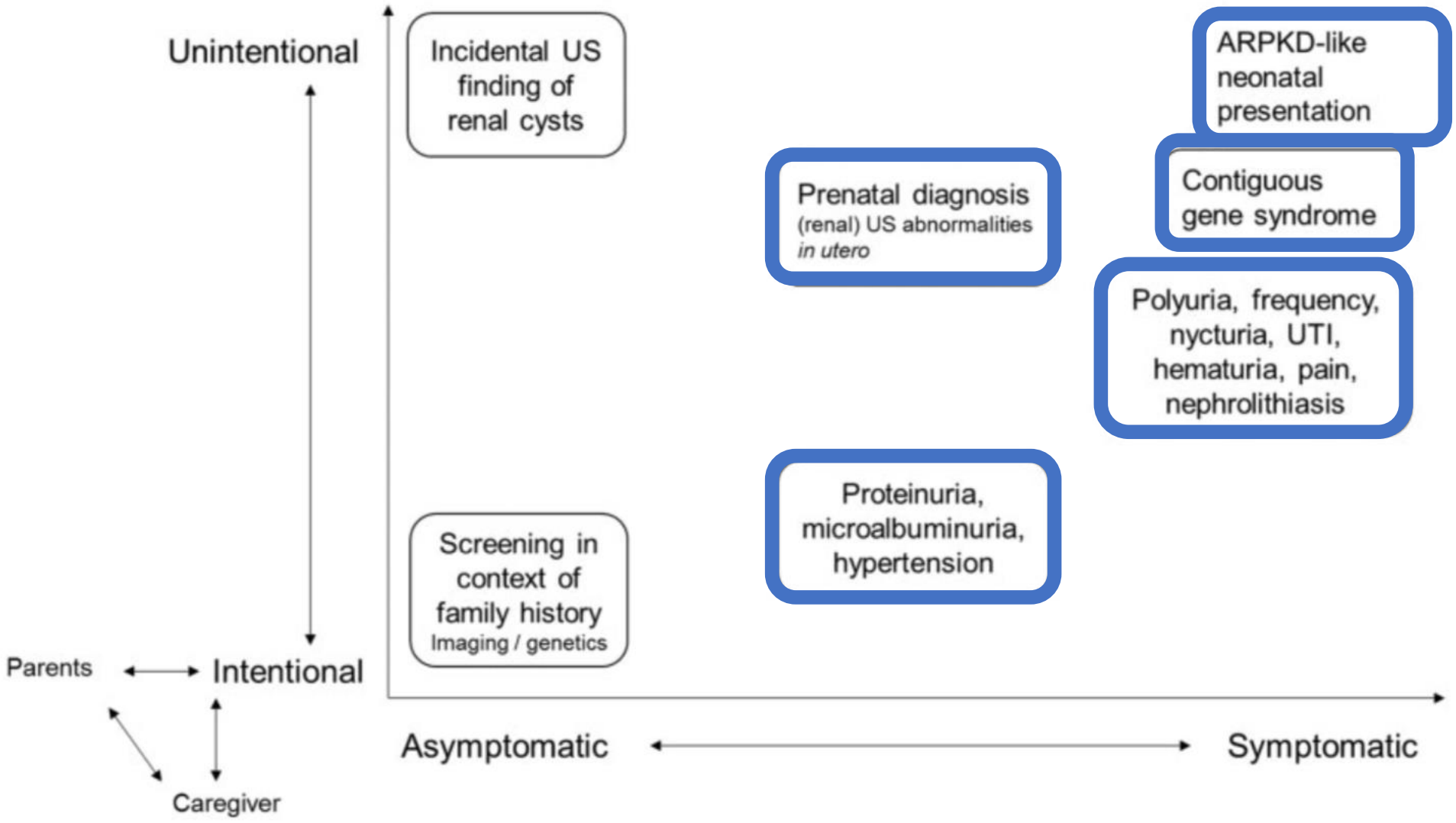
De Rechter et al. Clin Kidney J 11 (Suppl 1): i14-i16, 2018



Genotype-phenotype

e.g.
biallelic mutations with hypomorphic allele
ADPKD allele + allele other cystic nephropathy

De Rechter et al. Clin Kidney J 11 (Suppl 1): i14-i16, 2018



De Rechter et al. Clin Kidney J 11 (Suppl 1): i14-i16, 2018

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At adult age, much harm has already taken place.
→ It makes sense to identify early biomarkers of disease progression in children.
→ It makes sense to identify modifiable risk factors for progression in children.
→ It makes sense to search for early treatment options with acceptable side-effect profile.

De Rechter et al. Clin Kidney J 11 (Suppl 1): i14-i16, 2018

ADPedKD: A Global Online Platform on the Management of Children With ADPKD

www.ADPedKD.org

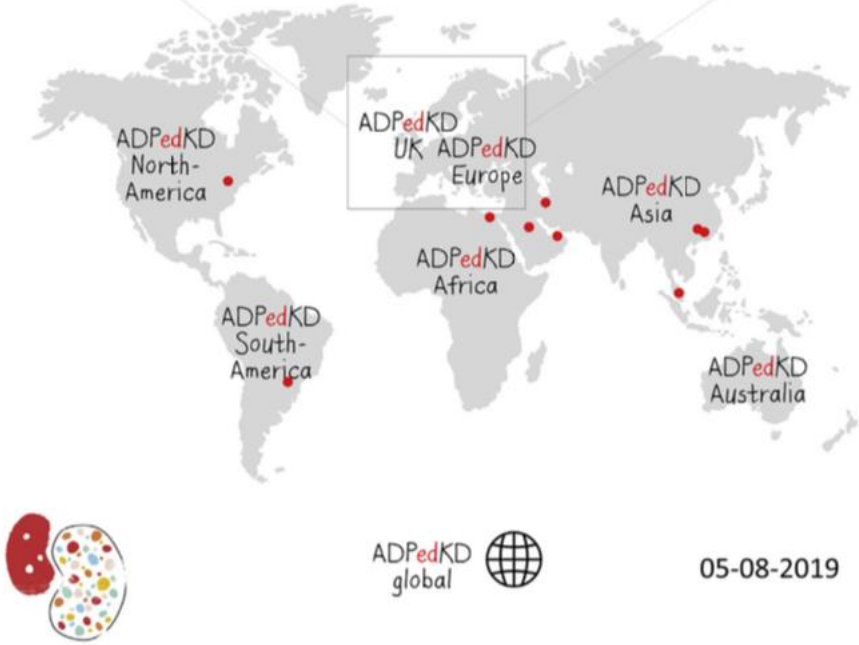
83 centers

29 countries

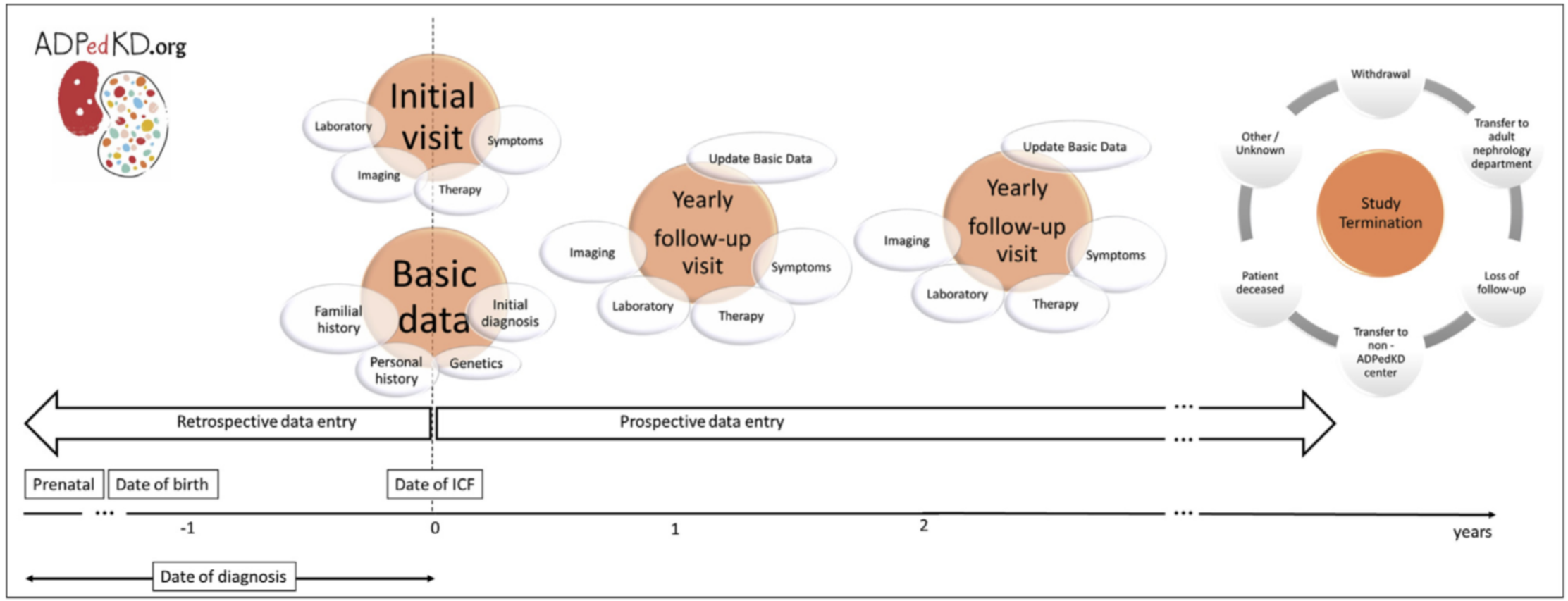
725 patients included

Djalila Mekahli

(in collaboration with Max Liebau, Franz Schaefer)



De Rechter et al. Kindey Int Rep 4: 1271-1284, 2019

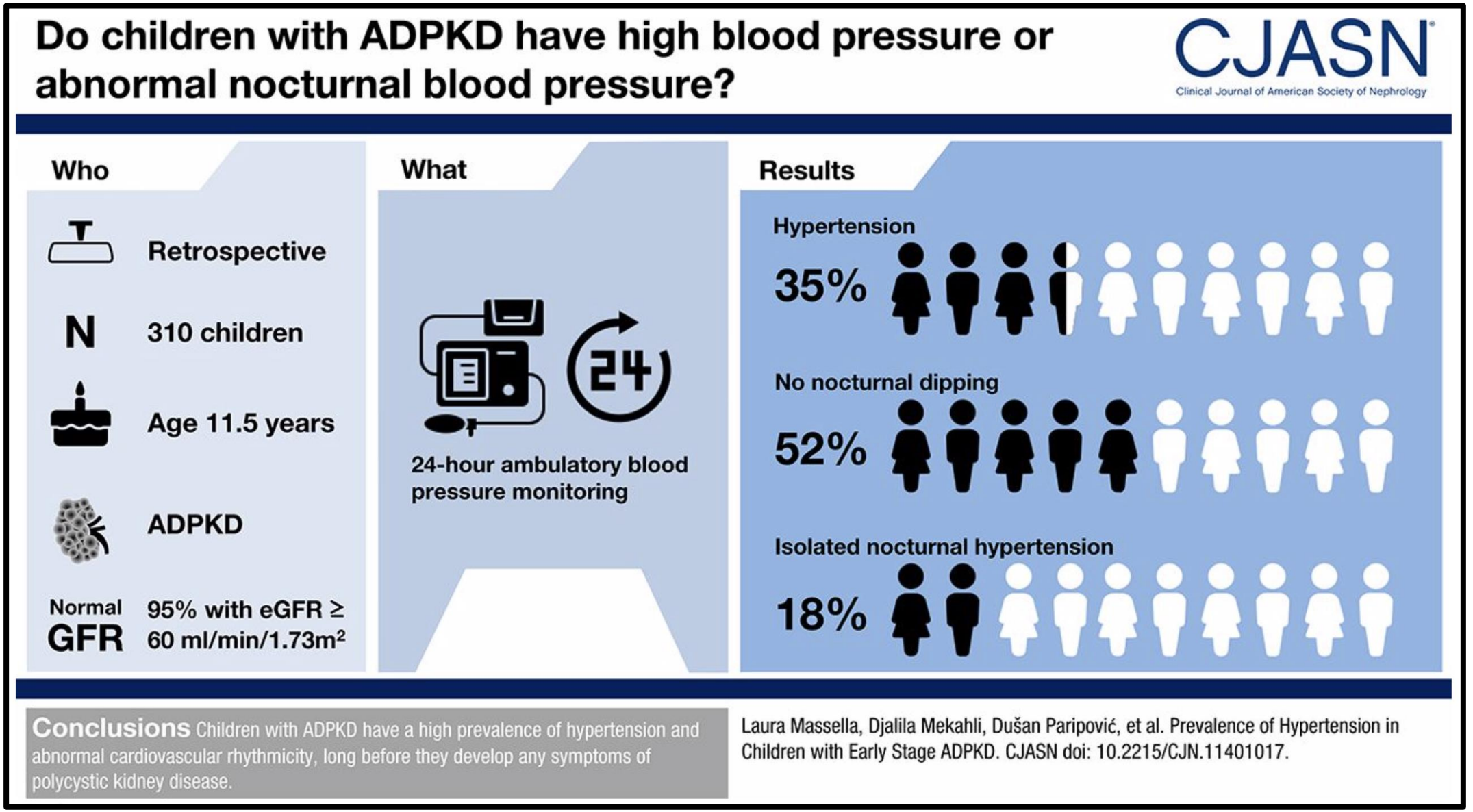


Genotype – phenotype correlation - Incidence and presentation of comorbidities - Current medical practice information - Familial recurrence risk

Recommendations regarding modifiable risk factors

Identification of clinical/biomarkers predicting early disease progression

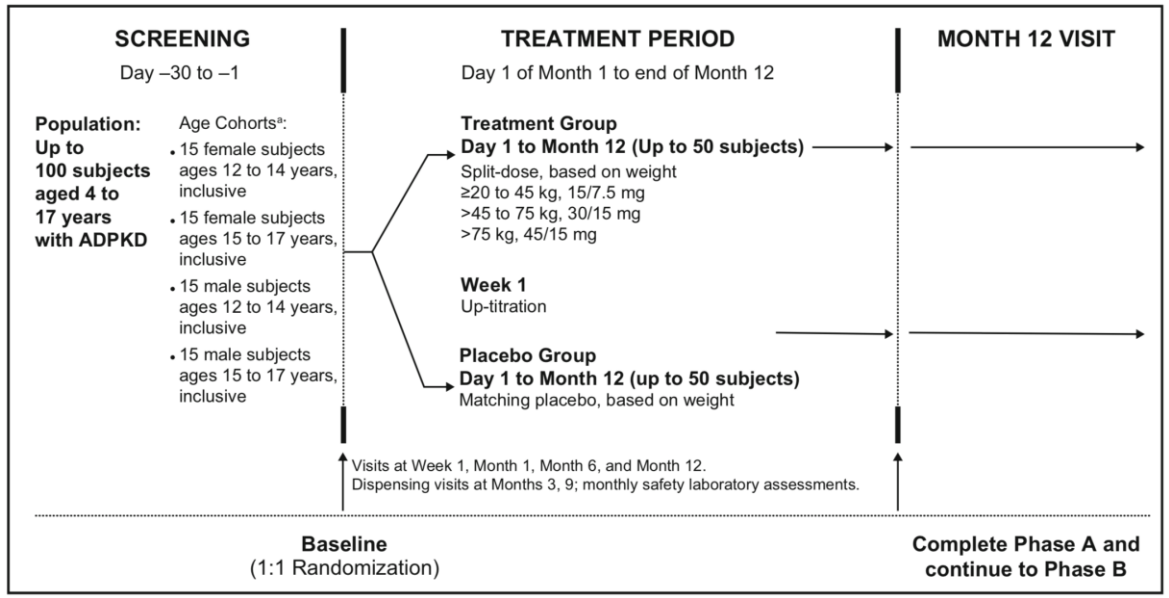
Talking about modifiable risk factors…

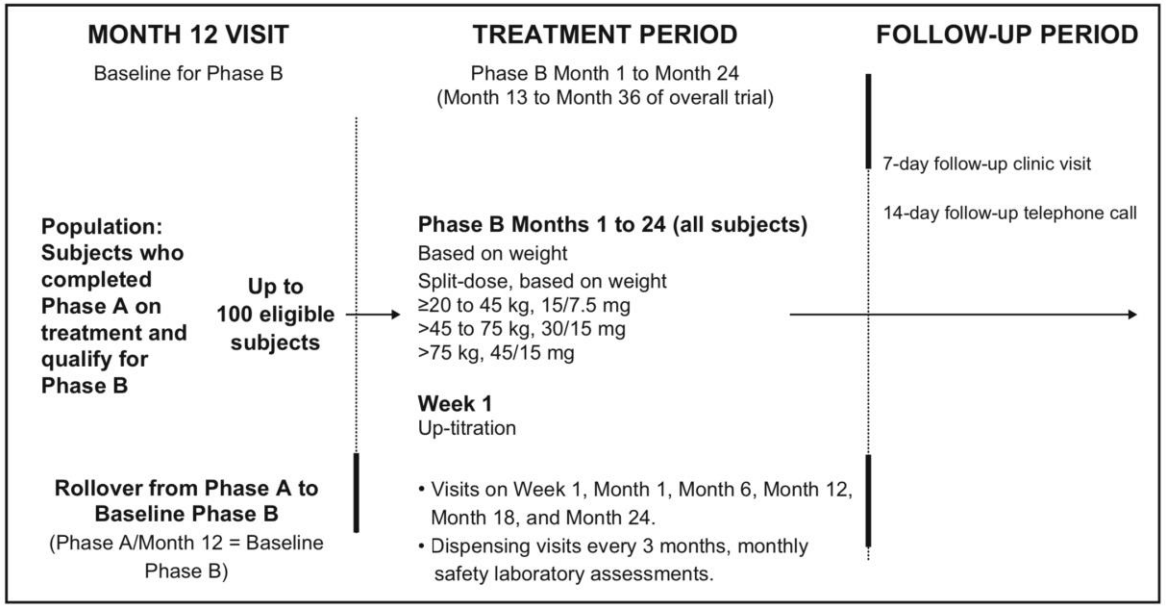


Massella et al. Clin J Am Soc Nephrol 13: 874-883, 2018

Talking about early treatment…

Tolvaptan use in children and adolescents with autosomal dominant polycystic kidney disease: rationale and design of a two-part, randomized, double-blind, placebo-controlled trial





Schaefer et al. Eur J Ped 178: 1013-1021, 2019

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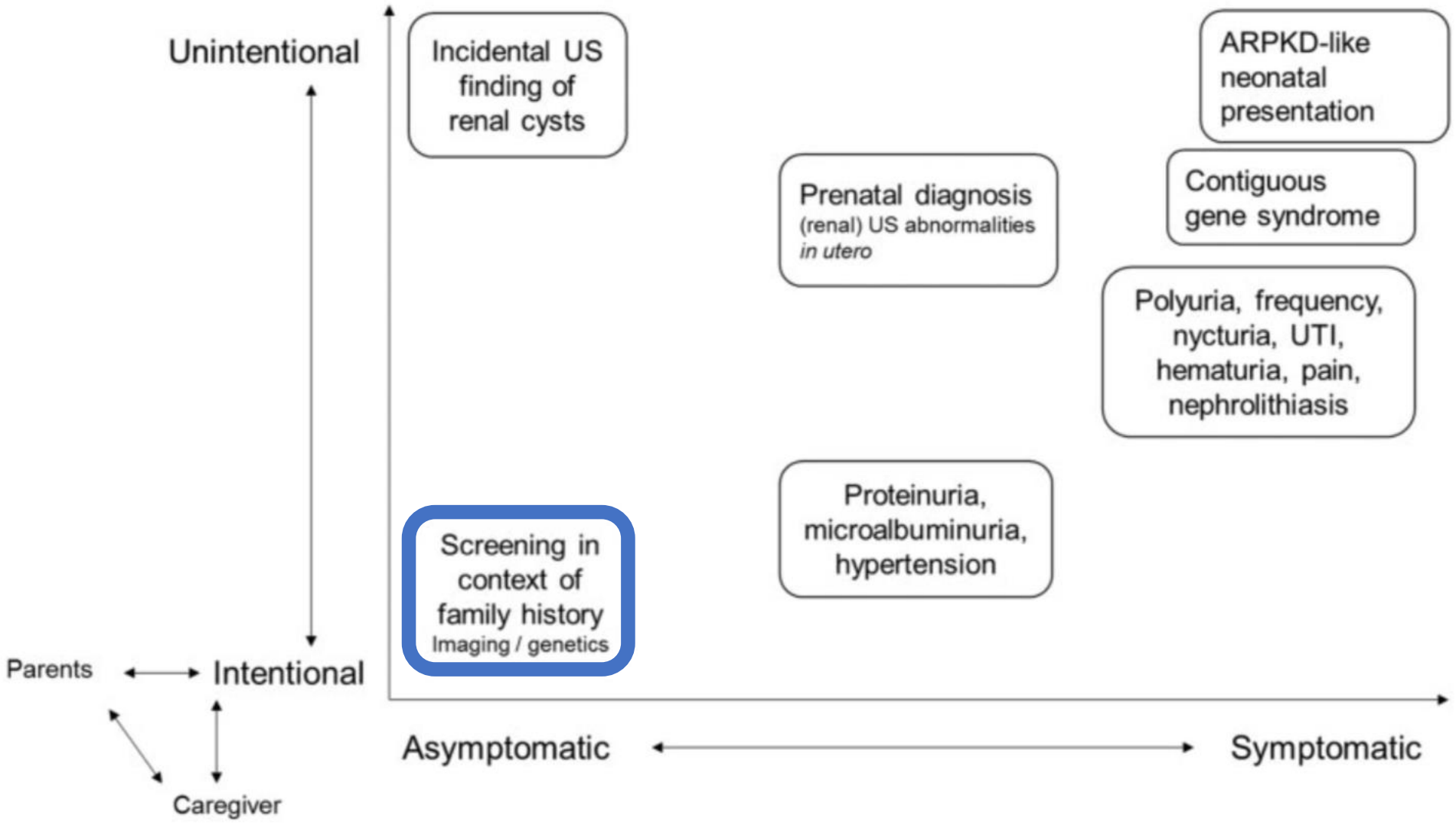
Screening of at-risk children: yes or no? Today (2020), still controversial.

De Rechter et al. Clin Kidney J 11 (Suppl 1): i14-i16, 2018



De Rechter et al. Clin Kidney J 11 (Suppl 1): i14-i16, 2018

Given the current scientific knowlegde and therapeutic armamentarium for ADPKD…

…all at-risk minors should have clinical evaluation for ADPKD.

…all at-risk minors should have genetic evaluation for ADPKD.

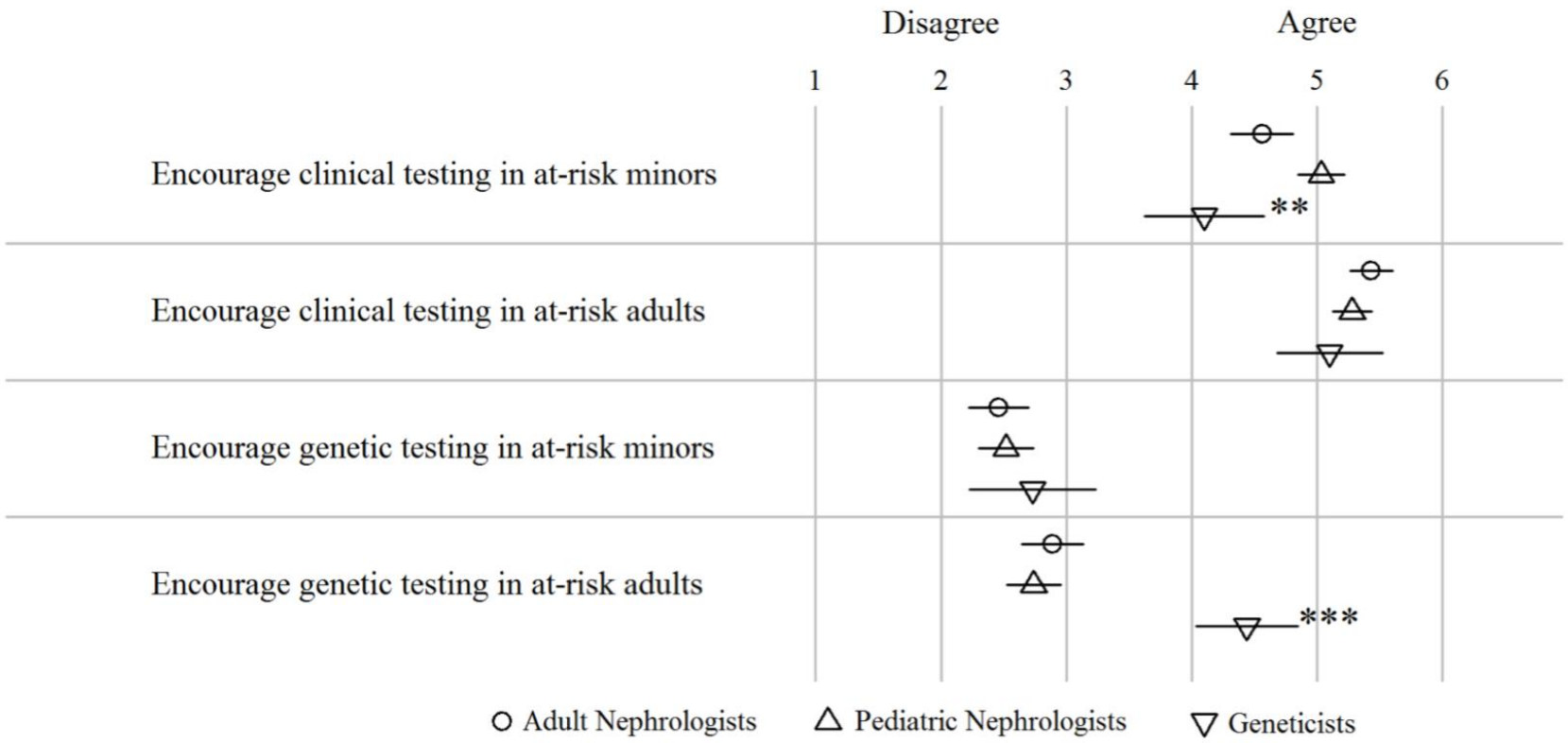
…all parents of at-risk minors should be advised to comply with general health measures for all of their children.*

…all parents of at-risk minors should be advised to comply with general health measures for all their children, with particular focus on blood pressure control.*

*Active screening only if symptomatic, when considering disease modifying treatment or for PGD. Or upon request of parents or at-risk minor after detailed counseling.

Which statement do you agree most?

Clinicians' attitude towards family planning and timing of diagnosis in autosomal dominant polycystic kidney disease



De Rechter et al. PloSONE 12(9): e0185779. https://doi.org/10.1371/journal. pone.0185779

Still a lot of work to be done!

ADPKD Clinics & Research @ UZ Leuven

Pediatric Nephrology Department UZ Leuven
PKD Group (Lab of Pediatrics)

Djalila Mekahli

ADPedKD

Lab of Ion Channel Research (KU Leuven/VIB)

VIB LICR

Rudi Vennekens

Center for Human Genetics UZ Leuven
Laboratory for Genetics of Human Development

Koen Devriendt

Adult Nephrology Department UZ Leuven
Nephrology & Renal Transplantation Research Group

Bert Bammens

Next Webinars

IPNA Clinical Practice Webinars

Date: **24 Sept 2020**

Speaker: **Francesco Emma**

Topic**: Clinical practice recommendations for the diagnosis and management of XLH**

ESPN/ERKNet Educational Webinars on Pediatric Nephrology & Rare Kidney Diseases

Date: **06 Oct 2020**

Speaker: **Olivier Devuyst**

Topic: **Autosomal dominant tubulointerstitial kidney disease**

ERKNet/ERA-EDTA Advanced Webinars on Rare Kidney Disorders

Date: **27 Oct 2020**

Speaker: **Rezan Topaloglu**

Topic: **Classification and physiopathology of vasculitis**

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