Otoantikorlar Transplantasyon İçin Önemli mi?

Doç.Dr. Sevim GÖNEN

Gazi Üniversitesi Tıp Fakültesi Doku Tipleme Laboratuvarı

OTOİMMÜNİTE

Öz antijenlere immünolojik yanıt…

- Genetik

- MHC/MHC dışı genler

- Protein değişiklikleri

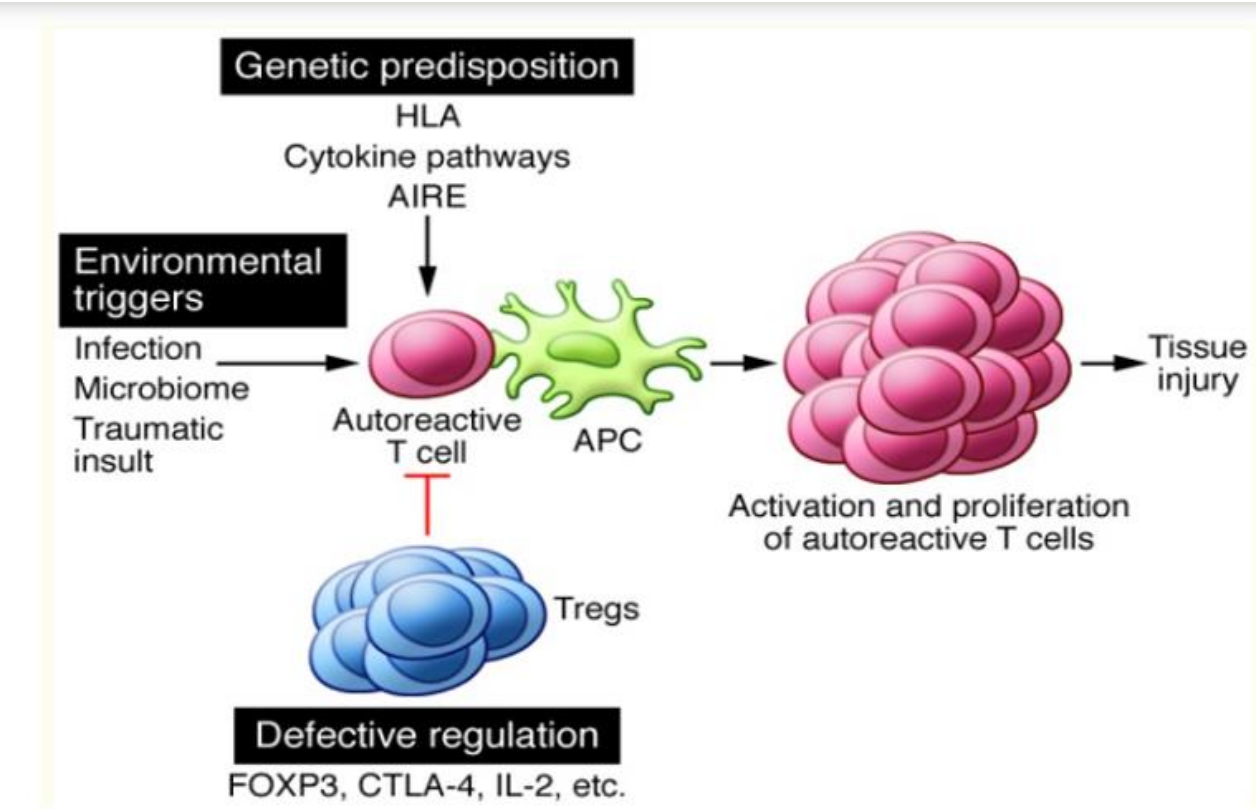
- Korunmuş proteinler

- Sitokinler

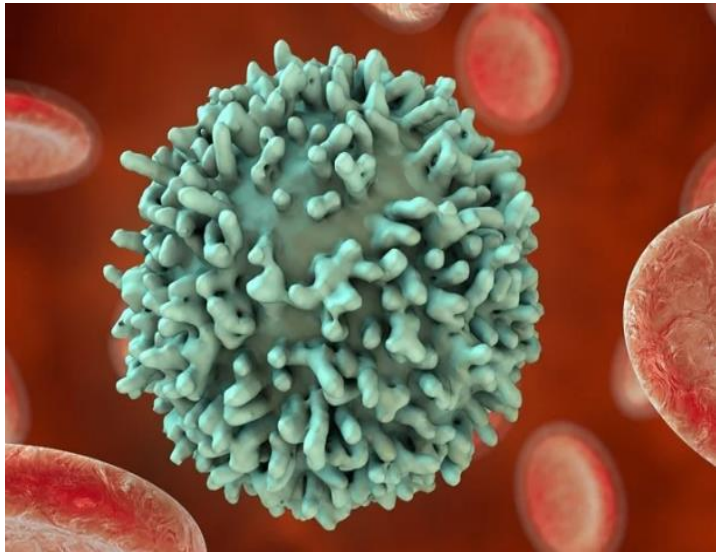
- Doğal otoantikorların rolü

- Çevresel etkenler

- Düzenleyici mekanizmalarda bozulma



OTOİMMUNİTENİN GELİŞME MEKANİZMASI



1. Anormal antijenik uyarma :

a) Çapraz reaksiyon,

b) Otoantijenlerin değişikliğe uğraması,

c) Gizli kalmış doku antijenlerinin açığa çıkması,

d) Otoantijen miktarının artması,

e) Ontojenik olarak geç gelişen doku antijenleri,

f) Adjuvantlar,

g) Greftin konağı reddi reaksiyonu .

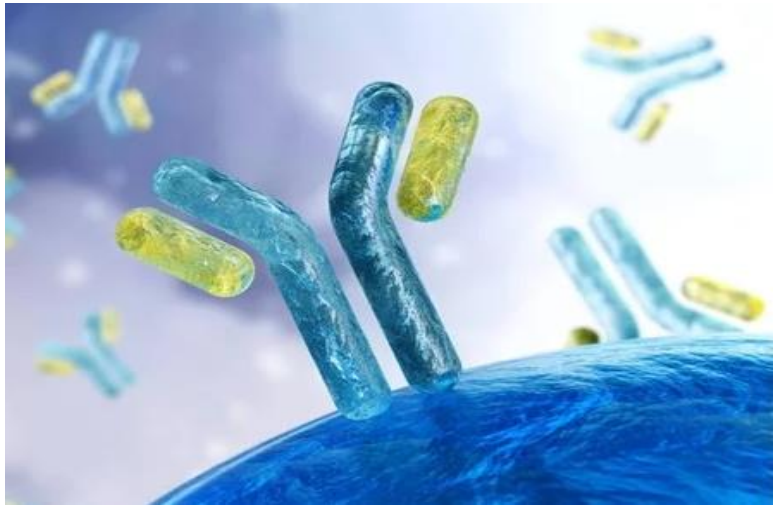
2. Anormal bağışık yanıt :

a) Otoantikor yapan hücre klonlarının mutasyonu,

b) Supresör T hücre yetersizliği veya yokluğu.

TANISAL BİYOBELİRTEÇ

- 1- Organa özgül otoantikorlar,
- 2- Organa özgül olmayan otoantikorlar.



TISSUE ANTIGENS
IMMUNE RESPONSE GENETICS

Tissue Antigens ISSN 0001-2815

REVIEW ARTICLE

Detection of anti-HLA antibodies by solid-phase assay in kidney transplantation: friend or foe?

S. Ferrari-Lacraz, J.-M. Tiercy & J. Villard

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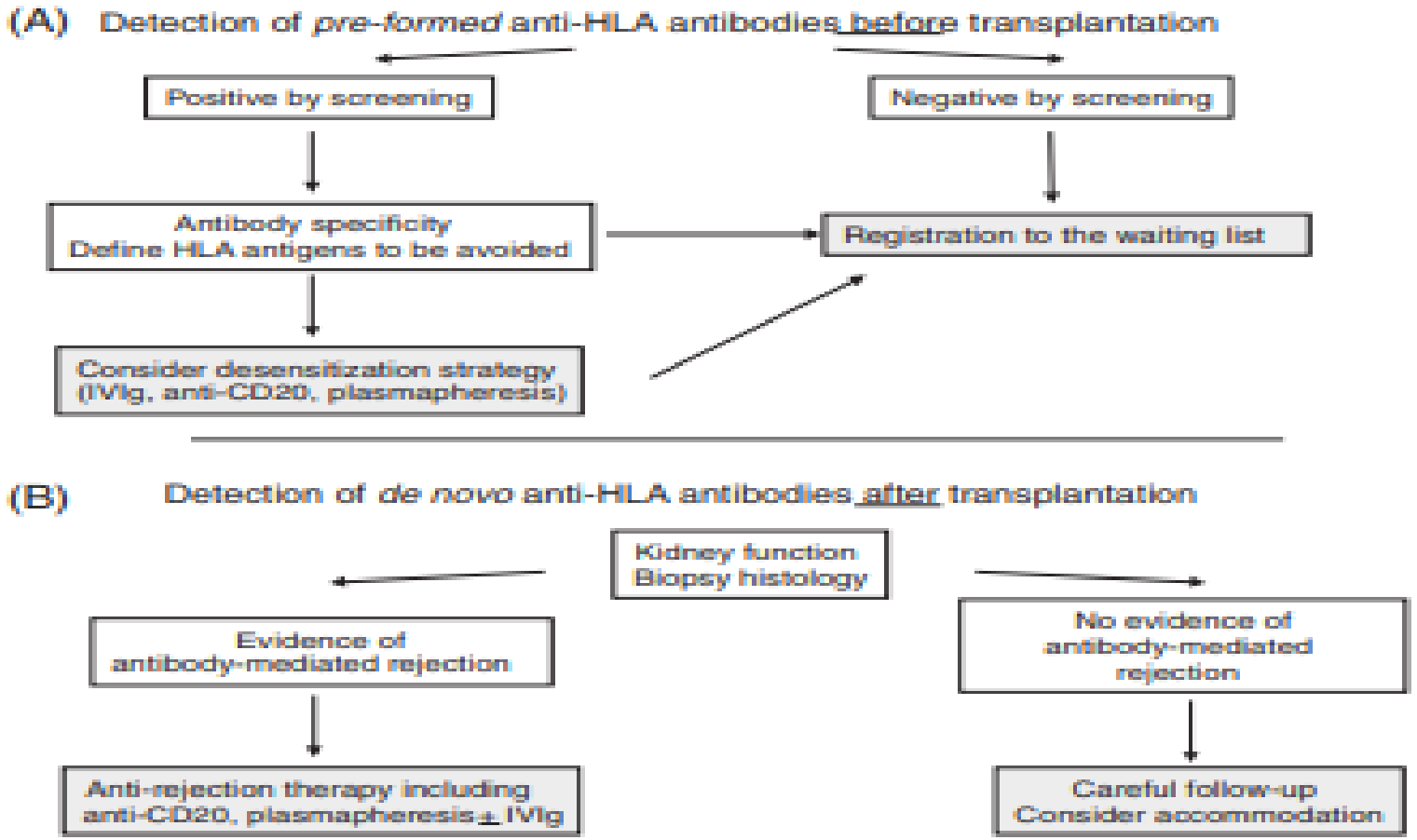

SPECIAL FEATURES

Consensus Guidelines on the Testing and Clinical Management Issues Associated With HLA and Non-HLA Antibodies in Transplantation

Tait, Brian D.[1]; Süsal, Caner[2]; Gebel, Howard M.[3]; Nickerson, Peter W.[4]; Zachary, Andrea A.[5]; Claas, Frans H.J.[6]; Reed, Elaine F.[7]; Bray, Robert A.[3]; Campbell, Patricia[8]; Chapman, Jeremy R.[9]; Coates, P. Toby[10]; Colvin, Robert B.[11]; Cozzi, Emanuele[12]; Doxiadis, Ilias I.N.[6]; Fuggle, Susan V.[13]; Gill, John[14]; Glotz, Denis[15]; Lachmann, Nils[16]; Mohanakumar, Thalachallour[17]; Suciu-Foca, Nicole[18]; Sumitran-Holgersson, Suchitra[19]; Tanabe, Kazunari[20]; Taylor, Craig J.[21]; Tyan, Dolly B.[22]; Webster, Angela[9]; Zeevi, Adriana[23]; Opelz, Gerhard[2,24]

Author Information

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Published in final edited form as:
Nat Rev Nephrol. 2016 August ; 12(8): 484–495. doi:10.1038/nrneph.2016.88.

The importance of non-HLA antibodies in transplantation

Qiuheng Zhang and **Elaine F. Reed**
UCLA Immunogenetics Center, Department of Pathology and Laboratory Medicine, David Geffen School of Medicine, University of California Los Angeles, 1000 Veteran Avenue, Los Angeles, CA 90095, USA

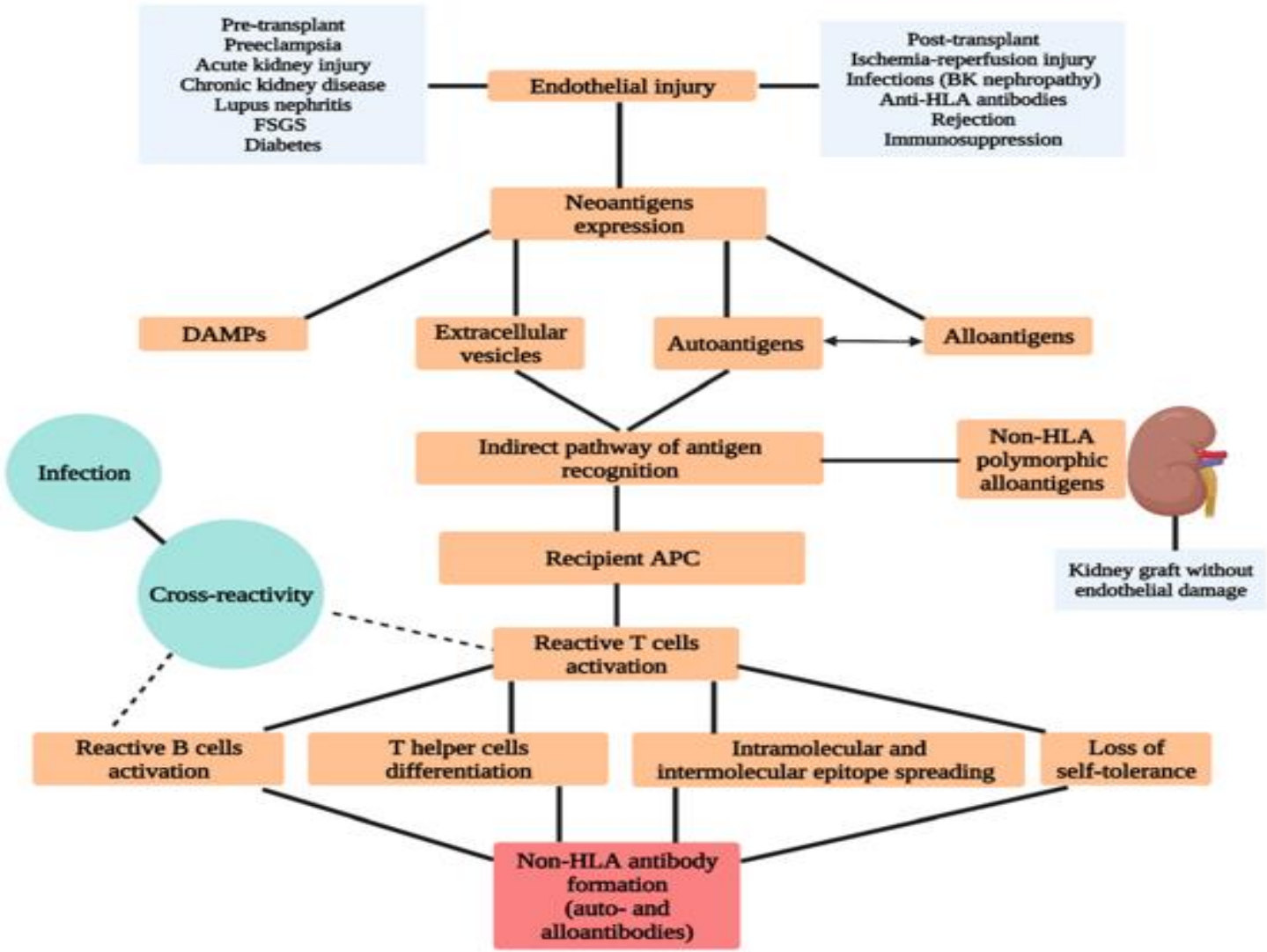
AT1R antibodies in human organ transplantation

Cohort	Time course	Key findings	Synergy with HLA-DSA	Ref.
Kidney recipients				
33 patients with steroid-refractory rejection: 16 with AT1R Ab$^+$, HLA-DSA$^-$ 13 with AT1R Ab$^-$, HLA-DSA$^+$	Post-TX	Biopsy samples from 11 of 16 AT1R Ab$^+$ patients did not show C4d deposition AT1R+, HLA DSA- patients had much rapid allograft loss as compared to AT1R-, HLA DSA+ recipients Treatment with plasmapheresis, IVIG and losartan improved allograft survival in AT1R ab+ recipients	NA	– 33
63 patients with no HLA-DSA or MICA-DSA, including 16 patients with AR (7 AMR, 9 ACR)	Pre-TX & post-TX	AT1R Ab associated with increased AMR but not ACR 6 of 7 patients with AMR did not have C4d deposition	NA	– 30
134 patients with abnormal biopsies[a] 217 control patients	Pre-TX & post-TX	AT1R Ab associated with abnormal biopsy samples *De novo* AT1R Ab associated with increased graft failure as compared to recipients without AT1R and HLA DSA	Yes	– 44
283 AT1R Ab+ patients 316 AT1R Ab- patients	Pre-TX	Pre-formed AT1R Ab associated with increased AR within 4 months and increased graft failure >3-year post-TX	NA	40
7 AT1R Ab+ patients 72 AT1R Ab− patients	Pre-TX	Pre-formed AT1R Ab associated with increased AMR but not ACR	NA	41
7 AT1R Ab+ patients 58 AT1R Ab− patients	Post-TX	Post-Tx AT1R Ab associated with Increased graft injury and graft failure	NA	– 46
11 patients with AR and no HLA-DSA	Pre-TX & post-TX	10 of 11 patients had pre-TX AT1R Ab 10 of 11 patients had no C4d deposition	NA	– 29
12 patients with AMR and no HLA-DSA	Pre-TX & post-TX	9 of 10 patients had AT1R Ab$^+$ sera before transplantation 10 of 12 patients were AT1R Ab$^+$ at the time of biopsy AT1R Ab increased the risk of AMR	NA	– 43
98 AT1R Ab+ patients 64 AT1R Ab− patients	Pre-TX	Pre-Tx AT1R Ab associated with increased AR	NA	– 42
Paediatric recipients: 10 autoAb$^+$ 10 autoAb$^-$	Pre & post Tx	50% of patients developed *de novo* Abs to AT1R, FN, or collagen IV in the first year after TX; development of these Ab did not correlate with graft function	NA	45
Heart recipients				
14 AT1R Ab+ patients 16 AT1R Ab− patients	Pre-TX Post-TX	Pre and post-transplant AT1R ab is associated with increased AMR, ACR and microvasculopathy	NA	55
76 AT1R Ab+ patients 124 AT1R Ab− patients	Pre-TX & post-TX	Pre-Tx and post-Tx AT1R Ab did not correlate with AMR, CMR and CAV AMR and CMR increased with HLA-DSA and AT1R	Yes	– 56

HLA dışı antikorların transplantasyondaki önemi

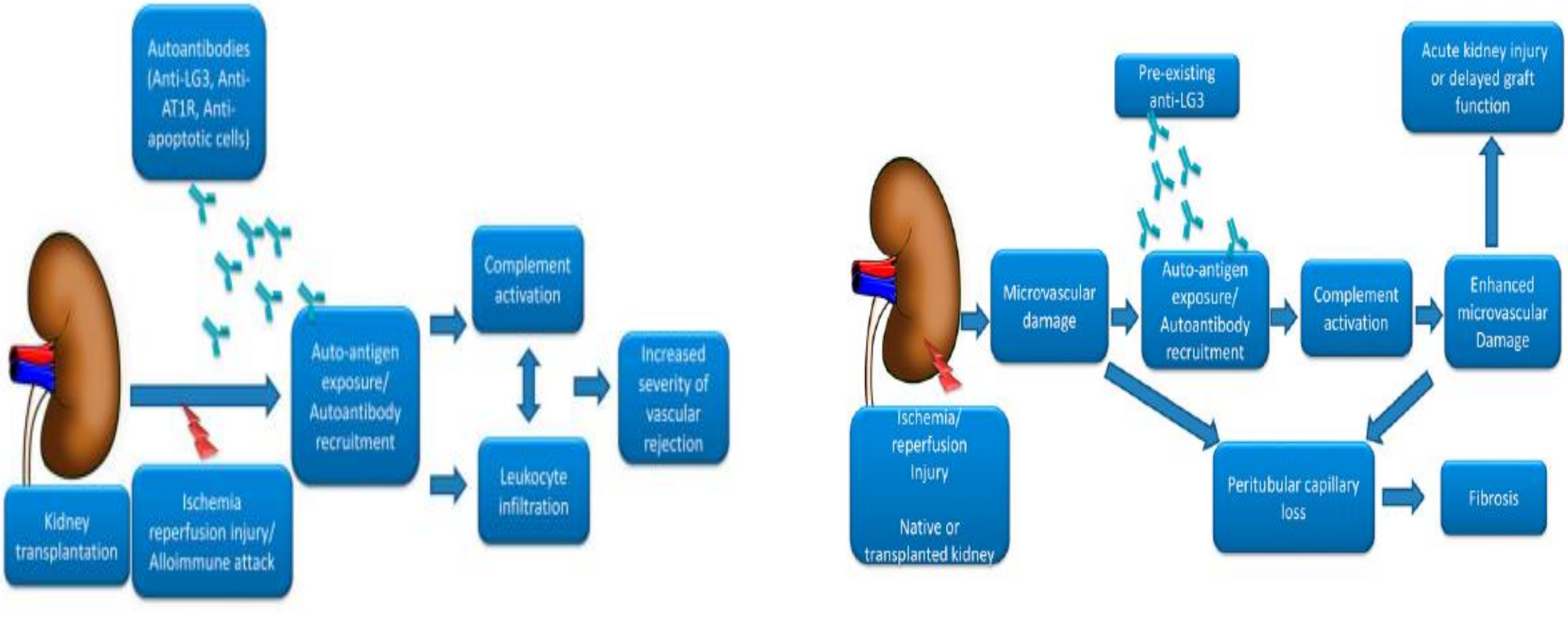
- Kompleman bağımlı/ komplemandan bağımsız yollar…
- Birçok mekanizma yoluyla üretim…
- IL-17 / TH_{17} üretimde merkezi bir rol...
- Daha fazla araştırmaya ihtiyaç…

HLA dışı antikorların oluşum mekanizmaları



The Emerging Importance of Non-HLA Autoantibodies in Kidney Transplant Complications.

Cardinal ve ark. Journal of the American Society of Nephrology 28(2):p 400-406, February 2017.



Non-HLA Abs in Solid Organ Transplantation.
María Gutiérrez-Larrañaga ve ark. Transplantology 2020, 1(1), 24-41

Table 2. Characteristics of non-HLA antibodies in organ transplants.

Antibody		Solid Organ Transplant			Detection Technique	Cellular Location	Proposed Mechanism
		Renal	Lung	Heart			
MICA	Pre	unclear association with graft survival and ABMR	-	unclear	Luminex	Ligand in NKG2D receptor	Complement-dependent cytotoxicity? [100]
	Post	unclear association with graft survival and ABMR	BOS, synergistic with HLA	ABMR synergistic with HLA, CAV			
AT1R	Pre	Acute rejection	Graft failure	Not associated with rejection	Commercially available ELISA	GPCR in endothelia, promoted by ischemic injury	Endothelial damage, vasoconstriction [44]
	Post	ABMR and decreased graft survival	Possible ABMR and CMR	ABMR and CMR			
Perlecan	Pre	AVR	-	-	Home made ELISA (also available for Luminex)	Vessel wall component acts as neoantigen after caspase-dependent cleavage	CD3, NK cell infiltrate and C4d deposition [67]
	Post	ABMR, impaired graft function	-	-			
Tubulin	Pre	-	BOS, PGD	-	Home made ELISA	Gap junctions, cytoskeletal protein	Complement activation, also HIF-1a and pro-fibrotic growth factors [101]
	Post	-	ABMR driven by donor HLA, BOS	CAV, ABMR			
Collagen	Pre	-	BOS, PGD	-	Home made ELISA	Connective tissues	In lung, Ab to collagen type V result in CD4 infiltrate and IL-17 production, resulting in BOS [102]
	Post	TG	ABMR driven by donor HLA, BOS	CAV, ABMR			
Vimentin	Pre	IFTA (IgG isotype)	-	Not associated with rejection	ELISA and/or LUMINEX	Cytoskeletal, intermediate filament	CD3, CD4, cell infiltrate and C3d deposition [103]
	Post	TG (IgG isotype), rejection	-	ABMR and CAV driven by donor HLA			

Abbreviations: MICA: MHC1-related chain A gene. NKG2D: Natural Killer Group 2 D. HLA: Human Leukocyte Antigen. ABMR: Antibody-Mediated Rejection. AVR: Acute Vascular Rejection. CMR: Cell-Mediated Rejection. AT1R: Angiotensin II Type 1 Receptor. GPCR: G-protein coupled receptor. BOS: Bronchiolitis Obliterans Syndrome. PGD: Primary Graft Dysfunction. CAV: Cardiac Allograft Vasculopathy. TG: Transplant Glomerulopathy. IFTA: Interstitial Fibrosis and Tubular Atrophy. ELISA: Enzyme-Linked ImmunoSorbent Assay. HIF-1a = Hypoxia-Inducible Factor 1 alpha.

En sık tespit edilen HLA dışı antikorları

- ***Anjiyotensin II Tip 1 Reseptör Antikorları (AT1R-Ab)***
- ***Anti-Endotelin A Reseptör Antikorları (Anti-Endothelin A Receptor Ab)***
- ***Majör histokompatibilite kompleksi (MHC) sınıf I-ilişkili zincir A (MICA) antikorları (Anti Mica Ab)***
- ***Anti-Perlekan antikorları (Anti-Perlecan/LG-3 Ab)***
- ***Anti kollajen antikorları (Anti-Collagen Type Iv, Type Iii, Type I and Anti-Fibronectin Ab)***
- ***Anti-Vimentin antikorları (Anti-Vimentin Ab)***
- *Anti-Agrin antikorları (Anti-Agrin Ab)*
- *Anti-H-Y antikorları (Anti-H-Y Ab)*
- *Anti-Rho Guanin Nükleotid Değişim Faktörü 2 Antikorları (Anti-ARHGDIB -Rho Guanine Nucleotide Exchange Factor 2 Ab)*
- *Anti-Peroksizomal Trans-2-Enoil-CoA Redüktaz Antikorları (Anti-PECR Peroxisomal Trans-2-Enoyl-CoA Reductase Ab)*
- *Anti-Protein Kinaz C Zeta Tipi Antikorlar (Anti-PRKCZ -Protein Kinase C Zeta Type Ab)*

Anjiyotensin II Tip 1 Reseptör Antikorları (AT1R-Ab)

Human Immunology 82 (2021) 89–96

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Research article

ARHGDIB and AT1R autoantibodies are differentially related to the development and presence of chronic antibody-mediated rejection and fibrosis in kidney allografts

Michiel G.H. Betjes [a,*], Kasia A. Sablik [a], Nicolle H.R. Litjens [a], Henny G. Otten [b], Annelies E. de Weerd [a]

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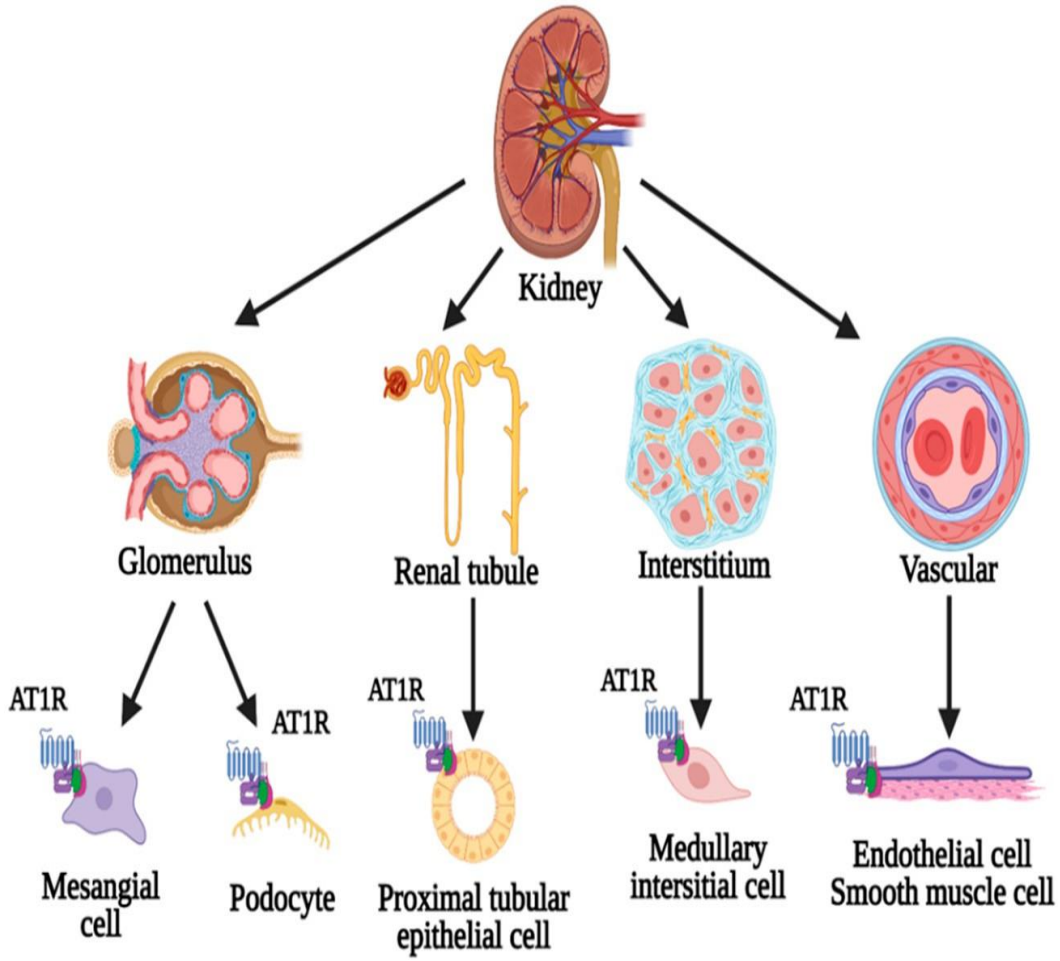
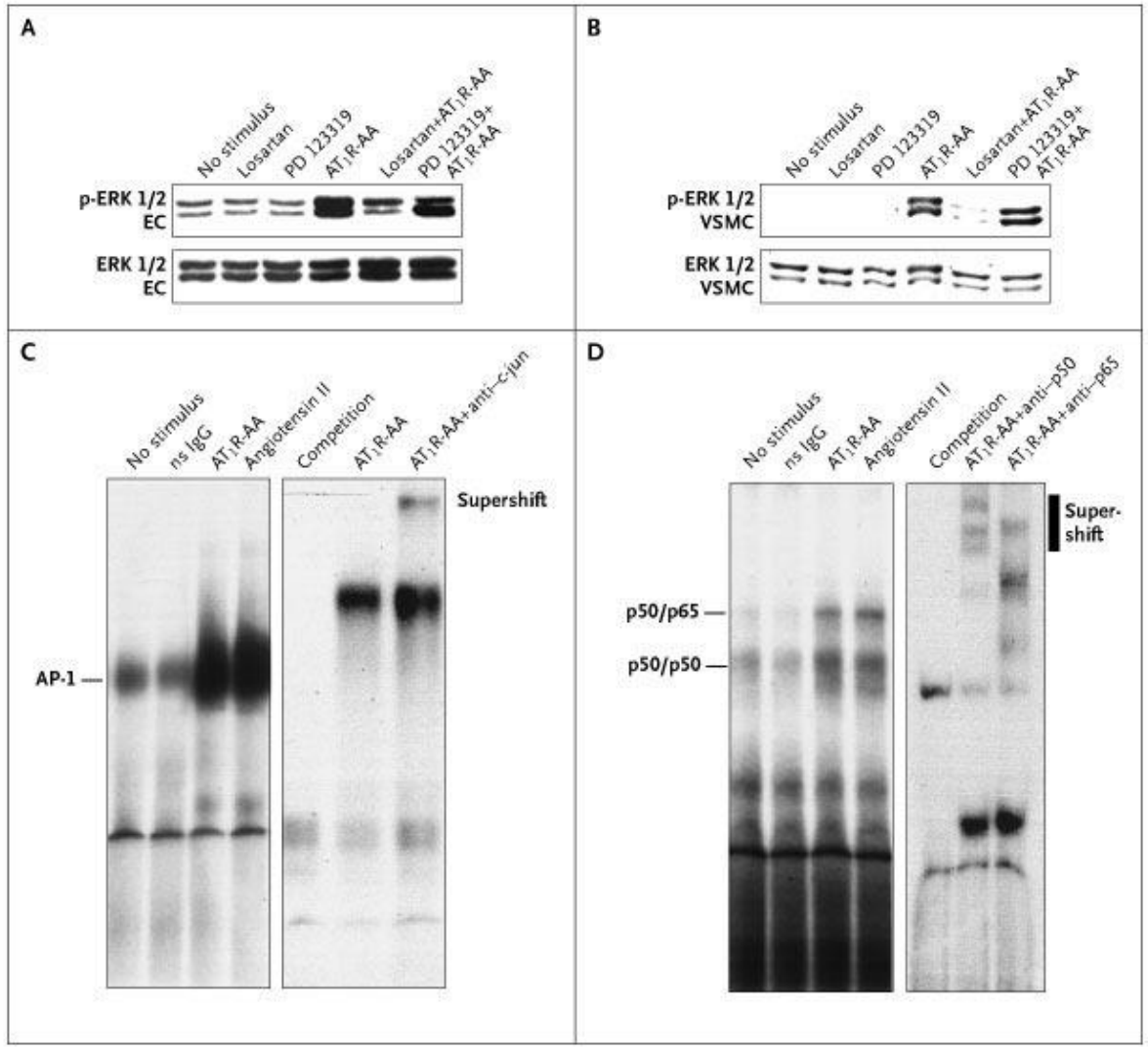
[b] Laboratory of Translational Immunology, University Medical Center Utrecht, Utrecht University, Utrecht, the Netherlands



› N Engl J Med. 2005 Feb 10;352(6):558-69. doi: 10.1056/NEJMoa035717.

Angiotensin II type 1–receptor activating antibodies in renal-allograft rejection

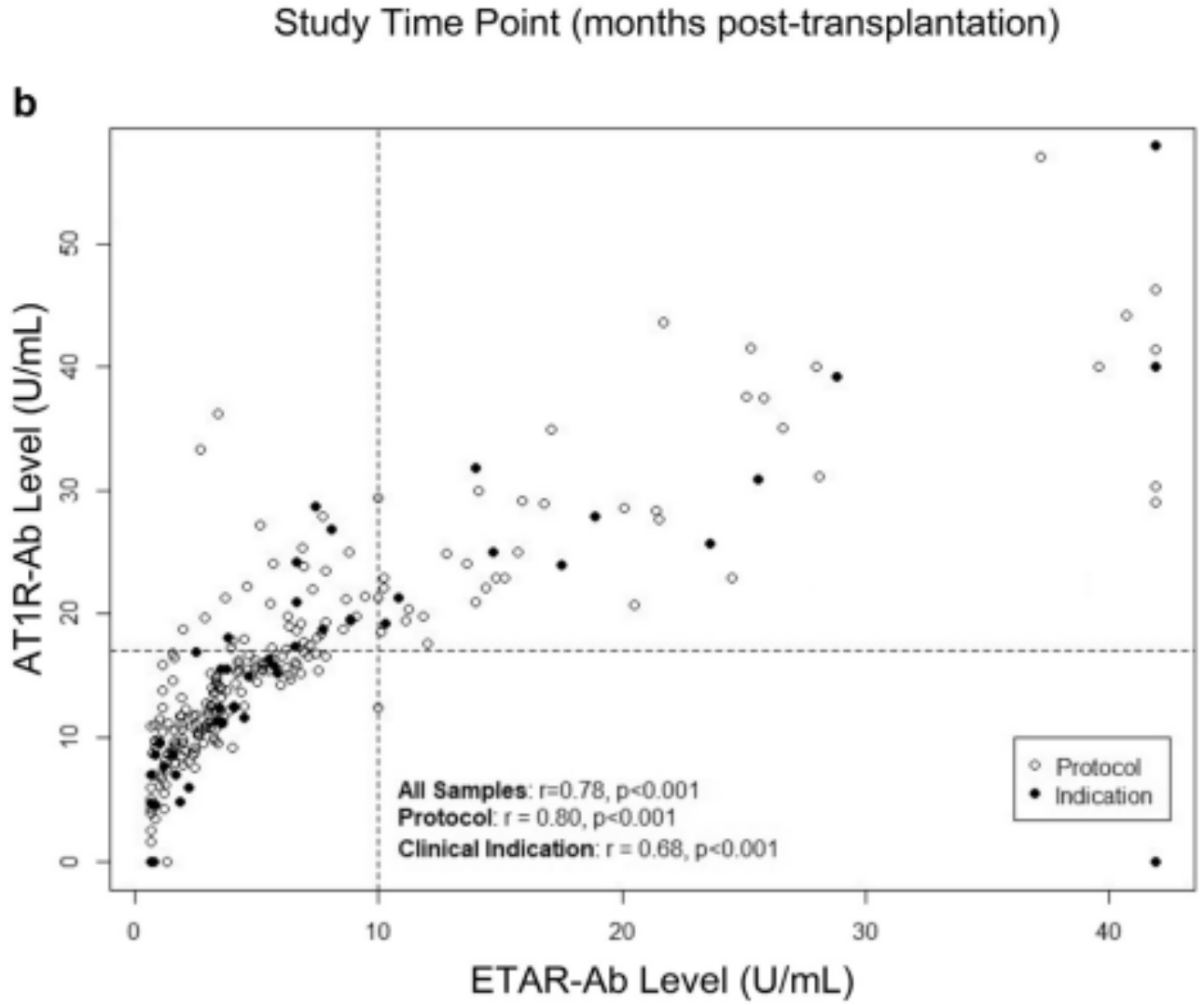
Duska Dragun [1], Dominik N Müller, Jan Hinrich Bräsen, Lutz Fritsche, Melina Nieminen-Kelhä, Ralf Dechend, Ulrich Kintscher, Birgit Rudolph, Johan Hoebeke, Diana Eckert, Istvan Mazak, Ralph Plehm, Constanze Schönemann, Thomas Unger, Klemens Budde, Hans-Hellmut Neumayer, Friedrich C Luft, Gerd Wallukat



Anti-Endotelin A Reseptör Antikorları (Anti-Endothelin a Receptor Ab)

Endothelin Type A Receptor Antibodies Are Associated With Angiotensin II Type 1 Receptor Antibodies, Vascular Inflammation, and Decline in Renal Function in Pediatric Kidney Transplantation

Meghan H. Pearl[1], Lucia Chen[2], Rim ElChaki[1], David Elashoff[2], David W.
Maura Rossetti[3], Patricia L. Weng[1], Qiuheng Zhang[3], Elaine F. Reed[3] an



Majör histokompatibilite kompleksi (MHC) sınıf I-ilişkili zincir A (MICA) antikorları (Anti Mica Ab)

nature medicine

ARTICLES

https://doi.org/10.1038/s41591-022-01725-2

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OPEN

The MHC class I *MICA* gene is a histocompatibility antigen in kidney transplantation

Raphael Carapito[1,2,3,4,5] ✉, Ismail Aouadi[1,2,3,5], Martin Verniquet[1,2,3,5], Meiggie Untrau[1,2,3,5], Angélique Pichot[1,2,3,5], Thomas Beaudrey[1,2,5,6], Xavier Bassand[1,2,5,6], Sébastien Meyer[1,2,3,5], Loic Faucher[2,7], Juliane Posson[8,9], Aurore Morlon[2,10], Irina Kotova[2,10], Florent Delbos[2,11], Alexandre Walencik[2,11], Alice Aarnink[12], Anne Kennel[12], Caroline Suberbielle[2,13], Jean-Luc Taupin[2,13], Benedict M. Matern[14], Eric Spierings[14], Nicolas Congy-Jolivet[2,15,16], Arnaud Essaydi[17], Peggy Perrin[1,2,5,6], Antoine Blancher[2,15,16], Dominique Charron[2,5,13], Nezih Cereb[18], Myriam Maumy-Bertrand[2,5,19], Frédéric Bertrand[2,5,19], Valérie Garrigue[2,20], Vincent Pernin[2,20], Laurent Weekers[21], Maarten Naesens[22], Nassim Kamar[2,23], Christophe Legendre[2,24], Denis Glotz[2,8,9], Sophie Caillard[1,2,5,6], Marc Ladrière[25], Magali Giral[2,7], Dany Anglicheau[2,24,26],

Table 3 | Impact of pre- and post-transplantation anti-MICA DSA on kidney graft loss and acute rejection[a]

Endpoint	Preformed anti-MICA DSA (*n* = 524)		Post-transplantation (1 year) anti-MICA DSA (*n* = 225)[b]		Post-transplantation (1 year) de novo anti-MICA DSA (*n* = 225)[b]	
	HR (95% CI)	*P* value	HR (95% CI)	*P* value	HR (95% CI)	*P* value
Overall graft loss	1.67 (1.28–2.18)	<0.001[c]	1.64 (1.11–2.42)	0.013	0.98 (0.39–2.47)	0.970
Death-censored graft loss	1.32 (0.82–2.10)	0.250	1.02 (0.73–1.42)	0.910	1.29 (1.05–1.58)	0.014
Acute rejection	2.28 (1.40–3.71)	<0.001[d]	1.98 (1.26–3.10)	0.003	1.94 (1.88–2.01)	<0.001[f]
TCMR	2.11 (1.01–4.42)	0.047	1.60 (1.01–2.53)	0.043	1.84 (1.68–2.01)	<0.001[g]
ABMR	3.79 (1.94–7.39)	<0.001[e]	9.92 (7.43–13.20)	< 0.001	3.30 (2.25–4.85)	<0.001[h]

[a]Multivariate Cox regression included all covariates listed in Table 1. Two-sided *P* values were calculated using Wald's test without correction for multiple testing. Exact *P* values: [b]The same 225 patients were analyzed for 1 year anti-MICA DSA and for 1 year de novo anti-MICA DSA. [c]1.32×10^{-4}; [d]8.78×10^{-4}; [e]9.49×10^{-6}; [f]1.57×10^{-10}; [g]5.71×10^{-17}; [h]2.48×10^{-6}.

Anti-Perlekan Antikorları (Anti-Perlecan/LG-3 Ab)

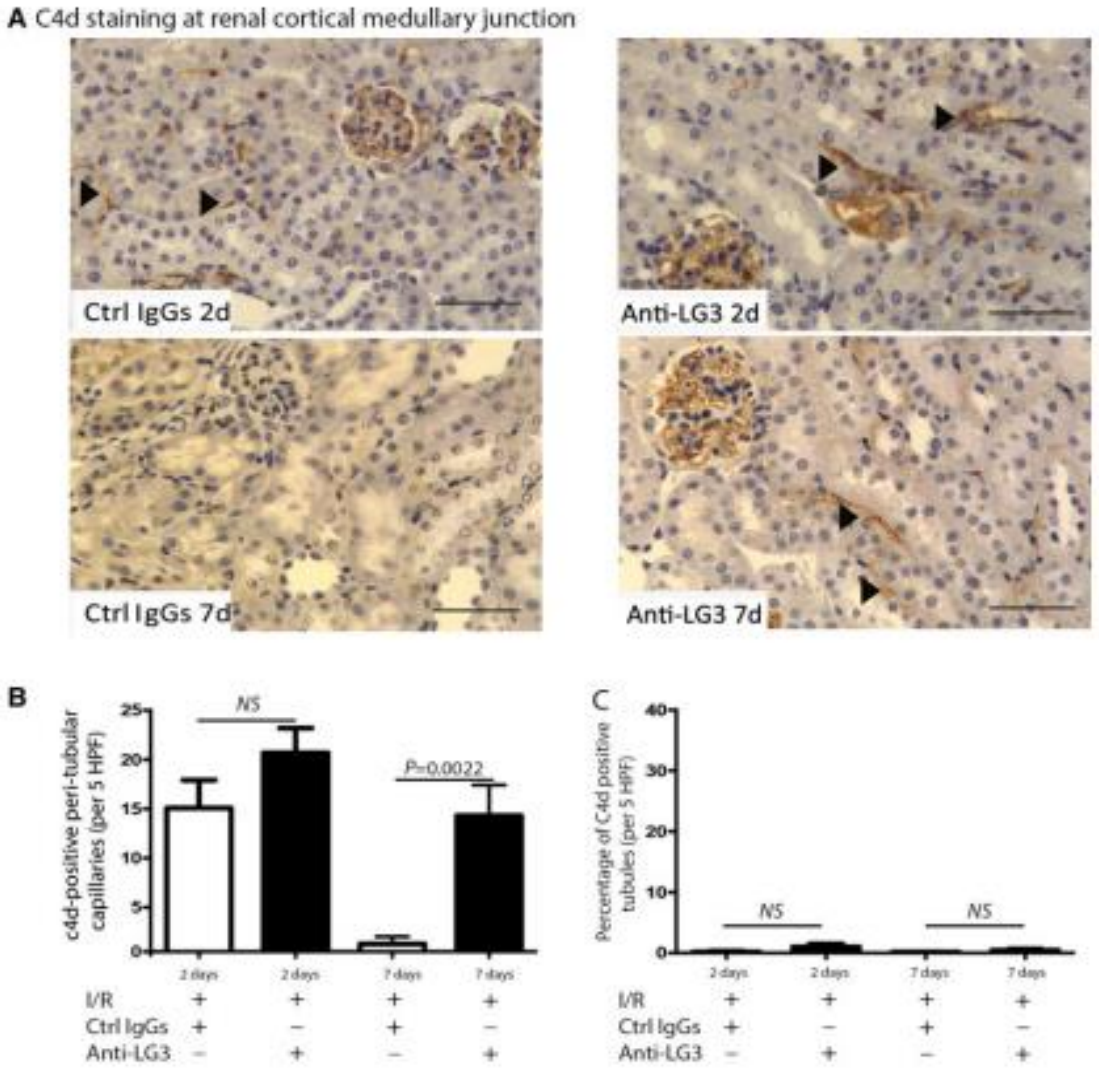
American Journal of Transplantation 2016; 16: 3416–3429
Wiley Periodicals Inc.

doi: 10.1111/ajt.13866

Anti-LG3 Antibodies Aggravate Renal Ischemia–Reperfusion Injury and Long-Term Renal Allograft Dysfunction

B. Yang[1,2,3], M. Dieudé[1,2,3], K. Hamelin[1,2,3], M. Hénault-Rondeau[1,2,3], N. Patey[1,2,3,4], J. Turgeon[1,2,3], S. Lan[1,2,3], L. Pomerleau[1], M. Quesnel[1], J. Peng[1], J. Tremblay[1], Y. Shi[1,3], J. S. Chan[1,3], M. J. Hébert[1,2,3,*,†] and H. Cardinal[1,2,3,*,†]

Abbreviations: α-SMA, α-smooth muscle actin; AKI, acute kidney injury; AT1R, angiotensin II type 1 receptor; BUN, blood urea nitrogen; CDC, complement-dependent cytotoxicity; CHUM, Centre Hospitalier de l'Université de Montréal; CI, confidence interval; COL, IVcollagen IV; cPRA, calculated panel reactive antibody; Ctrl, control; DGF, delayed graft function;



Anti Kollajen Antikorları (Anti-Collagen Type Iv, Type Iii, Type I and Anti-Fibronectin Ab)

Clinical significances of anti-collagen type I and type III antibodies in antibody-mediated rejection

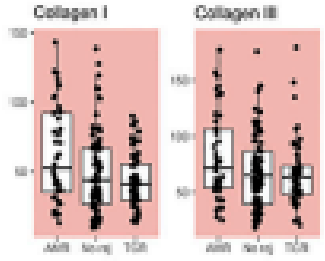
METHODS

Kidney transplant recipients from a tertiary center in Korea (2015-2019)

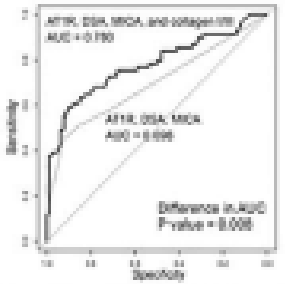
- **Antibody-mediated rejection** (N = 68)
- **T-cell mediated rejection** (N = 67)
- **Controls without rejection** (N = 86)

- Serum samples collected on biopsy
- measured titers for 39 non-HLA autoantibodies, Anti-AT1R, and MICA antibodies

RESULTS



Titers of anti-collagen I and collagen III antibodies were significantly higher in antibody-mediated rejection cases.



Considerations of the antibody titers increased the predictability for antibody-mediated rejection.

Death-censored graft failure risks

- Anti-collagen I antibody (1 SD)
- Adjusted HR 1.90 (1.32–2.75)
- Anti-collagen III antibody (1 SD)
- Adjusted HR 1.57 (1.15–2.16)

The higher titers of the antibodies were associated with worse prognosis of antibody-mediated rejection.

CONCLUSIONS Post-transplant anti-collagen I and collagen III antibodies may be novel non-HLA antibodies related to antibody-mediated rejection.

PARK S., et al. *Transpl. Int.* 2022
doi: 10.3389/ti.2022.10099

Anti-Vimentin Antikorları (Anti-Vimentin Antibodies)

ELSEVIER

Vimentin Antibodies: A Non-HLA Antibody as a Potential Risk Factor in Renal Transplantation

V. Carter, B.K. Shenton, B. Jaques, D. Turner, D. Talbot, A. Gupta, C.E. Chapman, C.J. Matthews, and G. Cavanagh

Table 1. Initial Screening of Samples

Antibodies identified	Number or Percentage	Antibody Classification	Number of Subjects
HLA class I (screen (donor-specific))	25/48 (23/48)	HLA class I only	3
HLA class II (screen (donor-specific))	37/48 (24/48)	HLA class II only	4
		HLA class I and II	11
Positive screen including vimentin	98%	HLA class I + vimentin	4
		HLA class II + vimentin	4
Positive donor-specific including vimentin	80%	HLA class I + II + vimentin	5
Vimentin	21/48	HLA (no specificity identified) +/− vimentin	11
No antibodies detected	1/48	Vimentin only	5
		No antibodies detected	1

The 48 patients were tested for the presence or absence of HLA and vimentin antibodies and for identifiable antibodies specific for donor antigens. Subjects were then classified by the combination of antibodies detected.

Anti -Agrin antikorları (Anti-Agrin Ab)

- Kronik ABMR'nin bir özelliği olan transplant glomerülopatili hastalarda agrine karşı antikorlar tanımlanmıştır.

American Journal of Transplantation 2005; 5: 383–393
Blackwell Munksgaard

doi: 10.1111/j.1600-6143.2005.00690.x

Antibody Response Against the Glomerular Basement Membrane Protein Agrin in Patients with Transplant Glomerulopathy

Simone A. Joosten[a], Yvo W.J. Sijpkens[a], Vanessa van Ham[a], Leendert A. Trouw[a], Johan van der Vlag[b], Bert van den Heuvel[c], Cees van Kooten[a,*] and Leendert C. Paul[a]

Introduction

Chronic allograft nephropathy (CAN) is one of the most important causes of graft failure after the first 6 months post-transplantation (Tx) and may affect over half of the

Anti-H-Y antikorları (Anti-H-Y Ab)

- H-Y antijeni, Y kromozomu üzerinde bulunan genler tarafından kodlanan minör bir doku uyumluluk antijenidir. Greft reddi ,kısa ve uzun süreli greft yetmezliği ile ilişkili .

J Am Soc Nephrol. 2009 Sep; 20(9): 2025–2033. PMCID: PMC2736773

doi: 10.1681/ASN.2008101110 PMID: 19541808

H-Y Incompatibility Predicts Short-Term Outcomes for Kidney Transplant Recipients

S. Joseph Kim* and John S. Gill†

Anti-Rho Guanin Nükleotid Değişim Faktörü 2 Antikorları (Anti-ARHGDIB -Rho Guanine Nucleotide Exchange Factor 2 Ab)

Received: 7 December 2018 | Revised: 3 April 2019 | Accepted: 4 May 2019

DOI: 10.1111/ajt.15493

ORIGINAL ARTICLE

AJT

Antibodies against ARHGDIB are associated with long-term kidney graft loss

Elena G. Kamburova[1] | Maartje L. Gruijters[2] | Tineke Kardol-Hoefnagel[1] | Bram W. Wisse[1] | Irma Joosten[3] | Wil A. Allebes[3] | Arnold van der Meer[3] | Luuk B. Hilbrands[4] | Marije C. Baas[4] | Eric Spierings[1] | Cornelis E. Hack[1] | Franka E. van Reekum[5] | Arjan D. van Zuilen[5] | Marianne C. Verhaar[5] | Michiel L. Bots[6]

TABLE 1 Multivariable analyses of the effect of antibodies against ARHGDIB on 10-year death-censored graft failure

	No. (%) of transplantations with anti-ARHGDIB antibodies	Hazard ratio	95% CI	P-value
Total cohort (N = 4770)	134 (2.8)	1.701	1.265-2.288	.0004
Deceased donors (N = 3276)	94 (2.9)	1.820	1.318-2.531	.0003
Living donors (N = 1494)	40 (2.7)	1.249	0.587-2.657	.5639

Anti-Peroksizomal Trans-2-Enoil-CoA Redüktaz Antikorları(Anti-PECR - Peroxisomal Trans-2-Enoyl-CoA Reductase Ab)

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doi: 10.1681/ASN.2010111183
PMCID: PMC3103737
PMID: 21566057

Antibodies Reactive to Non-HLA Antigens in Transplant Glomerulopathy

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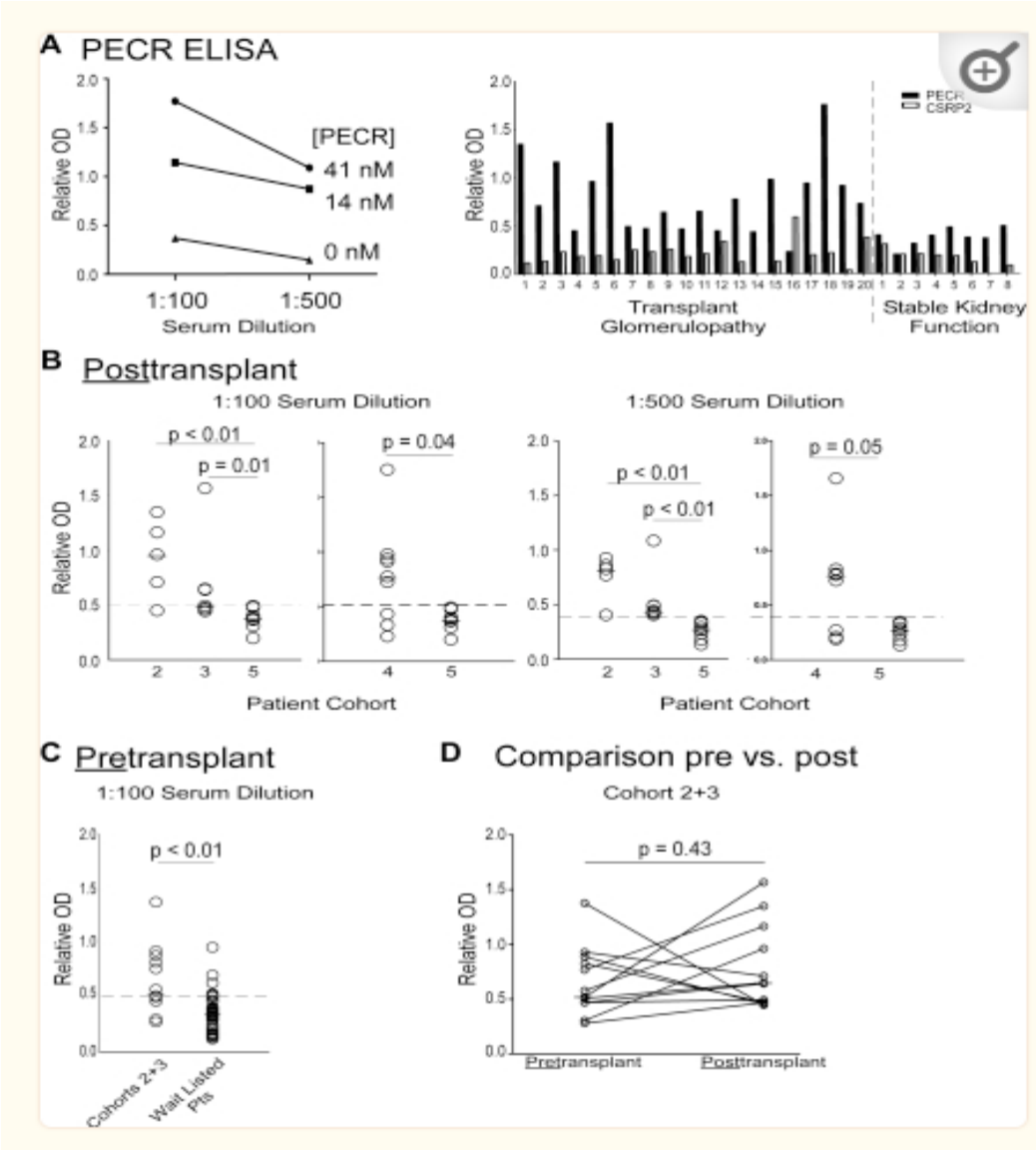

Anti-Protein Kinaz C Zeta Tipi Antikorlar (Anti-PRKCZ -Protein Kinase C Zeta Type Ab)

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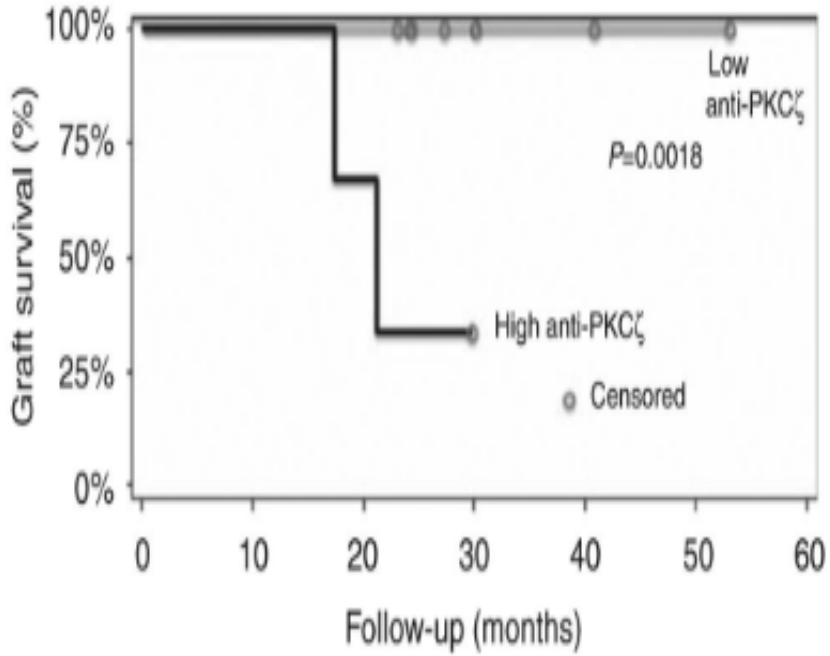
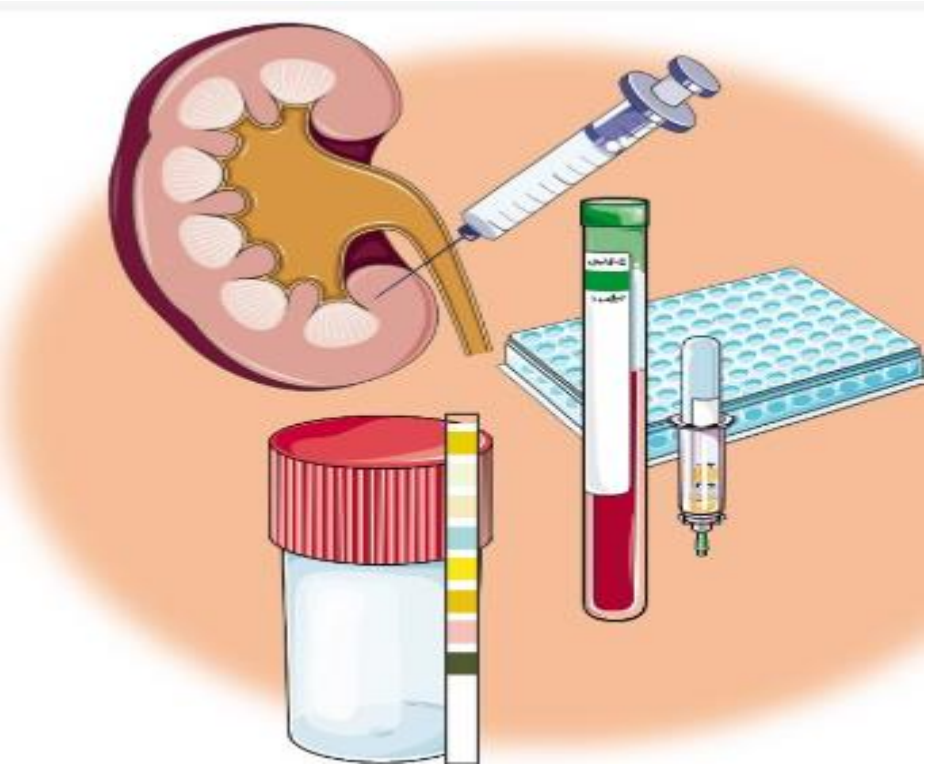
Protein microarrays identify antibodies to protein kinase Cζ that are associated with a greater risk of allograft loss in pediatric renal transplant recipients

Scott M. Sutherland[1], **Li Li**[1], **Tara K. Sigdel**[1], **Persis P. Wadia**[2], **David B. Miklos**[2], **Atul J. Butte**[3,4], and **Minnie M. Sarwal**[1,4]



- HLA dışı antikorlar immünolojik risk değerlendirmesine dahil edilmeli,
- Algoritma oluşturulmalı.



TEŞEKKÜRLER…