Kadavra Donörleri Nasıl Arttırabiliriz? Nasıl Daha Etkili Kullanabiliriz?

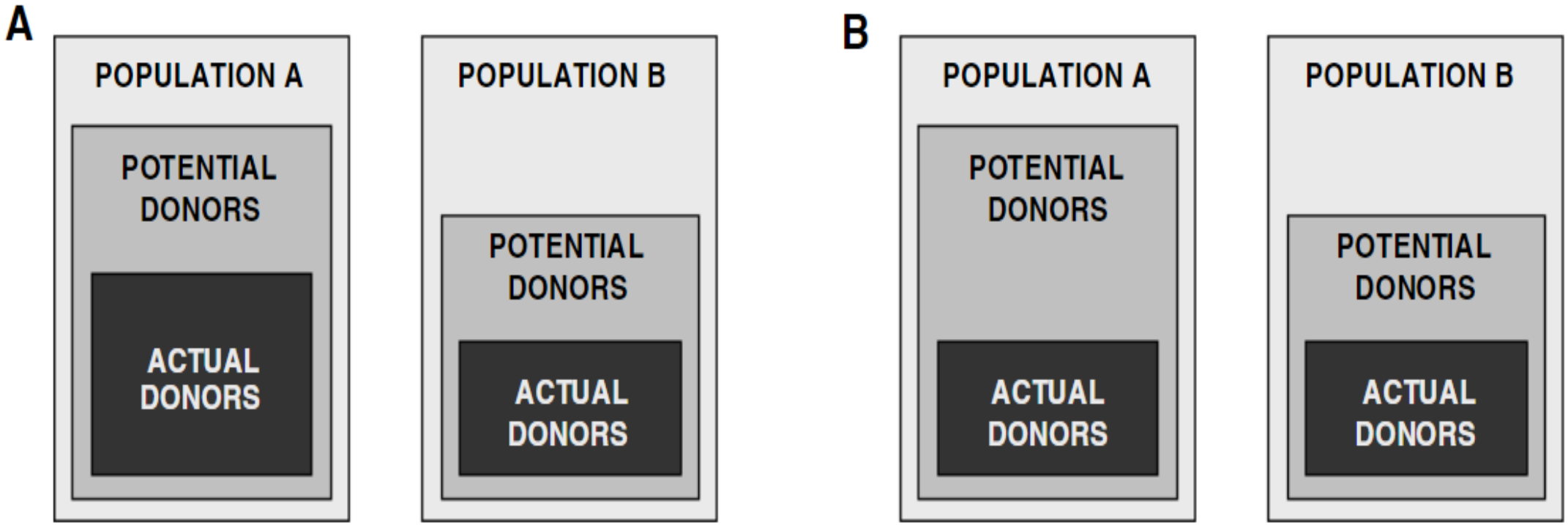
Dr. Hüseyin Koçak

Akdeniz Üniversitesi Tıp Fakültesi İç Hastalıkları A.B.D

Kadavra Organ Bağış Oranları

	Türkiye	USA	Avrupa
Organ Bağışı (Milyon Nufus Başına)	3.6	38	İspanya: 47 İngiltere: 20 Fransa: 25
Aile İzin Oranı	%17 (305/1840) 2018:%27 2020:%19 2022:%17 %10 Azalma	%60	İspanya: %85 İngiltere:%62
Tıbbi Nedenle Kullanılamayan Organ (TNK)	%57 2018:% 7 2020:%34, 2022:%39 6-7 Kat Artma	%20	Avrupa: %12

İspanya: Potansiyel Bağışçı, Gerçek Bağışçıya Dönüşmesi



Donation rate p.m.p. A > B
Donation conversion rate A = B

Donation rate p.m.p. A = B
Donation conversion rate A < B

American Journal of Transplantation 2017; 17: 1447–1454

ORGAN DONATION AND TRANSPLANT

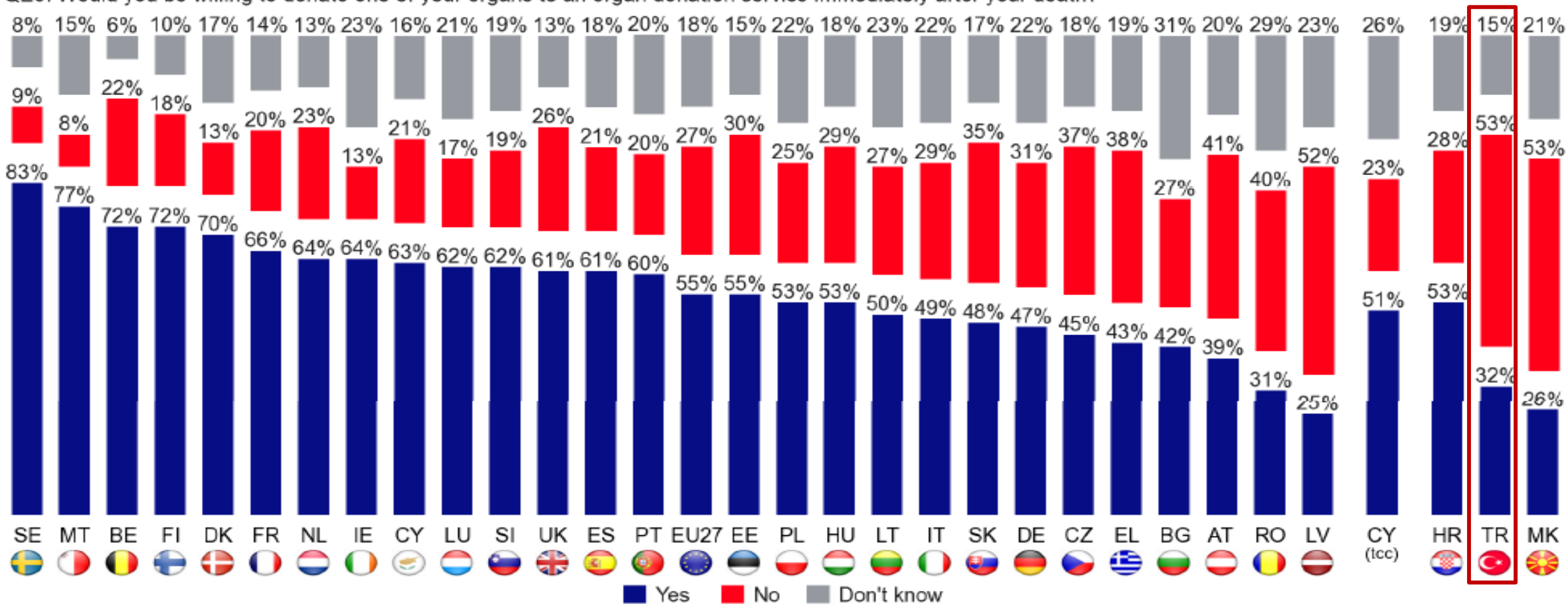
EUROBAROMETER

European Commission

EU27	Number of interviews: 26.788	Fieldwork: 02/10-19/10/2009
TR	Number of interviews: 1.004	Fieldwork: 02/10-18/10/2009

Methodology: face-to-face

QE3. Would you be willing to donate one of your organs to an organ donation service immediately after your death?



ORGAN DONATION AND TRANSPLANT

EUROBAROMETER

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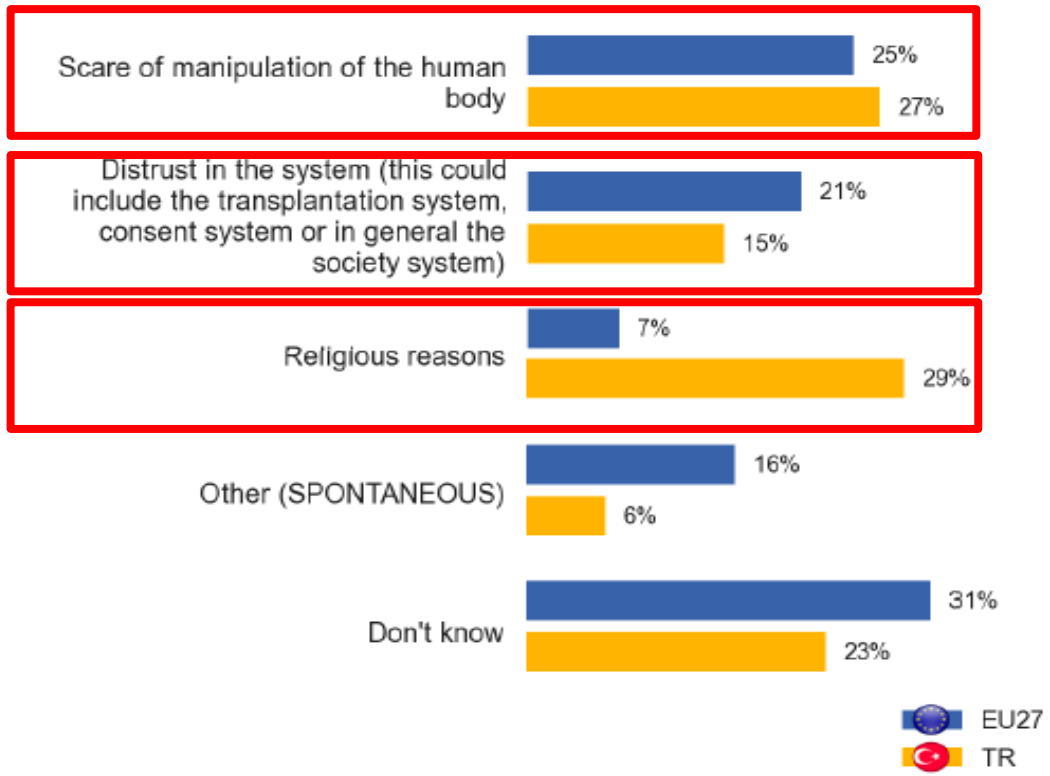
EU27	Number of interviews: 26.788	Fieldwork: 02/10-19/10/2009
TR	Number of interviews: 1.004	Fieldwork: 02/10-18/10/2009

Methodology: face-to-face

QE5. If you would be unwilling to donate your organs or those of a close family member what would these reasons be?



QE5. If you would be unwilling to donate your organs or those of a close family member what would these reasons be?
EU27 + Top ten countries for each item

Scare of manipulation of the human body		Distrust in the system		Religious reasons	
EU27	25%	EU27	21%	EU27	7%
CZ	45%	EL	45%	RO	17%
PL	36%	CZ	33%	AT	15%
LV	35%	SK	31%	SK	11%
SK	33%	IT	30%	IT	10%
CY	33%	PT	28%	EL	10%
AT	32%	DE	26%	PT	9%
EL	31%	LV	26%	CY	9%
IT	29%	BG	26%	LT	9%
BE	29%	AT	24%	HU	8%
LT	27%	HU	24%	EE	7%

Türk Halkı Kime Güveniyor Bilim Adamı, Din Adamı ?.

nature human behaviour

ARTICLES

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Check for updates

The Einstein effect provides global evidence for scientific source credibility effects and the influence of religiosity

Suzanne Hoogeveen[1]✉, Julia M. Haaf[1], Joseph A. Bulbulia[2], Robert M. Ross[3], Ryan McKay[4], Sacha Altay[5], Theiss Bendixen[6], Renatas Berniūnas[7], Arik Cheshin[8], Claudio Gentili[9], Raluca Georgescu[10], Will M. Gervais[11], Kristin Hagel[12], Christopher Kavanagh[13,14], Neil Levy[3,15], Alejandra Neely[16], Lin Qiu[17], André Rabelo[18], Jonathan E. Ramsay[19], Bastiaan T. Rutjens[1], Hugh Turpin[13], Filip Uzarevic[20], Robin Wuyts[1], Dimitris Xygalatas[21] and Michiel van Elk[22]

People tend to evaluate information from reliable sources more favourably, but it is unclear exactly how perceivers' worldviews interact with this source credibility effect. In a large and diverse cross-cultural sample ($N = 10{,}195$ from 24 countries), we presented participants with obscure, meaningless statements attributed to either a spiritual guru or a scientist. We found a robust global source credibility effect for scientific authorities, which we dub 'the Einstein effect': across all 24 countries and all levels of religiosity, scientists held greater authority than spiritual gurus. In addition, individual religiosity predicted a weaker relative preference for the statement from the scientist compared with the spiritual guru, and was more strongly associated with credibility judgements for the guru than the scientist. Independent data on explicit trust ratings across 143 countries mirrored our experimental findings. These findings suggest that irrespective of one's religious worldview, across cultures science is a powerful and universal heuristic that signals the reliability of information.

In a heated debate about the proximity of COVID-19 herd immunity, White House health advisor Dr Scott Atlas proclaimed 'You're supposed to believe the science, and I'm telling you the science'[1]. A group of infectious disease experts and former colleagues from Stanford, however, publicly criticized Dr Atlas, who is a radiologist, for spreading 'falsehoods and misrepresentation of science' through his statements about face masks, social distancing and the safety of community transmission[2]. In the 2020 pandemic crisis, all eyes turned to scientific experts to provide advice, guidelines and remedies; from COVID-19 alarmists to sceptics, appeal to scientific authority appeared a prevalent strategy on both sides of the political spectrum. Please see the Supplementary Information for a short commentary on how the current work might relate to the COVID-19 situation.

A large body of research has shown that the credibility of a statement is heavily influenced by the perceived credibility of its source[3–10]. Children and adults are sensitive to the past track record of informants[11–16], evidence of their benevolence toward the recipient of testimony[17–19], as well as how credible the information is at face value[20,21]. From an evolutionary perspective, deference to credible authorities such as teachers, doctors and scientists is an adaptive strategy that enables effective cultural learning and knowledge transmission[22–28]. Indeed, if the source is considered a trusted expert, people are willing to believe claims from that source without fully understanding them. We dub this 'the Einstein effect'; people simply accept that $E = mc^2$ and that antibiotics can help cure pneumonia because credible authorities such as Einstein and their doctor say so, without actually understanding what these statements truly entail.

Knowing that a statement originates from an epistemic authority may thus increase the likelihood of opaque messages being interpreted as meaningful and profound. According to Sperber[29], in some cases, incomprehensible statements from credible sources may be appreciated not just in spite of, but by virtue of their incomprehensibility, as exemplified by the speech of spiritual or intellectual gurus (the 'Guru effect'). Here, we investigate to what extent different epistemic authorities affect the perceived value of nonsensical information. To this end, we contrasted judgements of gobbledegook spoken by a spiritual leader with gobbledegook spoken

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Güven İndeksi

Din Adamı

Bilim Adamı

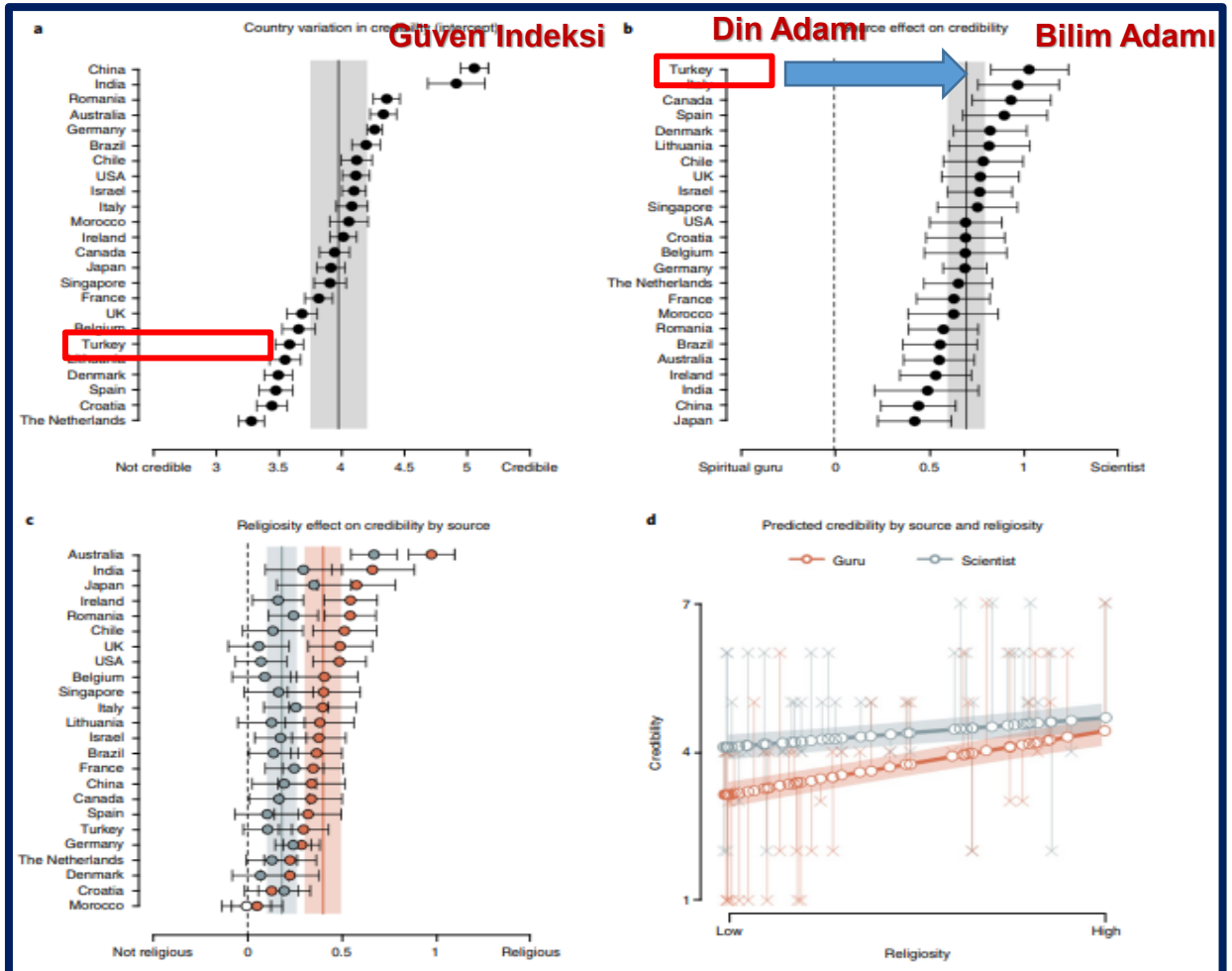
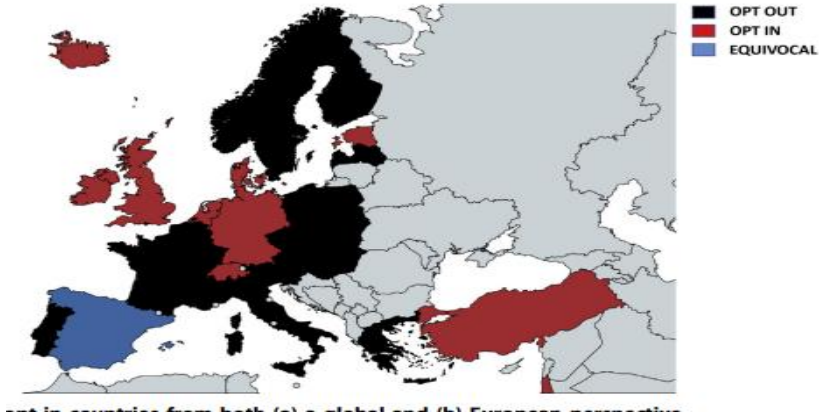


Fig. 2 | Summary of the multilevel-model (unconstrained) estimates per country and predicted overall effects. a,**b**, It is apparent that there is substantial variation across the 24 countries in (**a**) overall credibility judgements (that is, intercept) and (**b**) the effect of scientific versus spiritual source. **c**, Individual religiosity has a stronger effect on credibility judgements for the spiritual guru (red circles) than for the scientist (grey circles). The estimates are ordered from largest to smallest, and the open circles denote negatively valued effects. The error bars give the 95% CI for each country. The vertical lines denote the overall estimated effect with the 95% CI in the shaded bands. The dashed lines indicates zero. **d**, Predicted credibility as a function of source and individual religiosity, showing that the difference in credibility ratings for the scientist (grey lines) versus the guru (red lines) is less pronounced for high-religiosity individuals than low-religiosity individuals. The shaded bands reflects the 95% CI, crosses reflect the observed values for two randomly sampled participants per country, and circles reflect the corresponding estimated values. Crosses and circles are jittered to enhance visibility.

Bağışçı Rızasında Opt In ve Opt Out Sistemi

17 OECD Ülkesi Opt Out



opt in countries from both (a) a global and (b) European perspective

Table 1 | Snapshot of OECD countries used in the analysis with donation rates and transplantation activity (per million population)

Country	System	Deceased donation rates	Living donation rates	Total transplant activity
Australia	In	20.6	10.9	72.6
Austria	Out	24.8	7.9	89.3
Belgium	Out	30.7	9.6	90.0
Canada	In	20.0	14.9	79.6
Chile[a]	Out	7.4	5.3	24.5
Czech Republic	Out	25.1	4.4	75.4
Denmark	In	17.4	19.0	67.5
Estonia	In	16.8	3.1	43.5
Finland	Out	24.6	4.0	72.2
France	Out	28.6	8.9	90.8
Germany	In	10.4	7.9	45.0
Greece	Out	4.6	4.4	13.5
Hungary	Out	18.7	3.5	51.8
Iceland	In	26.9	14.9	14.9
Ireland[b]	In	16.2	10.5	58.8
Israel[c]	In	10.2	27.6	62.5
Italy	Out	24.9	4.8	63.6
Japan	In	0.8	14.6	29.9
Korea	In	11.4	42.4	79.3
Latvia	Out	15.4	5.1	31.8
Luxembourg	Out	5.1	n/a	n/a
Mexico	In	3.6	16.4	24.7
Netherlands[d]	In	14.7	33.9	75.4
New Zealand	In	13.0	18.1	55.7
Norway	Out	20.9	8.9	78.2
Poland	Out	14.2	2.0	42.6
Portugal	Out	32.6	6.3	82.2
Slovak Republic	Out	13.2	3.5	36.7
Slovenia	Out	20.2	1.0	52.4
Spain	Out/in[e]	43.6	8.0	103.9
Sweden	Out	20.3	14.4	80.8
Switzerland	In	13.1	15.5	60.4
Turkey	In	7.0	45.1	60.9
United Kingdom[f]	In	21.2	15.9	73.7
United States	In	30.7	18.4	106.1

Table 4 | Sensitivity analysis with Spain regarded as an opt-out country

Variable	Opt-out	Opt-in	P
Organ donation rates (per million population)			
Total deceased donors	20.6 (14.0–25.9)	14.7 (10.3–20.3)	0.062
Total living donors	5.0 (3.5–8.2)	15.9 (12.8–23.3)	**<0.001**
Organ-specific transplantation activity (per million population)			
Deceased kidney transplantation	31.0 (22.4–41.8)	21.8 (12.7–31.3)	**0.038**
Living kidney transplantation	4.6 (3.5–7.5)	15.4 (11.2–21.2)	**<0.001**
Deceased liver transplantation	14.9 (6.2–20.5)	10.1 (6.2–12.5)	0.219
Living liver transplantation	0.0 (0.0–0.3)	0.6 (0.0–1.6)	0.035
Heart transplantation	5.0 (2.4–6.6)	3.1 (0.6–4.9)	0.038
Lung transplantation	2.5 (0.0–6.4)	3.9 (1.1–6.6)	0.386
Pancreas transplantation	1.3 (0.2–2.6)	1.3 (0.2–1.8)	0.807
Small bowel transplantation	0.0 (0.0–0.1)	0.0 (0.0 0.1)	0.630
Overall solid organ transplantation activity (per million population)			
Overall kidney transplantation	39.2 (25.2–47.7)	42.3 (28.8–46.6)	0.782
Overall nonrenal transplantation	29.5 (9.7–35.5)	20.2 (17.2–24.4)	0.325
Overall solid organ transplantation	67.9 (35.5–84.0)	60.0 (44.3–74.5)	0.708

Values are median (interquartile range), with *P* values from Mann-Whitney U tests. Bold values are significant at $P < 0.05$.

Table 3 | Comparison of organ donation rates and solid organ transplantation activity between opt-out versus opt-in countries (latest year)

Variable	Opt-out	Opt-in	P
Organ donation rates (per million population)			
Total deceased donors	20.3 (13.7–25.0)	15.4 (10.4–20.7)	0.195
Total living donors	4.8 (3.5–8.4)	15.7 (10.8–21.2)	**<0.001**
Organ-specific transplantation activity (per million population)			
Deceased kidney transplantation	30.3 (22.0–40.7)	23.4 (14.1–33.8)	0.134
Living kidney transplantation	4.5 (3.5–7.0)	15.2 (10.8–20.1)	**<0.001**
Deceased liver transplantation	13.0 (5.6–20.3)	10.2 (6.9–13.0)	0.483
Living liver transplantation	0.0 (0.0–0.2)	0.6 (0.0–1.5)	0.025
Heart transplantation	4.5 (2.1–6.6)	3.1 (0.7–5.1)	0.083
Lung transplantation	2.5 (0.0–6.2)	4.1 (1.4–6.8)	0.219
Pancreas transplantation	1.1 (0.1–2.7)	1.4 (0.2–1.7)	0.961
Small bowel transplantation	0.0 (0.0–0.0)	0.0 (0.0–0.1)	0.309
Overall solid organ transplantation activity (per million population)			
Overall kidney transplantation	35.2 (24.2–46.5)	42.3 (30.4–48.0)	0.405
Overall nonrenal transplantation	28.7 (9.1–34.5)	20.9 (17.5–27.3)	0.606
Overall solid organ transplantation	63.6 (34.3–81.5)	61.7 (44.6–76.4)	0.909

Values are median (interquartile range), with *P* values from Mann-Whitney U tests. Bold values are significant at $P < 0.05$.

Kidney International (2019) 95, 1453–1460

Beyin Ölümü

MADDE 41 – 2238 sayılı Kanunun 11 inci maddesi aşağıdaki şekilde değiştirilmiştir.
"MADDE 11 – Bu Kanunun uygulanması ile ilgili olarak tıbbi ölümün gerçekleştiğine**, biri nörolog veya nöroşirürjiyen, biri de anesteziyolji ve reanimasyon veya yoğun bakım uzmanından oluşan iki hekim tarafından** kanıta dayalı tıp kurallarına uygun olarak oy birliği ile karar verilir."

EK-1

BEYİN ÖLÜMÜ TANISI

(1) Beyin ölümü klinik bir tanıdır ve tüm beyin fonksiyonlarının tam ve geri dönüşümü olmayan kaybıdır. Beyin ölümü tanısında gereken ön koşullar aşağıda belirtilmiştir.

a) Komanın nedeninin belirlenmiş olması,

b) Beyin hasarının yaygın ve geri dönüşümsüz olduğunun belirlenmiş olması,

c) Santral vücut ısısı ≥32 ^{o}C olması,

ç) Hipotansif şok tablosu olmaması,

d) Komadan geriye dönüşüm sağlanabilecek ilaç etkileri ve intoksikasyonların dışlanmış olması,

e) Beyin hasarından bağımsız şekilde klinik tabloyu açıklayabilecek metabolik, elektrolit ve asit-baz bozukluklarının olmaması.

(2) Birinci fıkrada yer alan tüm koşulların tespiti halinde beyin ölümü tanısı için aşağıdaki hususlar aranır.

a) Derin komanın olması (Tam yanıtsızlık hali; Santral ağrılı uyaranlara motor cevap alınamaması),

b) Beyin sapı reflekslerinin alınmaması;

1) Pupiller parlak ışığa yanıtsız, orta hatta ve dilatedir (4-9 mm),

2) Okülosefalik ve Vestibulo-oküler refleks yokluğu,

3) Kornea refleksi yokluğu,

4) Faringeal ve trakeal reflekslerin yokluğu.

c) Spontan solunum çabasının bulunmaması ve apne testinin pozitif olması.

Destekleyici Testler

- Beyin dolaşımının tamamen kesildiğini gösteren test:
  - Transkraniyel Dopler USG
  - Serebral Anjiyografi
  - Serebral perfüzyon sintigrafisi
- Biyoelektriksel aktivitenin kaybolduğunu gösteren testler:
  - EEG
  - Uyarılmış Potansiyeller

Ölüm Nedir ?, Beyin Ölümü, Dolaşımın Durmasına Bağlı Ölüm

ARTICLE

https://doi.org/10.1038/s41586-019-1099-1

Restoration of brain circulation and cellular functions hours post-mortem

Zvonimir Vrselja[1,2,18], Stefano G. Daniele[1,2,3,18], John Silbereis[1,2], Francesca Talpo[1,2,4], Yury M. Morozov[1,2], André M. M. Sousa[1,2], Brian S. Tanaka[5,6,7], Mario Skarica[1,2], Mihovil Pletikos[1,2,8], Navjot Kaur[1,2], Zhen W. Zhuang[9], Zhao Liu[9,10], Rafeed Alkawadri[6,11], Albert J. Sinusas[9,10], Stephen R. Latham[12], Stephen G. Waxman[5,6,7] & Nenad Sestan[1,2,13,14,15,16,17]*

The brains of humans and other mammals are highly vulnerable to interruptions in blood flow and decreases in oxygen levels. Here we describe the restoration and maintenance of microcirculation and molecular and cellular functions of the intact pig brain under ex vivo normothermic conditions up to four hours post-mortem. We have developed an extracorporeal pulsatile-perfusion system and a haemoglobin-based, acellular, non-coagulative, echogenic, and cytoprotective perfusate that promotes recovery from anoxia, reduces reperfusion injury, prevents oedema, and metabolically supports the energy requirements of the brain. With this system, we observed preservation of cytoarchitecture; attenuation of cell death; and restoration of vascular dilatory and glial inflammatory responses, spontaneous synaptic activity, and active cerebral metabolism in the absence of global electrocorticographic activity. These findings demonstrate that under appropriate conditions the isolated, intact large mammalian brain possesses an underappreciated capacity for restoration of microcirculation and molecular and cellular activity after a prolonged post-mortem interval.

Many mammalian species have large, energy-demanding brains that are highly susceptible to anoxia and cessation of blood flow[1–3]. Studies in both humans and experimental animals have shown that oxygen stores, global electrical activity, and consciousness are lost within seconds of interrupted blood flow, while glucose and ATP stores are depleted within minutes[4–8]. Unless perfusion is quickly restored, multiple deleterious mechanisms lead to widespread membrane depolarization, loss of ionic homeostasis, mitochondrial dysfunction, and excitotoxic accumulation of glutamate[9,10]. The convergence of these factors has been widely proposed to initiate a progressive, and largely irreversible, cascade of apoptosis, necrosis, and axonal damage[4–9].

However, several observations have questioned the inevitability

insult. Therefore, we postulate that, under appropriate conditions, certain molecular and cellular functions in the large mammalian brain may retain at least partial capacity for restoration after a prolonged post-mortem interval (PMI).

To test this hypothesis, we developed a surgical procedure, perfusate, and custom pulsatile-perfusion device that can restore and maintain microcirculation and cellular viability in the large mammalian brain under ex vivo normothermic conditions (37 °C) after an extended PMI. This system is herein referred to as Brain*Ex* (B*Ex*). To determine whether restoration and maintenance of cell viability is possible, we engineered a haemoglobin-based, acellular, echogenic, and non-coagulative cytoprotective B*Ex* perfusate. In order to develop all aspects

Domuz beyni ölümden 4 saat sonra, Beyin dolaşımının 6 saat özel solüsyonla sağlanması hücre yapısının koruyabilmekte, metabolik aktivite ve sinaptik aktivite gösterebilmektedir.

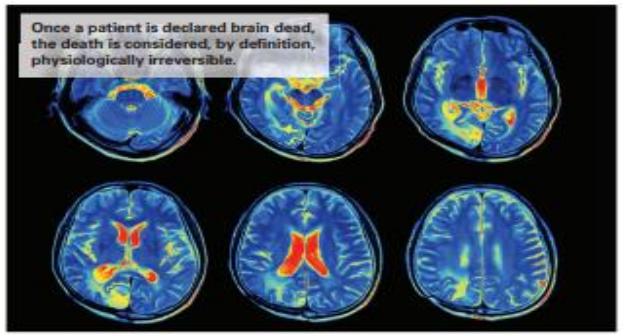
BY LARA C. PULLEN, PHD

The AJT Report

News and issues that affect organ and tissue transplantation

The Death Debate

As science advances, the transplantation community needs a more precise way to explain "brain death" to donors and their families



Once a patient is declared brain dead, the death is considered, by definition, physiologically irreversible.

First the reports hit *Nature* and then the general media: Scientists were able to "revive" dead pig brains and "keep them alive" for 10 hours after death.[1] In actuality, the report demonstrated some remnant cellular functions persisted when brains of dead pigs underwent ex vivo perfusion. The news reverberated throughout the lay community, sparking questions over its implications for transplantation. Does the scientists' work undermine the Dead Donor Rule, a standard that anchors the field's ethical foundation of voluntary deceased organ donation?

Plainly stating that vital organs should only be transplanted from dead patients, the Dead Donor Rule underscores the transplantation community's commitment to respect persons and human life. However, this newly reported science had some wondering: If dead pigs are not dead, are dead patients dead? The casual reader may easily imagine that the brains extracted from these dead pigs were at that point "brain dead," and that the revival of the brains was the porcine equivalent of reversing human brain death.

"Sometimes language is so important, especially in this field of organ transplantation," says Jim Gleason, president of Transplant Recipients International Organization in Beverly, New Jersey. He encourages others in the transplantation community to think carefully about the actual meaning of "brain death" and to employ precise language when discussing it.

Fear of death, particularly fear of a premature, mistaken declaration of death, pervades humanity, says James F. Childress, PhD, professor of ethics at the University of Virginia in Charlottesville. Perhaps because of this fear, death has historically tended to be up for debate, dramatized in macabre works by the likes of authors Edgar Allen Poe (1809–1849) and Bram Stoker (1847–1912), who leveraged the technical ambiguity of life versus death to great effect.

Decay is the only reliable indicator of the death of a system, as Dr. Childress points out. However, the transplant community cannot wait for the unfolding of this natural process, and the resulting tension between the human need for certainty and the imperative to save lives fuels a fear that organs may be removed following a mistaken declaration of death. Thus, explains Dr. Childress, the new data raise concerns for the transplantation community. The question is, he says, "If we have the public discourse, will we damage the public trust that is important for organ donation?" In other words, in the absence of a clear line between life and death, will more people say no to organ donation?

KEY POINTS

- **A recent report on the temporary revival of brain cells from dead pigs has confounded the usual definition of brain death.**
- **Some within the transplantation community are concerned that the common human fear of "mistaken death" will discourage people from becoming donors.**
- **A thorough, established protocol for determining brain death still preserves the viability of donor organs.**
- **It is important for physicians to be precise when explaining the irreversibility of brain death to donors and families, including awareness of new developments in neurobiology that might complicate discussions about death.**

Applying the Rule

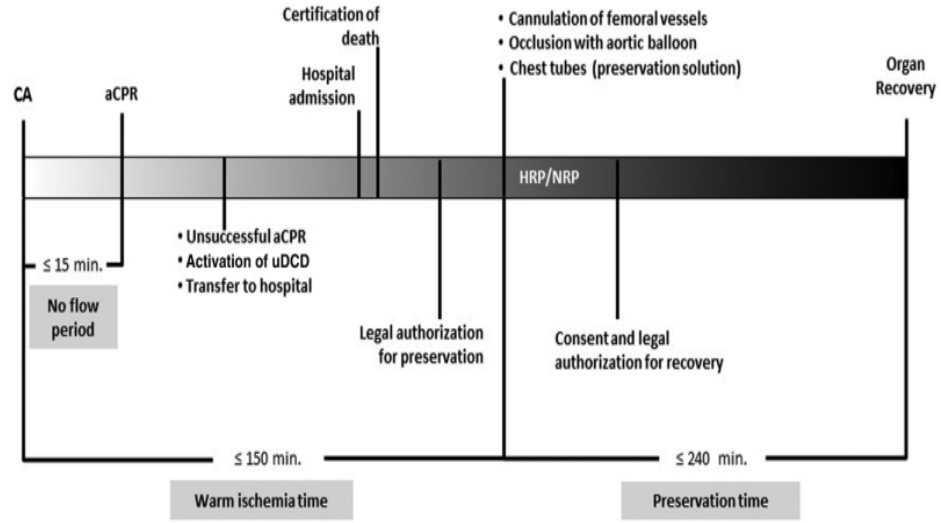
The Dead Donor Rule assumes that it is possible to reliably determine whether a person is living or dead. It also assumes that once an individual is declared dead, they cannot be harmed. When this assumption is applied, a declaration of death by an appropriate medical professional changes an individual's →

Dolaşımın Durmasına Bağlı Ölüm

Transplant International 2016; 29: 749–759

Maestrich Sınıflandırması

Kontrolsüz Ölüm	
1. Hastane dışında ölüm	Şahitli, şahitsiz (Sıcak İskemi zamanı 45 dk?)
2.Başarısız CPR	a. Yb'da beklenmedik ölüm b. Hastanede beklenmedik ölüm
3. Kalp durması beklenen	a. Yb'da beklenen ölüm kontrollü b. Ameliyathanede beklenen ölüm kontrollü destek tedavisi çekileli 30 dk geçen c. Ameliyathanede beklenen ölüm kontrollü destek tedavisi kesilmeden sonra ölüm 30 dk az
Kontrollu Ölüm	
4. Beklenen Beyin ölümü olan birinde	a. YB'da beklenmedik ani kardiyak Arest kontrolsuz b. Yb'da beklenen kardiyak arest kontrollu
5. Ötenazi	Kontrollu



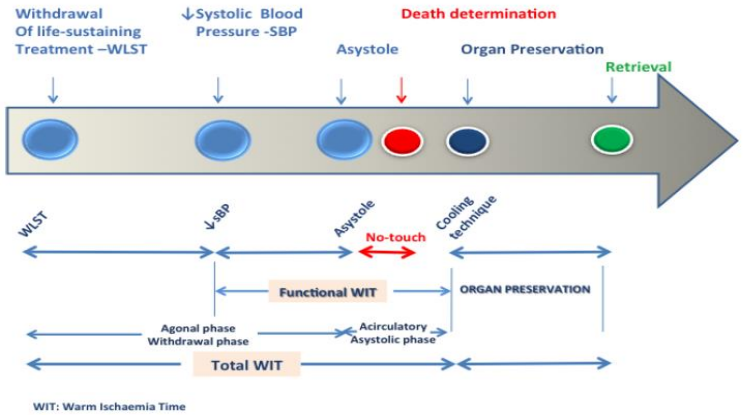
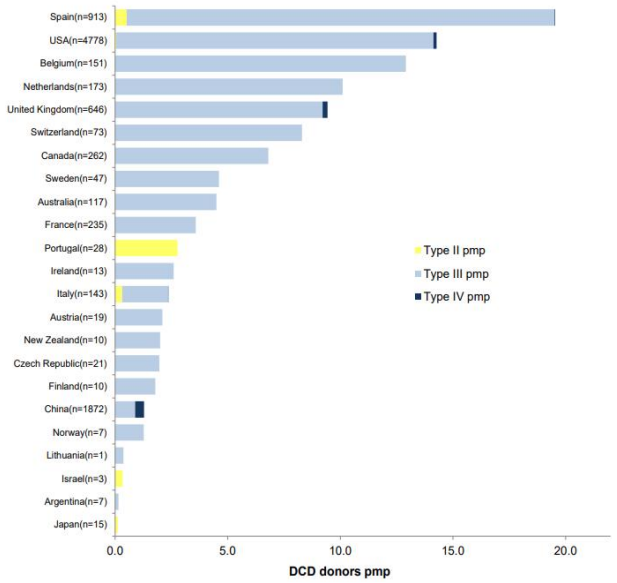


Figure 2 Controlled DCD process.
1 Functional WIT starts when SBP is ≤ 50 mmHg or ≤ 60 mmHg
2 No-touch period: 2 min to 20 min

DCD Kadaverik Donor



Actual DCD donors by Maastricht type (pmp)
2022

DCD donors: 9 544

Type II: 106 (1%)
Type III: 8 798 (92%)*
Type IV: 640 (7%)

DCD activities reported in 23/91 countries

Type II: Unexpected cardiac arrest, unsuccessfully resuscitated

Type III: Planned WLST with an expected cardiac arrest.

Type IV : Sudden or planned cardiac arrest after brain death.

**Includes type V DCD donors (donors after MAiD) for Belgium, Canada , Netherlands and Spain*

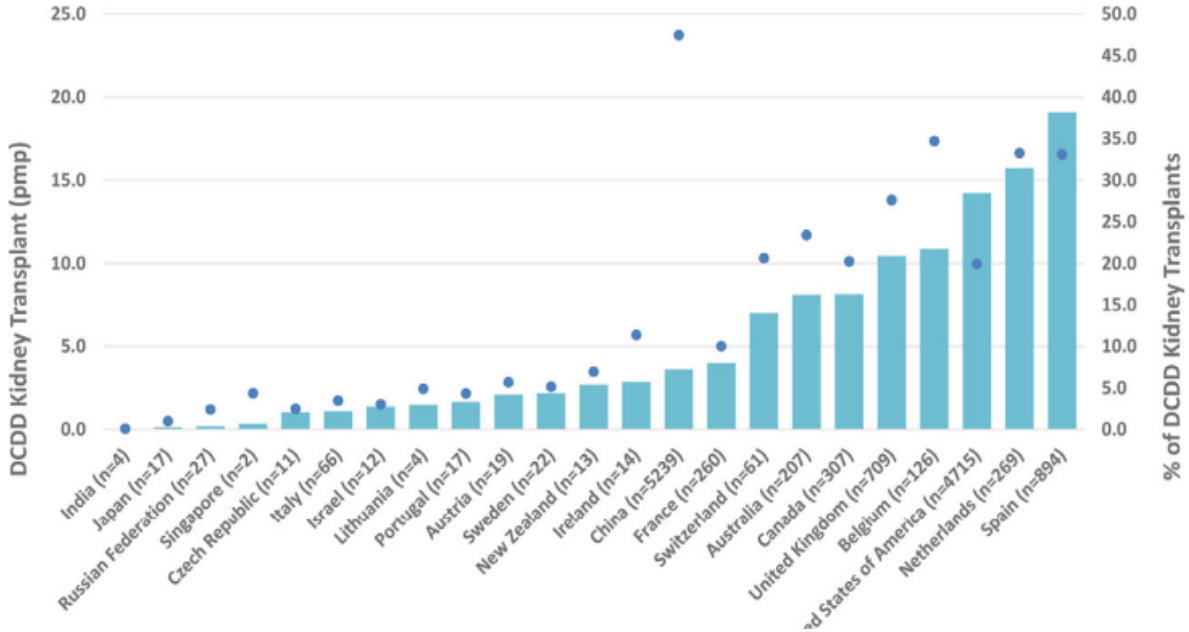


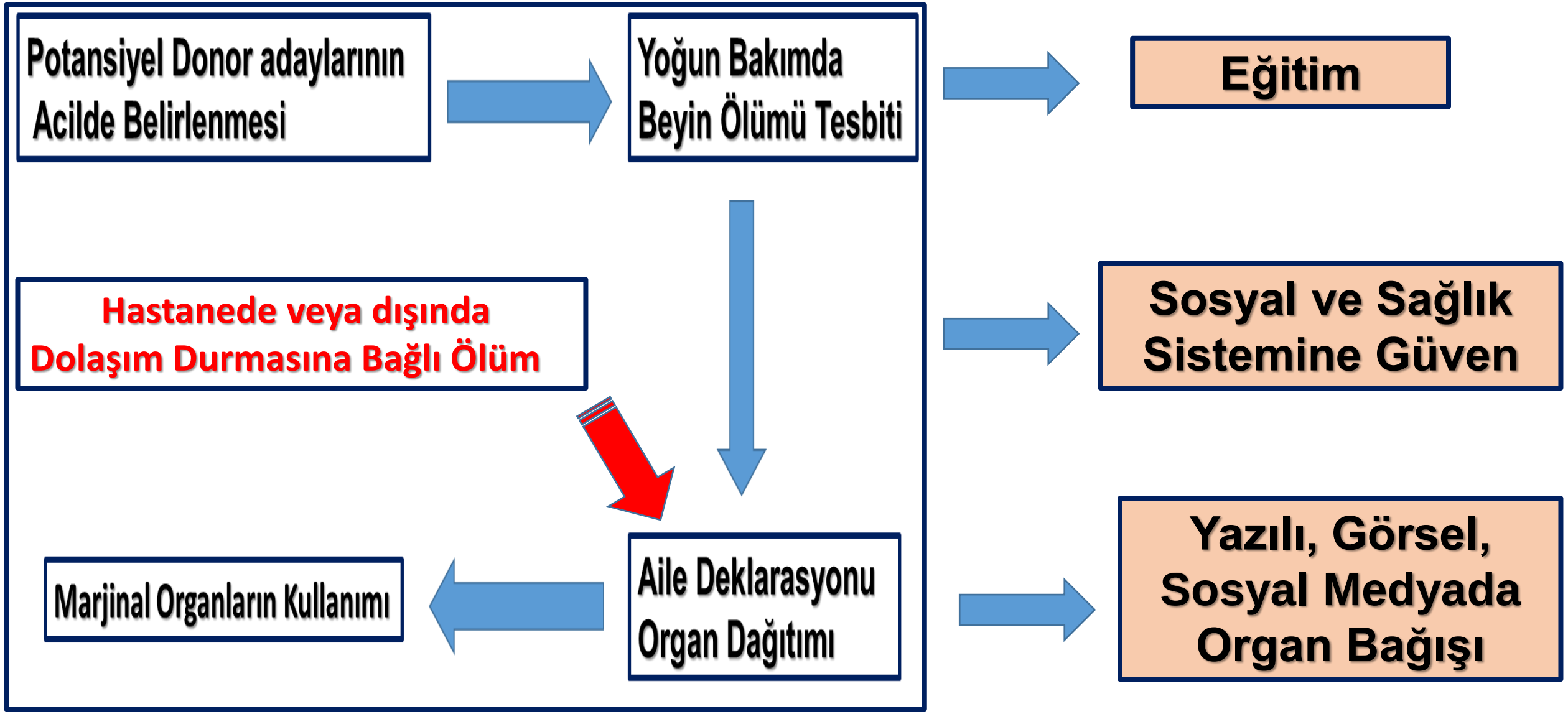
Table 1 | Comparison of transplant outcomes for DCD and DBD donor kidneys

	DCD $n = 3626$	DBD $n = 9684$	*P*-value	Risk-adjusted ratio (95% CI)	Risk ratio *P*-value
Primary non function	3.2% (115/3626)	2.6% (259/9684)	0.06[a]	OR 1.18 (0.9–1.5)	0.21[b]
Delayed graft function	48.5% (1417/2901)	24.9% (1745/5263)	<0.0001[a]	OR 2.81 (2.5–3.2)	<0.0001[b]
1-year eGFR	47.4 (35.6–61.2)	48.7 (37.3–61.1)	0.005[c]	RE −0.16 (−0.9–0.6)	0.69[d]
5-year eGFR	49.6 (35.1–64.7)	48.1 (35.8–62.2)	0.06[c]	RE 0.02 (−1.1–1.2)	0.97[d]
5-year death-censored graft survival	85.9%	84.5%	0.22[e]	HR 0.95 (0.8–1.1)	0.60[f]
5-year all-cause graft survival	76.8%	78.1%	0.15[e]	HR 0.97 (0.9–1.1)	0.55[f]
5-year patient survival	86.5%	89.4%	<0.0001[e]	HR 1.18 (0.8–1.1)	0.28[f]
10-year death-censored graft survival	74.9%	74.3%	0.20[e]	HR 0.95 (0.8–1.1)	0.42[f]
10-year all-cause graft survival	59.8%	60.7%	0.26[e]	HR 0.94 (0.9–1.0)	0.22[f]
10-year patient survival	71.7%	76.7%	<0.0001[e]	HR 0.95 (0.8–1.1)	0.42[f]

Kidney International (2015) 88, 241–249

Seminars in Nephrology, 2022-07-01

Kadaverik Bağışı Nasıl Artırabiliriz



Tıbbı Nedenle Kullanılmayan (TNK) Organ Nedenleri

1. İnfeksiyon (Yoğun Bakımda Bağışçının Ateşi Olması)

2. Marjinal Böbrek Olması (GFH düşük, Böbrek Bx)

3. Cerrahi Neden, Organ Transportunda hatalar

TNK Organ Nedeni İnfeksiyon

Viral İnf ← **İnfeksiyon %20 %0.1 Bulaş** → **Bakteriyal İnf % 5 Bakteremik**

↓

√ 48 Saat Yatış YB Kaynaklı İnfeksiyon → İK ve KK

√ HBsAg, Anti HBs, HBVcAB,(IgM, IgG) HCV AntikorHIV Antikor, EBV, CMV IgM,IgG, Toksoplazma IgG, Sifilis TPPA

√ Lokal İnfeksiyon Kontraendikasyon oluşturmaz

√ DTA Kolonizasyon Kontraendikasyon Oluşturmaz

√ Bakteremik Bağışçı en az 48 saat AB Tedavisi → Alıcı En az 7-11 gün

√ MDR Gram – Kolonizasyon konraendikasyon oluşturmaz

√ MDR Gram – İnfeksiyon vaka bazlı değerlendirilmeli

√ Bakterial Meningit Konraendikasyon oluşturmaz (Tbc,Listeria → ☠ ☠)

Clinical Transplantation. 2019;33:13548.

HCV, HIV + Donor Böbrek Kullanımı

HCV Pozitifden, HCV Negative Böbrek Nakli

The NEW ENGLAND JOURNAL of MEDICINE

CORRESPONDENCE

Trial of Transplantation of HCV-Infected Kidneys into Uninfected Recipients

TO THE EDITOR: Waiting times for kidney transplants exceed 3 to 5 years in many parts of the United States.[1] Yet more than 500 high-quality kidneys from deceased donors with hepatitis C virus (HCV) infection are discarded annually.[2,3] Direct-acting antiviral agents, which are associated with high HCV cure rates and manageable side effects, have created the potential to substantially increase the number of kidney transplants by making HCV-infected kidneys available to HCV-negative candidates on the waiting list.[4-7]

In this open-label, single-group, pilot trial at the University of Pennsylvania (Transplanting Hepatitis C Kidneys into Negative Kidney Recipients [THINKER]; ClinicalTrials.gov number, NCT02743897) we sought to determine the safety and efficacy of transplantation of kidneys from HCV genotype 1–viremic donors into HCV-negative patients, followed by elbasvir–grazoprevir (Zepatier) treatment. An external data and safety monitoring board reviewed all aspects of the trial. The authors vouch for the completeness and accuracy of the data and analysis and for the adherence of the trial to the protocol, available with the full text of this letter at NEJM.org.

Adults who were undergoing dialysis and who had long anticipated waiting times for a kidney transplant were eligible for inclusion in the trial, and patients with conditions that substantially elevate the risks of liver disease, allograft failure, or death were excluded. A physician-led, three-step, informed-consent process was implemented.

Deceased-donor criteria ensured selection of high-quality kidneys (see the Supplementary Appendix, available at NEJM.org). Since elbasvir–grazoprevir is not approved by the Food and Drug Administration (FDA) for patients with HCV genotypes 2 or 3, and a direct-acting antiviral agent for the treatment of patients with those genotypes who have renal failure has not been approved by the FDA, donors were limited to those who had positive qualitative HCV nucleic acid test results and HCV genotype 1. We developed a new protocol for donor genotyping concurrent with organ allocation (see the Supplementary Appendix).

Intravenous glucocorticoids and rabbit antithymocyte globulin were administered to all recipients, followed by oral tacrolimus, mycophenolate mofetil, and prednisone. The HCV viral load was measured in recipients on postoperative day 3; elbasvir–grazoprevir was initiated when the results became positive, and therapy was maintained for 12 weeks.

Among 38 patients who were potentially eligible to participate in the trial, 22 attended an educational presentation, and 14 provided writ-

THIS WEEK'S LETTERS

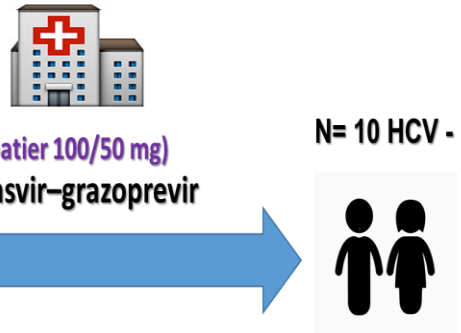
2394 Trial of Transplantation of HCV-Infected Kidneys into Uninfected Recipients

2396 Trial of Pregabalin for Acute and Chronic Sciatica

2397 Treatment of Benzodiazepine Dependence

2400 A Zimbabwean Man with a Severe Headache

2401 Prostate Cancer Screening



% 100 İlk 3 Gün HCV RNA (+), Tedavi ile HCV RNA (-)

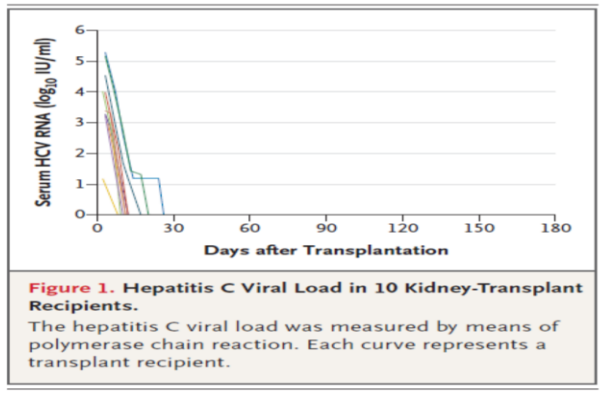
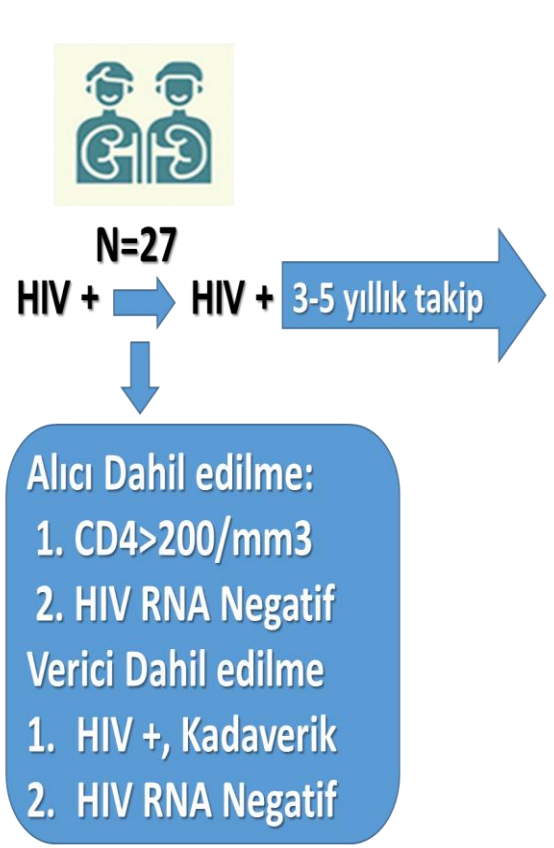


Figure 1. Hepatitis C Viral Load in 10 Kidney-Transplant Recipients.
The hepatitis C viral load was measured by means of polymerase chain reaction. Each curve represents a transplant recipient.

HIV Pozitifden, HIV Pozitife Böbrek Nakli



The New England Journal of Medicine,2015

Akut Böbrek Hasarlı Organ Kullanımı

A Review of Donor Acute Kidney Injury and Posttransplant Outcomes

Neel Koyawala, BA, BS[1] and Chirag R. Parikh, MD, PhD[2]

Abstract. Although over 90 000 people are on the kidney transplant waitlist in the United States, some kidneys that are viable for transplantation are discarded. Transplant surgeons are more likely to discard deceased donors with acute kidney injury (AKI) versus without AKI (30% versus 18%). AKI is defined using changes in creatinine from baseline. Transplant surgeons can use DonorNet data, including admission, peak, and terminal serum creatinine, and biopsy data when available to differentiate kidneys with AKI from those with chronic injury. Although chronic kidney disease is associated with reduced graft survival, an abundance of literature has demonstrated similar graft survival for deceased donors with AKI versus donors without AKI. Donors with AKI are more likely to undergo delayed graft function but have similar long-term outcomes as donors without AKI. The mechanism for similar graft survival is unclear. Some hypothesized mechanisms include (1) ischemic preconditioning; (2) posttransplant and host factors playing a greater role in long-term survival than donor factors; and (3) selection bias of transplanting only relatively healthy donor kidneys with AKI. Existing literature suggests transplanting more donor kidneys with stage 1 and 2 AKI, and cautious utilization of stage 3 AKI donors, may increase the pool of viable kidneys. Doing so can reduce the number of people who die on the waitlist by over 500 every year.

(*Transplantation* 2020;104: 1553–1559).

TABLE 1.
Discard rate by AKI stage

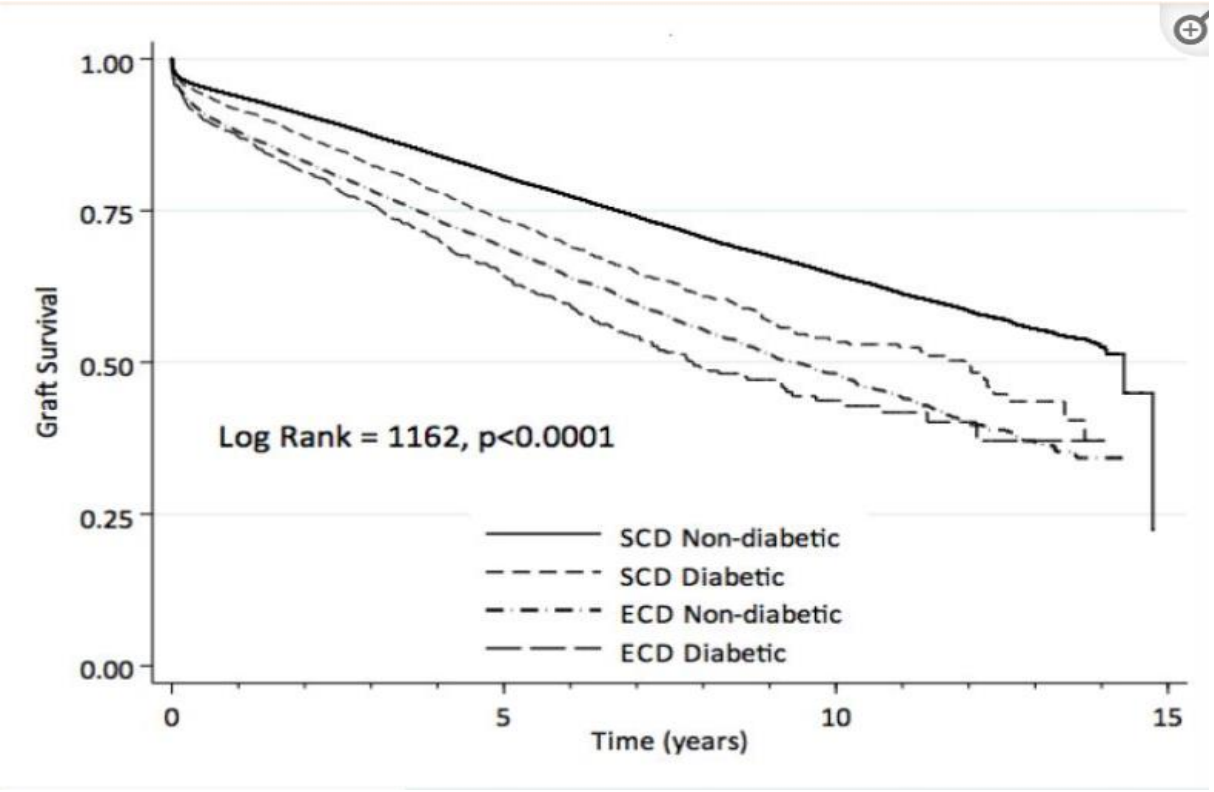
AKI status	Discard rate[a]	N discarded in Hall et al[8a]	National estimate of deceased donor kidneys discarded annually[b]	National estimate of discard annually assuming no AKI rate[c]	Estimated kidneys that could be saved annually[d]
No AKI	18%	433	2484	2484	
Stage 1	26%	145	832	572	260
Stage 2	35%	66	379	194	184
Stage 3	35%	99	304	159	145
		697	3998	3408	589

TABLE 2.
Number of studies demonstrating increase, no change, and decrease in posttransplant outcomes in AKI vs non-AKI donor group

Outcome	Overall effect of donor AKI on outcome[a]	No. of studies with outcome (N=36) (references)		
		Decreased incidence in donor AKI group	No change	Increased incidence in AKI group
Delayed graft function	Increased incidence	0	2 ([45,46])	23 ([8,14,24-44])
Acute rejection	No effect	0	9 ([27,37,41-43,45-48])	0
Graft function (eGFR, sCr)	No effect	2 ([33,49])	20 ([8,27,34-42,45-48,50-54])	0
Graft failure	No effect	0	25 ([7,9,14,24,25,27,28,30-32,34,36-41,44-48,53,55,56])	4 ([26,29,33,49])
Recipient survival	No effect	0	14 ([7,24,32,37-41,43,45-48,53])	0

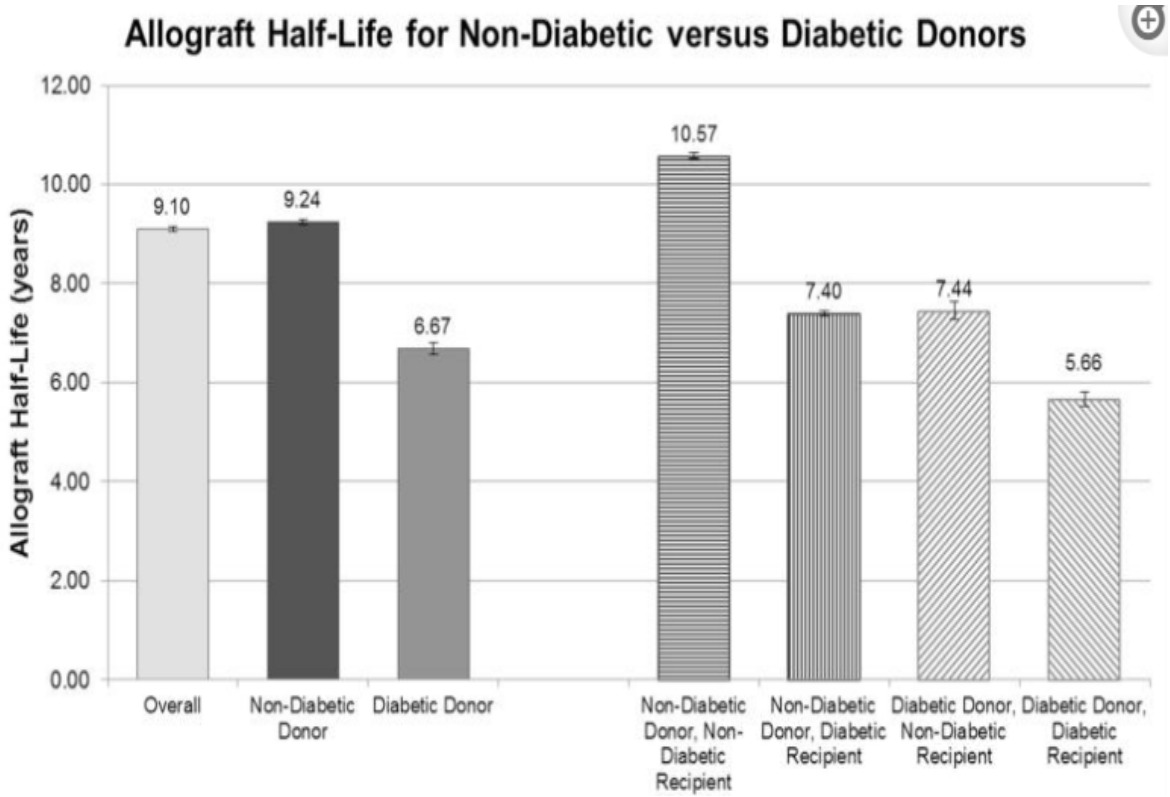
Kadavra Diyabetik Böbrek Kullanımı

N=1982 Diyabetik Donor,
N=11087 Non Diyabetik Donor
UNOS 1995-2004



Am J Transplant. 2012;12:2098–2105

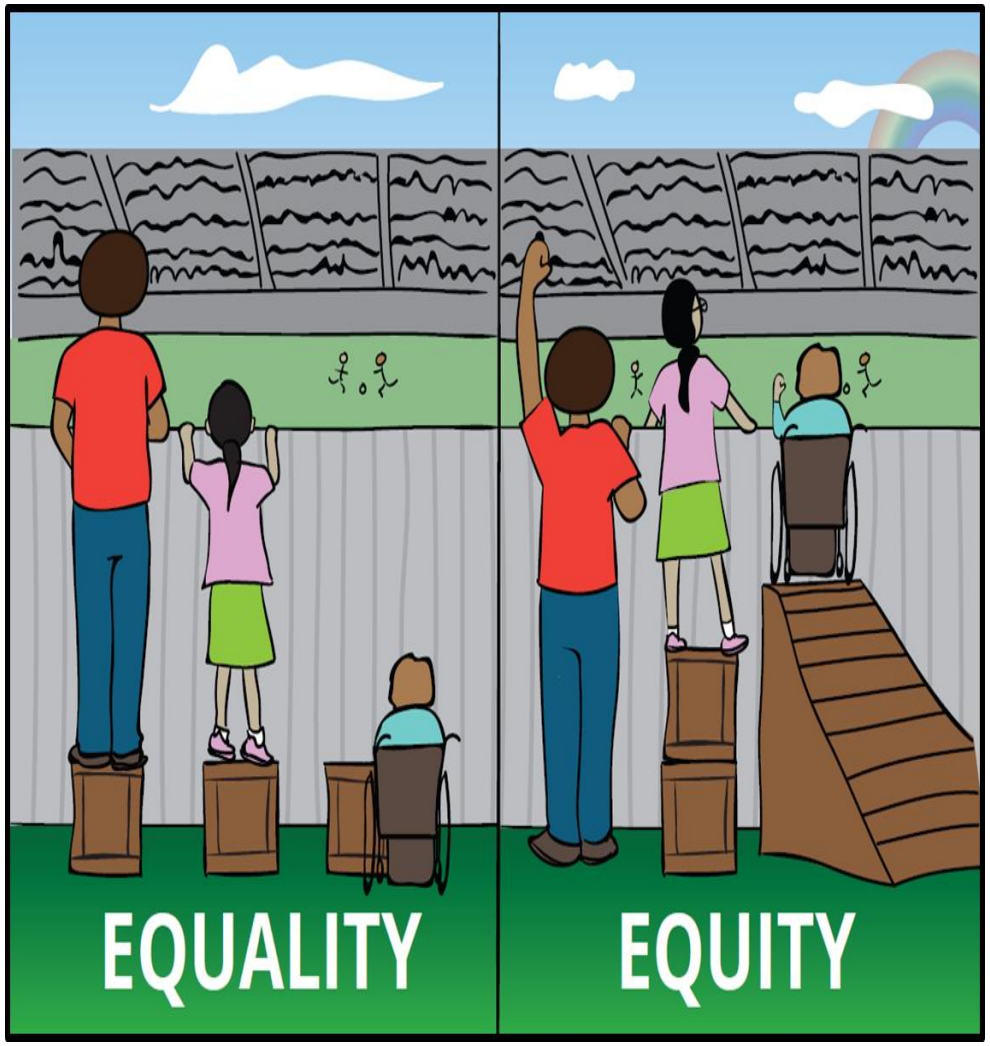
N=9074 Diyabetik donor,
N:152555 Non Diyabetik Donor
UNOS: 199-2014



Kidney Int. 2016;89:636–647.

Organ Dağıtımı ve Eşleştirme

Organ Dağıtımı



Eşitlik ve Adalet

Pediatrik Hasta

Bekleme Süresi

Medikal Aciller

Verimlilik

Sensitizasyon

HLA uyumu

Uzun ömürlü eşleştirme

Puanlama ve Eşleştirme

Türkiye

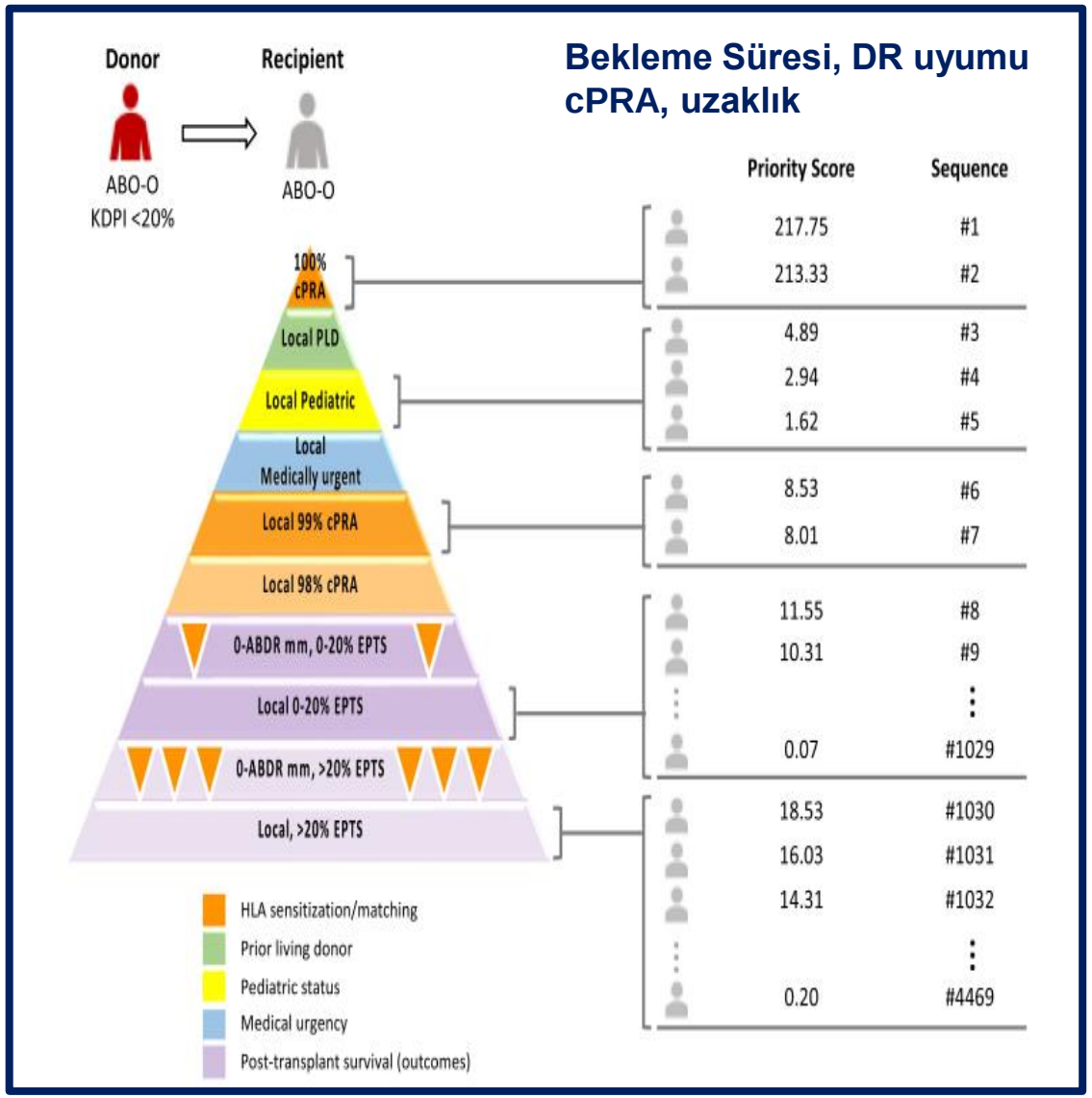
DEĞERLENDİRME KRİTERİ		PUAN
Doku Uyumu		Tam uyum (2A 2B 2DR uyumu) durumunda şarta bağlı olmaksızın alıcının olduğu yere gider Tam uyum dışındaki durumlarda uyumlu her DR antijeni için 150, B antijeni için 50, A antijeni için 5 puan verilir.
Donörün çıktığı bölge		1000
Donörün çıktığı merkez		250
Alıcı yaş grubu	11 ve altı	Doku uyumu puanı X 2.5
	12-17	Doku uyumu puanı X 1.5
	18 ve üzeri	Doku uyumu puanı X 1
Diyalize girme süresi		Her ay için 3 puan

USA

Table 2. Priority point system for new kidney allocation

Factor	Points Awarded
For qualified time spent waiting	1 per year (as 1/365 per day)
Degree of sensitization (CPRA)	0–202
Prior living organ donor	4
Pediatric candidate if donor KDPI<0.35	1
Pediatric candidate (age 0–10 yr at time of match) when offered a zero antigen mismatch	4
Pediatric candidate (age 11–17 yr at time of match) when offered a zero antigen mismatch	3
Share a single HLA-DR mismatch with donor	1
Share a zero HLA-DR mismatch with donor	2

These points will be used to rank candidates in each of the categories listed in



JASN 25;1842-1845, 2014

Puanlama ve Eşleştirmede Eksiklerimiz

Kadavra böbreği ve Alıcılarla ile ilgili veri analizimiz mevcut değil

Kaliteli böbrek yaşlı birisine, kalitesiz böbrek genç birisine nakil şansı

Canlı vericisi olan kombine nakil adaylarına öncelik

Liste Başındaki Sensitize hasta grubuna nakil yapamıyoruz

Eurotransplant Senior Program (ESP)

Amaç: Yaşlı donörlerden alınan böbreklerin daha verimli kullanılması ve yaşlı hastalara (yaşlıya yaşlı) transplantasyonun sunulmasıdır.

- ***≥65 yaşlı donörlerden*** HLA'ya bakılmaksızın ***≥ 65 yaşlı alıcılara*** *böbrek* tahsis eder.
- İskemik hasarı azaltmak için, böbrekler mümkün olan en kısa soğuk iskemi süresi
- Duyarlılaştırılmamış (PRA < %5) ilk transplant alıcıları sisteme dahil edildi.
- ESP, donör kreatinin klirensinin ***< 70 mL/dk*** olduğu durumlarda ***her iki böbreği tek bir alıcıya*** nakil.

Sonuç

- **Daha kısa** soğuk iskemiye ve **daha az** DGF yol açtı, ancak %5-10 daha yüksek ret oranları bildirilmiş.

Schachtner et al; Clinical Kidney Journal, sfz118 (2019)

Sensitize Hasta Grubuna Organ Eşleştirilmesi

Eurotransplant

Kabul edilebilir HLA Uyumsuzluğu
Acceptable Missmatch (AM)

UNOS

Kabul edilemez HLA Uyumsuzluğu
Unacceptable Missmatch

Eurotransplant Sensitize Hastaya Böbrek Eşleştirilmesi

Kadavra Böbrek

<65 YAŞ

AM Program
Acceptable Missmatch

>65 YAŞ

ESP

Sanal PRA>%85

Kriterler
√ Ortanca Bekleme süresi
√ 2 veya 1DR Uyum şart

Acceptable Missmatch Ag
√ CDC bazlı Tarama
√ Hla gene transfekte Hücre Dizisi
√ HLA Matchmaker
√ Luminex, >3000MFI,Sensitizasyon

Front İmmunology June 2021,Transplant Immunology 33 (2015) 51–57

UNOS Sensitize Hastaya Böbrek Eşleştirmesi

Kabul edilemez HLA Uyumsuzluğu
(Unacceptable HLA Missmatch)

↓

Hesaplanmıs PRA >%85

↓

Merkez Luminex DSA İstenmeyen Ag

↓

Sanal Kross Match

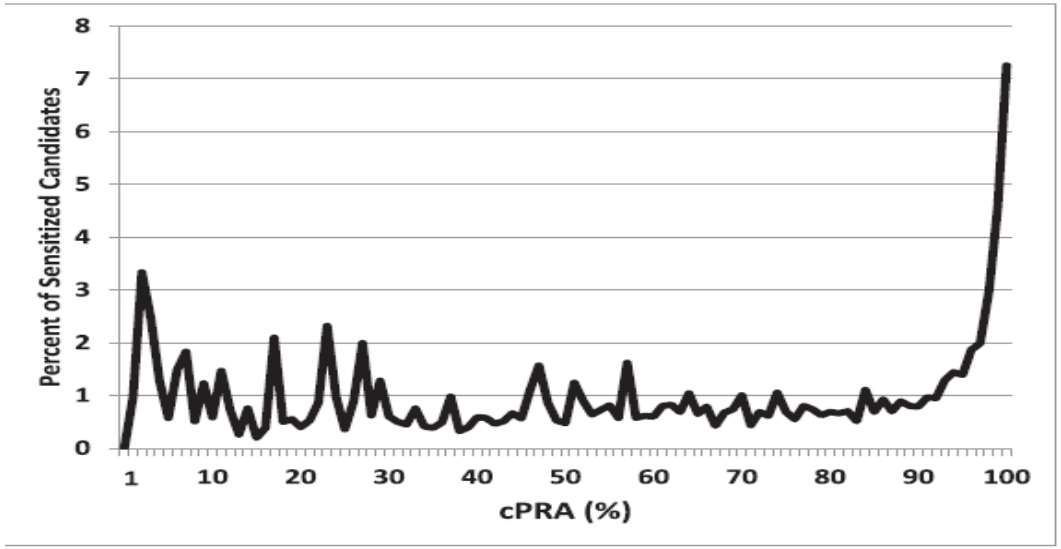


Table 3. Priority points awarded based on CPRA>19%

CPRA (%)	Points
0–19	0
20–29	0.08
30–39	0.21
40–49	0.34
50–59	0.48
60–69	0.81
70–74	1.09
75–79	1.58
80–84	2.46
85–89	4.05
90–94	6.71
95	10.82
96	12.17
97	17.3
98	24.4
99	50.09
100	202.1

Kidney International Reports:7:1179-1189

Clin J Am Soc Nephrol 11: 684–693, 2016

Marjinal Böbrek Kullanımı

Marjinal Kadaverik Donör Kriterleri (ECD)

Donör yaşı >60 yıl

Donör yaşı 50-59 yıl arasında olup

- Serum kreatinin >1.5 mg/dl veya,
- Ölüm nedeni serebrovasküler travma veya,
- Hipertansiyon öyküsü olması

} ***En az iki tanesi olması***

ECD'li böbrek nakiller kadaverik nakillerin %17'si oluşturuyordu

ECD'li olmayanlara göre greft yetmezliği riski %70, DGF ve mortalite riski daha yüksek

Transplantation 2002; 74: 1281 && Am J Transplant 2005; 5: 843

Marjinal Kadaverik Böbrek Değerlendirmede Biyopsi

Kidney Donor Risk Indeks

Referans Bağışçıya göre, posttransplant greft kaybının *rölatif riskini* belirler

Referans Bağışçı: 40 yaş, beyaz, 1.70 boyunda, 80 kğ, creatinin: 1.0 , normotansif, KŞ normal, HCV negatif, Ölüm nedeni kardiyovasküler dışı

Yaş	Diyabetes Mellitus öyküsü
Boy	Hipertansiyon öyküsü
Kilo	Serum kreatinin düzeyi
Etnik	HCV varlığı
Ölüm nedeni	DCD (Ölmüş Kalp Donasyon durumu)

KDPI>%85

Diyabet, HT ve veya ABH

Böbrek Biyopsisi

Transplantation 2009;88: 231–236

0041-1337/95/6004-334$03.00/0
TRANSPLANTATION
Copyright © 1995 by Williams & Wilkins

Vol. 60, 334–339, No. 4, August 27, 1995
Printed in U.S.A.

GLOMERULOSCLEROSIS AS A DETERMINANT OF POSTTRANSPLANT FUNCTION OF OLDER DONOR RENAL ALLOGRAFTS

LILLIAN W. GABER,[1] LINDA W. MOORE, RITA R. ALLOWAY, M. HOSEIN AMIRI, SANTIAGO R. VERA, AND A. OSAMA GABER

Department of Pathology and Division of Transplantation, Department of Surgery, The University of Tennessee—Memphis, Memphis, Tennessee

Transplantation of kidneys from older donors is being advocated to expand the organ donor pool. However, the prevalence of atherosclerosis and age-induced renal structural alterations account for the variable function of allografts procured from these older donors. Pretransplant biopsies are sometimes used to evaluate kidneys from older donors, but to date there are no defined criteria correlating the extent of structural alterations in these kidneys to subsequent function. We investigated the effect of glomerulosclerosis, a marker for nephrosclerosis, on graft outcome. Sixty-five baseline biopsies of kidney allografts were retrospectively analyzed to identify a referent point of glomerulosclerosis that correlated with inferior graft outcome. Age and death from nontraumatic cerebrovascular injuries were the main correlates for donor glomerulosclerosis ($P<0.001$). Allografts with poor function at 6 months defined as serum creatinine >2.5 mg/dl (n=13) or nephrectomy (n=4) had a mean of 20% glomerulosclerosis at the time of implantation compared with only 2% sclerosis in allografts with good function ($P<0.05$). Delayed graft function occurred in 22% and 33% of recipients with no glomerulosclerosis and those with less than 20% glomerulosclerosis, respectively. In contrast, patients receiving kidneys with >20% sclerosis had an 87% incidence of delayed function ($P<0.05$). Moreover, graft loss occurred in 7% of recipients of kidneys with less than 20% sclerosis and in 38% of recipients with >20% sclerosis ($P<0.04$). Measurements of serum creatinine in the donors did not distinguish the different degrees of glomerulosclerosis found on biopsy. Our data indicate that donor glomerulosclerosis greater than 20% increases the risk of delayed graft function and poor outcome of transplanted kidneys. Therefore, we advocate the use of routine biopsies of kidneys from older (>50 yrs) donors and those donors with nontraumatic cerebrovascular accidents, despite seemingly normal preprocurement serum creatinine.

Increasing demand for cadaveric kidneys has motivated transplant centers to consider alternatives for maximizing the rate of acceptance of cadaver donor organs. Acceptance of older donors has the potential of increasing the organ donor pool by 20% (*1*). However, data regarding the long-term function and survival of such kidneys remains unsettled. Although several studies have demonstrated comparable survival rates for kidneys from young and old donors, (*2–4*) others have expressed more caution in using old donor kidneys due to the increased risk of primary graft failure, delayed graft function (DGF),* rejection, and overall reduction of graft survival (*5, 6*). This discrepancy can be largely explained by the shortcomings of the current criteria used for screening old donors. Clinical criteria used for donor evaluation based on detailed medical and social history and laboratory investigations have been largely adequate for identifying high-risk donors or marginal kidneys but have not been age-discriminatory (*3*). For example, age-related decline in renal function is often masked by a normal serum creatinine in elderly individuals—therefore, such marginal kidneys will be identified as acceptable. In addition, estimation of nephrosclerosis by gross examination of the kidney is, at best, crude and is capable of only distinguishing extreme renal scarring. Accurate determination of the structural and functional status of the kidneys at the time of procurement is particularly important for aging kidneys, since the immunologic and hemodynamic changes induced by transplantation aggravate the preexisting lesions of aging. Taking these factors into account, it is essential to establish specific selection criteria for old donors that guarantee acceptance of grafts with no or with minimal preexisting pathology. Recently, structural-based criteria for acceptance of extrarenal allografts have been identified (*7*). To date, however, and despite sporadic use of renal biopsies for donor kidney evaluation, there have been no published reports of histologic features that identify high-risk kidney allografts from old donors.

Epidemiologic and biopsy studies of renal changes secondary to aging support the view that older donor kidneys are more likely to exhibit a greater degree of nephrosclerosis, reduction of renal plasma flow, and a decline in renal function (*8–12*). Furthermore, examination of donor kidney biopsies obtained at the time of transplantation has shown a greater prevalence of age-related pathology, with a striking 80% incidence of histologic manifestations of chronic nephron loss in kidneys procured from donors older than 50 years (*13*). The high prevalence of renal pathology in the older donors can be attributed in part to the mechanism of brain death in this group, which has been largely due to nontraumatic cerebrovascular hemorrhage (*14*), thus preselecting individuals with hypertension or vascular atherosclerosis, both highly associated with renal abnormalities (*11, 12*).

We therefore hypothesized that glomerulosclerosis, being a marker for nephron loss, will have a direct negative effect on

[1] Address correspondence to Lillian W. Gaber, M.D., Department of Pathology, University of Tennessee—Memphis, 899 Madison Ave., Room 576—Main, Memphis, TN 38163.

* Abbreviations: CVA, cerebrovascular accident; DGF, delayed graft function; MAP, mean arterial blood pressure.

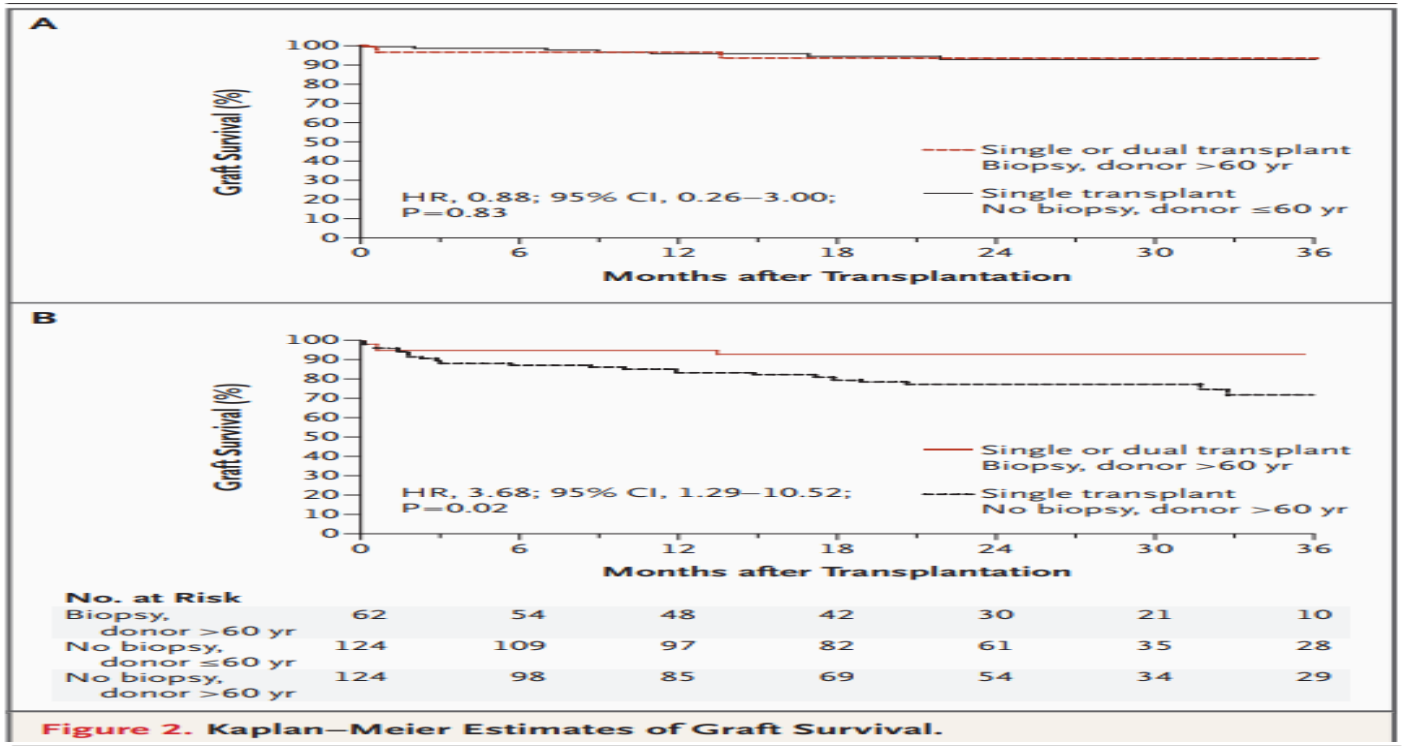


Figure 2. Kaplan—Meier Estimates of Graft Survival.

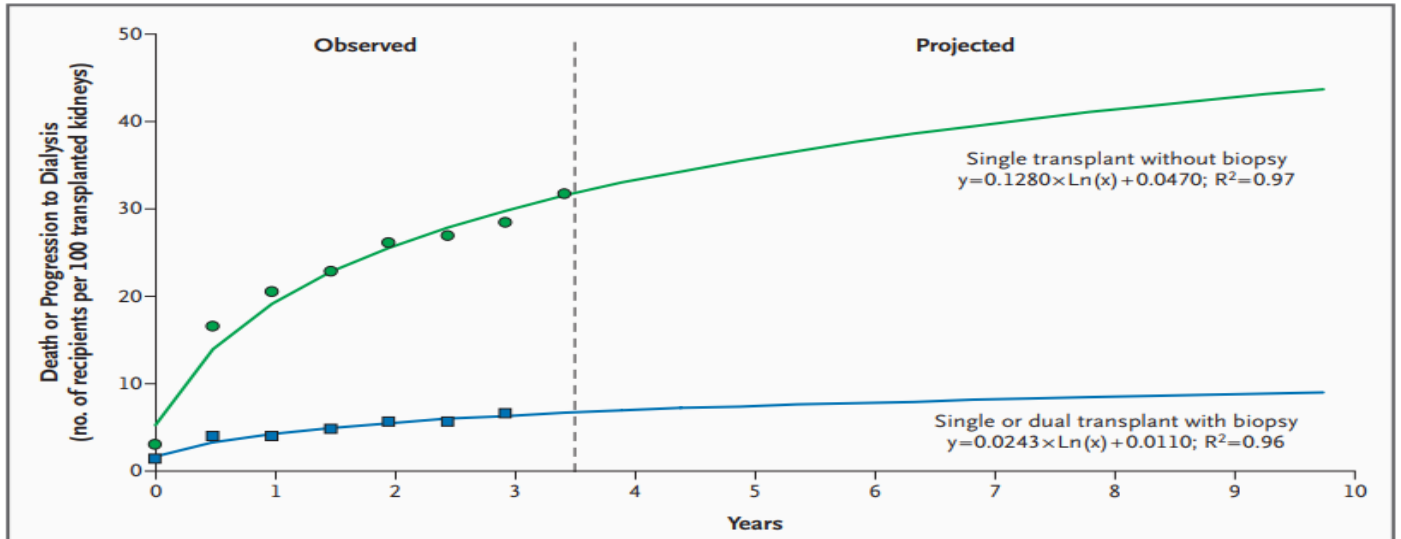


Figure 3. Death or Progression to Dialysis.

N Eng J Med. 2006 Jan 26;354(4):343-52

Böbrek Biyopsi

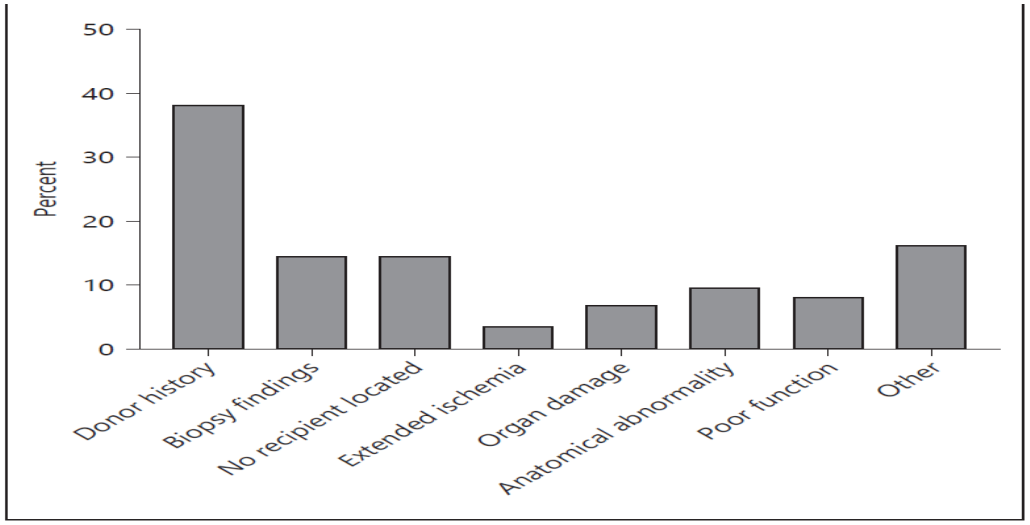
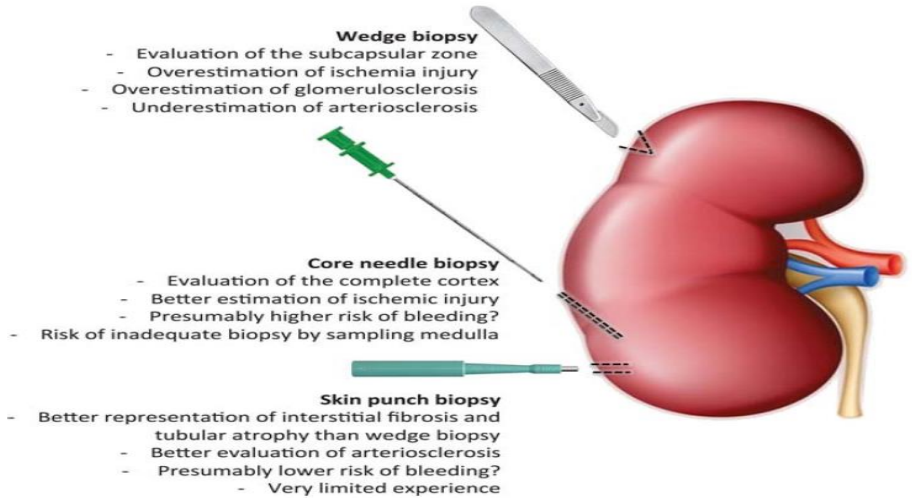


Fig. 2. Common causes of kidney discard in the US. Bars represent percent of 36,700 kidneys discarded between 2000 and 2015. From



Remuzzi Score

SCORING SYSTEM

Glomerular global sclerosis
- 0 = none
- 1+ = <20%
- 2+ = 20 to 50%
- 3+ = >50%

Tubular atrophy
- 0 = absent
- 1+ = <20% of tubuli affected
- 2+ = 20 to 50%
- 3+ = >50%

Interstitial fibrosis
- 0 = absent
- 1+ = <20% replacement by fibrous tissue
- 2+ = 20 to 50%
- 3+ = >50%

Arterial and arteriolar narrowing
- 0 = absent
- 1+ = increased wall thickness less than diameter of the lumen
- 2+ = wall thickness equal or slightly greater than diameter of the lumen
- 3+ = wall thickness far exceeds the diameter of the lumen

MARGINAL KIDNEY DONOR
- Age >60 yr
- History of diabetes or hypertension
- Clinical proteinuria (≤ 3 g/24 h)

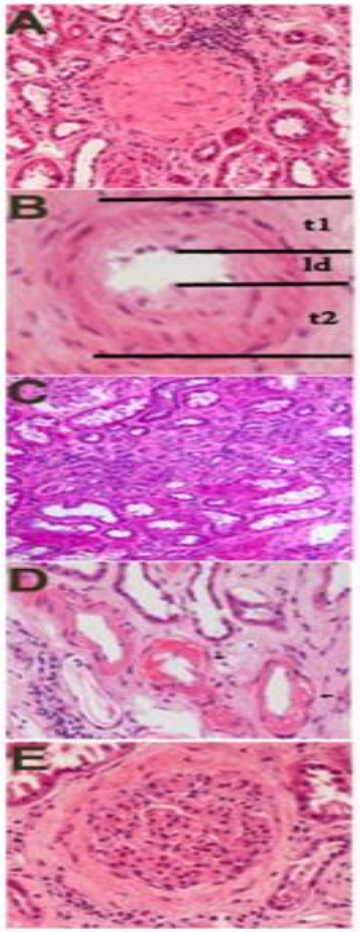
→ Macroscopic evaluation of the kidneys
→ Major vascular, ureteral, or parenchymal abnormalities
→ **Discard**

→ No abnormalities
→ Pre-transplant biopsy of both kidneys
→ Eligibility for double or single transplant according to a standardized score system

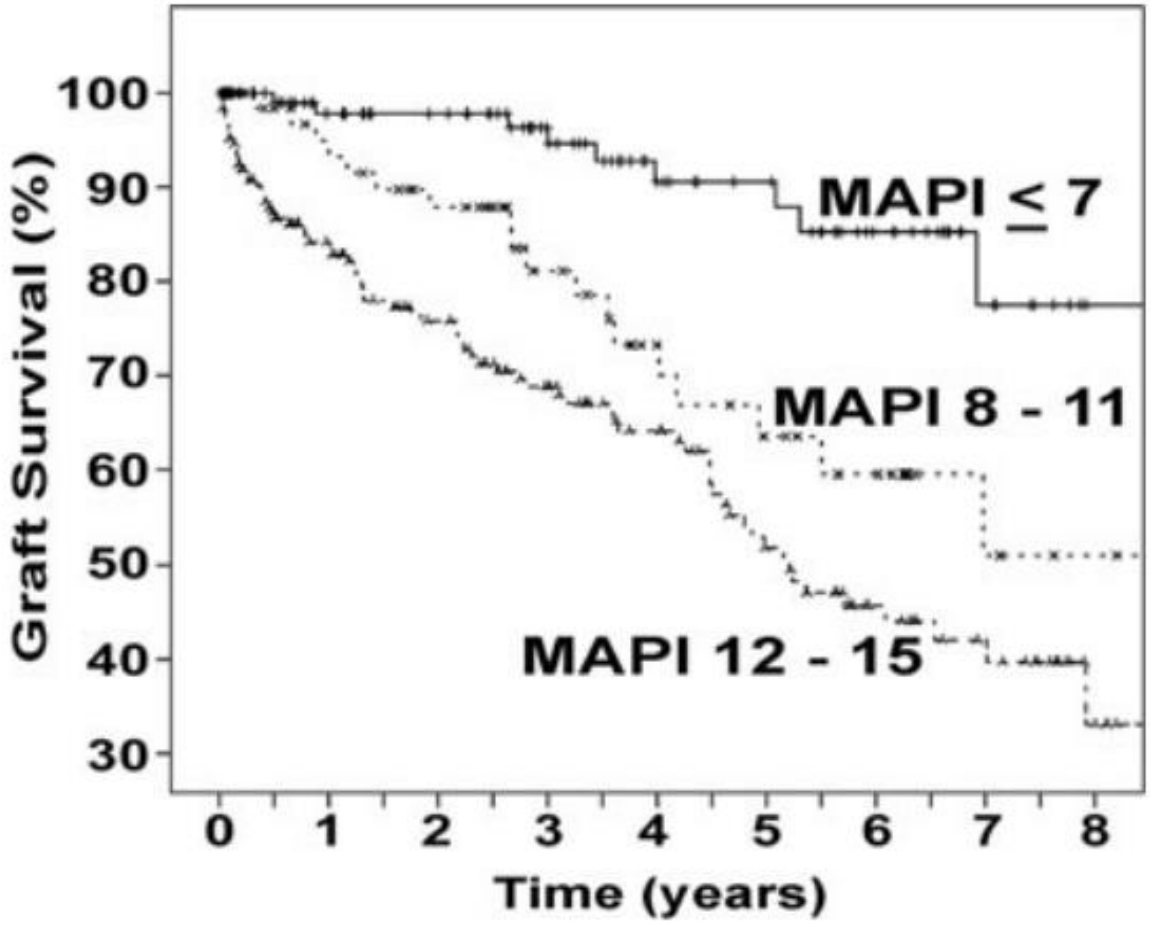
Final Grading
- 0 to 3 mild → ***single***
- 4 to 6 moderate → ***double***
- 7 to 12 severe → ***discard***

Böbrek Biyopsi

Maryland Aggregate Pathology Index



	MAPI Score
Global glomerulsclerosis (GS)>15%	2 points
Wall to Lumen Ratio (WLR) = (t1+t2)/ld WLR >0.5	2 points
Scar is a focus of sclerosis and renal parenchymal fibrosis and atrophy involving at least 10 tubules	3 points
Arteriolar hyalinosis, defined as amorphous, homogeneous eosinophilic deposits in the wall of arterioles	4 points
Periglomerular fibrosis, defined as thickening, wrinkling and reduplication of the Bowman's capsule	4 points



Clin Transplant 2014: 28: 897–905 DOI: 10.1111/ctr.12400

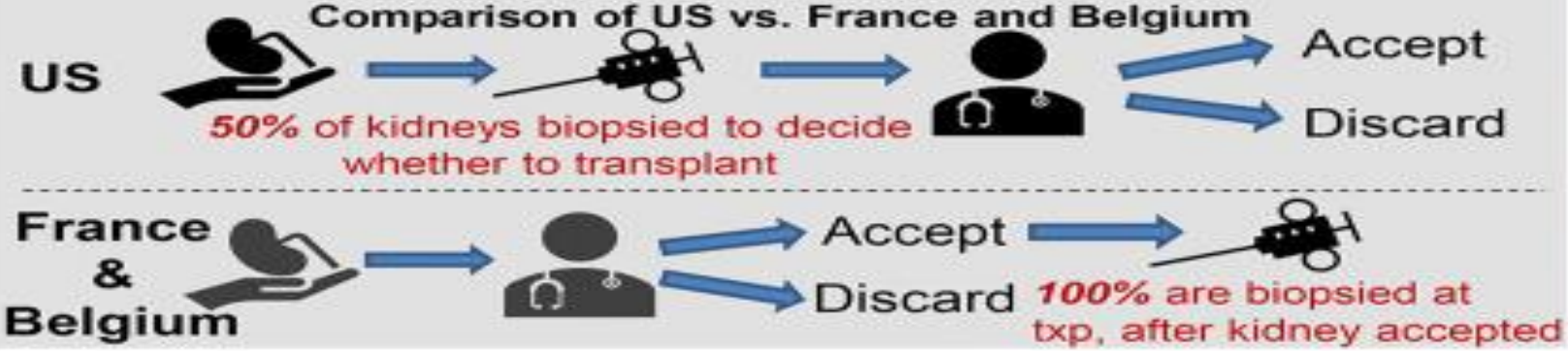
Klinik Kararda Sadece Böbrek Biyopsi Yeterli mi ?

Do Allocation Kidney Biopsies Add Incremental Value in Predicting How Long A Kidney Will Survive After Transplantation?

JASN
JOURNAL OF THE AMERICAN SOCIETY OF NEPHROLOGY

BACKGROUND

Comparison of US vs. France and Belgium



METHODS

*Multivariable Cox regression model of death-censored allograft failure in development set (2 French centers) and validation set (2 Belgian centers)

**Compared predictive accuracy between baseline model and then model with addition of biopsy data

*Matched kidneys discarded in US due to histology to nearly identical kidneys transplanted in France

RESULTS

*1,629 kidney recipients at 2 French centers:
- C-stat without histology: 0.635
- C-stat with histology added: 0.646

*Similar results at Belgian centers

*493 kidneys (45%) discarded in 2015 – 2016 in the US matched to 493 transplanted French kidneys

*Those matched and transplanted kidneys had acceptable allograft survival: 93.1%, 80.7%, and 68.9% at 1, 5, and 10 years, respectively

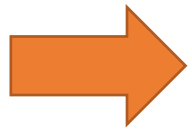
CONCLUSION

Kidney histology did not provide additional value in determining organ quality. Many kidneys discarded due to biopsy findings would have benefitted US wait-listed patients.

doi: 10.1681/ASN.2020040464

Çift (Dual) veya Tek Böbrek

- Donor Yaşı >60
- Giriş GFH <65 ml/dk
- Kreatinin > 2.5 mg/dl Çıkarımda
- Glomerulosklerozu %15-50
- Uzun Süreli HT veya DM +



2 Kriter +

Dual Böbrek

Kadavra Donorleri Nasıl Artırabiliriz
Nasıl Daha Etkili Kullanabiliriz

√ Organ bağış oranlarının arttırılması
(Sosyal Farkındalık, Eğitim, Bilime Olan inanç, Şeffaflık)

√ Data analizi, Puanlamanın gözden geçirilmesi, simülasyon
√ A2B → B Kan grubuna

√ Yaşa göre organ dağıtımının yeniden değerlendirilmesi
√ Sensitize Hasta Grubu Programı

√ Marjinal Böbrek kullanımının artırılması
√ Non-heart beating donor için
→ Hukuki altyapının hazırlanması ve kuralların konması

Kadavra Donorleri Nasıl Artırabiliriz
Nasıl Daha Etkili Kullanabiliriz

√ Organ bağış oranlarının arttırılması için gerekli önlemlerin alınması

√ Marjinal Böbrek kullanımının artırılması

√ Data analizi, Puanlamanın gözden geçirilmesi, simülasyon çalışması

√ PRA, sensitize hasta, immunolojik düzenleme

√ Yaşa göre organ dağıtımının yeniden değerlendirilmesi (Genç ve Yaşlı hasta programı)

√ Non-heart beating donor için hukuki altyapının hazırlanması ve kuralların konması

UNOS Yeni Organ Eşleştirmenin Sonuçları

Pozitif

- √ Diyaliz >10 yıl, %13 azaldı
- √ cPRA>%99, %17 azaldı
- √ cPRA>98% nakil oranları arttı, yüksek sensitize hastalar azaldı
- √ KDPI<20%, böbreklerin %80'I <40 yaş alıcılara takıldı
- √ Alıcı-verici yaş farkı azaldı
- √ Uzun süredir diyalizde olan hastaların nakil şansı arttı (diyalize başlama süresi hesaplandığı için)
- √ AA nakil arttı, hispanik azaldı

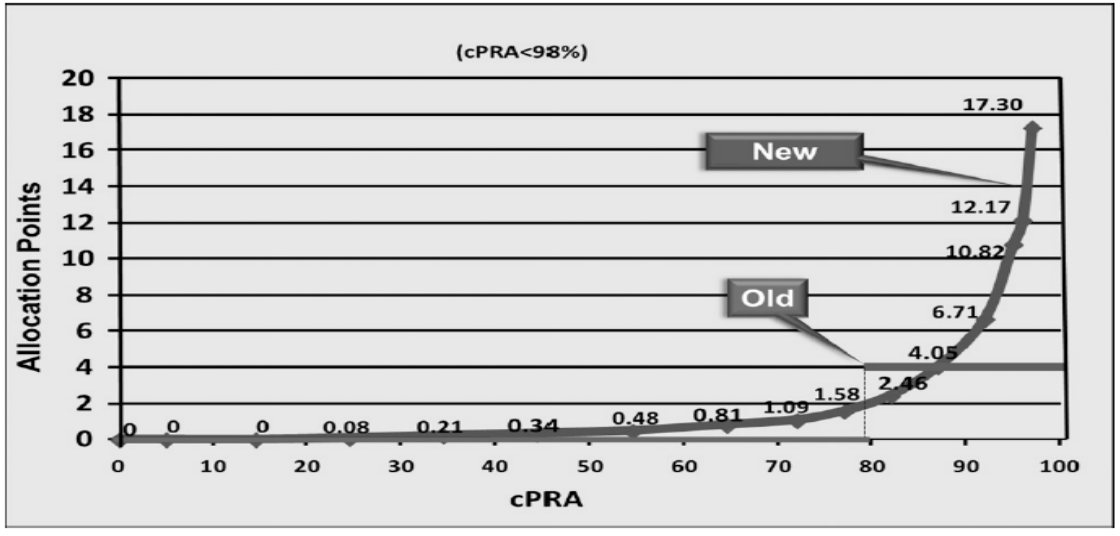
Negatif

- √ Non-fonksiyone graft oranları değişmedi
- √ Bekleme listesi mortalitesi değişmedi
- √ 0 MM %8.2 den %4.7'ye
- √ 0 DR MM %19.8'den %16.8'e (cPRA nedeni ile)
- √ Pediatrik nakiller azaldı
- √ CIT uzadı
- √ DGF %25'den %30'a çıktı
- √ Böbrek kullanmama oranları arttı

Human Immunology:78:9-15,2016

HLA tam uyumlu 60 Yaşında Hasta

25 Yaşında 2DR, 1A, 1B hasta alam



Wait-Listed Candidates			
KDPI≤0.20	KDPI 0.21–0.34	KDPI 0.35–0.85	KDPI>0.85
Local CPRA 100%	Local CPRA 100%	Local CPRA 100%	Local CPRA 100%
Regional CPRA 100%	Regional CPRA 100%	Regional CPRA 100%	Regional CPRA 100%
National CPRA 100%	National CPRA 100%	National CPRA 100%	National CPRA 100%
Local CPRA 99%	Local CPRA 99%	Local CPRA 99%	Local CPRA 99%
Regional CPRA 99%	Regional CPRA 99%	Regional CPRA 99%	Regional CPRA 99%
Local CPRA 98%	Local CPRA 98%	Local CPRA 98%	Local CPRA 98%
0 HLA mm top 20	0 HLA mm	0 HLA mm	0 HLA mm
Prior living donors	Prior living donors	Prior living donors	Local, regional adult
Local pediatric	Local pediatric	Local	National adult
Local top 20	Local adult	Regional	
0 HLA mm bottom 80	Regional pediatric	National	
Local bottom 80	Regional adult		
Regional pediatric	National pediatric		
Regional top 20	National adult		
Regional bottom 80			
National pediatric			
National top 20			
National bottom 80			

Table 2. Priority point system for new kidney allocation

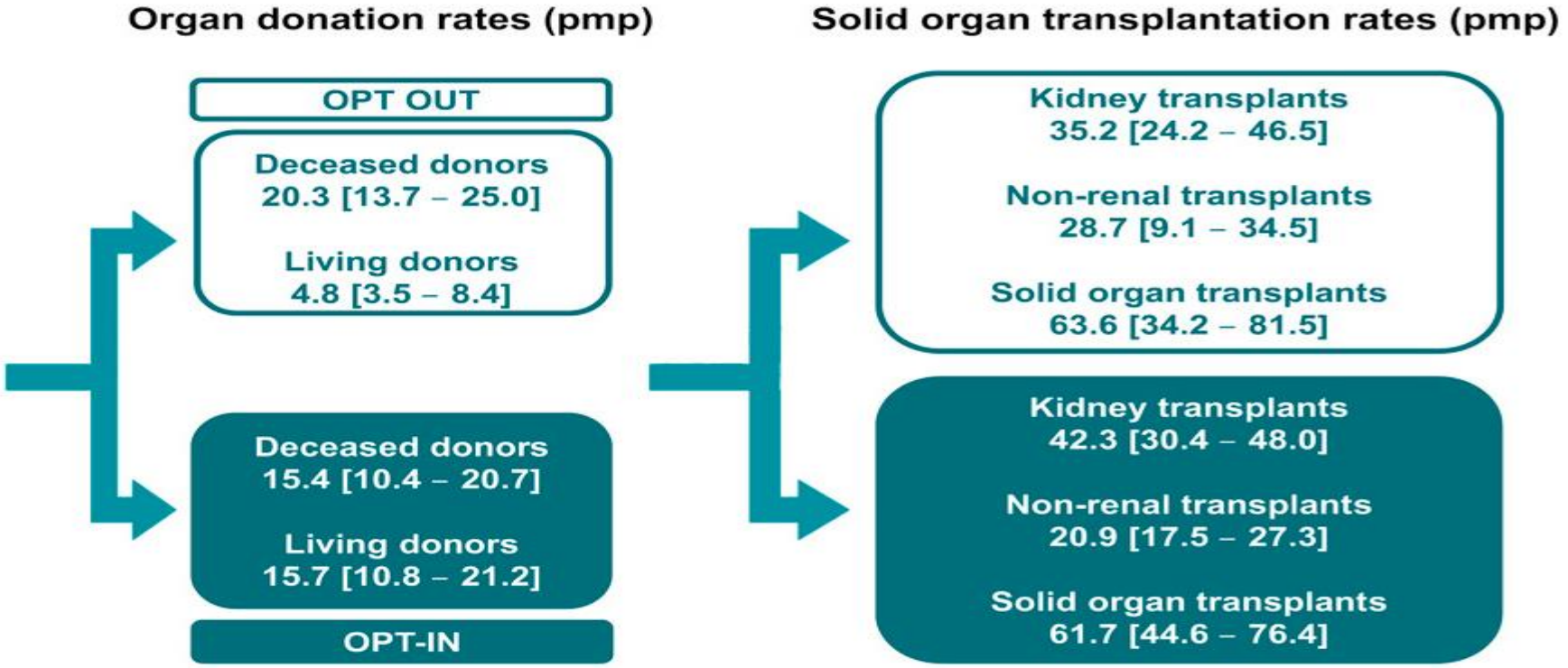
Factor	Points Awarded
For qualified time spent waiting	1 per year (as 1/365 per day)
Degree of sensitization (CPRA)	0–202
Prior living organ donor	4
Pediatric candidate if donor KDPI<0.35	1
Pediatric candidate (age 0–10 yr at time of match) when offered a zero antigen mismatch	4
Pediatric candidate (age 11–17 yr at time of match) when offered a zero antigen mismatch	3
Share a single HLA-DR mismatch with donor	1
Share a zero HLA-DR mismatch with donor	2

These points will be used to rank candidates in each of the categories listed in

Aile İzininde Opt In ve Opt Out Sistemi

Data sources

- Organisation for Economic Co-operation and Development (OECD, www.oecd.org).
- Global Observatory for Donation and Transplantation (GODT, www.transplant-observatory.org)
- International Registry in Organ Donation and Transplantation (IRODaT, www.irodat.org).
- World Health Organisation
- Pew Research Centre
- UN Department of Economic and Social Affairs.



Arshad et al, 2019

CONCLUSION:

Apart from less living-donors, our data demonstrates no difference in deceased-donors or solid organ transplantation activity between opt-out versus opt-in countries.

ISN kidney INTERNATIONAL
OFFICIAL JOURNAL OF THE INTERNATIONAL SOCIETY OF NEPHROLOGY

STUDY POPULATION

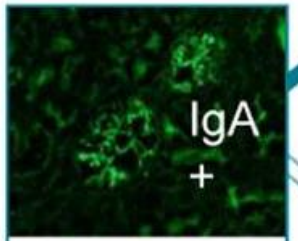
Kidney Transplants: 1,607

T_0 biopsies: 802

IgA+ 164

IgA- 638

METHODS



Living Donors: 44

Deceased Donors: 101

IgA-

Living Donors: 289

Deceased Donors: 311

HR, hazard ratio; CI, confidence interval; cPRA, calculated panel reactive antibodies; eGFR, estimated glomerular filtration rate; IgA, Immunoglobulin A

FINDINGS

Characteristics associated with death-censored graft failure at 5 years post-transplant

Variable	Adjusted HR (95%CI)	P-value
Donor kidney IgA presence	1.70 (0.80, 3.62)	0.17
Recipient age (years)	0.97 (0.94, 1.00)	0.04
Systemic lupus erythematosus	1.57 (0.44, 5.59)	0.49
eGFR at last follow-up (mg/dL)	0.95 (0.93, 0.97)	<0.001
cPRA at transplant	1.02 (1.00, 1.03)	0.01
Multi-organ transplant	9.69 (2.55, 36.77)	0.001
Expanded-criteria donors (ECDs)	8.13 (2.87, 23.02)	<0.001

C-statistic = 0.85, Cox proportional hazards modeling

CONCLUSION:

Incidental IgA in donor kidneys resolves with time in the majority of cases and is not associated with adverse graft outcome. Living donors of IgA+ kidneys should be followed closely post-donation

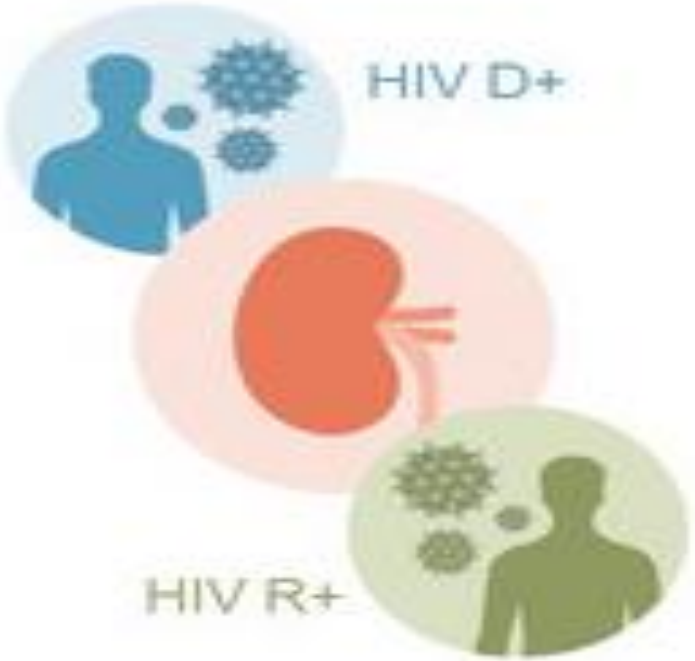
KI REPORTS

Gaber LW, 2020

Organ Bağışını Nasıl Arttırabiliriz

A prospective multicenter pilot study of HIV-positive deceased donor to HIV-positive recipient kidney transplantation: HOPE in action

What are the risks attributable to an HIV+ donor for HIV+ recipients?



Mar 2016–July 2019
14 centers

75 Kidney Txs

HIV D+/R+ (n=25)

HIV D−/R+ (n=50)

Median follow-up:
1.7 years

Between the two cohorts there were **no deaths, nor differences in:**

- 1-year graft survival
- 1-year mean eGFR
- HIV breakthrough
- infectious hospitalizations or opportunistic infections

One-year rejection was **higher for D+ recipients** (50% vs 29%) but did not reach statistical significance

10.1111/ajt.16205 AJT

HIV POZİTİF KADAVERİK İLK BÖBREK NAKLİ 2008 DE GÜNEY AFRİKADA

Afrikada bekleme listesindeki hastaların % 10'u HIV POZİTİF

Renal Transplantation between HIV-Positive Donors and Recipients

Table 1.

Characteristic	Patient 1	Patient 2	Patient 3	Patient 4
Age (yr)	47	56	37	29
Sex	Male	Male	Male	Female
Before transplantation				
Diagnosis on renal biopsy	HIV-associated nephropathy	HIV-associated nephropathy and hypertensive nephropathy	Malignant hypertension	HIV-associated nephropathy
Creatinine (μmol/liter)	678	582	1712	725
CD4 count (cells/mm³)	288	258	132	147
HIV viral load (copies/ml)	<50	<50	<50	<50
Antiretroviral regimen	Tenofovir, lamivudine, and lopinavir–ritonavir	Stavudine, lamivudine, and efavirenz	Stavudine, lamivudine, and nevirapine	Zidovudine, lamivudine, and nevirapine
After transplantation				
Antiretroviral regimen	Tenofovir, lamivudine, and lopinavir–ritonavir	Tenofovir, lamivudine, and lopinavir–ritonavir	Tenofovir, lamivudine, and lopinavir–ritonavir	Tenofovir, lamivudine, and lopinavir–ritonavir
CD4 count (cells/mm³)				
At 6 mo	129	113	140	140
At 12 mo	253	119	112	220
HIV viral load (copies/ml)				
At 6 mo	<50	<50	<50	<50
At 12 mo	<50	<50	<50	<50
Creatinine (μmol/liter)				
At 6 mo	114	119	181	101
At 12 mo	87	104	110	85
Diagnosis on renal biopsy				
At 3 mo	Normal kidney	Normal kidney	Acute tubular necrosis	Normal kidney
At 9 mo	Normal kidney	Calcineurin toxicity	Early collapsing glomerulonephritis	Normal kidney
Tacrolimus				
Average dose (mg/wk)	1.00	1.25	0.75	0.50
Average trough level at 0–12 mo (ng/ml)	12.10	11.86	7.50	11.85

* To convert values for creatinine to milligrams per deciliter, divide by 88.4. HIV denotes human immunodeficiency virus.

effectiveness of transplanting a kidney from an HIV-positive donor into an HIV-infected recipient is undetermined.

At our hospital, we undertook four renal transplantations involving HIV-positive recipients and HIV-positive donors, from September through November 2008 (Table 1). The recipients had ESRD, were receiving antiretroviral therapy, had stable disease (defined as an HIV viral load of <50 copies per milliliter for >6 months), and had no previous opportunistic infections other than fully treated pulmonary tuberculosis (Patient 2). None had access to dialysis or an HIV-negative donor transplant within the state sector, because HIV was an exclusion criterion. The four transplants were from two deceased donors who had not received antiretroviral therapy, did not have a history of serious opportunistic infection or cancer, and had normal renal biopsies without evidence of proteinuria.

Recipients received antithymocyte globulin as induction therapy, prednisone, mycophenolate mofetil, and tacrolimus. One patient receiving tacrolimus had calcineurin toxicity and was switched to sirolimus. At 12 months after transplantation, all patients had good renal function, did not have clinically significant graft rejection, and have not needed dialysis since the procedure.

Transplantation programs in resource-limited settings cannot offer renal replacement to all patients who are in need. The use of HIV-infected donors would increase the donor pool, providing organs that otherwise would be discarded to recipients who would otherwise die of ESRD. The suitability of recipients depends on therapeutic, physical, and social attributes. All recipients must have proven adherence, virologic suppression, and immune reconstitution. Donor suitability is defined as HIV infection (confirmed with the use of enzyme-linked immunosorbent assay), absence of proteinuria, and a normal kidney as assessed with post hoc renal biopsy. To combat high rates of early acute rejection, antithymocyte globulin should be used. Prospective

protease inhibitor in all recipients would increase the likelihood of suppressing any virus that is transplanted along with the kidney.

This report of four successful renal transplantations involving HIV-positive donors and recipients offers a new therapeutic approach to treating selected HIV-infected patients who have ESRD.

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Marc Mendelson, M.D., Ph.D.
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Cape Town, South Africa
elmi.muller@uct.ac.za

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

1. Han TM, Naicker S, Ramdial PK, Assounga AG. A cross-sectional study of HIV-seropositive patients with varying degrees of proteinuria in South Africa. Kidney Int 2006;69:2243-50.
2. Fabian J, Katz T, Gerntholtz T, Goetsch S, Naicker S. Chronic kidney disease in human immunodeficiency virus infection. Panminerva Med 2007;49:51-66.
3. Roland ME, Barin B, Carlson L, et al. HIV-infected liver and kidney transplant recipients: 1- and 3-year outcomes. Am J Transplant 2008;8:355-65. [Erratum, Am J Transplant 2008;8:1081.]
4. Streeck H, Li B, Poon AF, et al. Immune-driven recombination and loss of control after HIV superinfection. J Exp Med 2008; 205:1789-96.

INSTRUCTIONS FOR LETTERS TO THE EDITOR

Letters to the Editor are considered for publication, subject to editing and abridgment, provided they do not contain material that has been submitted or published elsewhere. Please note the following:

- Letters in reference to a *Journal* article must not exceed 175 words (excluding references) and must be received within 3 weeks after publication of the article.
- Letters not related to a *Journal* article must not exceed 400 words.
- A letter can have no more than five references and one figure or table.
- A letter can be signed by no more than three authors.
- Financial associations or other possible conflicts of interest must be disclosed. Disclosures will be published with the letters. (For authors of *Journal* articles who are responding to letters, we will only publish new relevant relationships that have developed since publication of the article.)
- Include your full mailing address, telephone number, fax number, and e-mail address with your letter.
- All letters must be submitted at authors.NEJM.org.

The NEW ENGLAND JOURNAL of MEDICINE

CORRESPONDENCE

Trial of Transplantation of HCV-Infected Kidneys into Uninfected Recipients

TO THE EDITOR: Waiting times for kidney transplants exceed 3 to 5 years in many parts of the United States.[1] Yet more than 500 high-quality kidneys from deceased donors with hepatitis C virus (HCV) infection are discarded annually.[2,3] Direct-acting antiviral agents, which are associated with high HCV cure rates and manageable side effects, have created the potential to substantially increase the number of kidney transplants by making HCV-infected kidneys available to HCV-negative candidates on the waiting list.[4,5]

In this open-label, single-group, pilot trial at the University of Pennsylvania (Transplanting Hepatitis C Kidneys into Negative Kidney Recipients [THINKER]; ClinicalTrials.gov number, NCT02743897) we sought to determine the safety and efficacy of transplantation of kidneys from HCV genotype 1–viremic donors into HCV-negative patients, followed by elbasvir–grazoprevir (Zepatier) treatment. An external data and safety monitoring board reviewed all aspects of the trial. The authors vouch for the completeness and accuracy of the data and analysis and for the adherence of the trial to the protocol, available with the full text of this letter at NEJM.org.

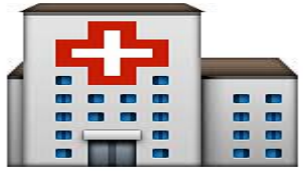
Adults who were undergoing dialysis and who had long anticipated waiting times for a kidney transplant were eligible for inclusion in the trial, and patients with conditions that substantially elevate the risks of liver disease, allograft failure, or death were excluded. A physician-led, three-step, informed-consent process was implemented.

Deceased-donor criteria ensured selection of high-quality kidneys (see the Supplementary Appendix, available at NEJM.org). Since elbasvir–grazoprevir is not approved by the Food and Drug Administration (FDA) for patients with HCV genotypes 2 or 3, and a direct-acting antiviral agent for the treatment of patients with those genotypes who have renal failure has not been approved by the FDA, donors were limited to those who had positive qualitative HCV nucleic acid test results and HCV genotype 1. We developed a new protocol for donor genotyping concurrent with organ allocation (see the Supplementary Appendix).

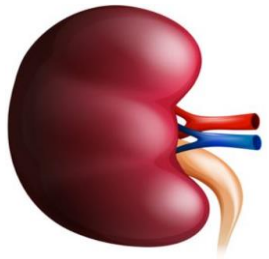
Intravenous glucocorticoids and rabbit antithymocyte globulin were administered to all recipients, followed by oral tacrolimus, mycophenolate mofetil, and prednisone. The HCV viral load was measured in recipients on postoperative day 3; elbasvir–grazoprevir was initiated when the results became positive, and therapy was maintained for 12 weeks.

Among 38 patients who were potentially eligible to participate in the trial, 22 attended an educational presentation, and 14 provided writ-

THIS WEEK'S LETTERS



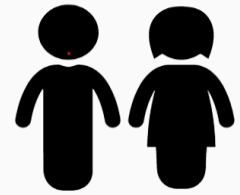
N= 10 HCV +



(Zapatier 100/50 mg)

Elbasvir–grazoprevir

N= 10 HCV -



% 100 İlk 3 Gün HCV RNA (+), Tedavi ile HCV RNA (-)

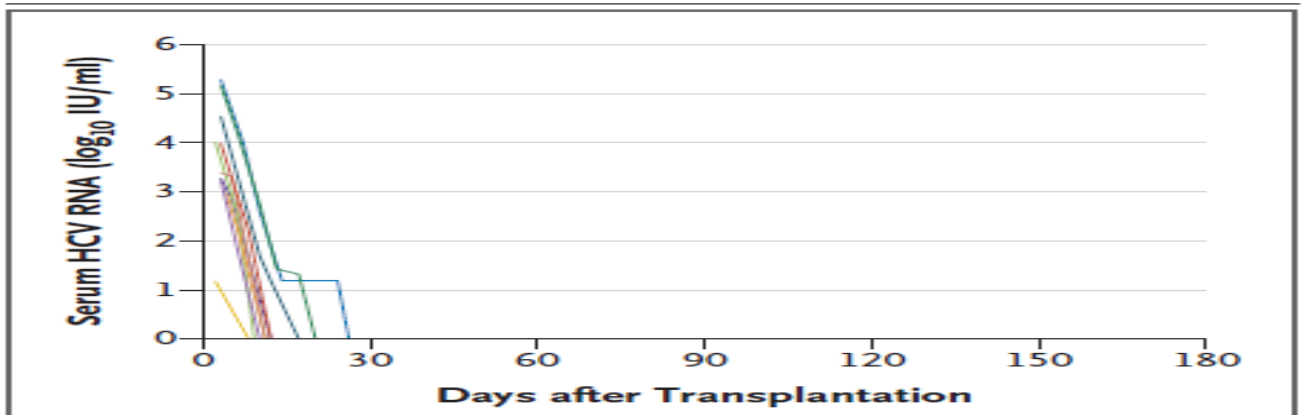


Figure 1. Hepatitis C Viral Load in 10 Kidney-Transplant Recipients.

The hepatitis C viral load was measured by means of polymerase chain reaction. Each curve represents a transplant recipient.

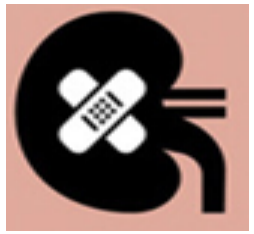
HIV Pozitifden, HIV Pozitife Böbrek Nakli

N=27

HIV + → HIV + 3-5 yıllık takip

Alıcı Dahil edilme:
1. CD4>200/mm3
2. HIV RNA Negatif

Verici Dahil edilme
1. HIV +, Kadaverik
2. HIV RNA Negatif



Akut rejeksiyon:
1.Yıl % 8.3
3.Yıl % 22

Hasta Sağkalım:
1.ve 3.Yıl: %84
5. Yıl: %74

Ölüm Nedenleri N=5
Sepsis, MI, Duedonal perforation, N=2 İnfeksiyon

Greft Sağkalım:
1.ve 3.Yıl: % 93 ve %84
5. Yıl: %84

The New England Journal of Medicine,2015

HCV, HIV İle İnfekte Donor Böbrek Kullanımı

HCV Pozitifden, HCV NegatifeBöbrek Nakli

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THIS WEEK'S LETTERS

2394 Trial of Transplantation of HCV-Infected Kidneys into Uninfected Recipients
2396 Trial of Pregabalin for Acute and Chronic Sciatica
2397 Treatment of Benzodiazepine Dependence
2400 A Zimbabwean Man with a Severe Headache
2401 Prostate Cancer Screening

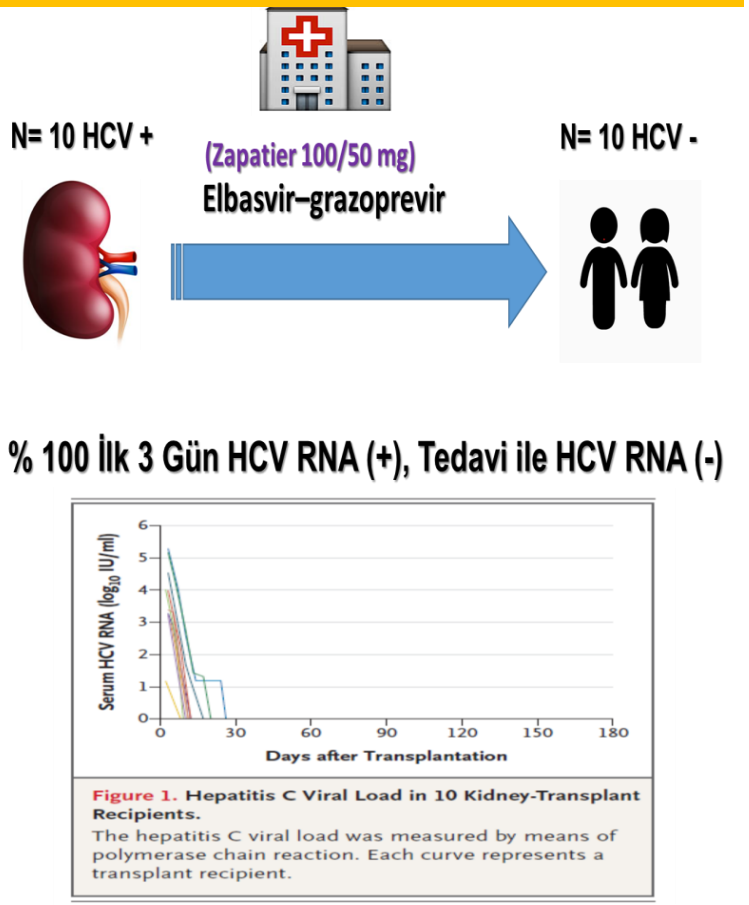
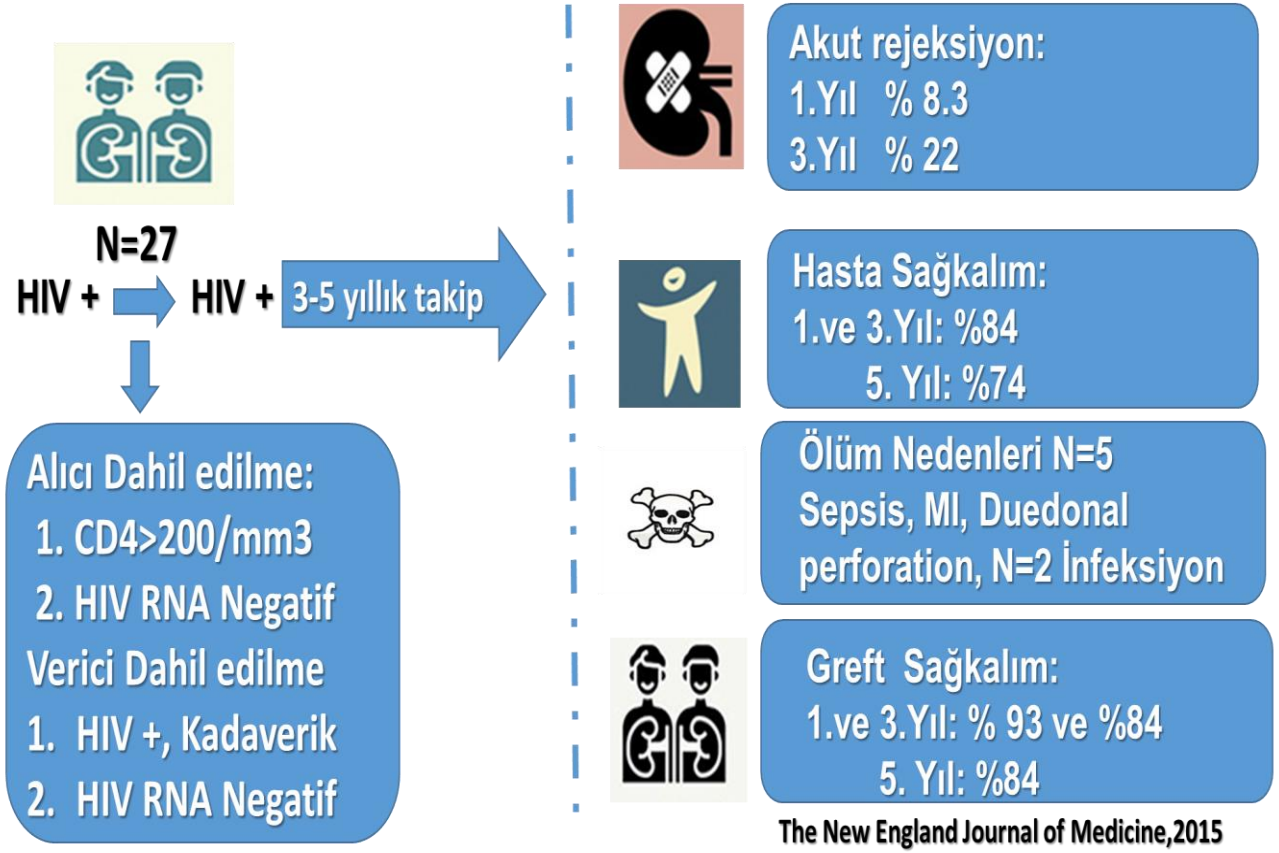


Figure 1. Hepatitis C Viral Load in 10 Kidney-Transplant Recipients.
The hepatitis C viral load was measured by means of polymerase chain reaction. Each curve represents a transplant recipient.

HIV Pozitifden, HIV Pozitife Böbrek Nakli



The New England Journal of Medicine,2015

Nakil Sonrası HCV Tedavisi

frontiers | Frontiers in Medicine

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Efficacy and Safety of Direct-Acting Antivirals in Kidney Transplantation From HCV-Viremic Donors to Negative Recipients: A Meta-Analysis

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Background: With the development of direct-acting antiviral agents (DAAs), the research on kidney transplantation from Hepatitis C virus (HCV)-viremic donors to HCV-negative recipients has grown. The objective of this comprehensive analysis was to evaluate the efficacy and safety of DAAs in kidney transplantation from HCV-viremic donors to negative recipients.

Methods: Multiple databases were searched for a systematic and comprehensive up to March 2022. The primary outcomes included the percentage of sustained virological response at week 12 after the end of treatment (SVR12), adverse events (AEs; any grade), and severe adverse events (SAEs) as the endpoints. Publication bias was examined by using the funnel plots and Egger's test.

Results: In total, 16 studies with 454 subjects were included in the study and the pooled estimate of SVR12, AEs, and SAEs rates were 100.0% (95% CI: 99.2-100.0), 1.9%(95%CI: 0.0-4.9), and 0.0% (95%CI: 0.0-1.5). Subgroup analysis showed that pooled SVR12 rates were 100.0% (95%CI: 99.6-100.0) for genotype (GT)1a and 96.3% (95%CI: 83.3-100.0) for GT2; 100.0% (95%CI: 98.9-100.0) for DAAs treatments; and 100.0% (95%CI: 98.2-100.0) for prophylaxis subgroup. Egger's tests showed that no publication bias was found in this study.

Conclusion: This comprehensive analysis showed the high efficacy and safety of DAAs in kidney transplantation from HCV-viremic donors to HCV-negative recipients.

Systematic Review Registration: https://www.crd.york.ac.uk/PROSPERO/display_record.php?RecordID=246541.

Keywords: antiviral agents, HCV-viremia, Hepatitis C, kidney donors, kidney transplant, meta-analysis

INTRODUCTION

Hepatitis C virus (HCV) infection affects around 180 million individuals worldwide, of which around 71 million people develop chronic HCV infections (1, 2). HCV may develop cirrhosis, hepatocellular carcinoma, and liver-related deaths (3), and 40% of the infected population may have

16 Çalışma, 454 böbrek nakilli hasta

↓

3 Vakada genotipten dolayı tedavi başarısızlığı, Genotipe uygun DAA uygulaması ile tedavi başarılı

↓

Nakil öncesi veya sonrası DAA uygulaması arasında tedaviye yanıtta fark yok

American Journal of Transplantation 2010; 10: 2453–2462
Wiley Periodicals Inc.

doi: 10.1111/j.1600-6143.2010.03280.x

Long-Term Experience With Kidney Transplantation From Hepatitis C-Positive Donors Into Hepatitis C-Positive Recipients

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Kidney transplantation from hepatitis C virus (HCV) antibody positive donors (HCVD+) into HCV antibody positive recipients (HCVR+) is controversial. We implemented this policy in our units in 1990. Herein, we report the long-term safety of this strategy. From March 1990 to March 2007, 162 HCVR+ received a kidney from HCVD+ (group 1) and 306 from HCVD− (group 2) in our units. Mean follow-up was 74.5 months. Five-and 10-year patient survival was 84.8% and 72.7% in group 1 *vs.* 86.6% and 76.5% in group 2 (p = 0.250). Three deaths in group 1 and two in group 2 were liver-disease related. Five- and 10-year graft survival was 58.9% and 34.4% versus 65.5% and 47.6% respectively (p = 0.006) while death-censored graft survival was 69% and 47% versus 72.7% and 58.5% (p = 0.055). Decompensated chronic liver disease was similar: 10.3% versus 6.2%. Cox-regression analysis could not identify the donor's HCV serology as a significant risk factor for death, graft failure and severe liver disease in HCVR+. In conclusion, long-term outcome of HCVR+ transplanted with kidneys from HCVD+ seems good in terms of patient survival, graft survival and liver disease. HCVD+ was not a significant risk factor for mortality, graft failure and liver disease among HCVR+. These data strongly suggest that the use of kidneys from HCVD+ in HCVR+ is a safe long-term strategy that helps to prevent kidney loss.

Key words: Hepatitis C, kidney transplantation, liver disease, organ donation

Abbreviations: ALT, Alanine Aminotransferase; APRD, Adult Polycystic kidney disease; CLD, Chronic Liver Disease; DGF, Delayed Graft Function; HCV, Hepatitis C virus; HCVD+, Hepatitis C virus Antibody Positive Donor; HCVD−, Hepatitis C virus Antibody Negative Donor; HCVR+, Hepatitis C virus Antibody Positive Recipient; IT, induction with antilymphocyte antibodies; KDIGO, Kidney Disease: Improving Global Outcomes; MMF, Mycophenolate Mofetil; NODAT, New Onset of Diabetes After Transplantation; PCR, Polymerase Chain Reaction; PRA, Peak Panel Reactive Antibodies; RNA, Ribonucleic Acid; sCr, Serum Creatinine; UNOS, United Network for Organ Sharing.

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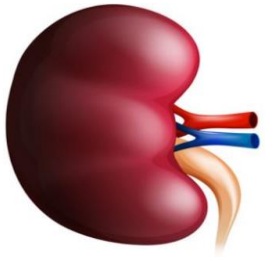
Introduction

The waiting list for renal transplantation has increased at a higher rate than the number of donors and organs. Even in Spain, with the highest deceased donation rates ever described, approximately 4000 patients were on the waiting list for kidney transplantation last year. However, only about 2200 procedures are performed annually (www.ont.es). Therefore, the use of expanded criteria donors and donors with potentially transmissible diseases has been established as a way to mitigate this global organ shortage.

Because hepatitis C virus (HCV) infection is transmitted through organ transplantation (1–5) there is almost universal consensus on rejecting kidneys from HCV antibody positive donors (HCVD+) for transplantation in HCV antibody negative recipients (6). However, there is still controversy regarding the use of these kidneys for HCV antibody positive recipients (HCVR+), and some countries even have legal and/or technical provisions in force which preclude the use of these organs.

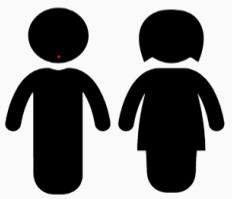
In 1990, our two Spanish centers adopted the policy of using kidneys from HCVD+ in HCVR+. This strategy, approved by the *Nephrology and Transplantation Departments*, was safe in the short term (7). However, it was modified in July 1993, after it was observed that four out of five HCVR+ with a negative HCV RNA before transplantation became HCV RNA positive after receiving a kidney graft from an HCV RNA positive donor, two of them



HCV Pozitif Kadaverik Böbrek

Ülkemizde

HCV Pozitif Alıcı Adayı



UNOS Böbrek Biyopsi Endikasyonu

KDPI>%85

Diyabet, HT ve veya ABH

Böbrek Biyopsisi

Böbrek Verici Risk indeksi (Kidney Donor Risk Index)

Referans Bağışçıya göre, posttransplant greft kaybının *rölatif riskini* belirler
Referans Bağışçı: 40 yaş, beyaz, 1.70 boyunda, 80 kğ, creatinin: 1.0 , normotansif, KŞ normal, HCV negatif, Ölüm nedeni kardiyovasküler dışı

Yaş	Diyabetes Mellitus öyküsü
Boy	Hipertansiyon öyküsü
Kilo	Serum kreatinin düzeyi
Etnik	HCV varlığı
Ölüm nedeni	DCD (Ölmüş Kalp Donasyon durumu)

Transplantation 2009;88: 231–236

Böbrek Verici Profil İndeksi (KDPI)

ole 2. Estimated Kidney Graft Survival Rates for Single Kidney Transplants in the U.S. in 2007-2017, by KDPI

			Estimated Single Kidney Graft Survival Rates					
KDPI	$KDRI_{RAO}$*	$KDRI_{MEDIAN}$*	1 Year	2 Years	3 Years	5 Years	8 Years	10 Years
1%	0.70	0.58	97.6%	95.4%	92.7%	83.2%	70.3%	63.2%
5%	0.77	0.64	95.0%	91.7%	89.0%	80.9%	70.2%	62.5%
10%	0.83	0.68	96.5%	94.0%	90.7%	82.9%	71.2%	63.9%
20%	0.92	0.76	96.5%	92.9%	90.1%	80.2%	66.1%	56.2%
30%	1.01	0.83	95.1%	92.2%	88.7%	78.4%	64.7%	49.7%
40%	1.11	0.91	94.6%	92.0%	88.0%	79.1%	62.2%	54.1%
50%	1.22	1.00	94.5%	90.7%	87.7%	81.4%	63.5%	58.3%
60%	1.35	1.11	92.9%	89.5%	85.4%	75.2%	62.3%	52.6%
70%	1.49	1.22	92.1%	88.3%	83.8%	72.1%	55.6%	46.4%
80%	1.67	1.37	89.6%	84.5%	79.7%	69.5%	50.6%	38.3%
90%	1.94	1.60	87.7%	81.2%	76.0%	63.1%	43.7%	26.1%
95%	2.24	1.84	87.4%	80.5%	75.6%	60.9%	36.4%	26.5%
99%	2.71	2.23	81.8%	73.3%	69.8%	57.5%	36.1%	19.6%

* Maximum of the range of KDRI rounded to 2 decimal places.

Donor reference population: All deceased kidney donors recovered for transplant in 2017.

Based on OPTN data including primary, adult, deceased donor, kidney alone transplants, as of May 10, 2019.

TNK Böbrek ve Marjinal Böbrek İlişkisi

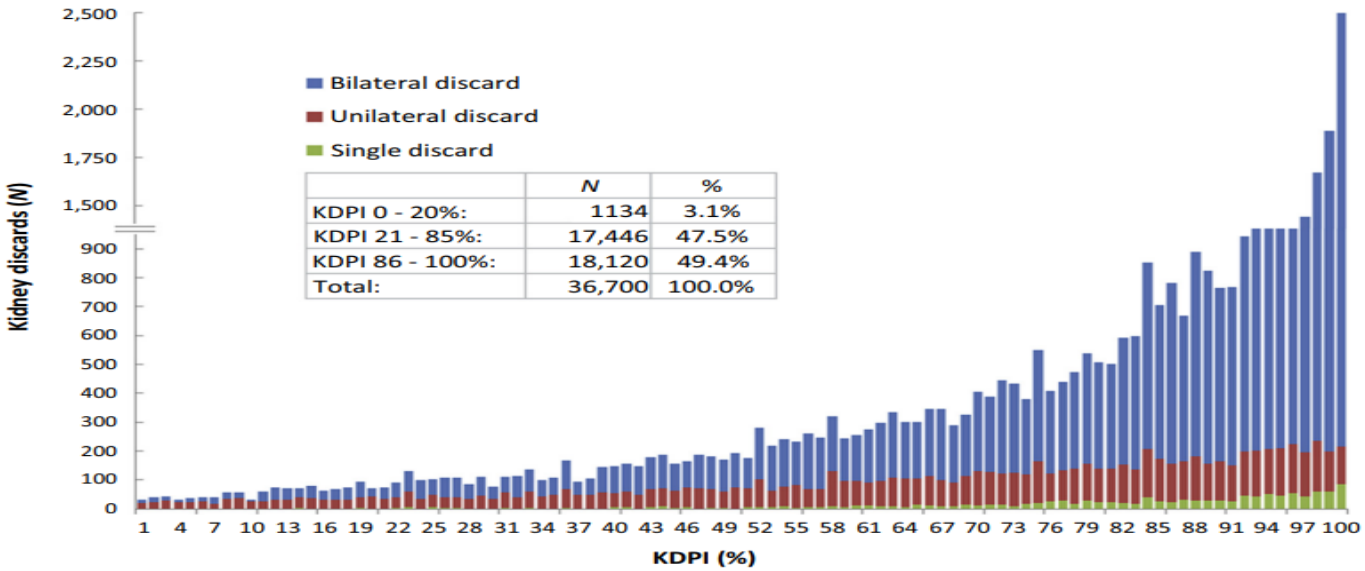


Figure 4 | US organ quality (Kidney Donor Profile Index [KDPI]) of deceased donor kidney discards stratified by discard type (*n* =36,700), 2000 to 2015.

Table 2 | Common causes of kidney discard by discard quality and type of organs procured in the US between 2000 and 2015 (*N* = 36,700)

	Extended ischemia	Organ damage	Anatomical abnormality	Poor function	Donor history	Biopsy findings	No recipient located	Other	
N (row %)	912 (2.5)	1333 (3.6)	2527 (6.9)	3534 (9.6)	3019 (8.2)	14,032 (38.2)	5368 (14.6)	5975 (16.3)	*P* value
Discard type									
Single	1.9	6.5	9.6	10.0	7.2	29.0	18.0	18.0	<0.001
Bilateral	1.8	1.6	5.2	9.8	8.8	43.7	15.1	14.1	
Unilateral	5.0	10.2	12.4	9.2	6.5	20.6	12.4	23.8	
Organ quality									
Median KDRI (IQR)	1.59 (0.61)	1.29 (0.71)	1.66 (0.75)	1.73 (0.73)	1.65 (0.74)	1.90 (0.72)	1.83 (0.74)	1.64 (0.75)	<0.001
Median KDPI (IQR)[a]	76.5 (32.5)	57 (54)	80 (37)	84 (31)	80 (35)	89 (22)	87 (25)	79 (36)	<0.001
Median terminal sCr (mg/dl) (IQR)	1.10 (0.70)	1.0 (0.70)	1.10 (0.70)	1.40 (1.34)	1.10 (0.80)	1.30 (0.90)	1.20 (0.98)	1.10 (0.90)	<0.001
Biopsy performed	2.3	1.8	4.9	9.3	5.8	46.4	15.8	13.9	<0.001
Discarded locally									
Yes	2.0	3.8	7.2	9.0	9.8	37.2	17.2	14.0	<0.001
No	4.4	3.5	6.4	11.5	5.0	43.8	3.7	21.7	
Unknown	2.0	3.2	6.4	9.8	6.3	34.8	19.4	18.2	

IQR, interquartile range; KDPI, Kidney Donor Profile Index; KDRI, Kidney Donor Risk Index; sCr, serum creatinine; UNOS, United Network of Organ Sharing.
[a]KDPI is calculated based on a scaling factor of 1.2175005163, a median KDRI value among all deceased donor kidneys procured during 2015.

Kidney International (2018) 94, 187–198