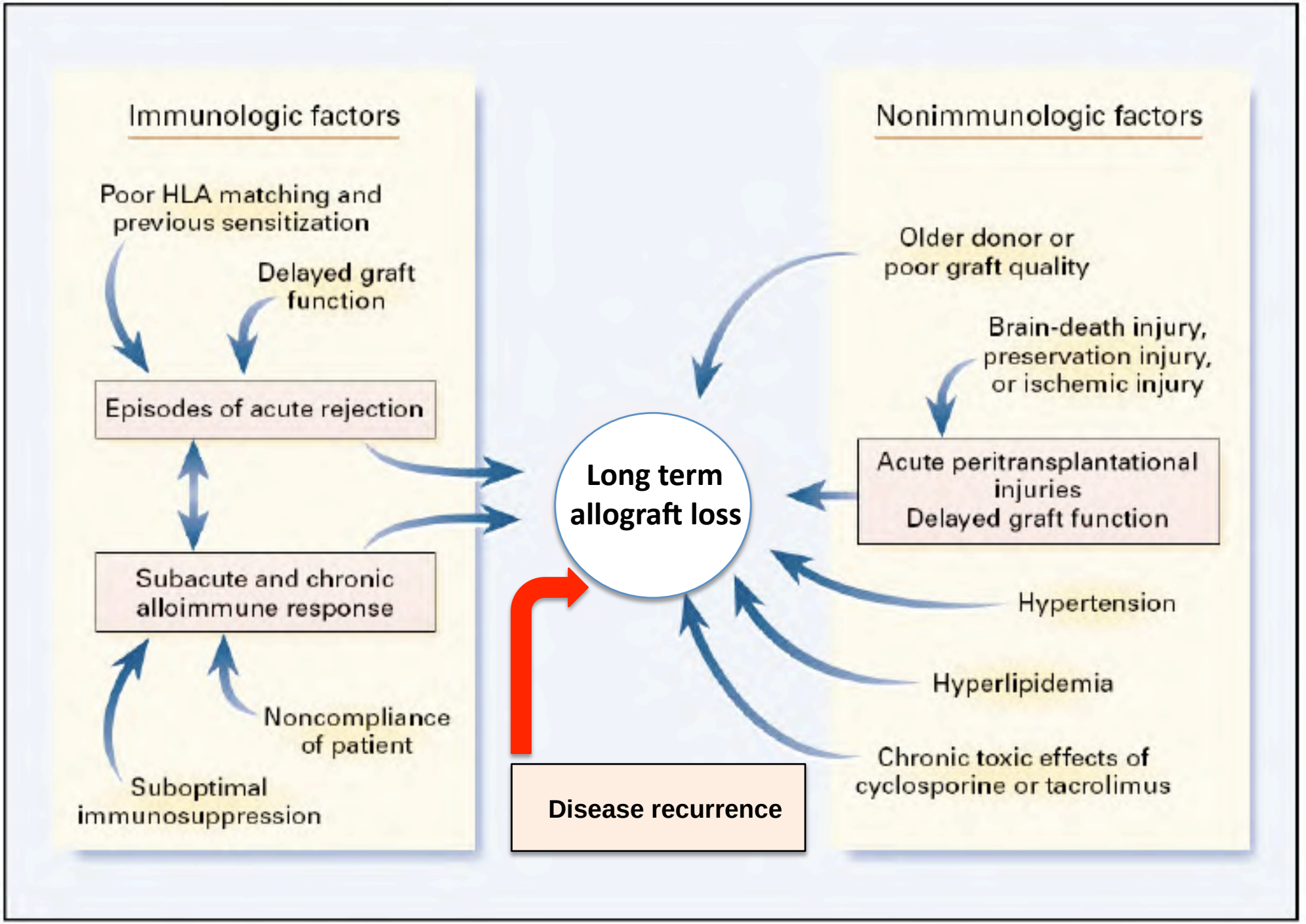
Le rein greffé: une victime des maladies systémiques.

Christophe Legendre

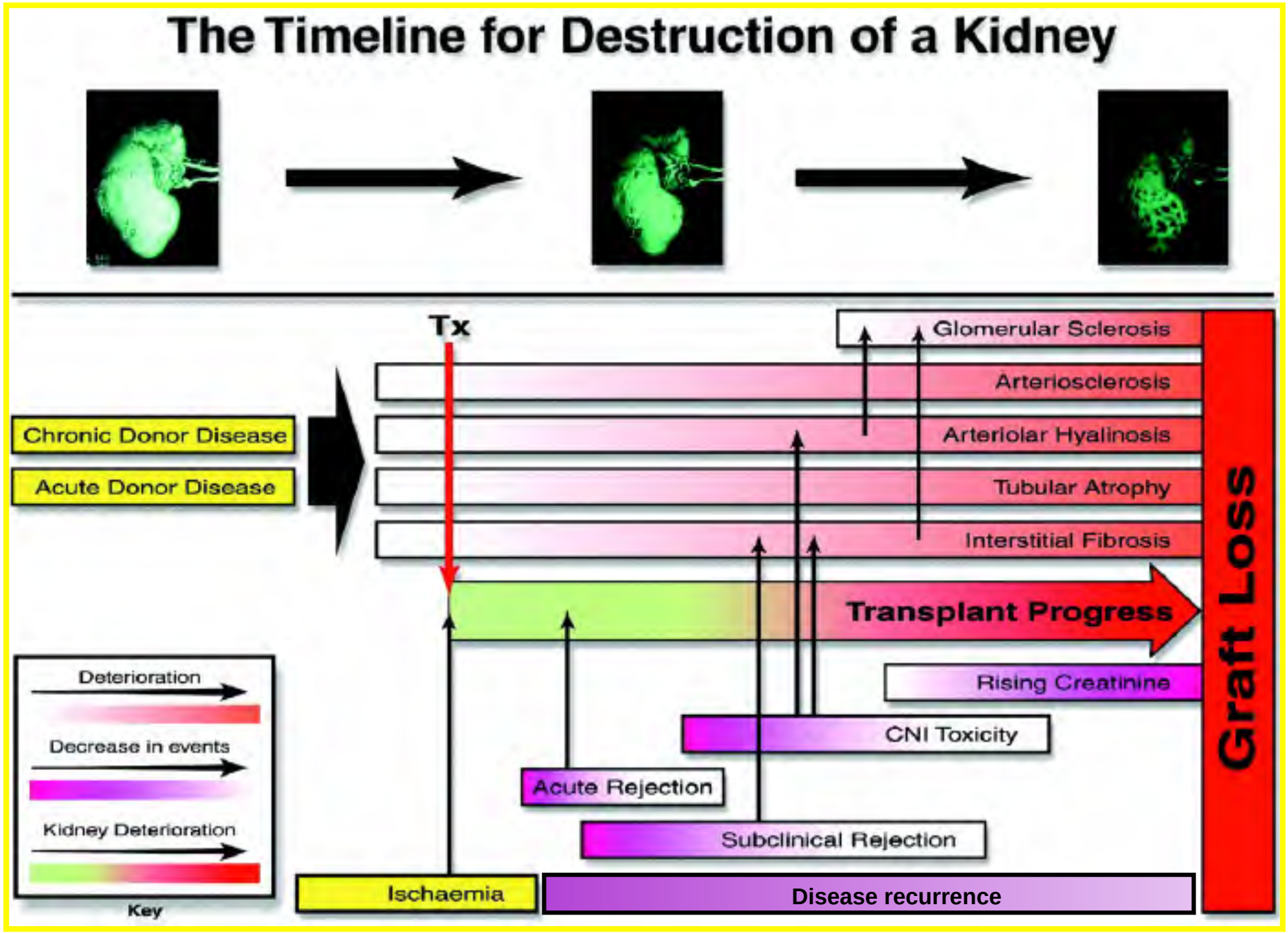
Université Paris Descartes, Sorbonne Paris Cité

& Hôpital Necker, Paris, France.

73ème Journée Française de Médecine, Paris, 16 mars 2012



Pascual M et al, N Engl J Med 2002



Chapman J et al, J Am Soc Nephrol 2006

Post-transplant kidney disease recurrence

Primary glomerular diseases

Systemic diseases

Metabolism diseases

Post-transplant kidney disease recurrence

Primary glomerular diseases

- Focal and segmental glomerulosclerosis +++
- IgA nephropathy +
- Henoch-Schönlein nephritis +
- Type 1 mesangiocapillary GN +
- Type 2 mesangiocapillary GN ++
- HUS +++
- Membranous glomerulonephritis+

Post-transplant kidney disease recurrence

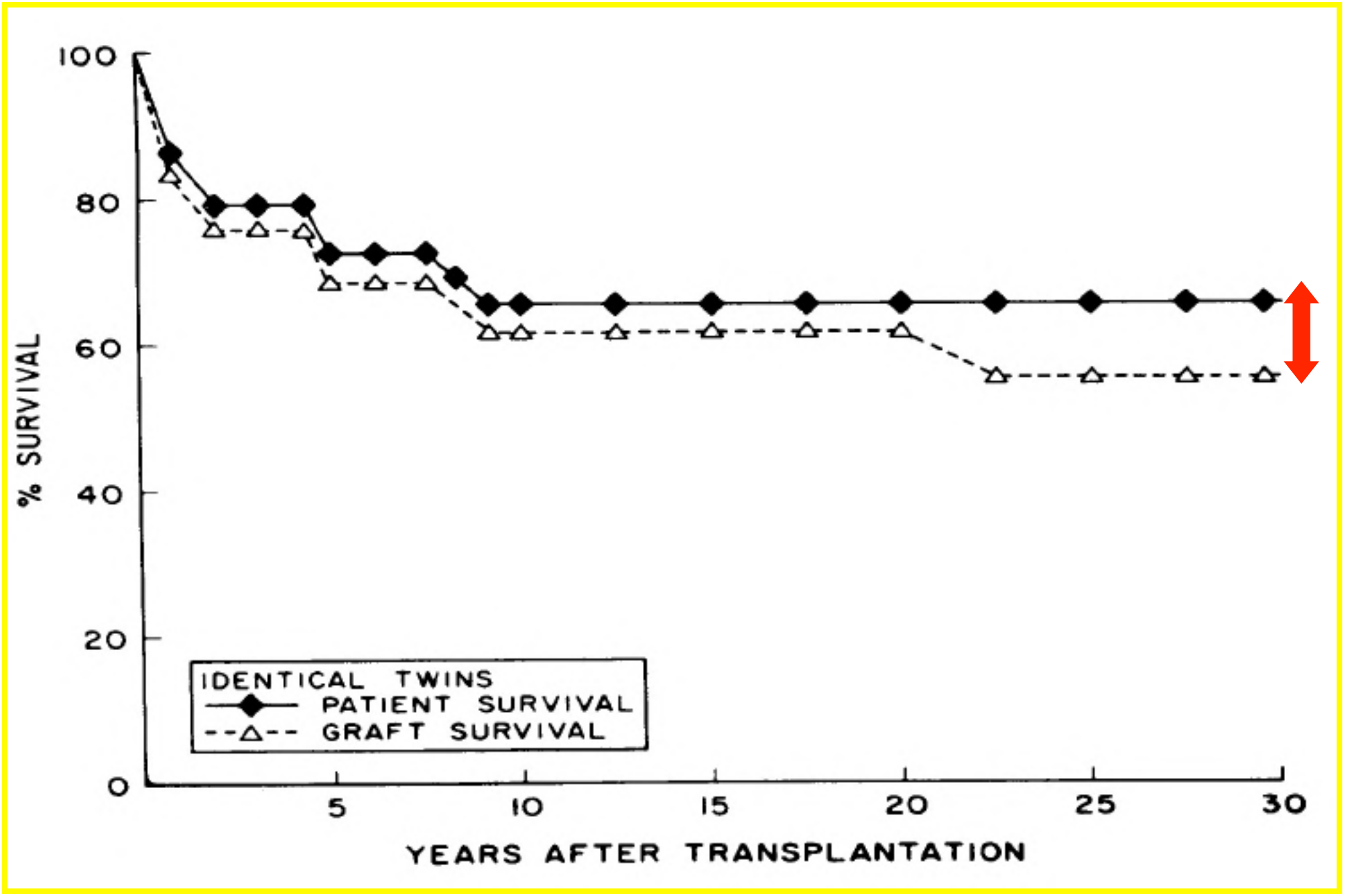
Systemic diseases

- Lupus +
- Angeitis:
    - ANCA-associated GN +
    - Wegener granulomatosis +
- Goodpasture syndrome +
- Diabetes mellitus +
- Amyloidosis +
- Sickle cell disease +

Post-transplant kidney disease recurrence

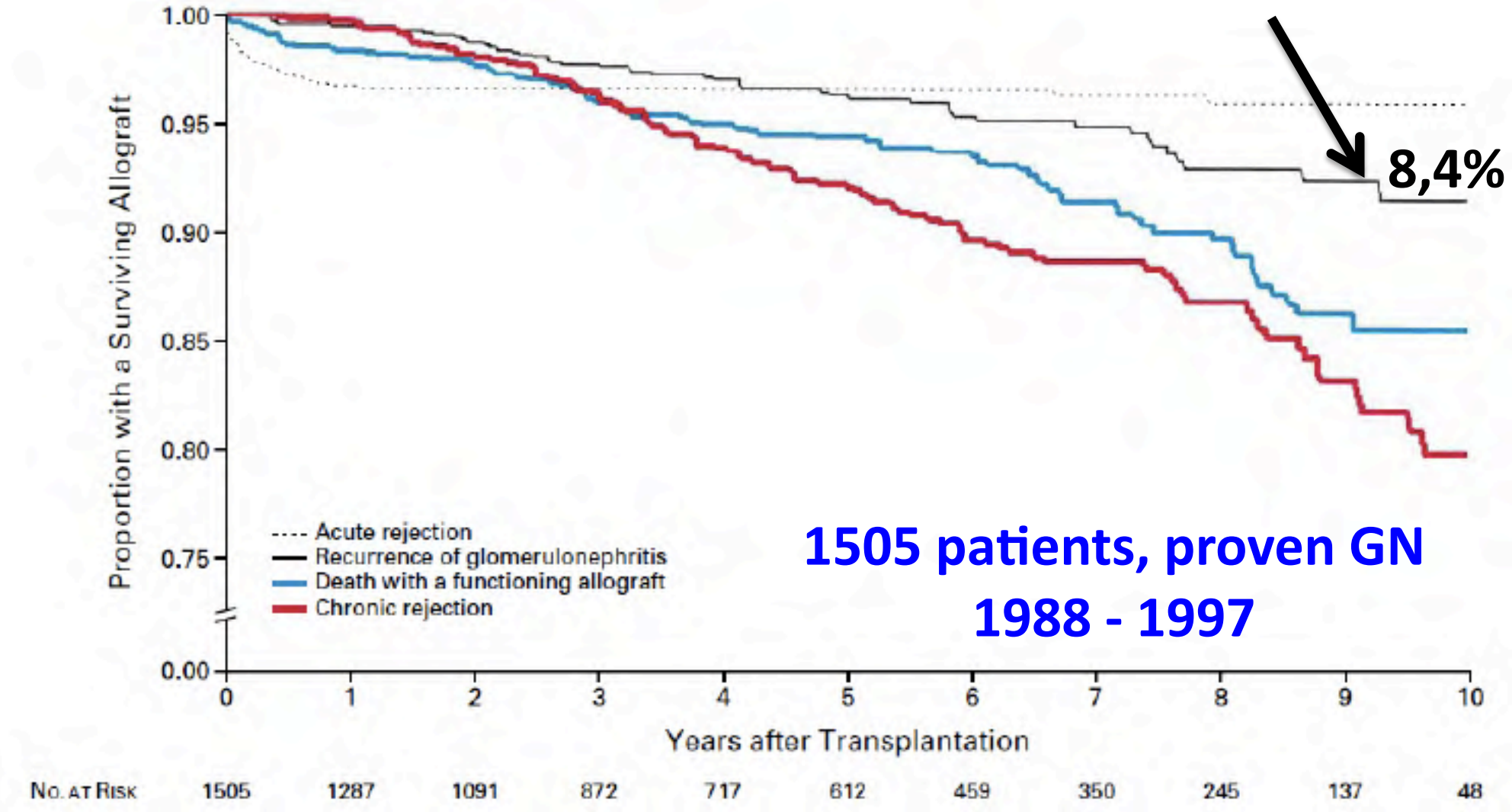
Metabolism diseases

- Type 1 et 2 hyperoxaluria +++
- Cystinosis +
- Fabry's disease +



Tilney N et al, World J Surg 1986

Graft loss due to recurrent GN



Hariharan S et al, Transplantation 1999

Briganti EM et al, N Engl J Med 2002

Outline

- **Definitions,**
- **Limitations in recurrent disease diagnosis,**
- **Current data:**
  - **From registries,**
  - **From single center studies,**
- **Therapeutical perspectives.**

Classifications of recurrent glomerular diseases

- **Clinical classification**:
  - True recurrence: native and recurrent disease are the same confirmed by histology,
  - Potential recurrence: occurrence of a post-transplant glomerular disease confirmed by biopsy without histological knowledge of native kidney disease,
  - De novo glomerulonephritis: occurrence of a new glomerular disease in the transplant kidney.

Classifications of recurrent glomerular diseases

- **Histological classification**:
  - Recurrence of a primary glomerular disease: FSGS, IgAN, MN, MPGN,
  - Recurrence of a secondary glomerular disease: SLE, Henoch-Schönlein purpura, HUS/TTP, crescentic GN, anti-GBM GN,
  - Glomerular recurrence of a systemic disease: diabetes mellitus, amyloidosis, fibrillary GN etc
  - Glomerular recurrence of Alport syndrome as anti-GBM GN.

Limitations in recurrent glomerular disease Δ

- **Native kidney disease**:
  - Unknown in many patients with ESRD,
  - Difficulties at late stage of chronic kidney disease,
  - Primary versus secondary FSGS.
- **Indication for post-transplant renal biopsy**:
  - Protocol versus for cause biopsy,
  - Immunofluorescence and EM not routinely applied.

Golgert WA et al, Clin J Am Soc Nephrol 2008

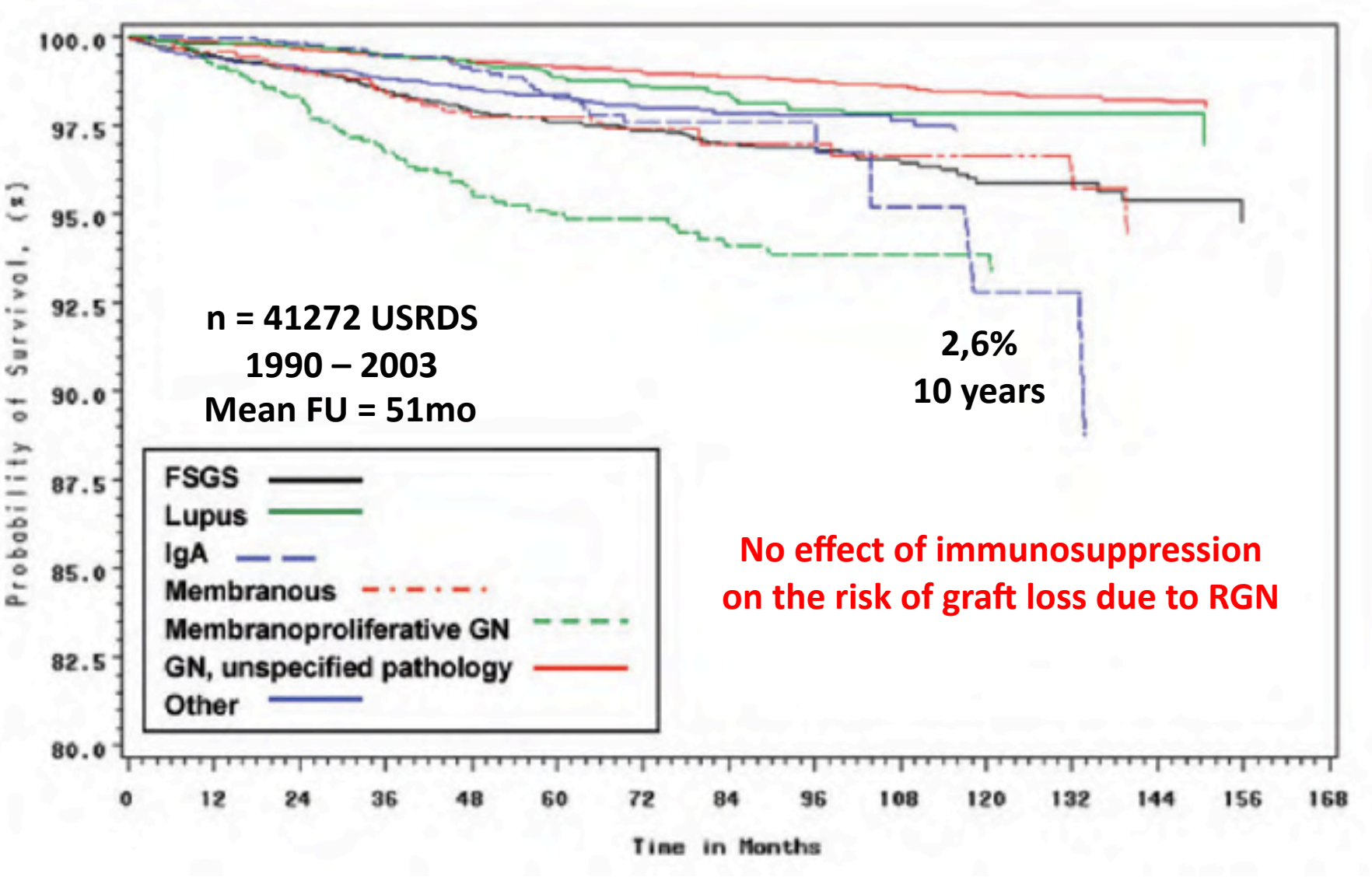
Limitations in recurrent glomerular disease Δ

- **Diagnosis of post-transplant GN**:
  - Early diagnosis of FSGS,
  - Incidence increases with time,
  - Recurrence may cause graft loss or not,
  - Difficulties in diagnosing MPGN versus TG,
  - Difficulties in diagnosing primary versus secondary IGAN,
  - Difficulties in diagnosing the cause of HUS/TTP…

Golgert WA et al, Clin J Am Soc Nephrol 2008

Data from registry

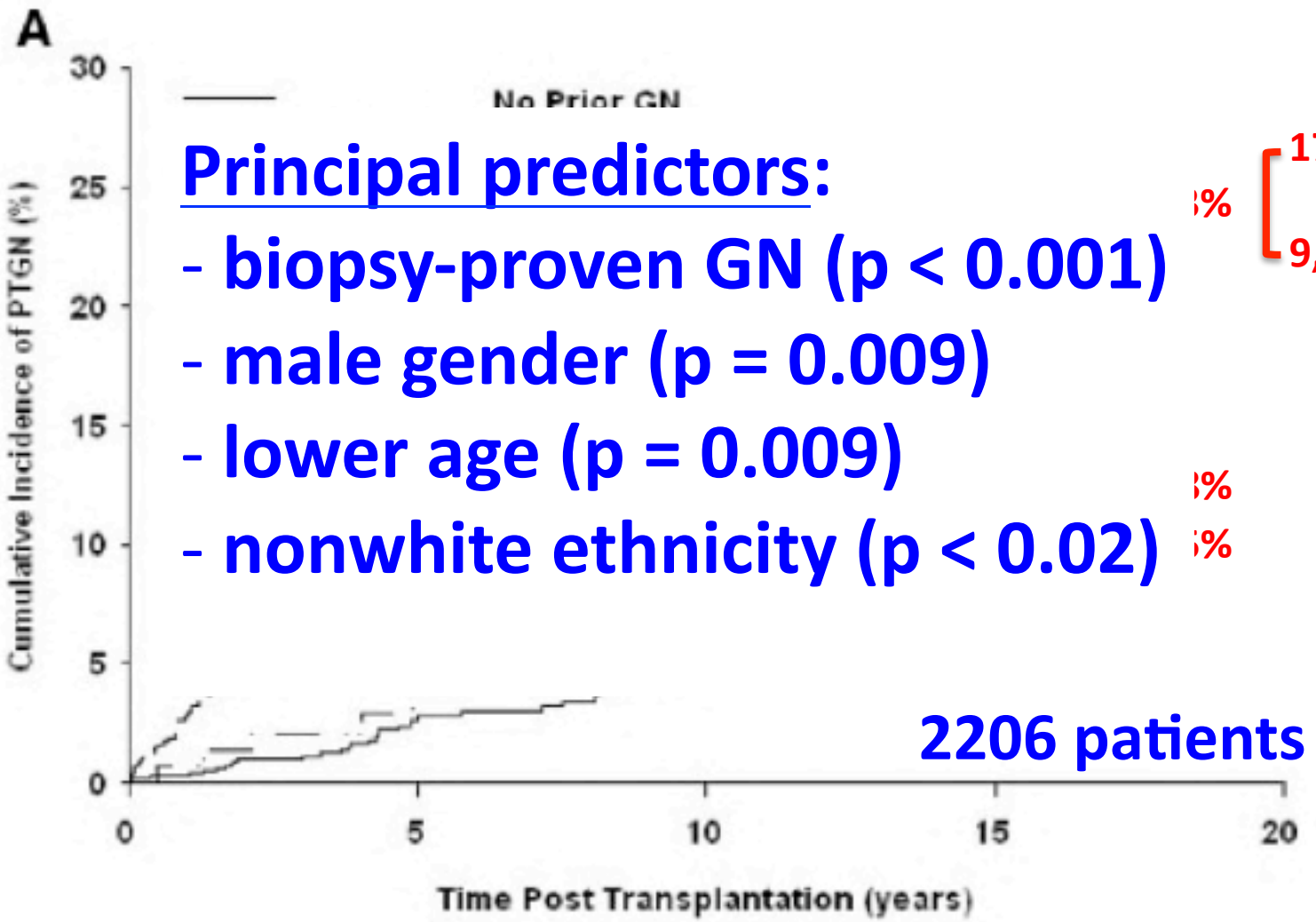
Graft loss due to post-RT disease recurrence



Mulay AV et al, Am J Transplant 2009

Data from single center

Probability of post-RT glomerulonephritis



Principal predictors:

- biopsy-proven GN (p < 0.001)
- male gender (p = 0.009)
- lower age (p = 0.009)
- nonwhite ethnicity (p < 0.02)

17,8% REC

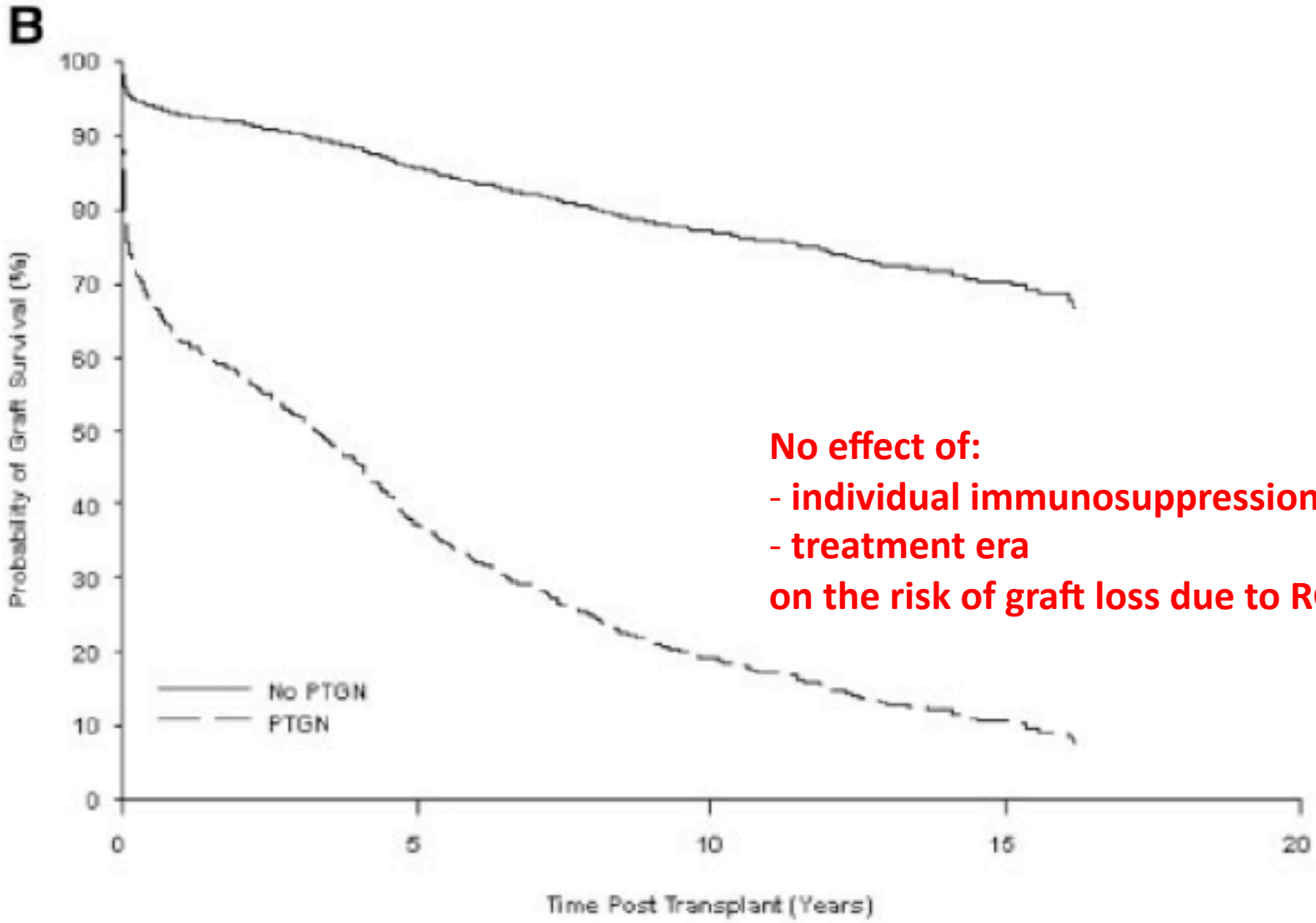
9,6% DE NO

2206 patients

Chailimpamontree W et al, J Am Soc Nephrol 2009

Data from single center

Graft loss due to post-RT disease recurrence



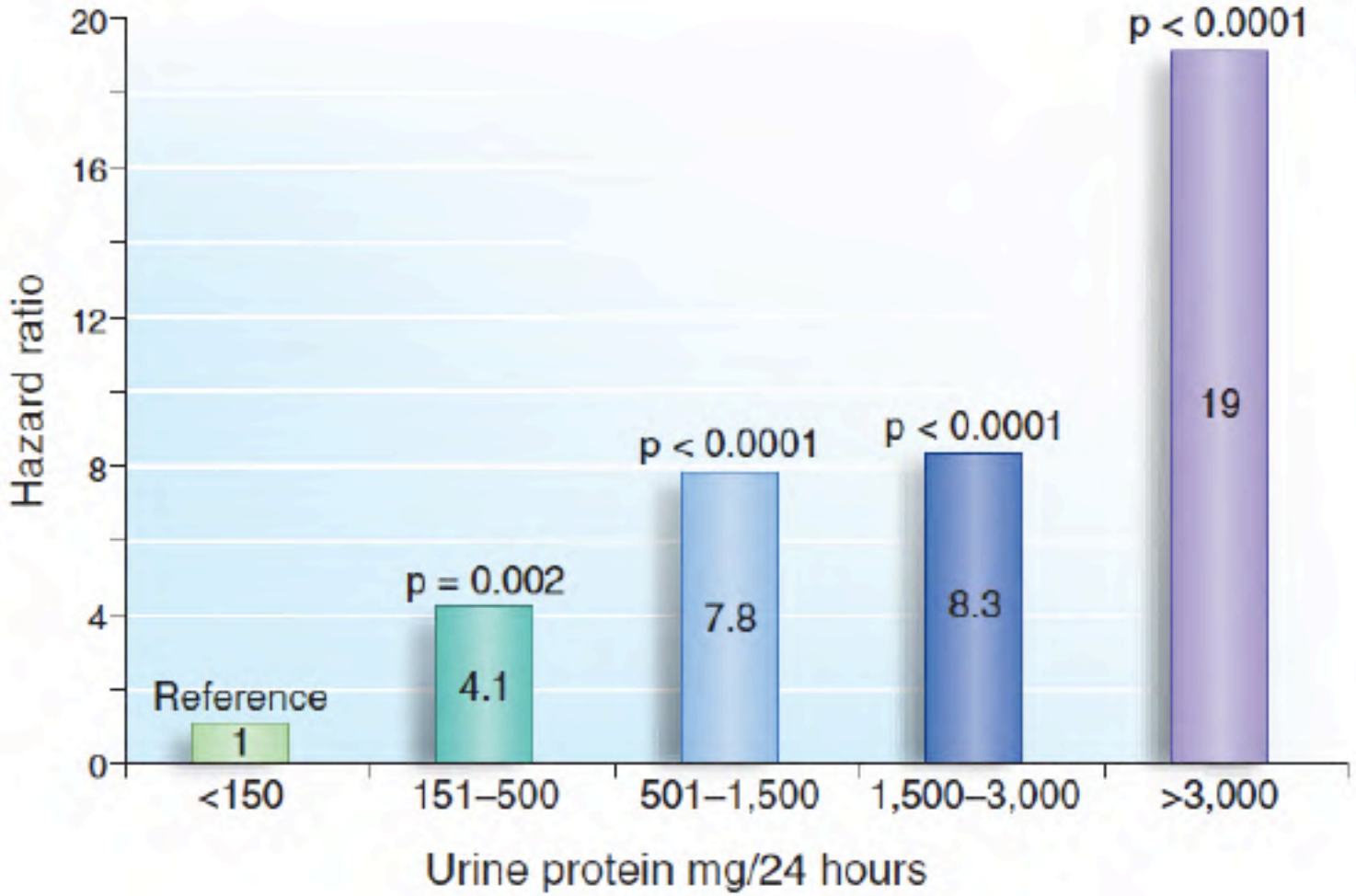
No effect of:
- individual immunosuppression,
- treatment era
on the risk of graft loss due to RGN.

Chailimpamontree W et al, J Am Soc Nephrol 2009

Data from single center

Proteinuria due to post-RT disease recurrence

80% with a glomerular disease



Hamer H et al, Am J Transplant 2007

Data from single center

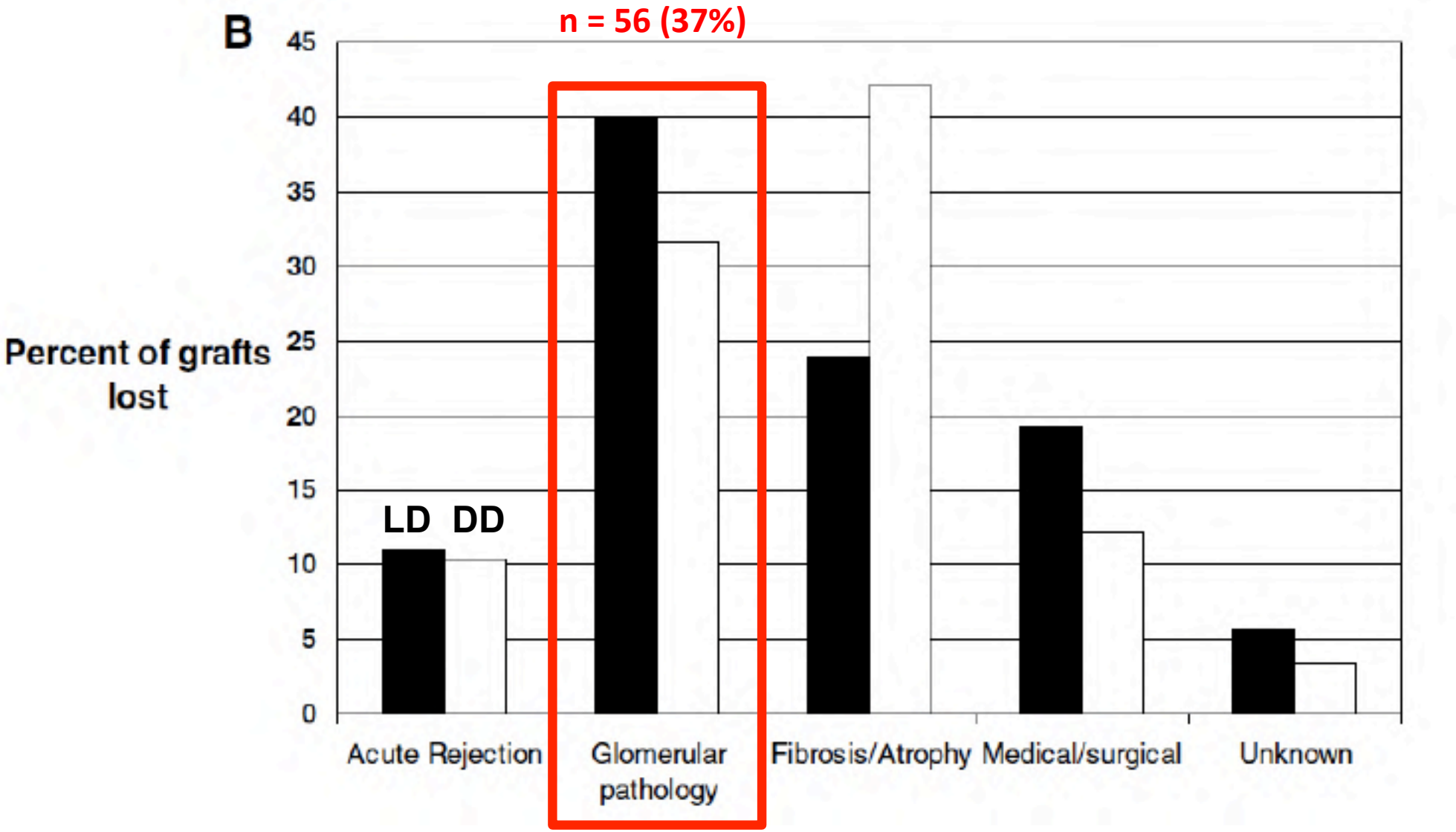
Graft loss due to post-RT disease recurrence

- Causes of kidney graft losses at one center,
- 1317 kidney recipients studied (mean FU = 50,3 ± 32,6 months),
- **330 grafts lost (25%) during follow-up**:
  - 39 (2,9%) due to primary non function,
  - 138 (10,4%) due to death with function,
  - **153 (11,6%) due to graft failure censored for death.**

EL-Zoghby ZM et al, Am J Transplant 2009

Data from single center

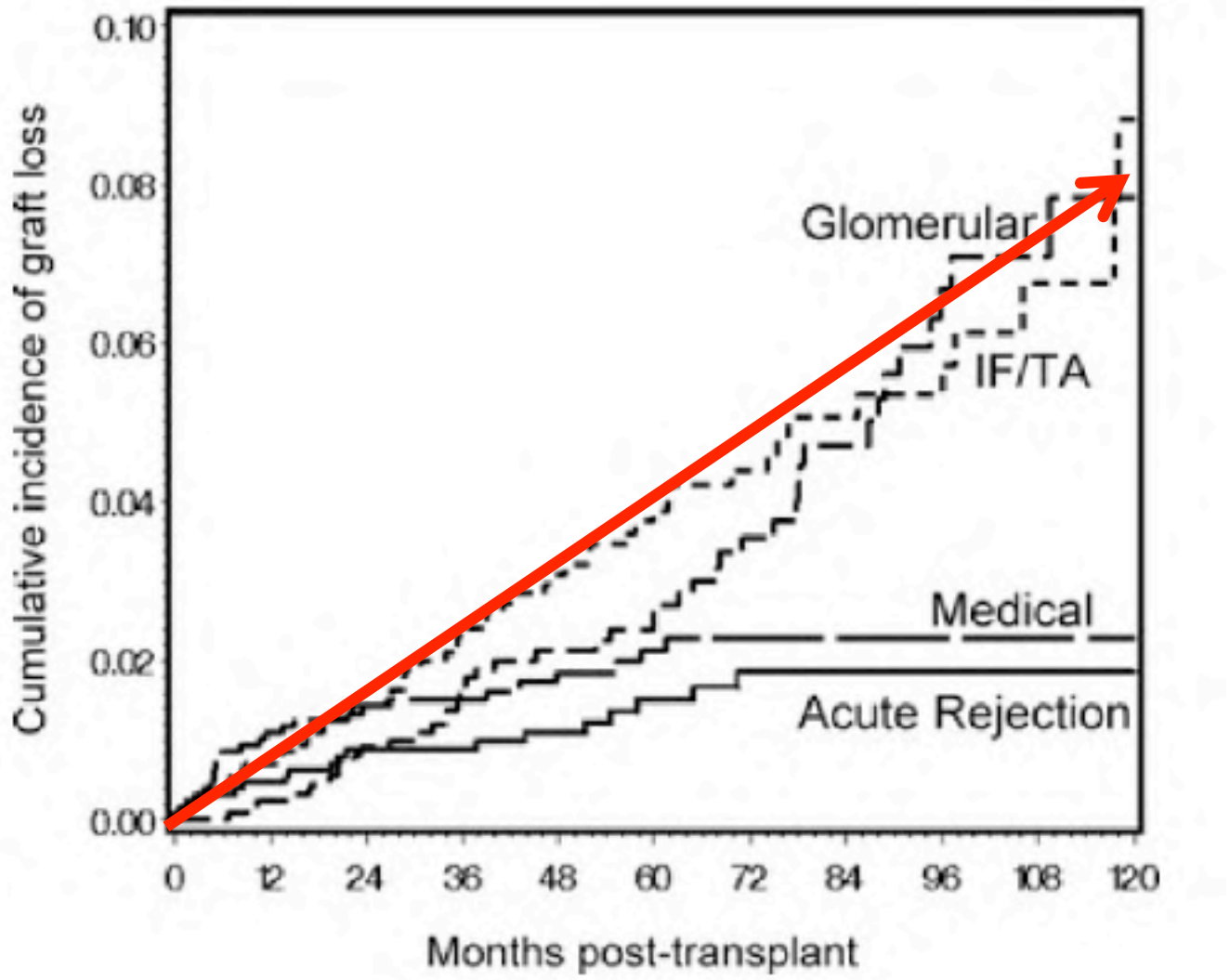
Graft loss due to post-RT disease recurrence



EL-Zoghby ZM et al, Am J Transplant 2009

Data from single center

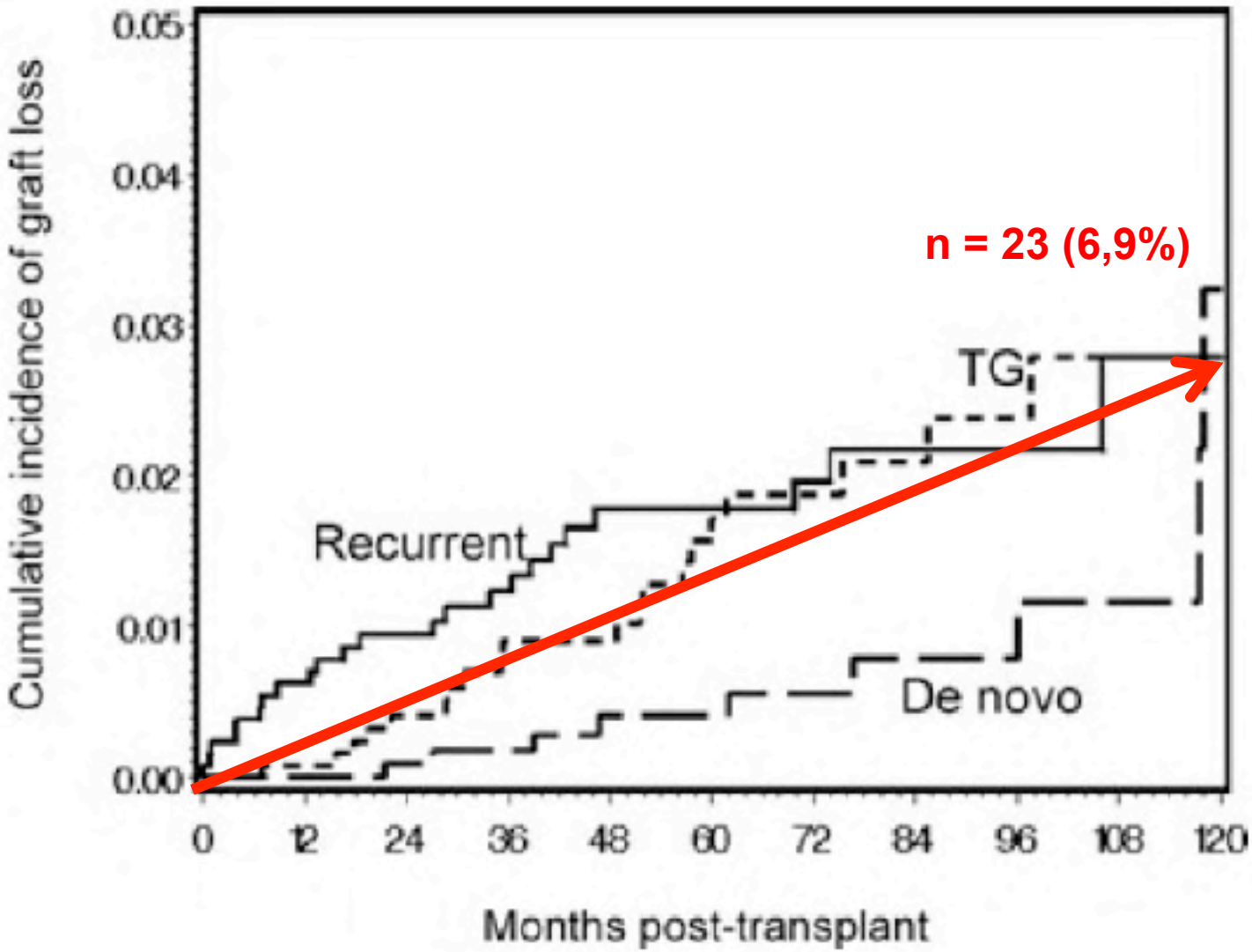
Graft loss due to post-RT disease recurrence



EL-Zoghby ZM et al, Am J Transplant 2009

Data from single center

Graft loss due to post-RT disease recurrence



EL-Zoghby ZM et al, Am J Transplant 2009

1. Antiphospholipid syndrome

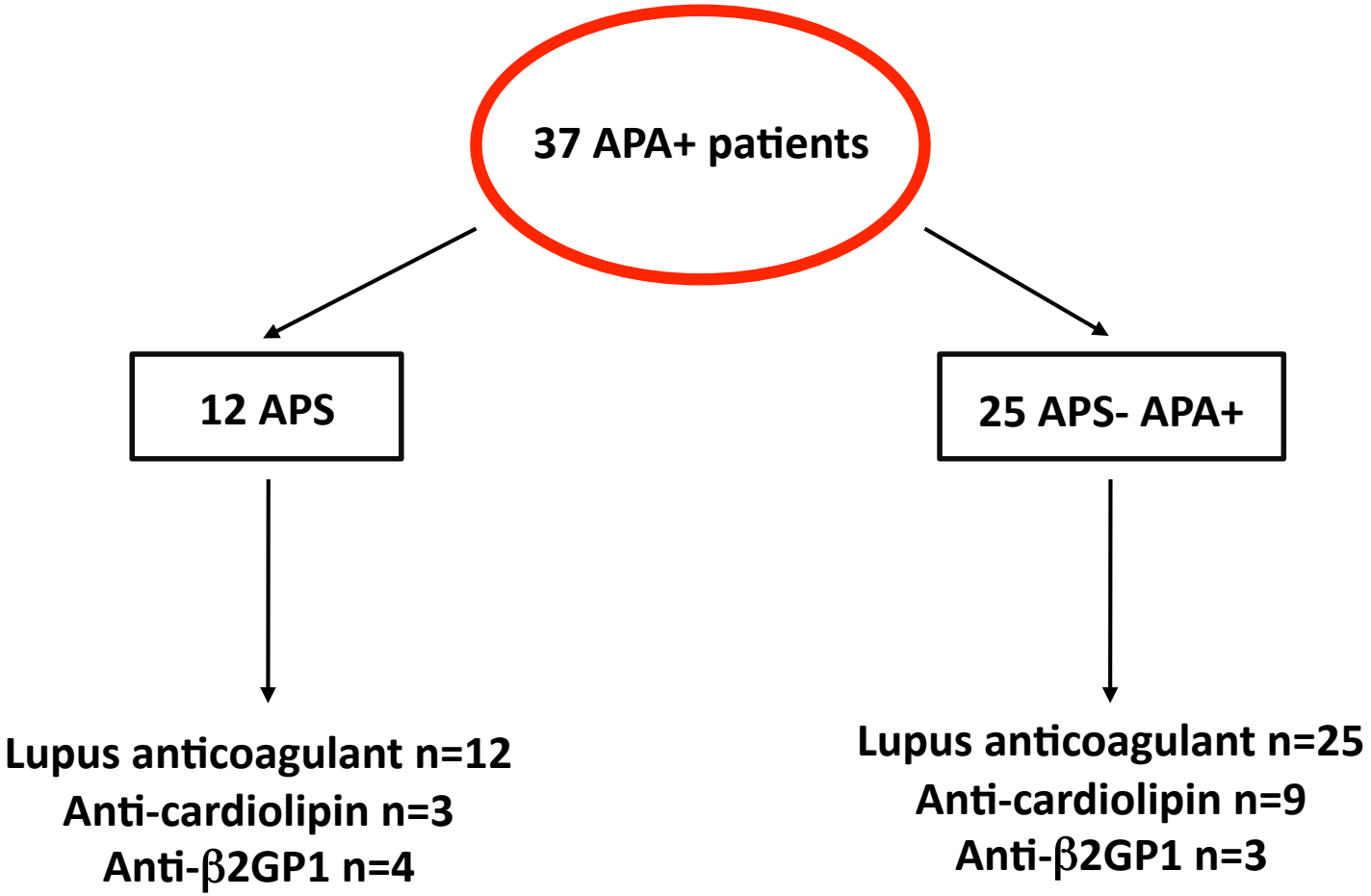
2. Focal and segmental glomerulosclerosis

3. Atypical Hemolytic Uremic Syndrome

4. Membranous nephropathy

5. IgA nephropathy

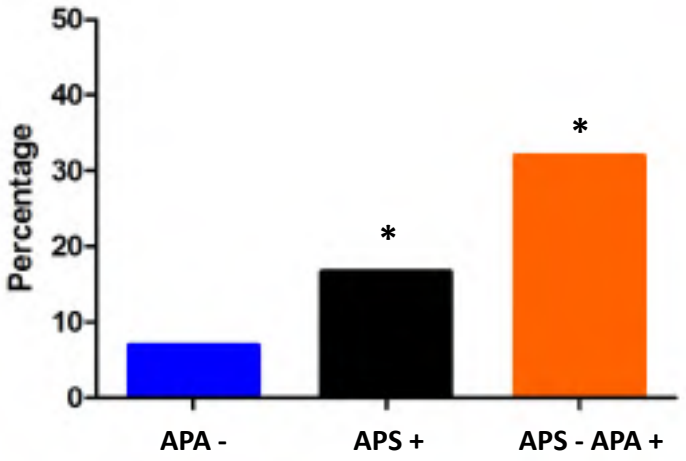
Antiphospholipid syndrome (APS)



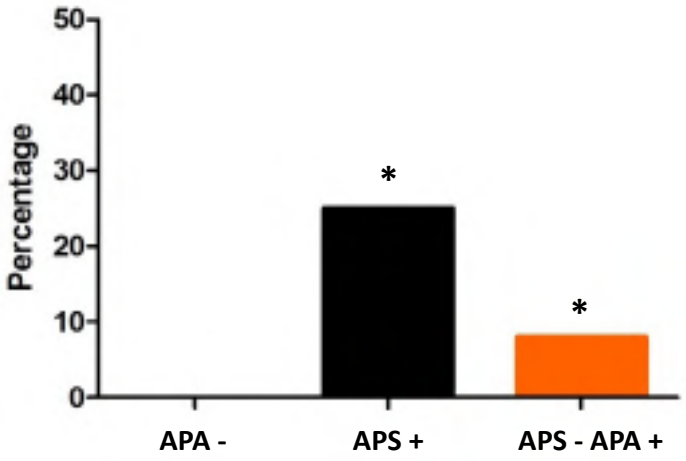
Canaud G et al, Am J Transplant 2010

Antiphospholipid syndrome (APS)

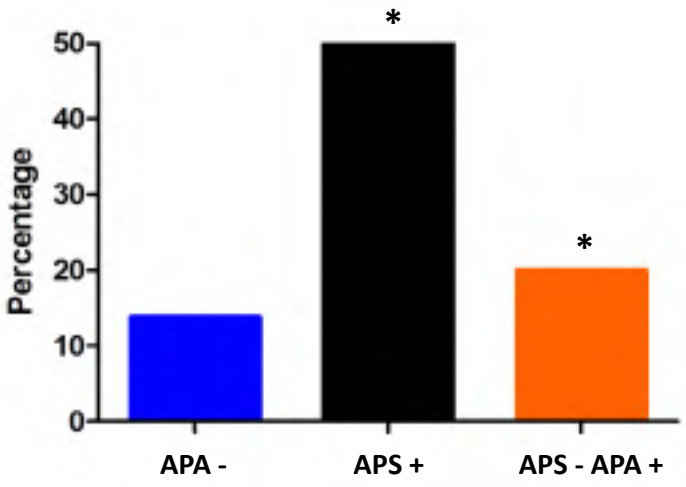
Allograft thrombosis



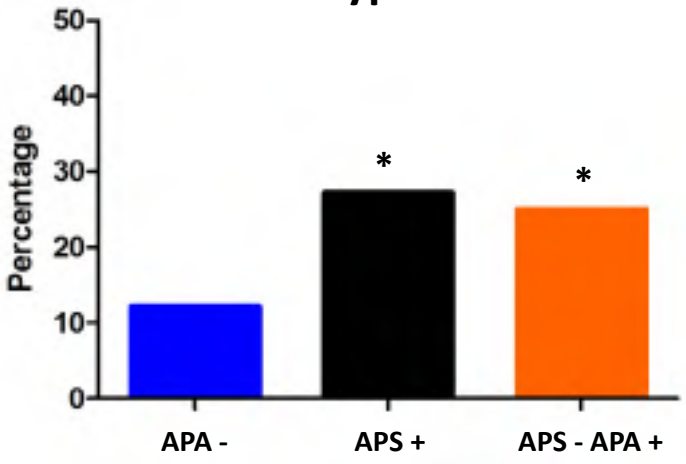
Cortical necrosis



Deep venous thrombosis post Tx



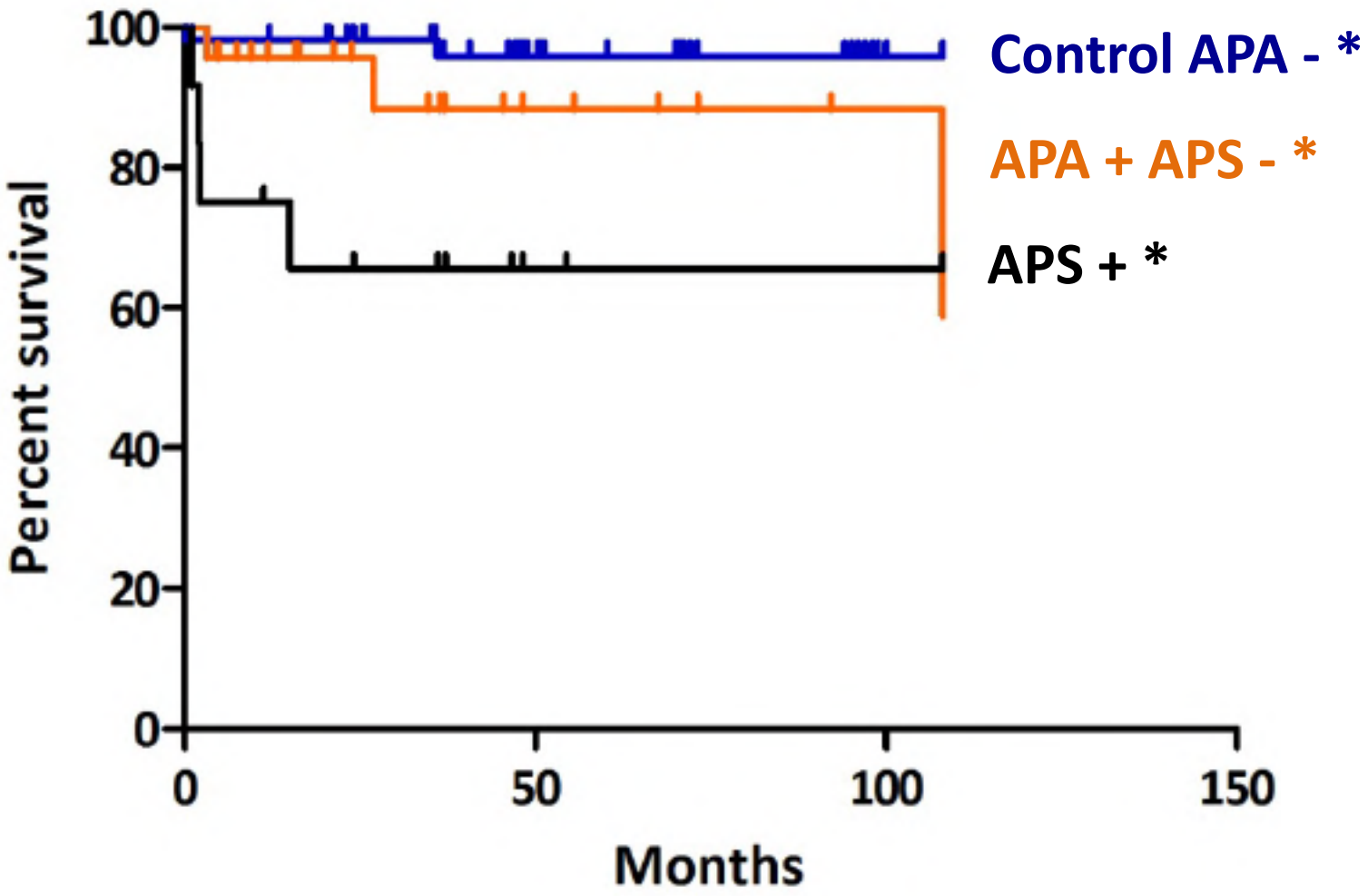
Severe hypertension



Canaud G et al, Am J Transplant 2010

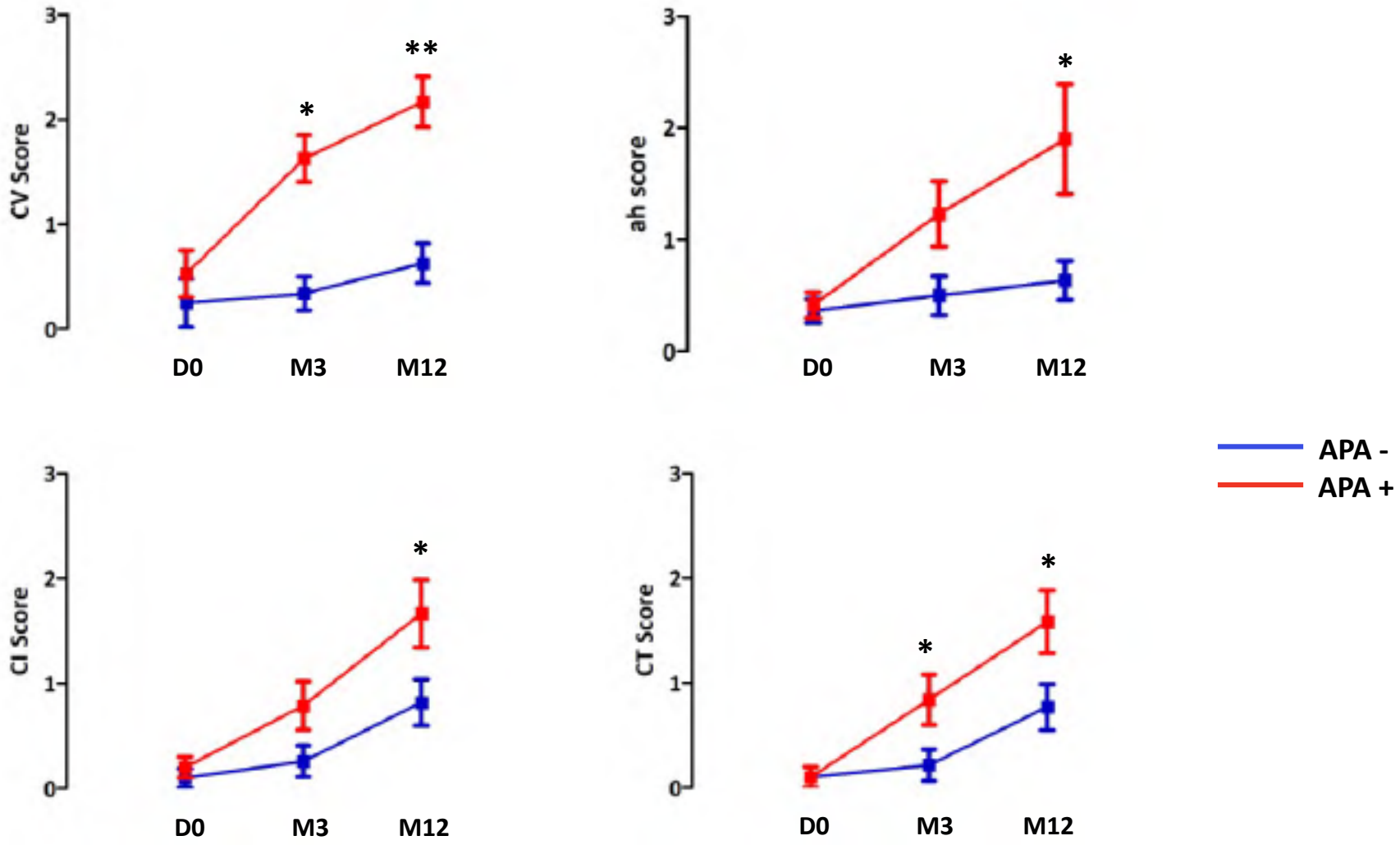
APS

Antiphospholipid syndrome (APS)



Canaud G et al, Am J Transplant 2010

Antiphospholipid syndrome (APS)

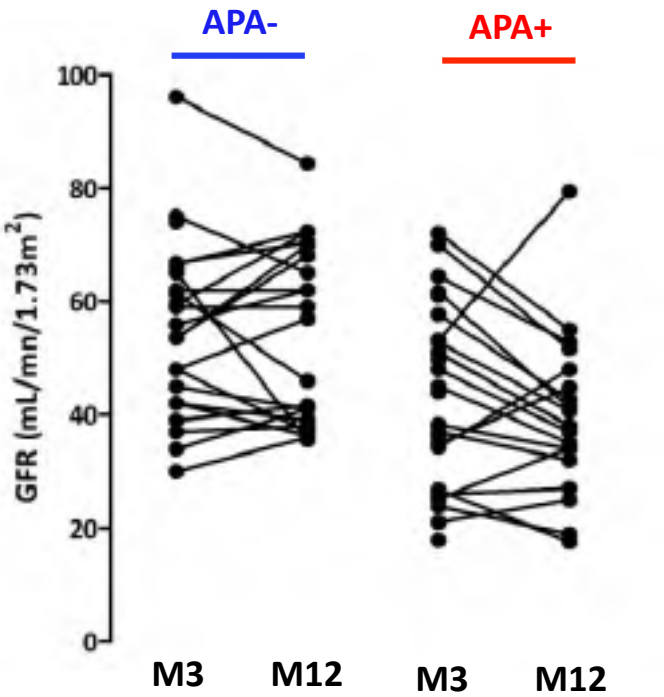


Canaud G et al, Am J Transplant 2010

APS

Antiphospholipid syndrome (APS)

Measured GFR (mL/min/1.73m^2)	APA -	APA +	p
Month-3	55 ± 15.3	42.8 ± 16.6	<0.01
Month-12	53.9 ± 15.6	39.6 ± 13.9	<0.01



Canaud G et al, Am J Transplant 2010

APS

Antiphospholipid syndrome (APS)

American Journal of Transplantation
Wiley Periodicals Inc.

doi: 10.1111/j.1600-6143.2011.03696.x

Case Report

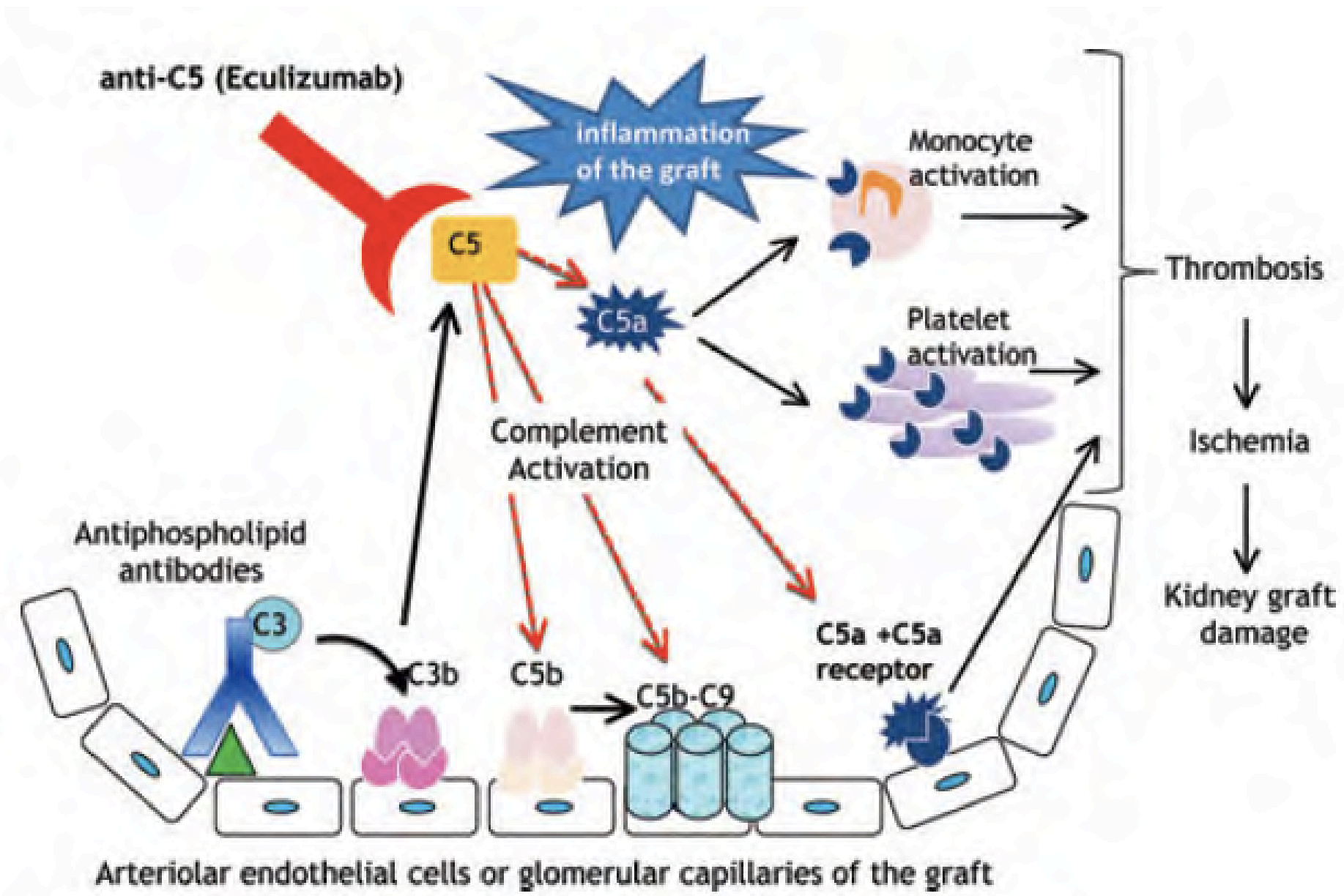
Eculizumab in Acute Recurrence of Thrombotic Microangiopathy After Renal Transplantation

K. Hadaya[a,b], S. Ferrari-Lacraz[c,d], D. Fumeaux[a], F. Boehlen[e], C. Toso[b,c], S. Moll[f], P-Y. Martin[a] and J. Villard[c,d,*]

Received 26 April 2011, revised 08 June 2011 and accepted for publication 01 July 2011

Hadaya K et al, Am J Transplant 2011

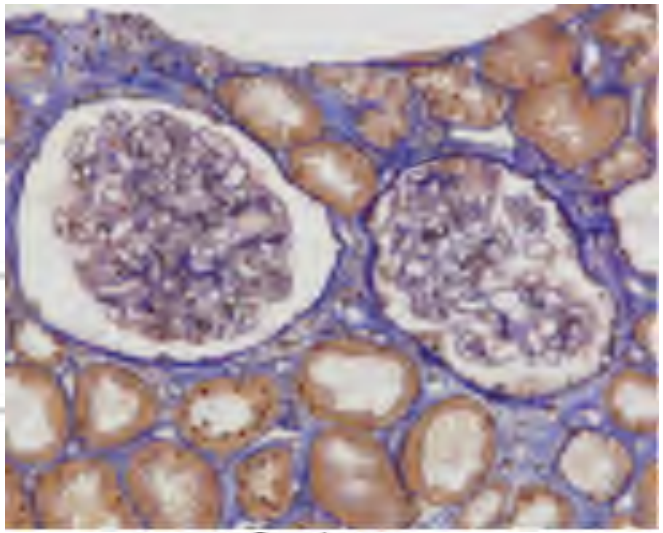
APS



Hadaya K et al, Am J Transplant 2011

APS

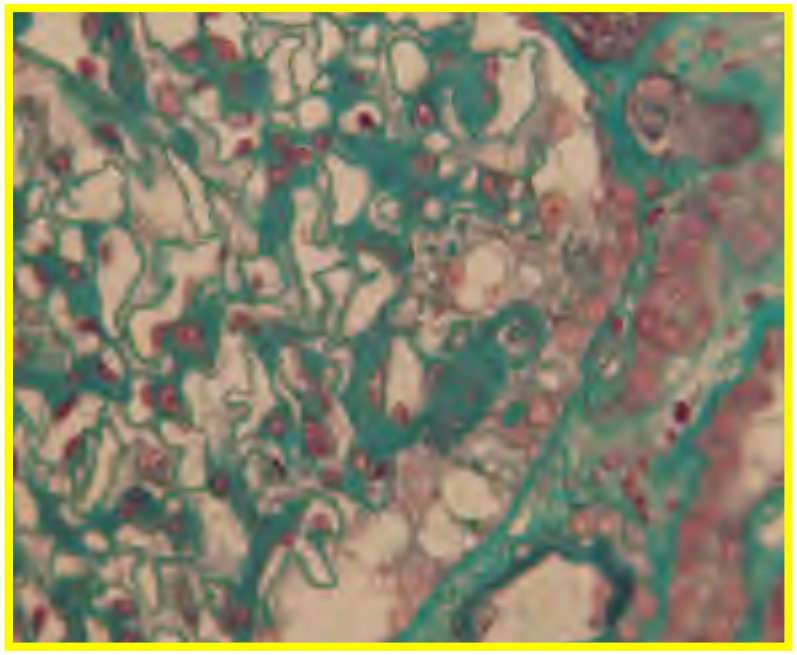
Antiphospholipid syndrome (APS)

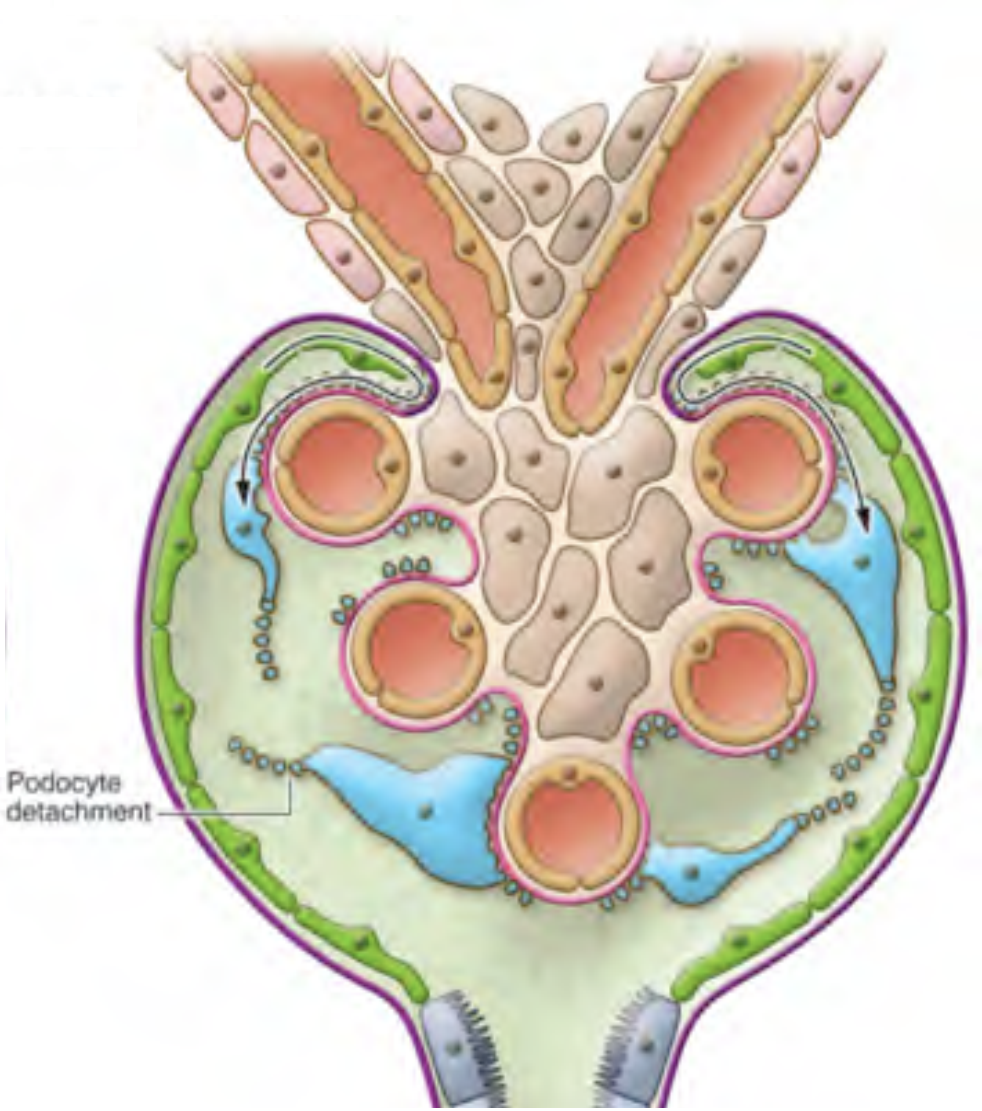


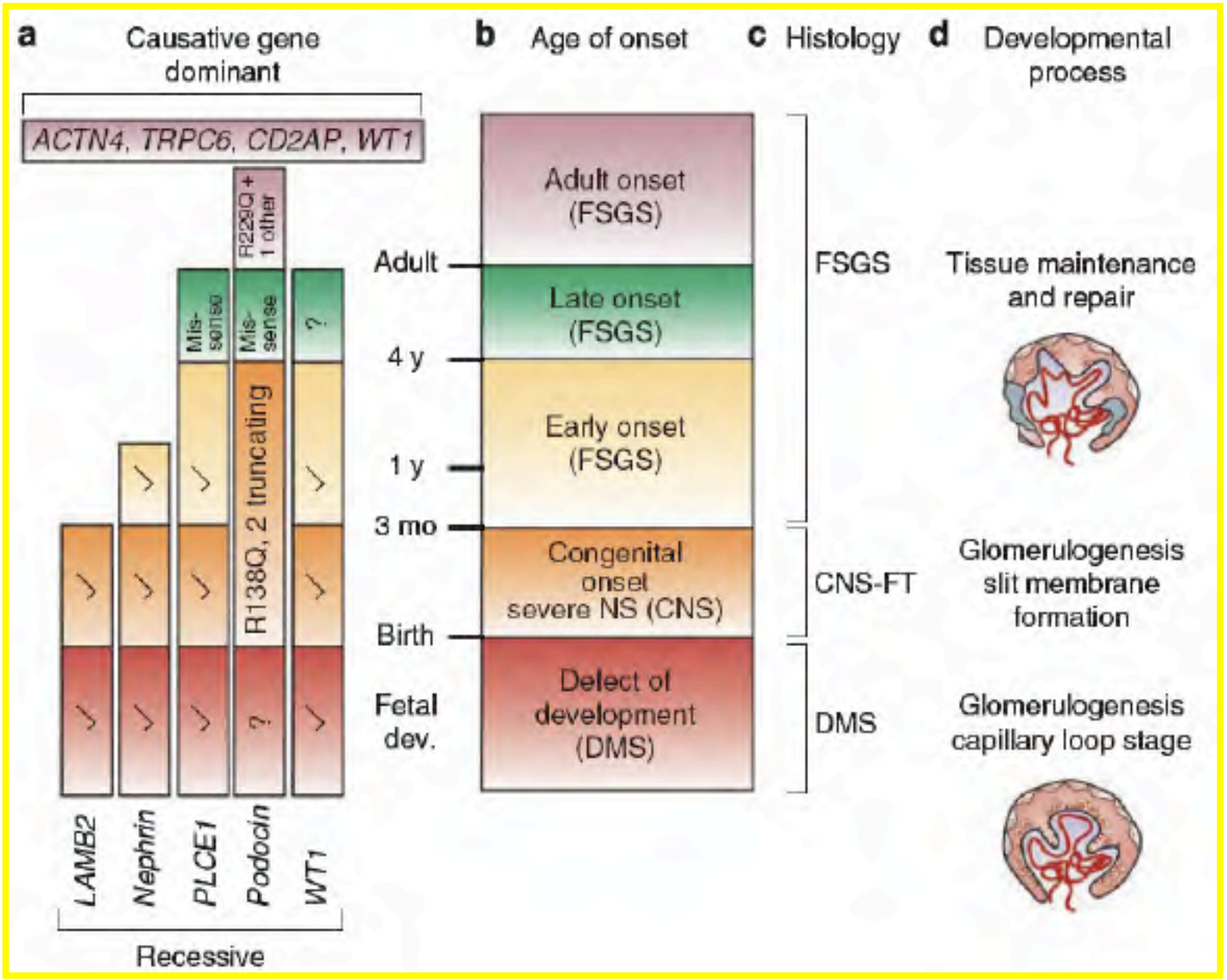
Hadaya K et al, Am J Transplant 2011

APS

Focal and segmental glomerulosclerosis







Hildebrand F et al, Kidney Int 2009

Primary FSGS

Secondary FSGS

D'Agati VD et al,

Curr Opin Nephrol Hypertens 2008

1. Familial/genetic

- Mutations in nephrin
- Mutations in podocin
- Mutations in α-actinin-4
- Mutations in transient receptor potential cation 6 channel
- Mutations in CD2AP
- Mutations in WT-1
- Mutations in PLCE1
- Mutations in SMARCAL1 (Schimke immuno-osseous dysplasia)
- Mutations in mitochondrial proteins (mitochondrial cytopathies)
- Mutations in β4 integrin (epidermolysis bullosa)
- Mutations in tetraspanin 24 (epidermolysis bullosa, deafness)
- Mutations in laminin β2 (Pierson syndrome)

2. Virus-associated

- HIV-1 ('HIV-associated nephropathy')
- Parvovirus B-19
- SV40
- Cytomegalovirus

3. Drug-induced

- Heroin ('heroin nephropathy')
- Interferon-α
- Lithium
- Pamidronate
- Sirolimus

4. Mediated by adaptative structural-functional responses

- Reduced renal mass
  - Oligomeganephronia
  - Unilateral renal agenesis
  - Renal dysplasia
  - Reflux nephropathy
  - Sequela to cortical necrosis
  - Surgical renal ablation
  - Chronic allograft nephropathy
  - Any advanced renal disease with reduction in functioning nephrons
- Initially normal renal mass
  - Hypertension
  - Atheroemboli or other acute vaso-occlusive processes
  - Obesity
  - Cyanotic congenital heart disease
  - Sickle cell anemia

	n	Traitements	Remissions
Laufer J., Transplantation, 1988	**2**	**9 EP**	**Complètes puis rechutes**
Ingulli E., Transplantation, 1990	**3**	**Ciclosporine**	**-1 Complète puis rechute -1 partielle**
Cochat P., Pedia Nephro,1993	**3**	**Bolus Solumedrol + 10EP puis 1/s pendant 2 mois + Cyclophosphamide**	**-Complètes puis rechute pour 1**
Dantal J., NEJM, 1994	**8**	**ATG+Ciclosporine+corticoïdes+ Immunoadsorption**	**-7 complètes, puis rechutes systématiques**
Dall'Amico R., AJKD, 1999	**11**	**10-15 EP + Cyclophosphamide**	**-7 complètes (suivi 32 mois) soit 63%**
Cheong HI., NDT, 2000.	**6**	**10 EP pendant 2 mois + Cyclophosphamide+ Ciclosporine IV (C0 150-200ng/ml)**	**-3 complètes -3 partielles**
Salomon R., Transplantation, 2003.	**16**	**Ciclosporine IV EP pour 4**	**-14 complètes 68% -2 partielles**
Raafat RH., AJKD, 2004.	**16**	**Ciclosporine orale 6-25mg/kg selon la rémission (C0 200-1000ng/ml) EP pour 7**	**-11/16 complètes mais 2 ont perdu leur greffons d'autres causes -2/11 partielles (suivi 0.8-12 ans)**
Deegens JK., Transplant Int, 2004	**13**	**EP dès la récidive**	**-7 complètes (18 EP/patients) -4 partielles avec EP prolongés (58 EP/patients) -2 pertes de greffons précoces**
Valdivia P., Transplant Proc, 2005.	**10**	**17 EP+ Losartan**	**-6 complètes (suivi 10 mois) -3 partielle (suivi de 10 mois)**

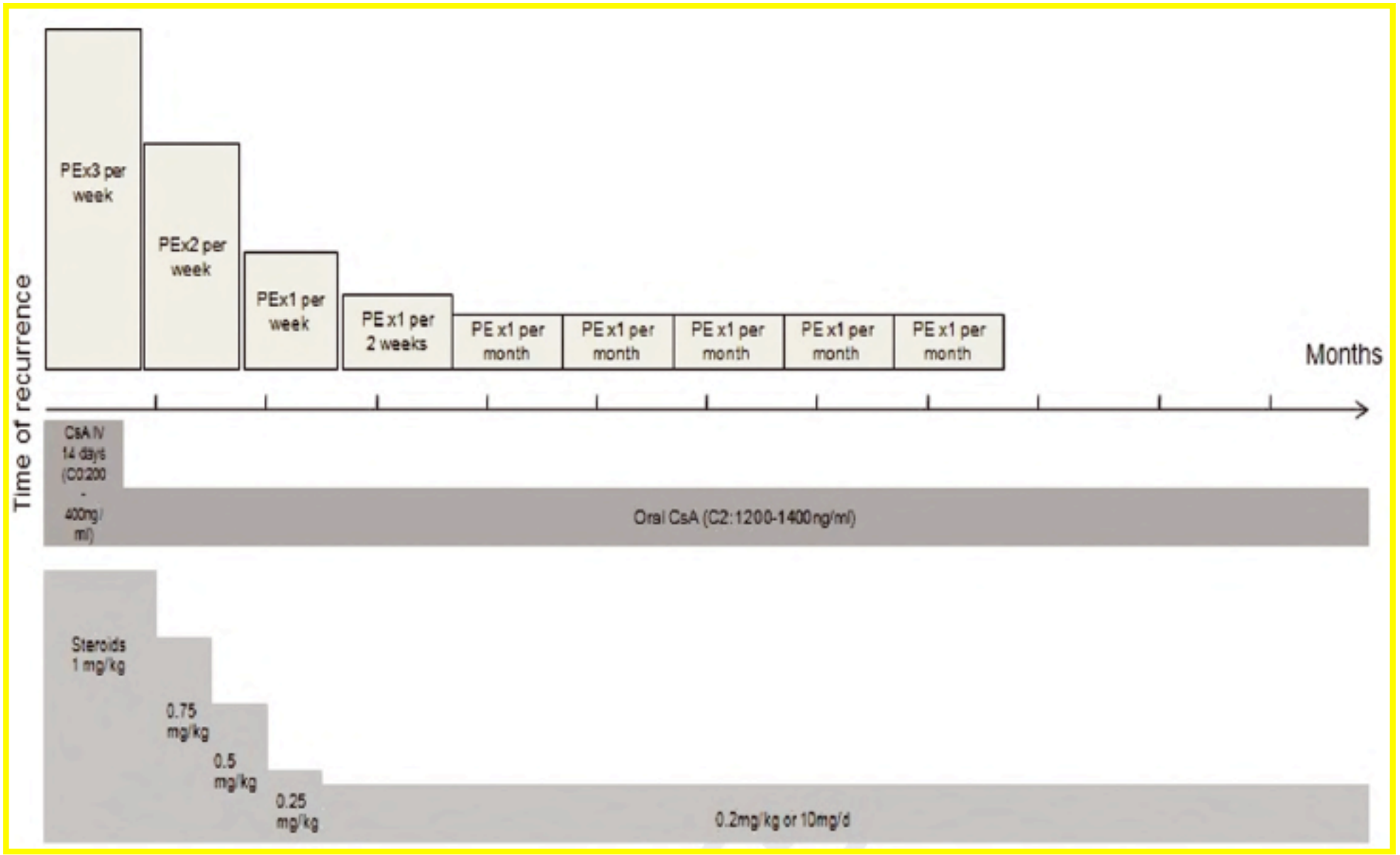
Table 2: Patients treated for FGGS recurrence during the1997–2005 time period (control group)

Patients	Treatment	Histological finding	Outcome	Proteinuria remission
1	CsA IV	FSGS on M12	Return on dialysis on M24	No
2	Cyc + PE	Normal kidney on light microscopy	Last follow-up M80	Complete and sustained
3	FK + PE	Normal kidney on light microscopy	Last follow-up M47	Complete and sustained
4	Steroids + FK	FSGS on M12	Last follow-up M52	No
5	CsA IV + PE	FSGS on M18	Return on dialysis on M36	No
6	Steroids + PE	Normal kidney on light microscopy	Last follow-up M61	Partial
7	CsA oral +PE	FSGS on M6	Return on dialysis M24	No
8	Steroids + PE	FSGS M12	Return on dialysis on M34	No
9	CsA oral + PE	Normal kidney on light microscopy	Last follow-up M92	Partial
10	CsA IV + PE	FSGS on M6	Return on dialysis on M6	No
11	CsA IV + PE	Normal kidney on light microscopy	Last follow-up M55	Complete and sustained
12	PE + Cyc + Rituximab	FSGS on M12	Return on dialysis M18	No
13	CsA IV	FSGS on M9	Return on dialysis on M48	No
14	CsA oral + PE	FSGS M18	Return on dialysis on M40	No
15	PE + Steroids + FK	FSGS M24	Return on dialysis on M24	No
16	FK+ PE	Normal kidney on light microscopy	Last follow-up M60	Complete and sustained
17	PE + Steroids + Rituximab	Normal kidney on light microscopy	Last follow-up M38	Partial
18	PE + Steroids	Normal kidney on light microscopy	Last follow-up M85	Complete and sustained
19	CsA IV + PE	FSGS on M24	Return on dialysis on M48	No

42% complete remission at 3 months
27% complete remission at one year

Canaud G et al, Am J Transplant 2009

FSGS



Combined IV CsA 14d + high dose steroids + plasma exchanges

Canaud G et al, Am J Transplant 2009

FSGS

Table 3: FSGS recurrence and treatment characteristics

Patient	Previous graft	Day of recurrence	Proteinuria at time recurrence (g/day)	Delay to remission (day)	Proteinuria month 3 (g/day)	Proteinuria month 12 (g/day)	Ioexhol GFR at 1 year (mL/min)	Follow-up after remission (months)	Total of PE sessions
1	0	2	4	18	0.05	0.05	86	21	25
2	0	12	5.4	24	0.1	0.1	58	19	25
3	0	55	7.1	28	0.3	0.3	75	16	25
4	0	1	7.9	29	0.15	0.07	84	18	25
5	0	2	5.6	18	0.20	0.05	94	17	25
6	0	4	7.7	20	0.22	0.1	41	14	25
7	0	4	22	10	0.3	0.05	61	16	25
8	2	1	8.7	23	0.04	1	85	15	39
9	0	1	40	33	0.05	0.1	56	13	25
10	0	1	12	26	0.2	0.1	45	9	25
Mean		8.3	12.0	22.9	0.16	0.19	68.5	15.8	
SD		16.8	11.1	6.7	0.09	0.29	18.6	3.3	

42% complete remission at 3 months
27% complete remission at one year

→

100% complete remission at 3 months
90% complete remission at one year
90% complete remission at 2 years

Canaud G et al, Am J Transplant 2009

FSGS

The FSGS permeability factor?

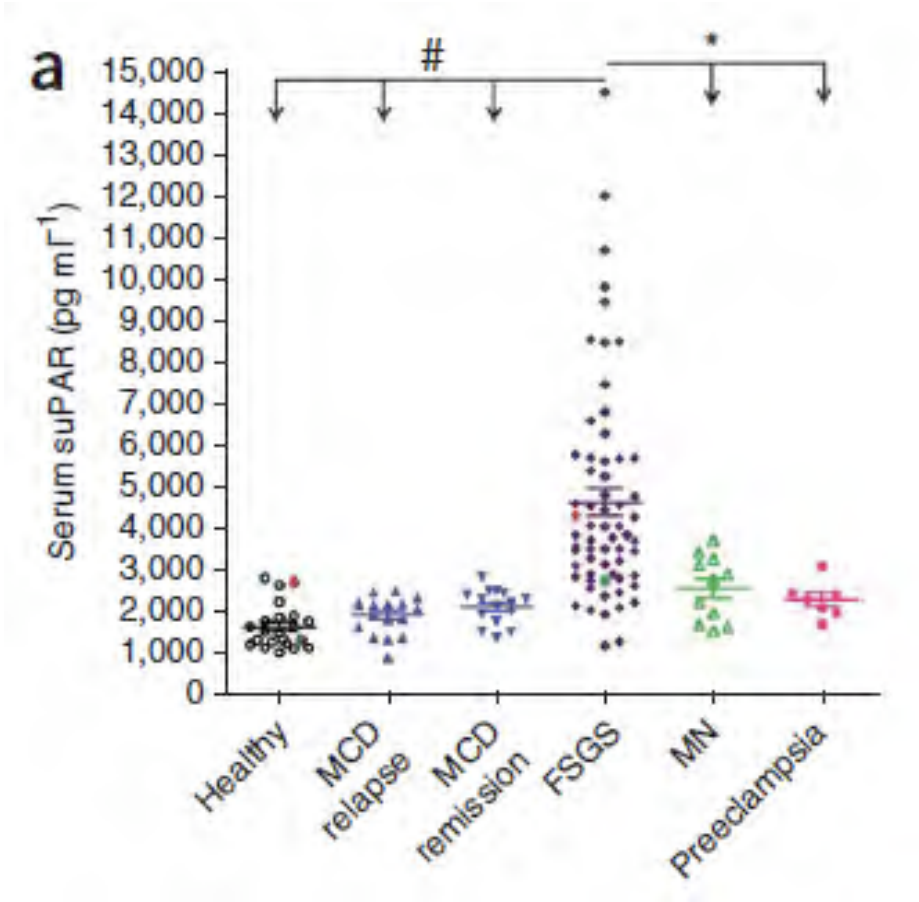
ARTICLES

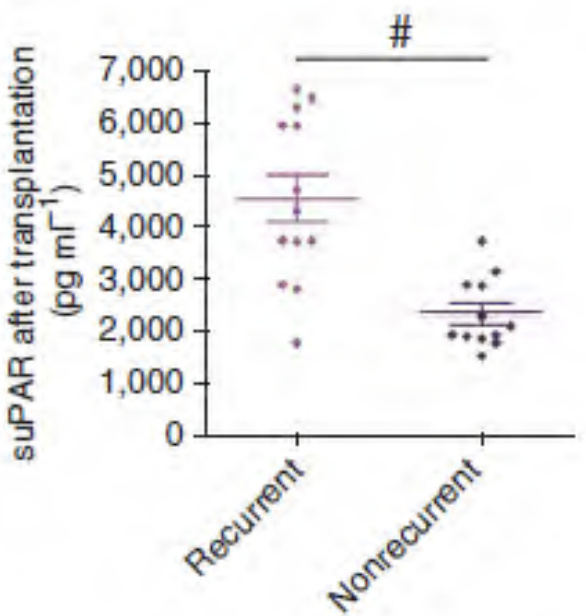
nature medicine

Circulating urokinase receptor as a cause of focal segmental glomerulosclerosis

Changli Wei[1], Shafic El Hindi[1,18], Jing Li[1,18], Alessia Fornoni[1,2,18], Nelson Goes[3], Junichiro Sageshima[4], Dony Maiguel[1], S Ananth Karumanchi[5], Hui-Kim Yap[6], Moin Saleem[7], Qingyin Zhang[8], Boris Nikolic[3], Abanti Chaudhuri[9], Pirouz Daftarian[10,11], Eduardo Salido[12], Armando Torres[12], Moro Salifu[13], Minnie M Sarwal[9], Franz Schaefer[14], Christian Morath[15], Vedat Schwenger[15], Martin Zeier[15], Vineet Gupta[1], David Roth[1], Maria Pia Rastaldi[16], George Burke[4], Phillip Ruiz[4,17] & Jochen Reiser[1]

Wei C et al, Nature Medicine 2011

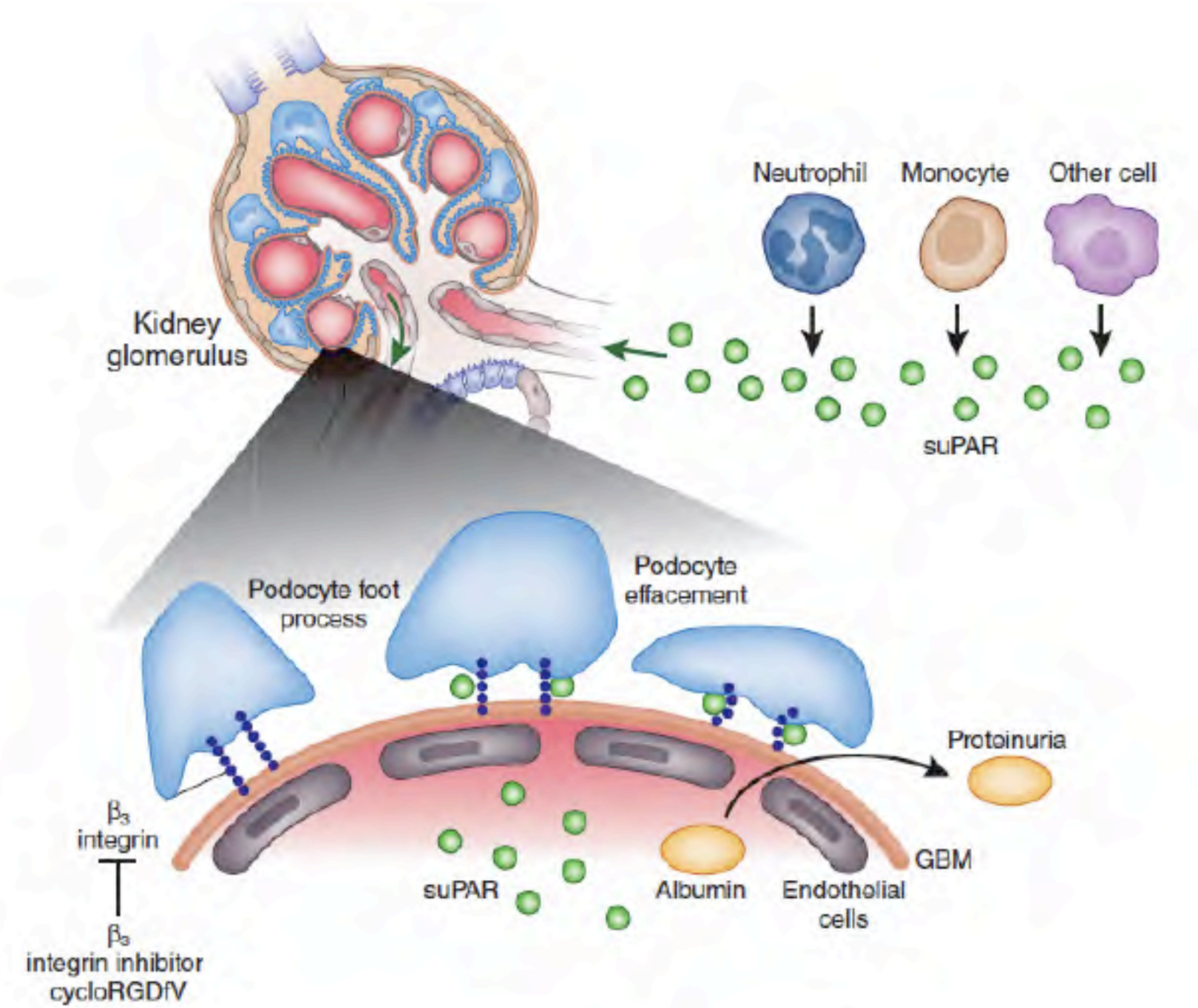




Native kidney

Transplanted kidney

Wei C et al, Nature Medicine 2011



Wei C et al, Nature Medicine 2011

A case of non recurrence!

- **Mrs N. DEL., 5/11/1952,**
- **FSGS diagnosed in 1996 (steroids).**
- **Hemodialysis in 07/05.**
- **Kidney transplantation on 22/05/08 with a 60 year old cadaver kidney donor (diuresis # 1000cc/d, Pu = 14g/l).**
- **At month one: screat = 130mmol/l,Pu = 4g/d,**
- **Salb = 27,9g/l**

A case of non recurrence!

- Mrs N. DEL., 5/11/1952,
- At month 3: screat = 132mmol/l, Pu = 4g/d, Salb = 30g/l
- Screening biopsy = no glomerular lesion.
- At month 6, both ureters were ligatured.
- 2 days later: Pu = 0.15g/d!
- At last FU= screat = 124mmol/l, Pu = 0.15g/d.

Atypical Hemolytic Uremic Syndrome

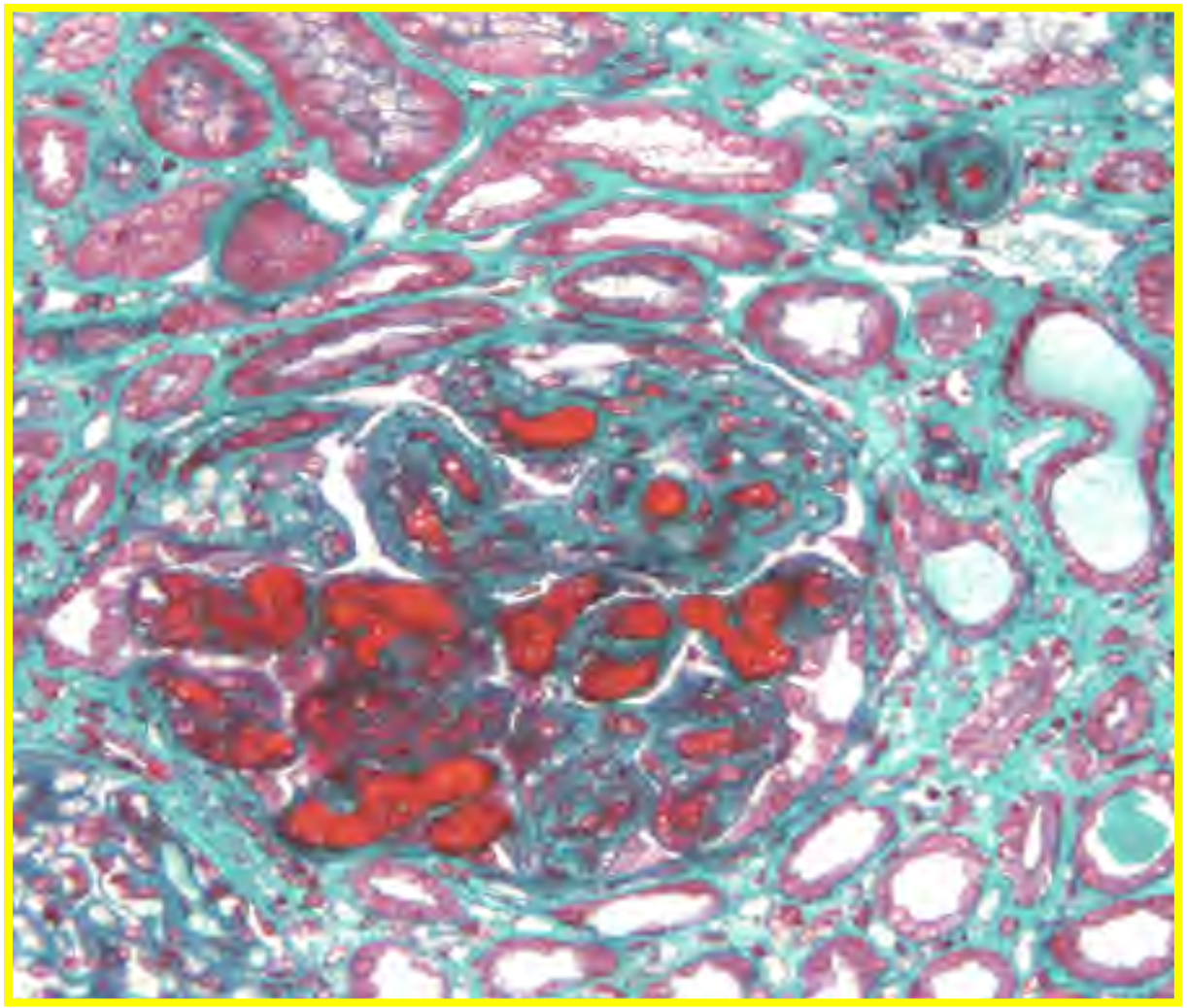


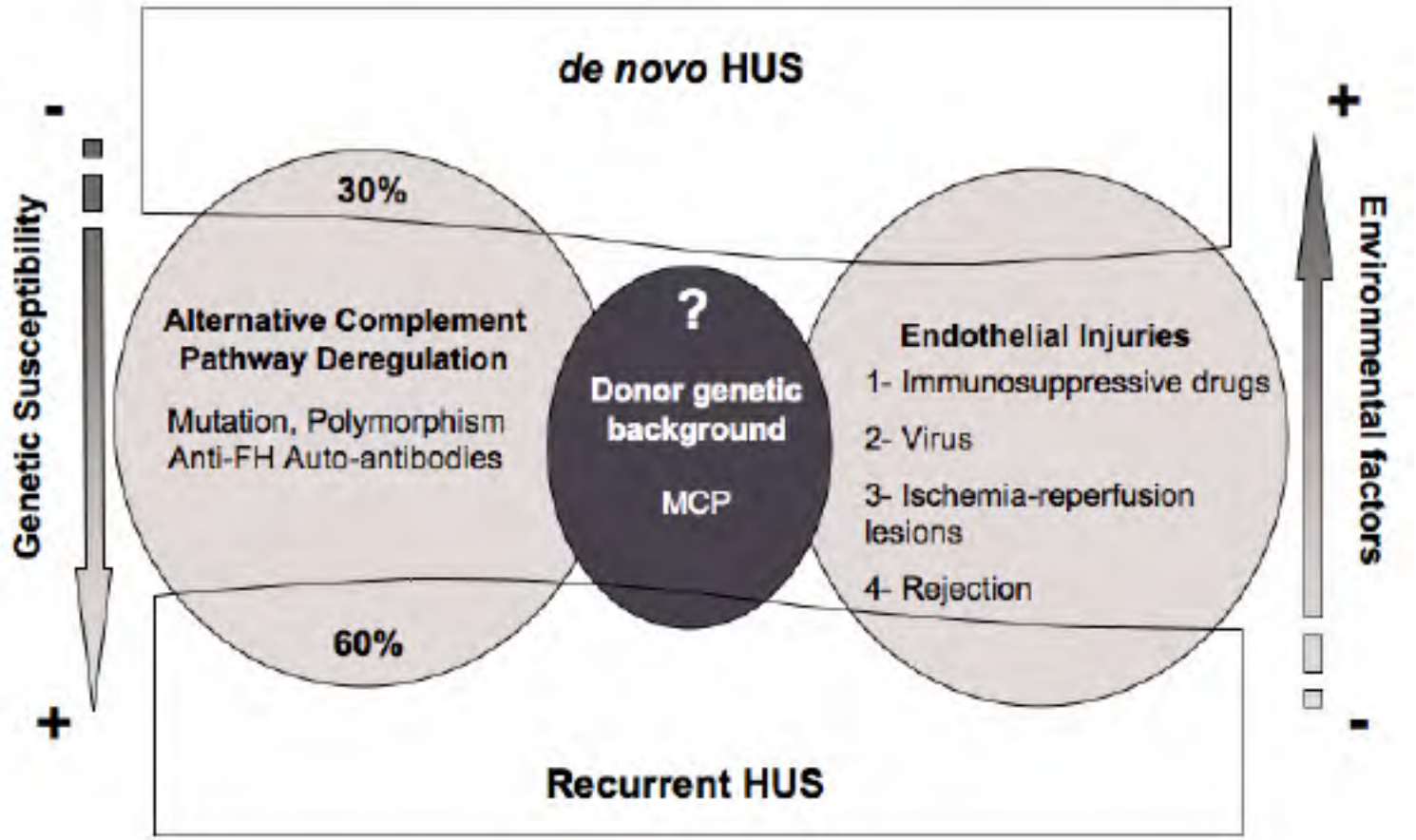
Table 1 | Classification of HUS, TTP, and related disorders

Part 1: etiology advanced	
1.i	*Infection induced* (a) Shiga and verocytotoxin (shiga-like toxin)-producing bacteria; enterohemorrhagic *Escherichia coli*, Shigella dysenteriaen type 1, *Citrobacter* (b) *Streptococcus pneumoniae*, neuraminidase, and T-antigen exposure
1.ii	*Disorders of complement regulation,* (a) Genetic disorders of complement regulation (b) Acquired disorders of complement regulation, for example anti-FH antibody
1.iii	*von Willebrand proteinase, ADAMTS13 deficiency* (a) Genetic disorders of ADAMTS13 (b) Acquired von Willebrand proteinase deficiency; autoimmune, drug induced
1.iv	*Defective cobalamine metabolism*
1.v	*Qinine induced*
Part 2: Clinical associations: etiology unknown	
2.i	HIV
2.ii	Malignancy, cancer chemotherapy and ionizing radiation
2.iii	Calcineurin inhibitors and transplantation
2.iv	Pregnancy, HELLP syndrome and oral contraceptive pill
2.v	Systemic lupus erythematosis and antiphospholipid antibody syndrome
2 vi	Glomerulopathy
2.vii	Familial, not included in part 1
2.viii	Unclassified

Post-diarrheal (D+) HUS

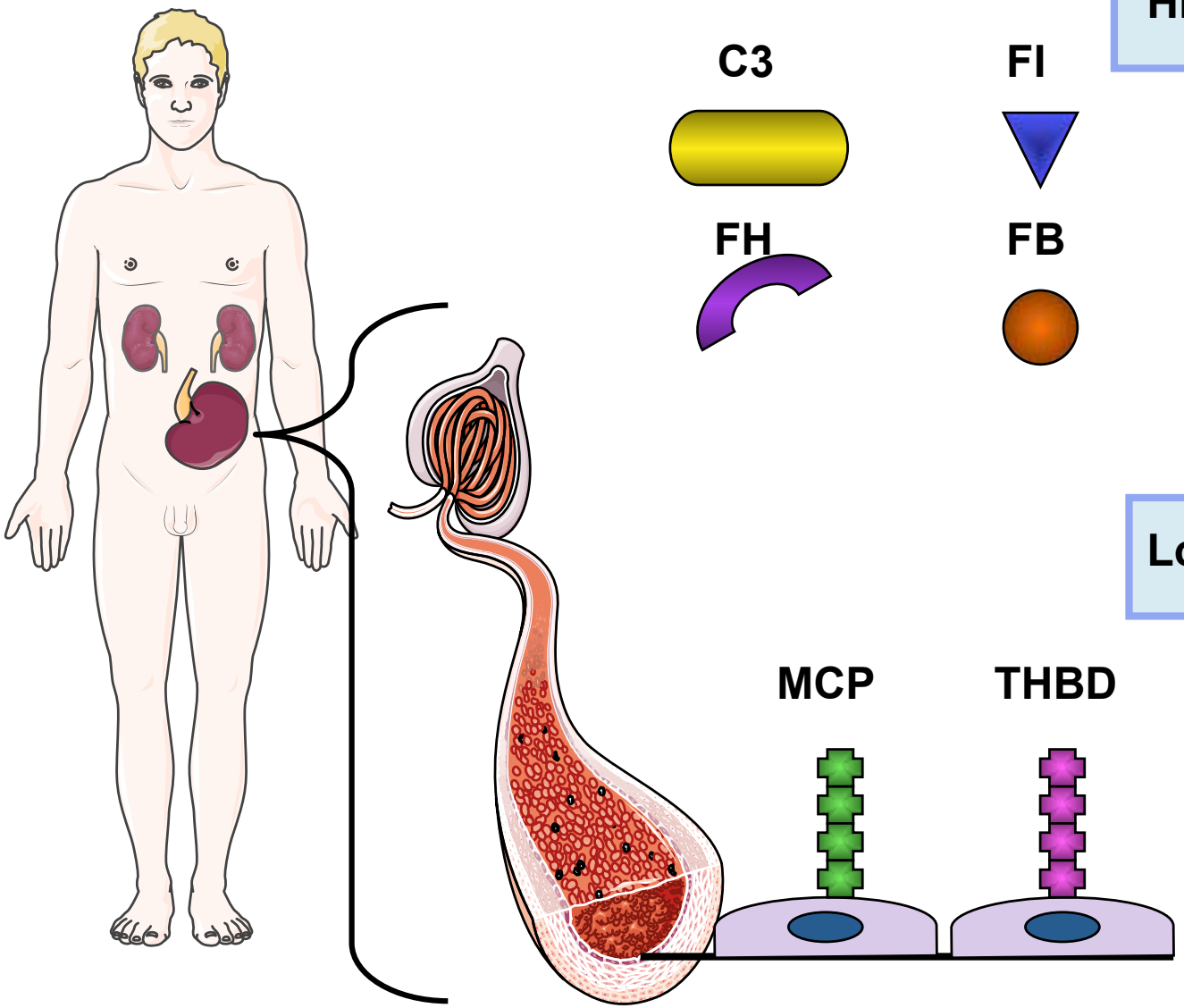
- **No or very rare cases of recurrence (<1%).**
- **<u>But:</u>**
  - **Retrospective classification may be misleading,**
  - **STEC (Shiga toxin E Coli) infection criteria may be negative (15%),**
  - **D+ HUS without diarrhea,**
  - **Gastroenteritis is the triggering event of aHUS in about 28% of cases.**
- **Thus: it is recommended to <u>investigate complement</u> in case of HUS of uncertain etiology.**

Paradigm for post-transplant HUS



Zuber J et al, Nat Rev Nephrol 2011

Membrane versus circulating factor



Zuber J et al, Nat Rev Nephrol 2011

HUS

- **Alternative complement pathway deregulation** is involved in **60% of aHUS** cases and in **30% of *de novo*** post-transplant HUS

- aHUS related to mutations in genes encoding **circulating complement regulators** has a **high recurrence rate** following renal transplantation

- So far, post-transplant recurrence of aHUS has been asociated with an extremely **poor prognosis**

- Innovative therapeutic avenues have emerged, including **preemptive plasma therapy, combined LKT and anti-C5 Ab**, which are extremely promising for the prevention or even the cure of aHUS recurrence

Zuber J et al, Nat Rev Nephrol 2011

aHUS

- **Risk of HUS recurrence was not influenced by:**
  - **pre-transplant bilateral nephrectomy,**
  - **avoidance of calcineurin inhibitors,**
  - **familial or sporadic occurrence of HUS,**
  - **CFH or C3 plasma levels,**
  - **type and position of the mutation in the CFH gene.**

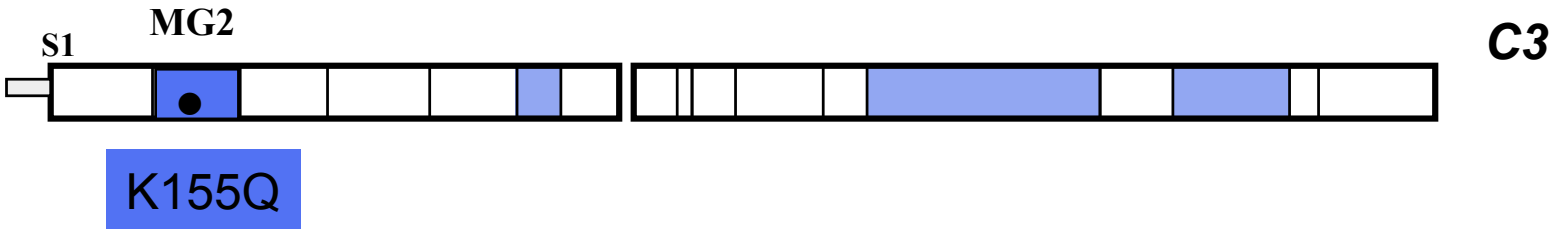
Preemptive Plasma therapy

- Aug 1998: late-onset aHUS in a **42 year-old female**
- Sept 1998: starts hemodialysis

- Mar 2000: **1rst deceased-donor renal transplantation**
  HUS recurrence at 5 months post-transplant
  Intensive plasma therapy (PE and plasma infusions)

- Feb 2004: Return to hemodialysis

- 2006-2007: Identification of a **new mutation in *C3* gene**



Personal data

Preemptive Plasma therapy

Mar 2007: **2nd deceased-donor renal transplantation** (51 year-old)

Renal biopsy	**Normal**			**Normal**	**Normal**	
Creatinine levels (μmol/L)	**75**	**50**	**65**	**68**	**61**	**67**
FPI	**15 ml/kg/dy**			**10 ml/kg/15dy**	**10 ml/kg/30dy**	
PE		**1 EP/7dy**	**1 EP/15dy**			

Personal data

Anti-FH antibodies

Table 2: Characteristics of the anti-FH-associated form of aHUS

Anti-FH autoantibodies lead to an **acquired functional FH deficiency**, in the context of an **alternative pathway activation**. However, normal antigenic C3 and Factor B levels do not exclude the diagnosis.

Frequency has been determined to be between **6% and 10% of aHUS** cases but this may be an underestimation.

Anti-FH IgG-associated aHUS occurs **mainly in children** with onset between 3 and 17 years.

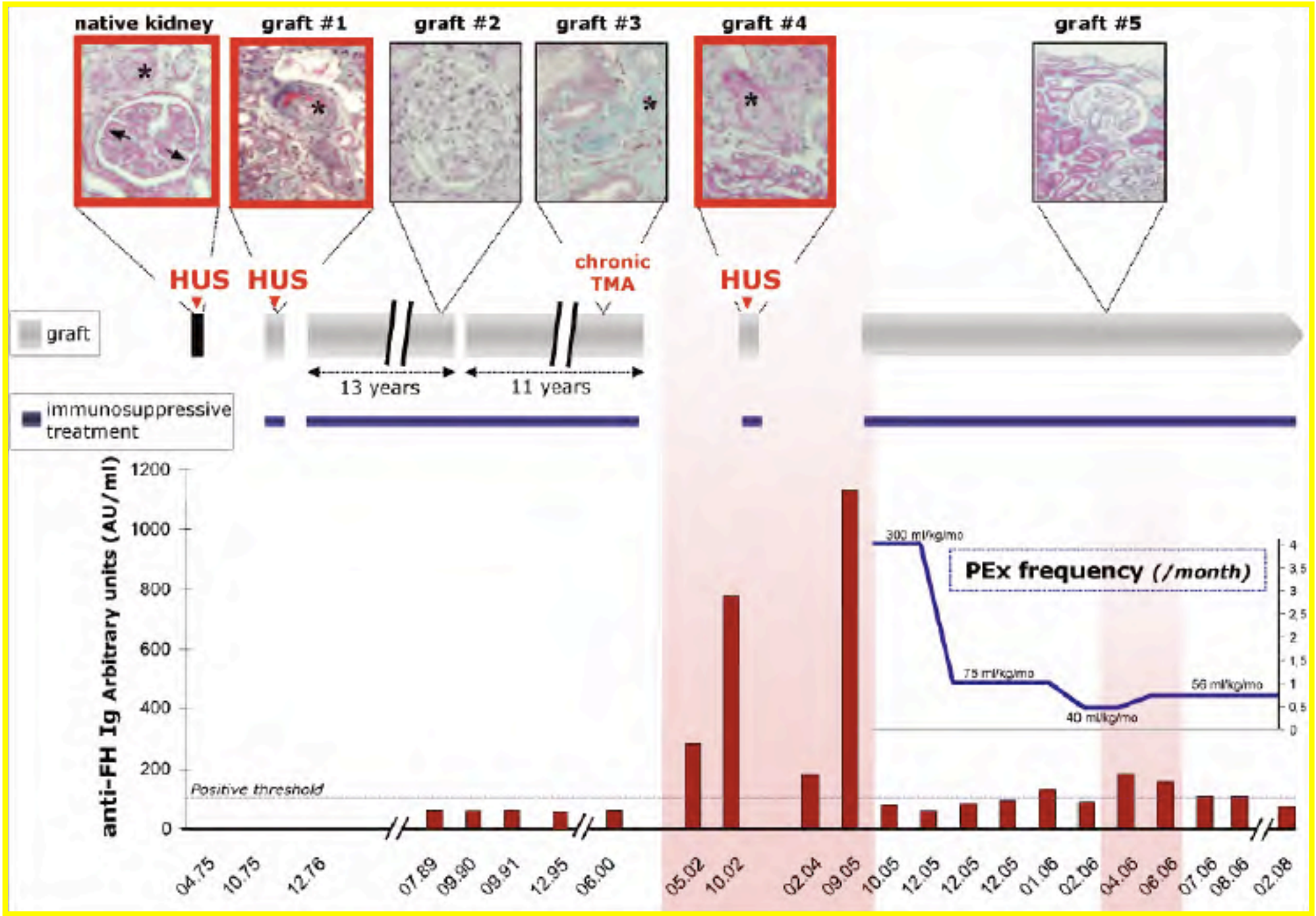
It is particularly prone to **relaps** and can **recur on transplanted graft** and cause **graft loss**.

Anti-CFH IgG represents a **diagnostic marker** and the titer determination is useful for assessing **disease evolution**, because changes precede clinical symptoms, and for **monitoring of treatment.**

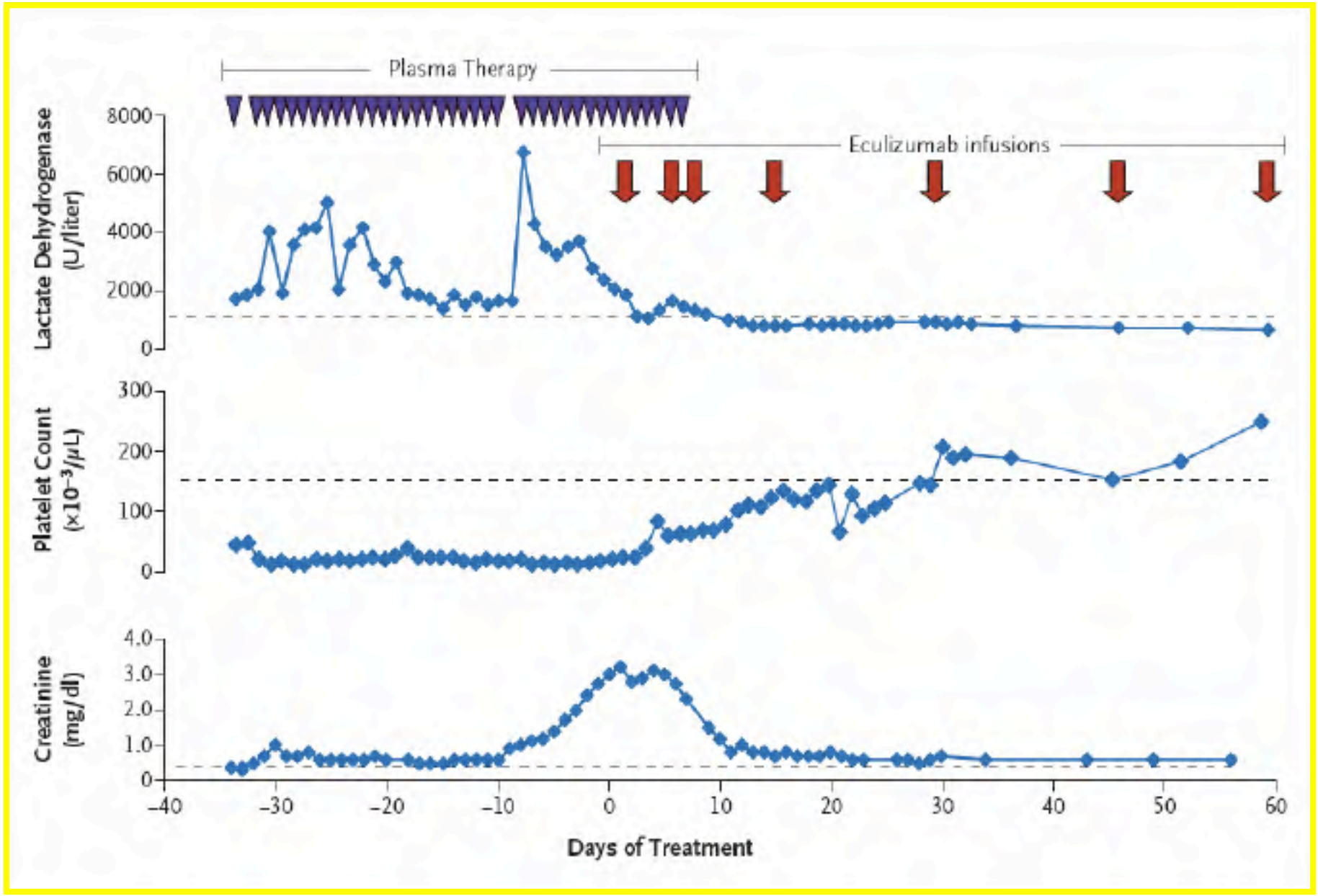
This form of aHUS may benefit from **specific treatments**, such as **plasma exchange therapy** and **immunosuppression,** which can be effective on native as well as grafted kidneys.

Patients who develop these antibodies present a **high frequency of homozygous deletion of two genes related to Factor H**: the Complement Factor H-related genes CFHR1 and CFHR3.

Le Quintrec M et al, Am J Transplant2009



Le Quintrec M et al, Am J Transplant 2009

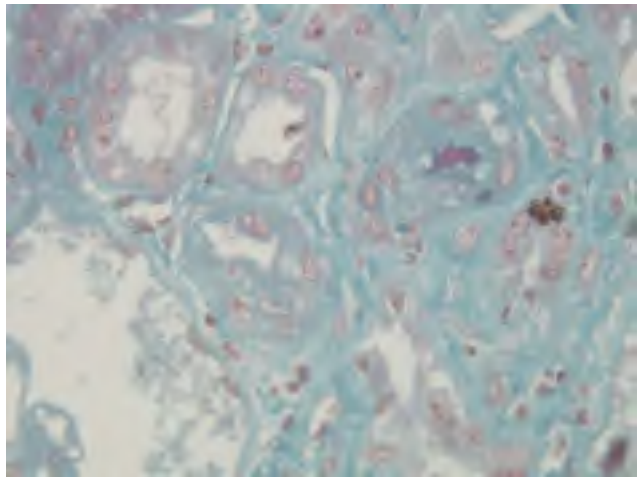


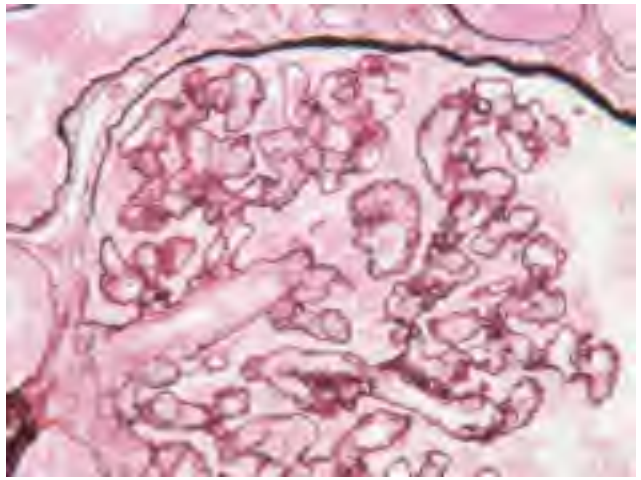
Gruppo RA al, N Engl J Med 2009

Eculizumab

- 1987: early-onset aHUS in a **6 month-old female**
  starts peritoneal dialysis

- 1990: **1rst deceased-donor renal transplantation**
  HUS recurrence within the following days
  Intensive plasma therapy poorly tolerated



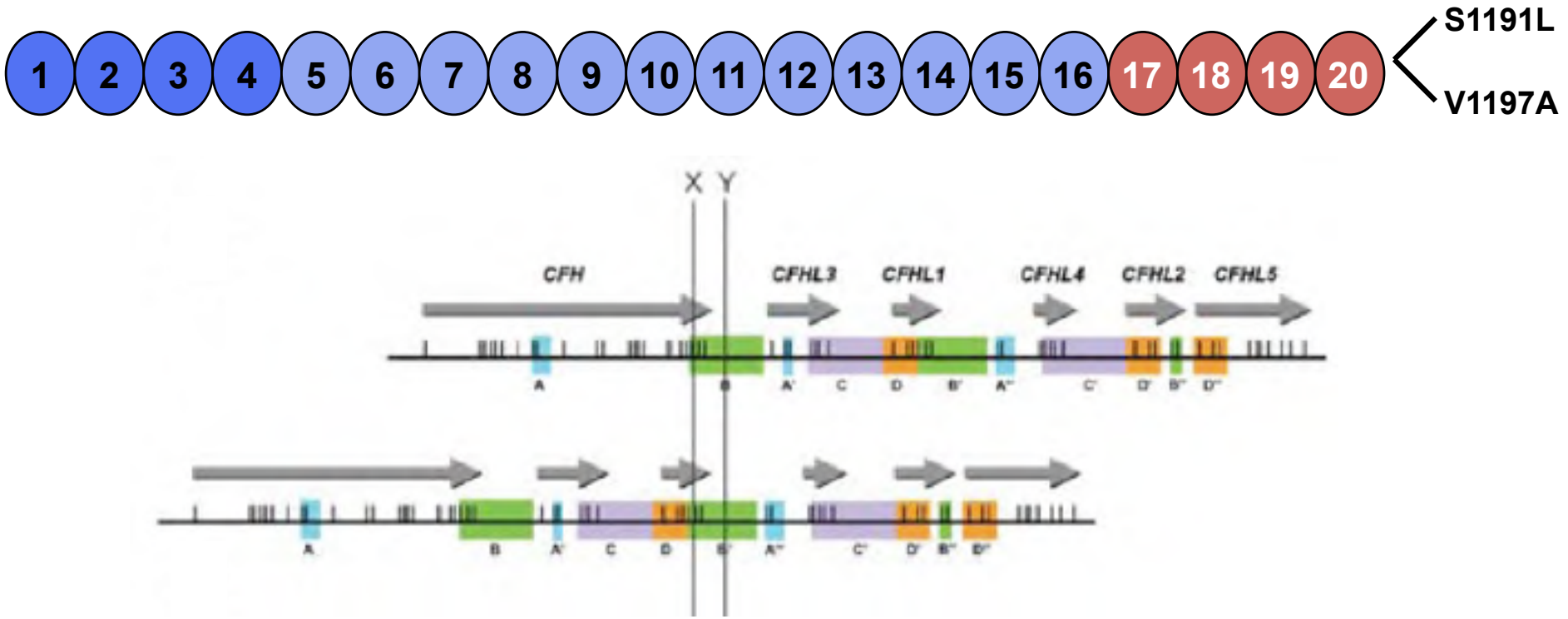


- 1995: Return to hemodialysis

Zuber J et al, Nat Rev Nephrol 2011

Eculizumab

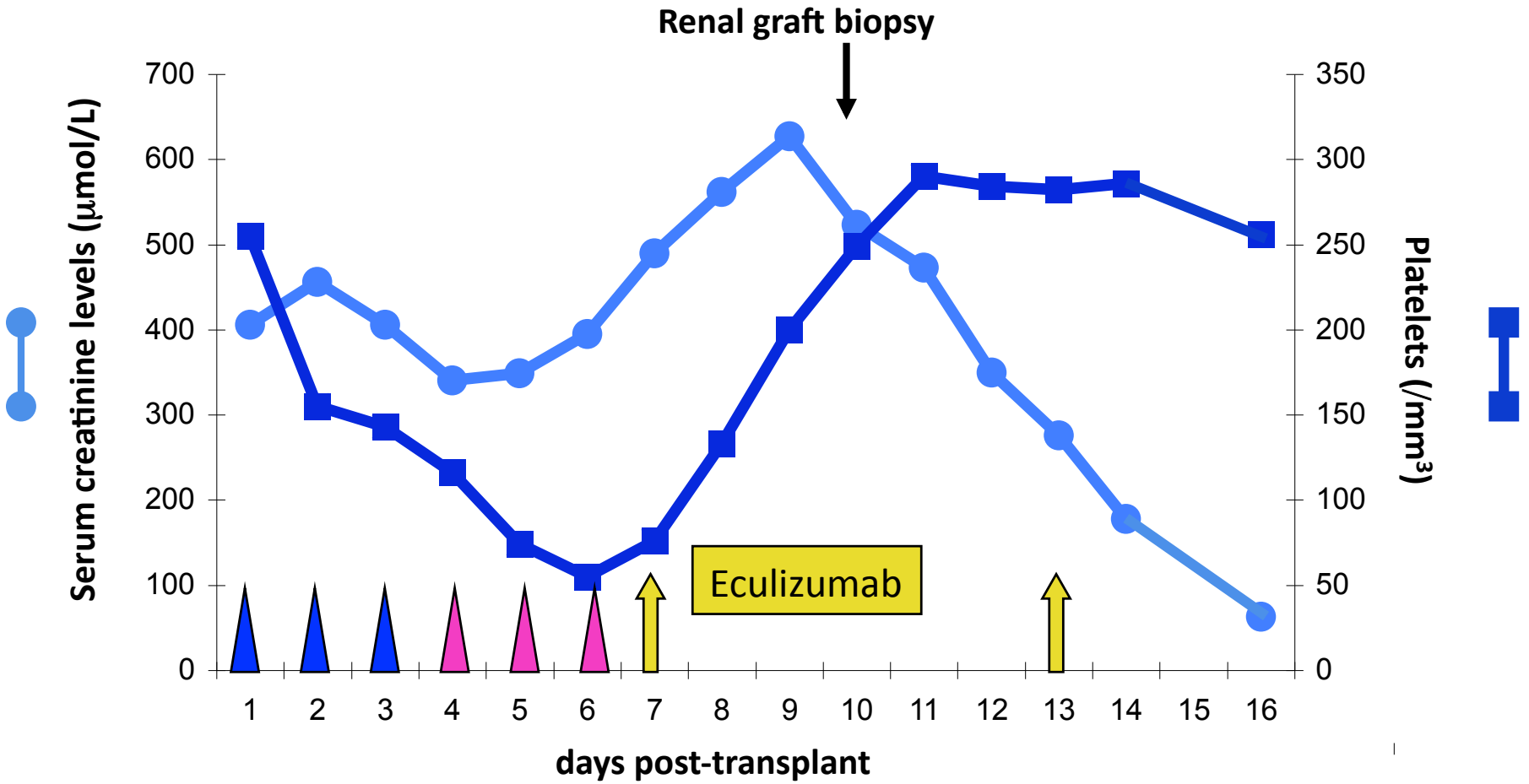
2006: Identification of **two heterozygous mutations in *CFH***



Zuber J et al, Nat Rev Nephrol 2011

HUS

Eculizumab

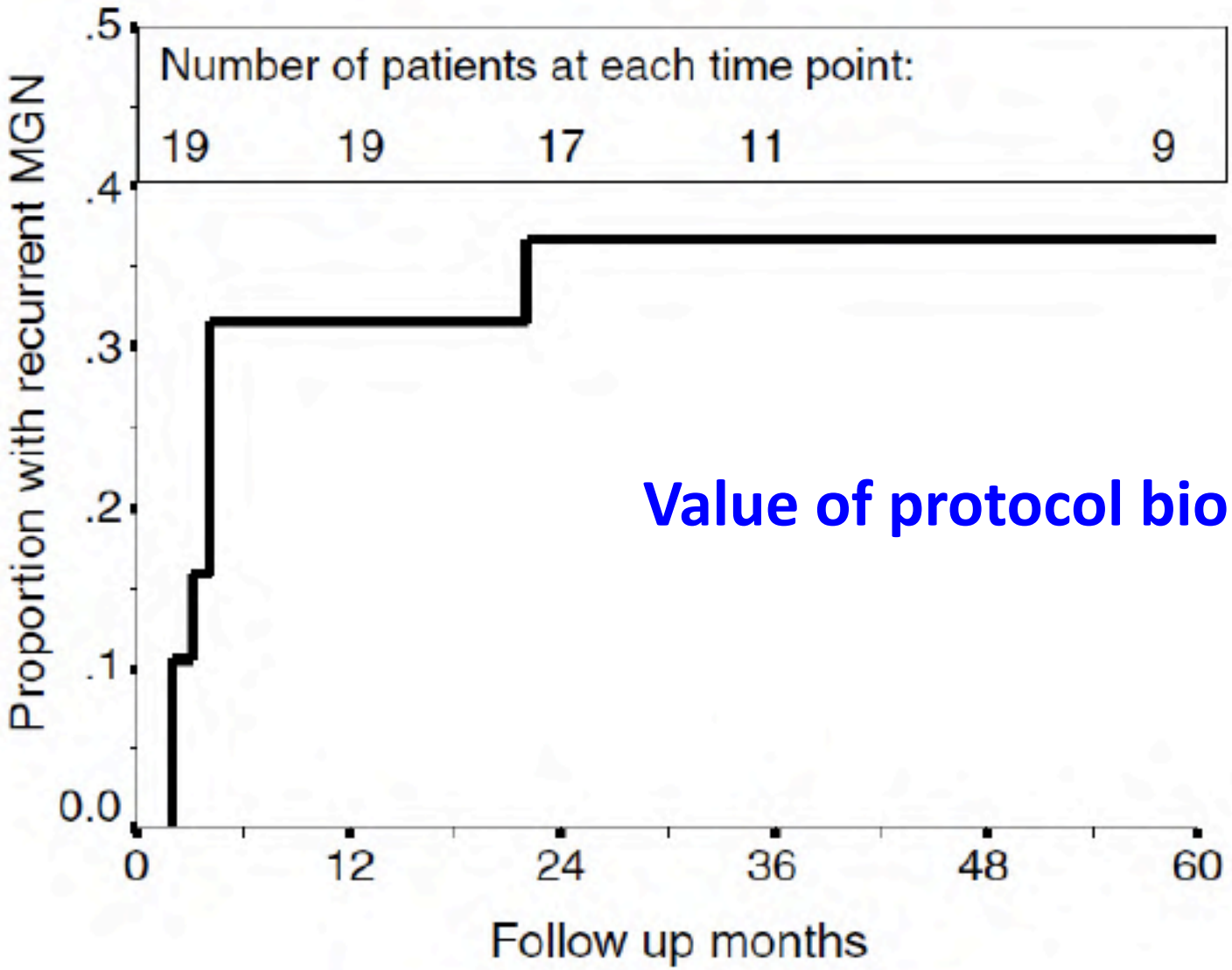


Chatelet V et al, Am J transplant 2009

Fernandes-de Larrea C et al, Transplantation 2010

PI PE

Membranous nephropathy



Value of protocol biopsies

Dabade TS et al, Am J Transplant 2008

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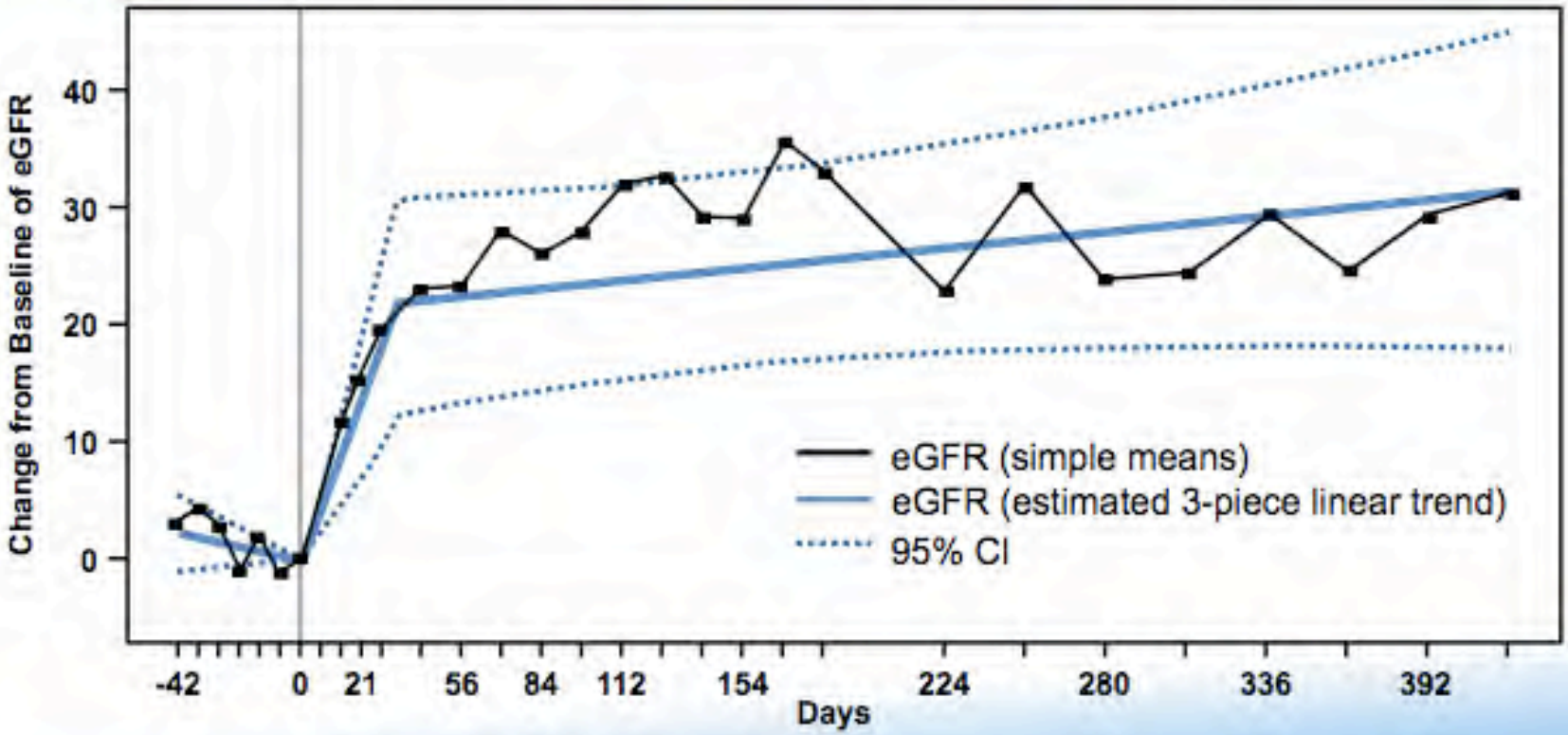
C08-002

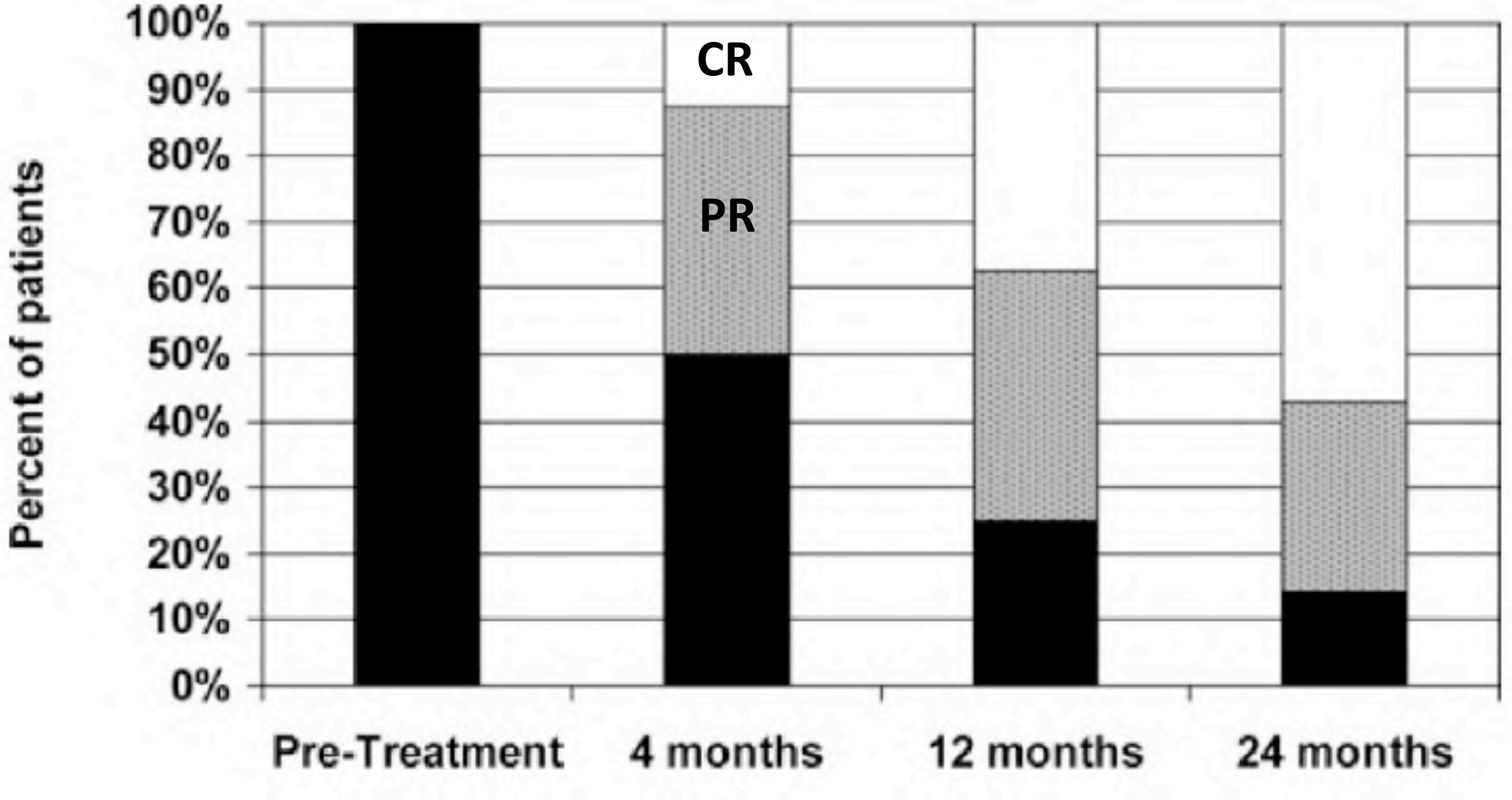
Chronic Eculizumab Treatment Significantly Improved eGFR

- Sustained eculizumab treatment showed a statistically significant, time-dependent increase in eGFR
  - 31 mL/min/1.73m^2 (95%CI, 17–45) through week 26 (p=0.0001)
  - 31 mL/min/1.73m^2 (95%CI, 14–44) through median duration 64 weeks (p=0.0003)



Mean eGFR increased from 22.8(13) mL/min/1.73m^2 at baseline to 60.4(22) mL/min/1.73m^2 by day 420

Membranous nephropathy: tt anti-CD20



EL-Zoghby ZM et al, Am J Transplant 2009

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The NEW ENGLAND JOURNAL of MEDICINE

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M-Type Phospholipase A_2 Receptor as Target Antigen in Idiopathic Membranous Nephropathy

Laurence H. Beck, Jr., M.D., Ph.D., Ramon G.B. Bonegio, M.D., Gérard Lambeau, Ph.D., David M. Beck, B.A., David W. Powell, Ph.D., Timothy D. Cummins, M.S., Jon B. Klein, M.D., Ph.D., and David J. Salant, M.D.

Human Idiopathic Membranous Nephropathy — A Mystery Solved?

Richard J. Glassock, M.D.

Beck L et al, N Engl J Med 2009

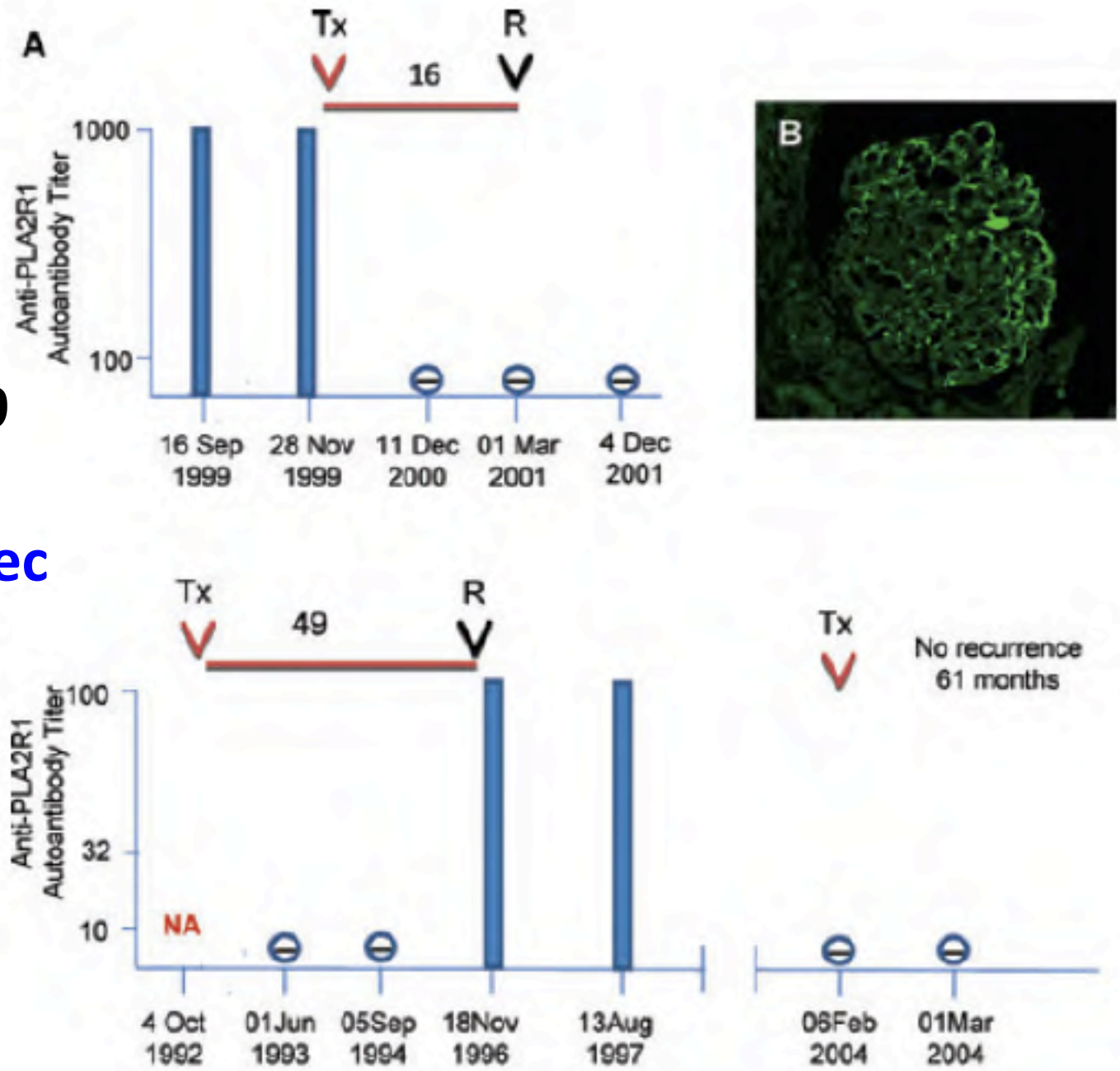
MN

Rec MN n = 10

De novo MN n = 9

PLA2R1 in 5/10 Rec

None in de novo



Debiec H et al, Am J Transplant 2011

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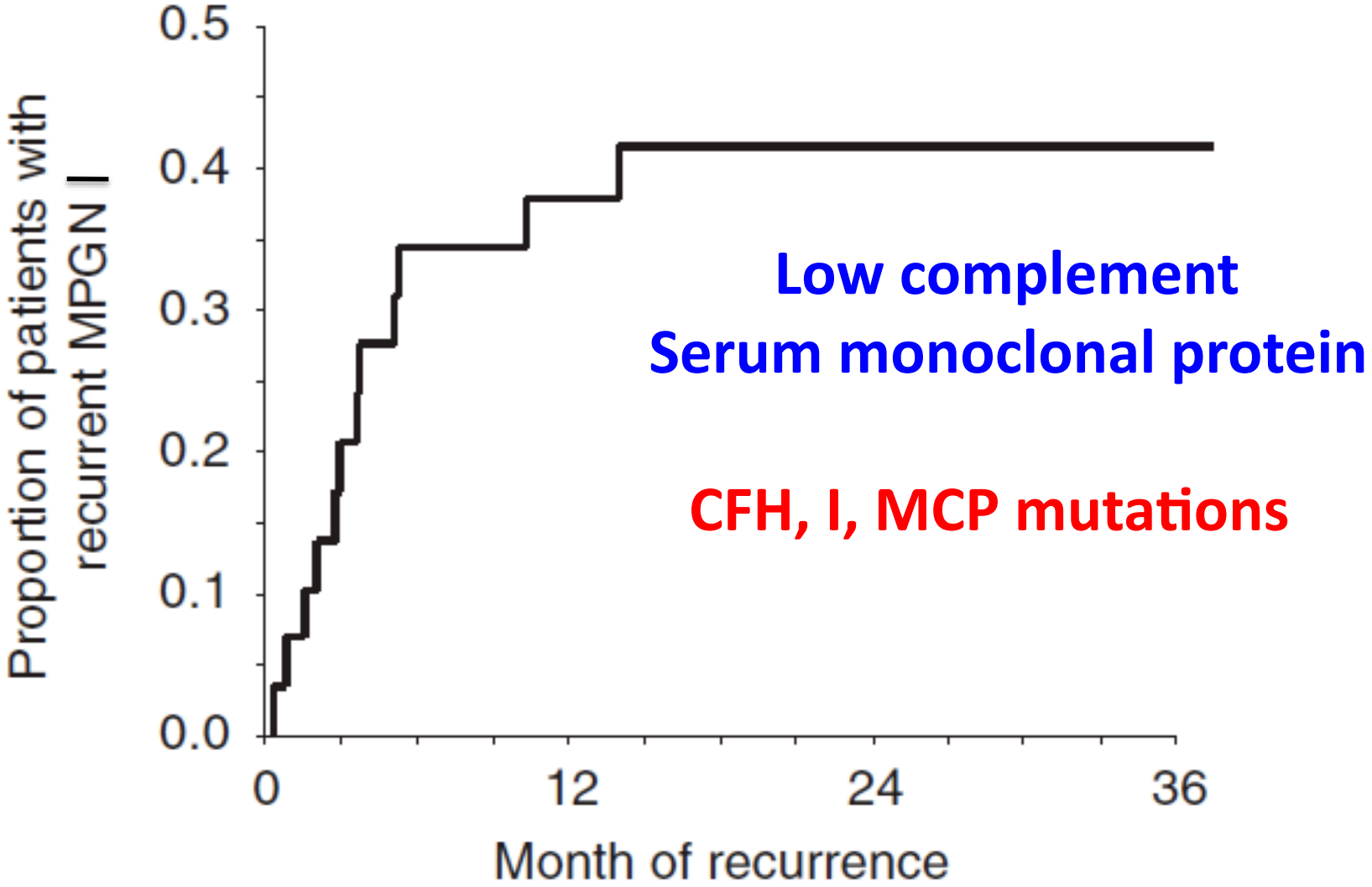
Membrano-proliferative glomerulopathy



Low complement

Serum monoclonal protein

CFH, I, MCP mutations

Lorenz EC et al, Kidney Int 2010

MPGN

VIEWPOINT

www.nature.com/clinicalpractice/neph

Recurrence of lupus nephritis after renal transplantation: if we look for it, will we find it?

Francis Weng and Simin Goral*

Lupus recurrence post-RT

- ***Recurrence of lupus nephritis is widely regarded as rare (1-4%),***
- ***But several studies report incidences between 10 and 43%! Why?***
- ***Several reasons:***
  - ***small number of patients in most studies,***
  - ***recurrence may be late, so follow-up must be too,***
  - ***definition of recurrence varies (recurrence of lupus is different from graft loss due to lupus recurrence),***
  - ***definitive diagnosis requires a biopsy with IF ± EM,***
  - ***Incidence is higher in studies with screening biopsies.***

Weng F et al, Nat Clin Pract Nephrol 2005

Lupus recurrence post-RT

- **Recurrence of lupus nephritis is usually not severe:**
  - **no or few class IV.**
- **Very few kidneys are lost due to lupus recurrence.**

Weng F et al, Nat Clin Pract Nephrol 2005

Conclusions

- Post-transplant disease recurrence is a significant cause of graft loss, specially in younger recipients.
- Modern immunosuppression has not changed these figures in the last decade.
- However, there are very promising therapeutical perspectives:
  - Combined IV-CSA/high dose steroids/PE = FSGS
  - Eculizumab = aHUS
  - Anti-CD20 = MN.


1952 - 2012
Merci de votre attention!