Centre universitaire de santé McGill
McGill University Health Centre

Les meilleurs soins pour la vie
The Best Care for Life

DIVISION DE NEPHROLOGIE
NEPHROLOGY DIVISION

Diagnosis and Management of Metabolic Problems in Kidney Transplant Recipients (CKD-CMBD-Tx)

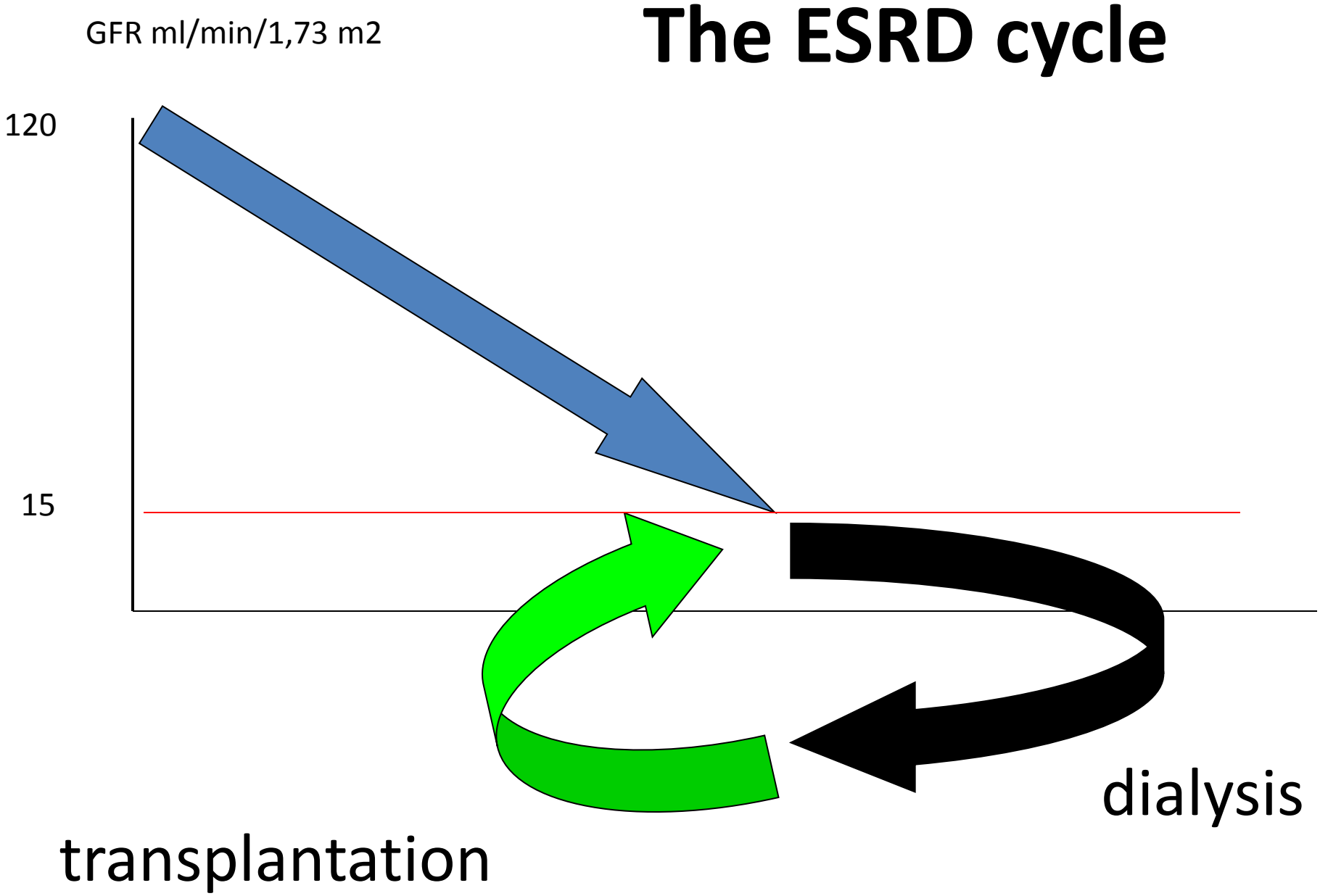
Istvan Mucsi MD, PhD
associate professor

McGill University Health Centre, Montreal, Quebec, Canada

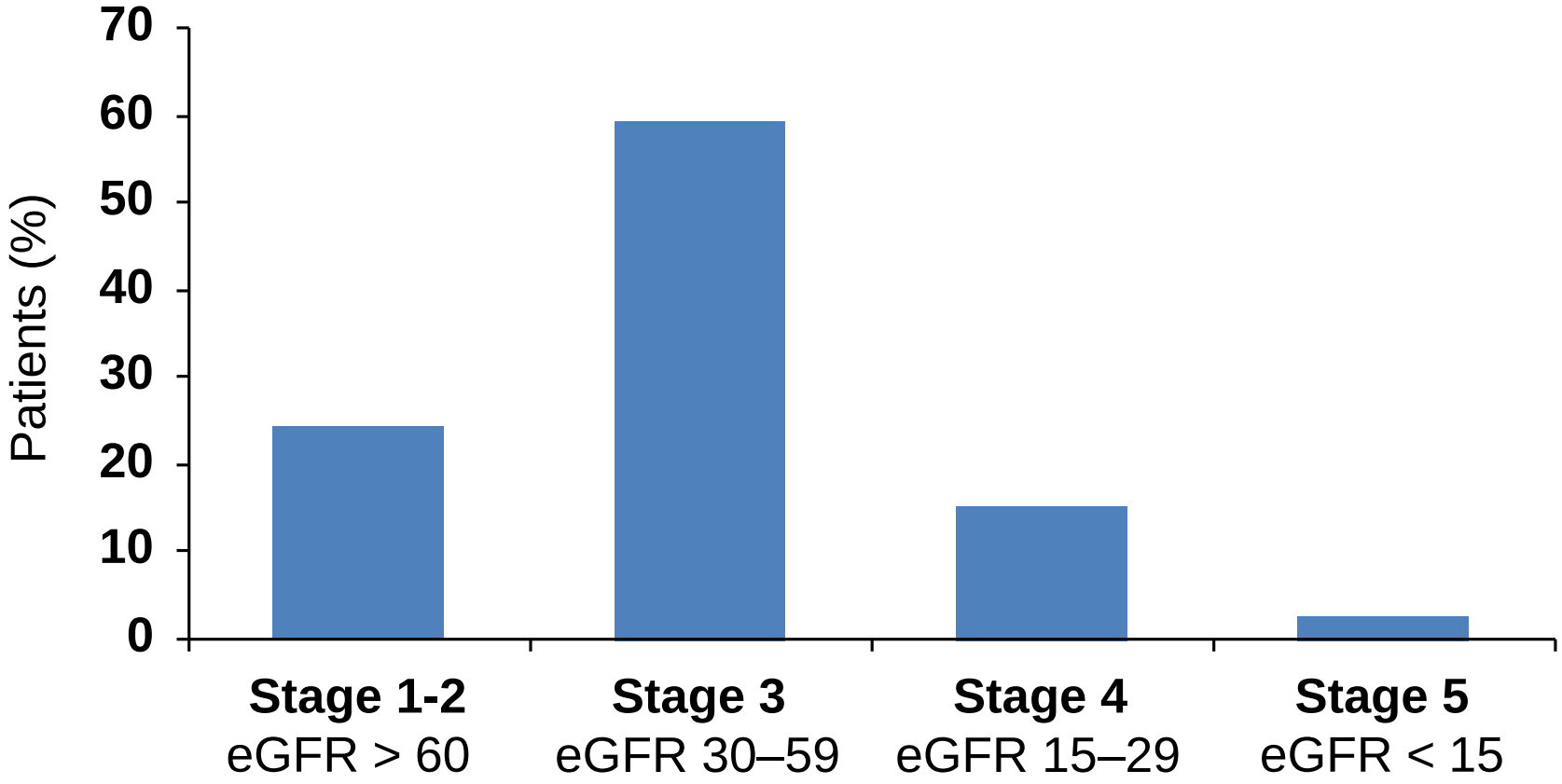
Semmelweis University Budapest, Hungary

- CKD in Tx
- DM
- Lipids
- Obesity
- Malnutrition/inflammation
- Bone
- How to manage this…

The ESRD cycle



Majority of transplant recipients have kidney function equivalent to stage 3 CKD or worse (UK data)



19,074 adult patients with a functioning kidney transplant at the end of 2005

UK Renal Registry Report 2006. Chapter 1.

The Burden of Chronic Kidney Disease in Renal Transplant Recipients

Vanji Karthikeyan[a], Jolanta Karpinski[a], Rama C. Nair[b] and Greg Knoll[a,*]

American Journal of Transplantation 2003; 4: 262–269

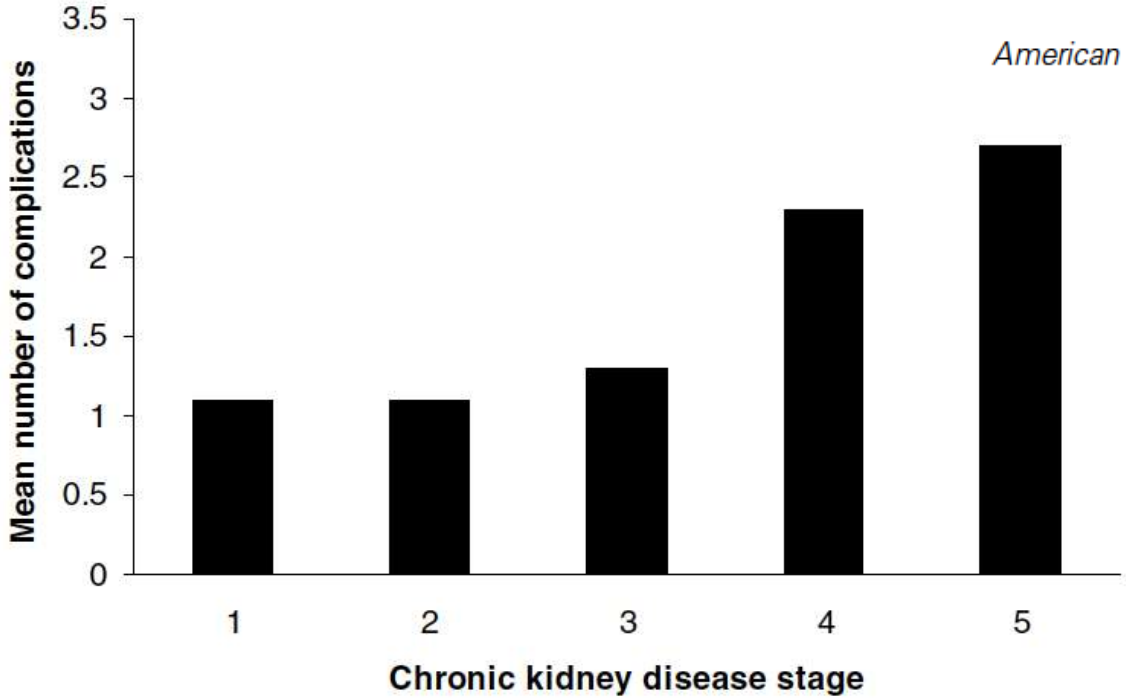


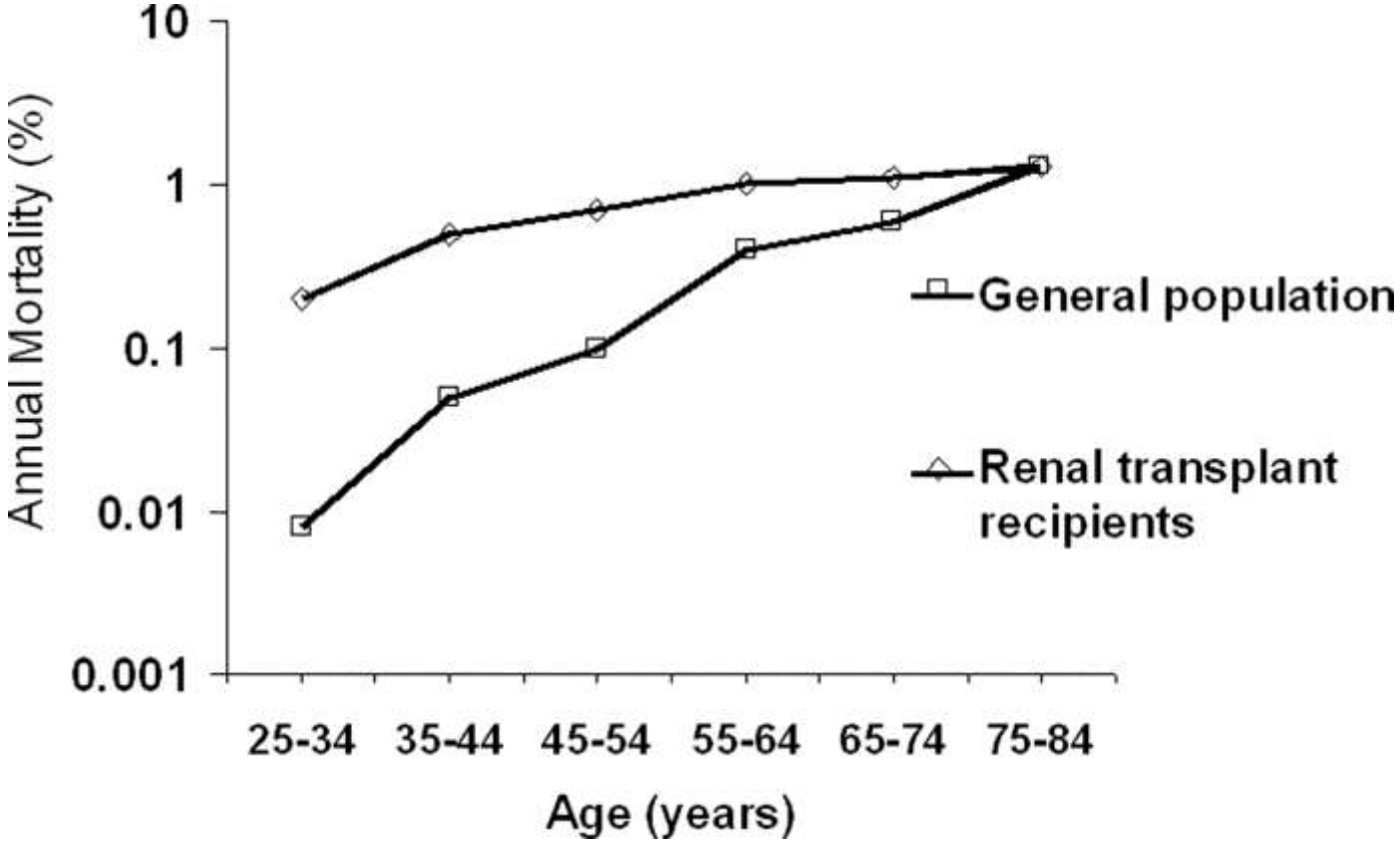
Figure 1: The mean number of complications per patient according to chronic kidney disease stage. The following complications were counted: hypertension (blood pressure $\geq$ 140/90 mmHg), serum calcium $<$ 8.5 mg/dL, serum phosphorous $>$ 4.5 mg/dL, hemoglobin $<$ 11 g/dL, serum albumin $<$ 3.5 g/dL, LDL $>$ 100 mg/dL and total CO_2 $<$ 22 mEq/L.

Kidney Disease : Improving Global Outcomes (KDIGO) Guidelines

Levey As, et al. Kidney Int 67:2089, 2005

- Consider all kidney transplants recipients to have CKD, irrespective of GFR level or presence or absence of markers of kidney damage.
- The rationale for this is based on damage to native kidneys, presumed damage to the kidney transplant based on studies of "protocol biopsies," and need for life-long care caused by complications of prior CKD and chronic allograft nephropathy

Figure 2. Cardiovascular mortality in kidney transplant recipients



Djamali, A. et al. Clin J Am Soc Nephrol 2006;1:623-640

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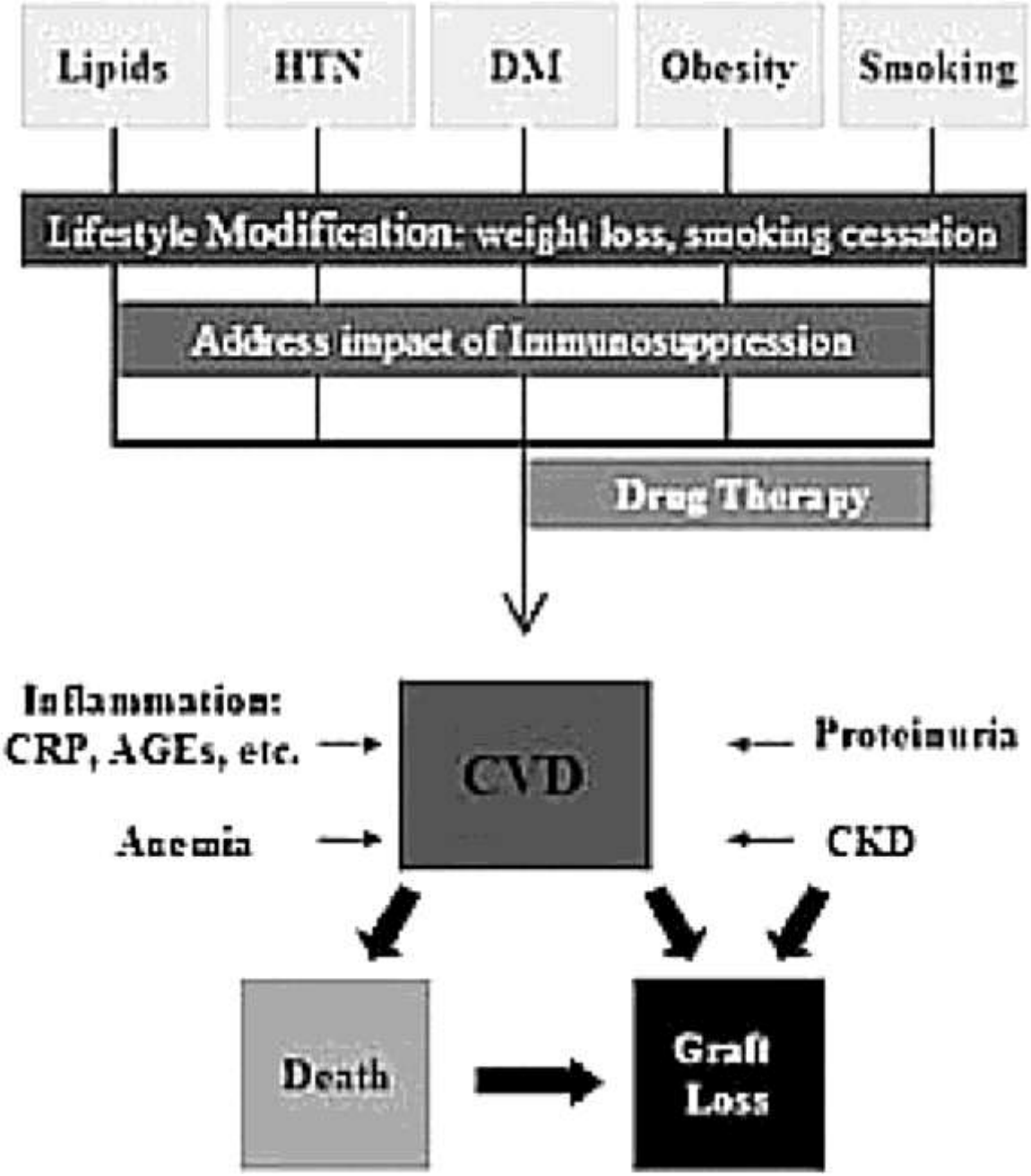

cardiovascular disease management after renal transplantation

Shirali, A. C. et al. Clin J Am Soc Nephrol 2008;3:491-504

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Metabolic effects of common immuno-suppressive agenst

	CSA	TAC	SRL	MMF	AZA	Steroid
Dyslipidaemia	++	+	+++	-	-	++
Hypertension	++	+	-	-	-	++
NODAT	+	+ (+)	-	-	-	++

Shirali, A. C. et al. Clin J Am Soc Nephrol 2008;3:491-504

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How to improve outcome in kidney transplanted patients?

An important issue for long term patient outcomes is to reduce ISU toxicity and to manage CV disease.

- Before Tx :
  - Dialysis vintage
  - CV management
  - CV interventions

- After Tx: medical management
  - DM
  - Dyslipidaemia
  - Obesity
  - Smoking
  - Inflammation
  - Anemia
  - Bone
  - …

Diabetes mellitus (new and old)

Patient Survival and Cardiovascular Risk After Kidney Transplantation: The Challenge of Diabetes

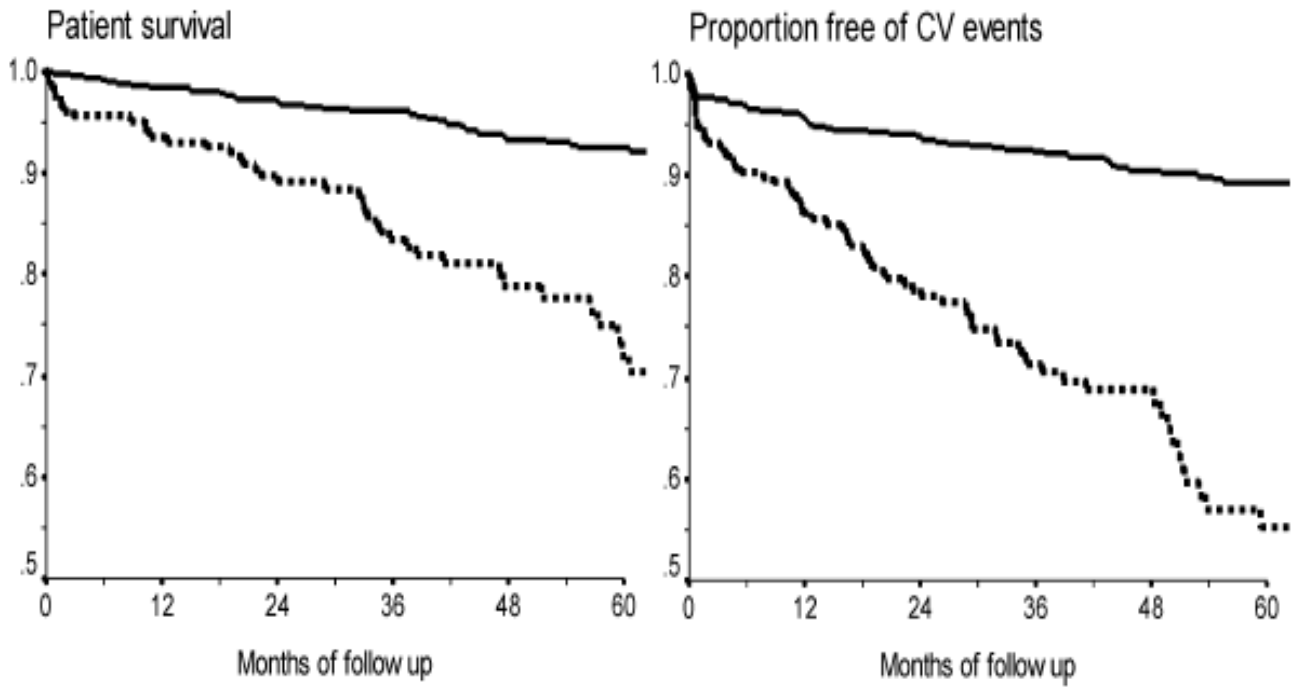

Figure 1: Left: Kaplan–Meier plots of patient survival after transplantation in recipients without DM (—) and those with DM (.....) (log-rank, $p < 0.0001$). Right: Kaplan–Meier plots of the incidence of fatal and nonfatal posttransplant CV events in recipients without DM (—) and those with DM (.....) (log-rank, $p < 0.0001$).

Cosio et al.:

American Journal of Transplantation 2008; 8: 593–599
Blackwell Munksgaard

Table 5. Risk factors for NODAT[a]

Recipient characteristics
older age (>45 yr)
higher body mass index (≥30)
black race
family history of diabetes
Hispanic ethnicity
education (no college degree)
Donor
deceased donor
male gender
Transplant era (after 1995)
Tacrolimus use
HLA mismatch
Acute rejection
HCV infection

[a]HCV, hepatitis C virus; NODAT, new-onset diabetes after transplantation.

NODAT risk factors

Djamali, A. et al. Clin J Am Soc Nephrol 2006;1:623-640

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Unmasking Glucose Metabolism Alterations in Stable Renal Transplant Recipients: A Multicenter Study

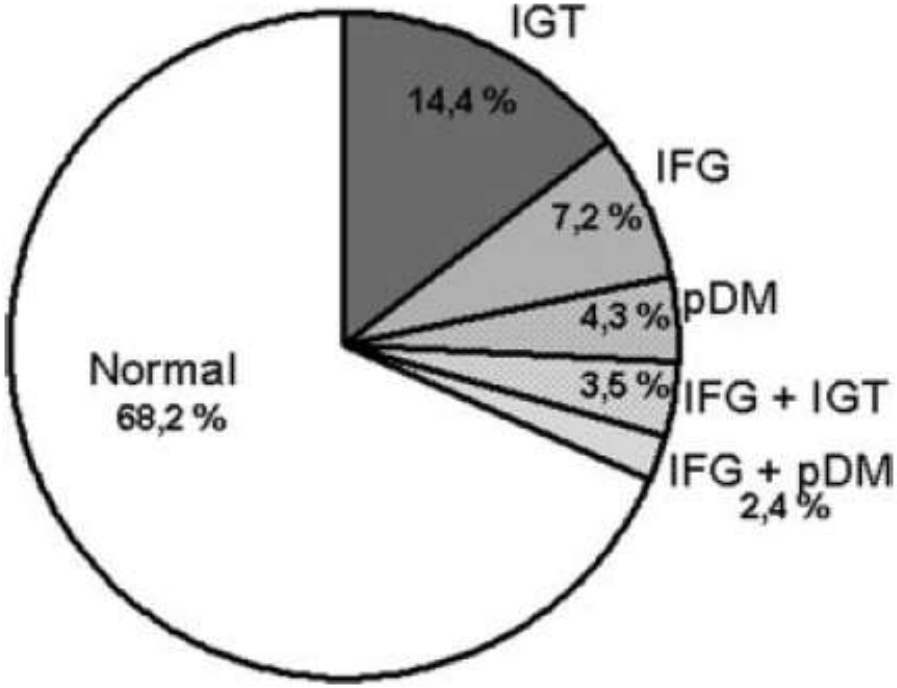


Figure 1. Distribution of various glucose metabolism alterations in stable renal transplant patients. IFG, impaired fasting glucose; IGT, impaired glucose tolerance; pDM, provisional diabetes.

P. Delgado et al.: Clin J Am Soc Nephrol 3: 808-813, 2008.

KDIGO clinical practice guideline for the care of kidney transplant recipients

15.1: SCREENING FOR NEW-ONSET DIABETES AFTER TRANSPLANTATION

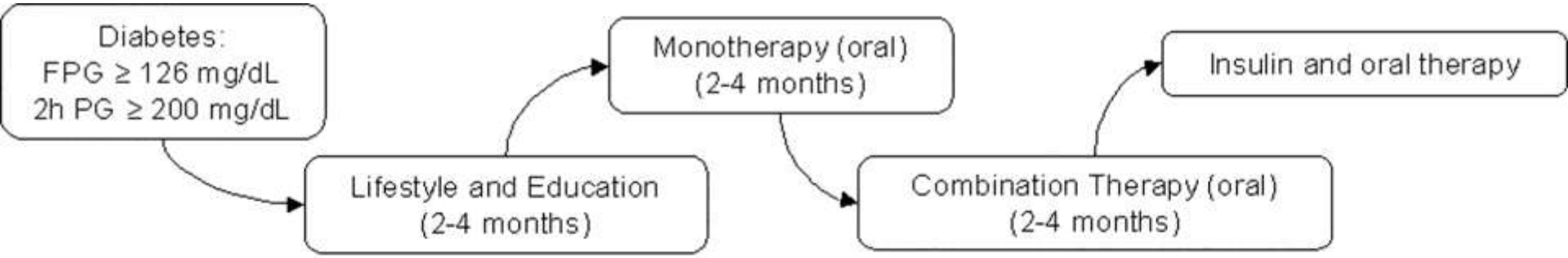
15.1.1: We recommend screening all nondiabetic KTRs with fasting plasma glucose, oral glucose tolerance testing, and/or HbA_{1c} *(1C)* at least:

- **weekly for 4 weeks *(2D)*;**
- **every 3 months for 1 year *(2D)*; and**
- **annually, thereafter. *(2D)***

15.1.2: We suggest screening for NODAT with fasting glucose, oral glucose tolerance testing, and/or HbA_{1c} after starting, or substantially increasing the dose, of CNIs, mTORi, or corticosteroids. *(2D)*

American Journal of Transplantation 2009; 9 (Suppl 3): S1–S157

Diabetes management after the first posttransplant year



Oral Agent	Target Population	Advantage	Disadvantage
Sulfonylurea	DM2 < 5 year duration	↓ cost Rapid effect	↑ Weight ↑ hypoglycemia
Meglitinides	Recent DM2 ↑ PPG	↓ hypoglycemia short acting	↑ cost
Biguanides	Overweight/Obese Insulin resistance	No ↑ weight ↓ hypoglycemia	GI side-effects Lactic acidosis (rare)
Thiazolidinediones	Overweight/Obese Insulin resistance	↓ Insulin requirement ↓ hypoglycemia	↑Cost, weight ↑Liver toxicity Slow onset of action
α-glucosidase inhibitor	↑ PPG	↓ hypoglycemia	GI side-effects ↑ cost

Djamali, A. et al. Clin J Am Soc Nephrol 2006;1:623-640

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Dyslipidemia

Dyslipidemia Following Kidney Transplantation:
Diagnosis and Treatment

Table 2. Effect of immunosuppressive drugs on lipid parameters

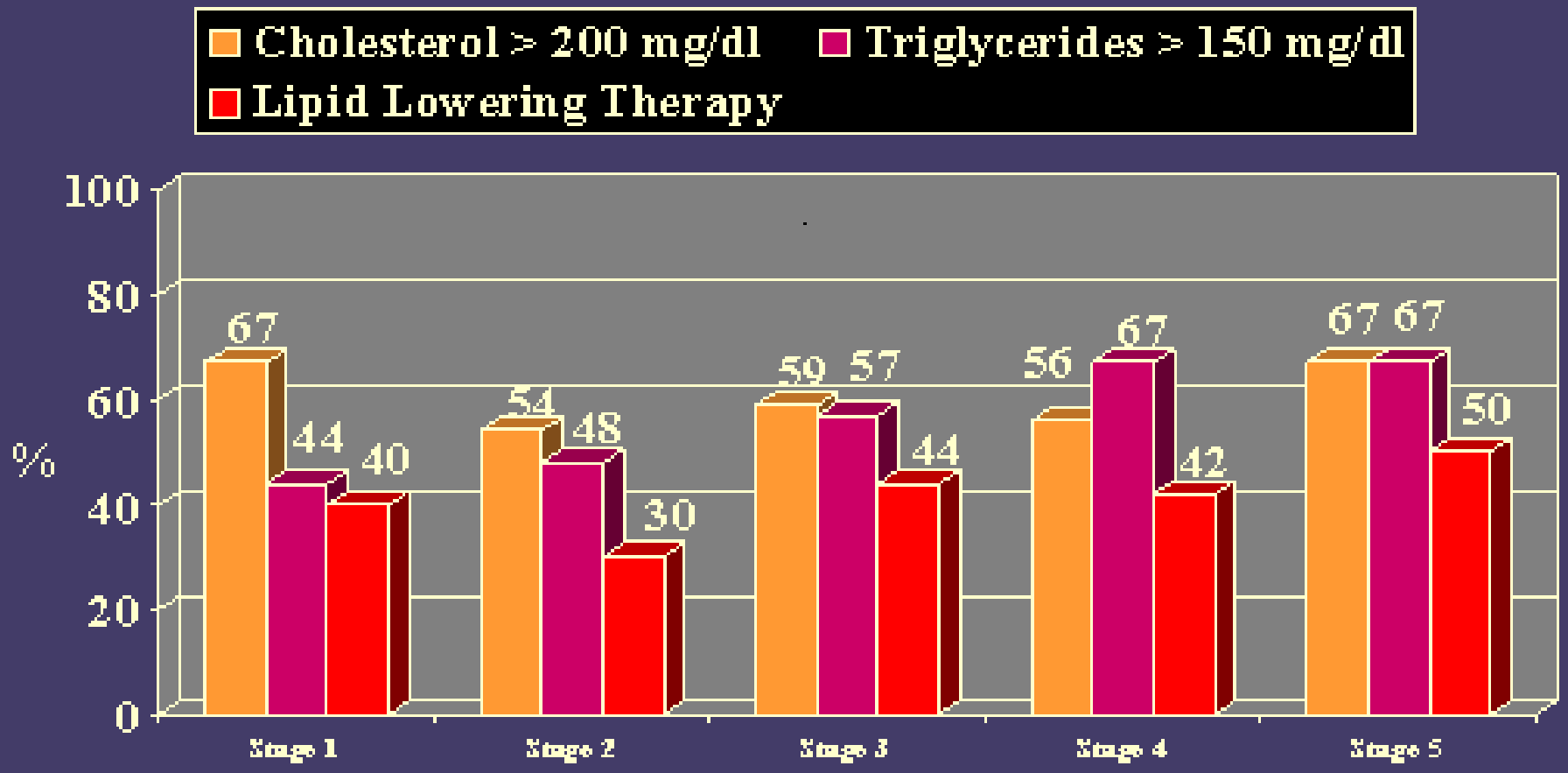
Drug	TC	LDL-C	HDL-C	TG
Cyclosporine	↑↑	↑↑	↓	↑↑
Tacrolimus	↑	↑	↓	↑
Sirolimus	↑↑	↑↑	↓	↑↑↑
Everolimus	↑↑	↑↑	↓	↑↑↑
Mycophenolate mofetil	–	–	–	–
Azathioprine	–	–	–	–
Prednisone	↑	↑	↑	↑
Deflazacort	↑	↑	↑↑	↑

HDL-C—high-density lipoprotein cholesterol; LDL-C—low-density lipoprotein cholesterol; TC—total cholesterol; TG—triglcyeride.

S. Badiou et al.: Current Diabetes Reports 2009, 9:305–311

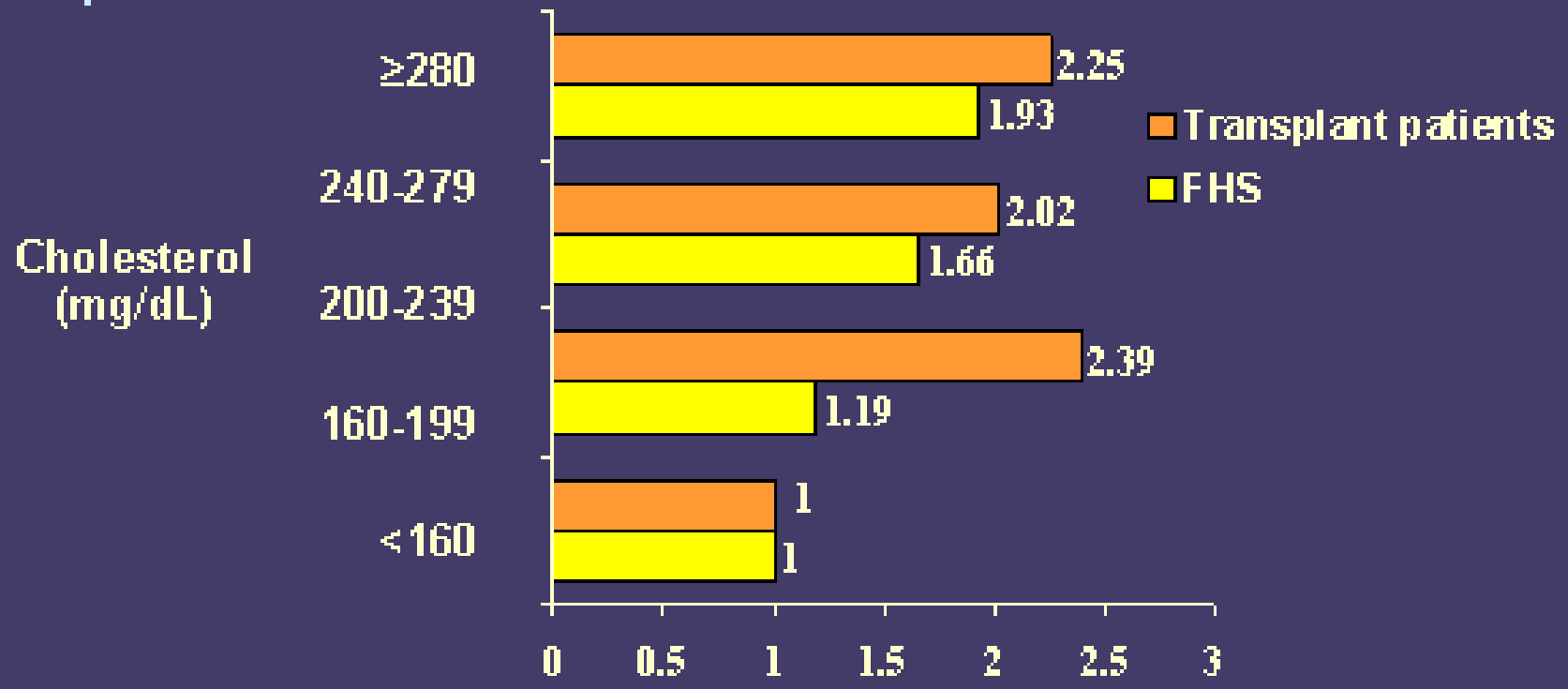
Prevalence of Hyperlipidemia in Renal Transplant Patients Based on CKD Stage

Karthikeyan V, Am J Transplant 4:262-269,2004



Hypercholesterolemia: Relative Risk for Ischemic Heart Disease in Patients More Than One Year After Renal Transplantation

Relative Risk of IHD in Males From the Framingham Heart Study (FHS) or Transplant Patients



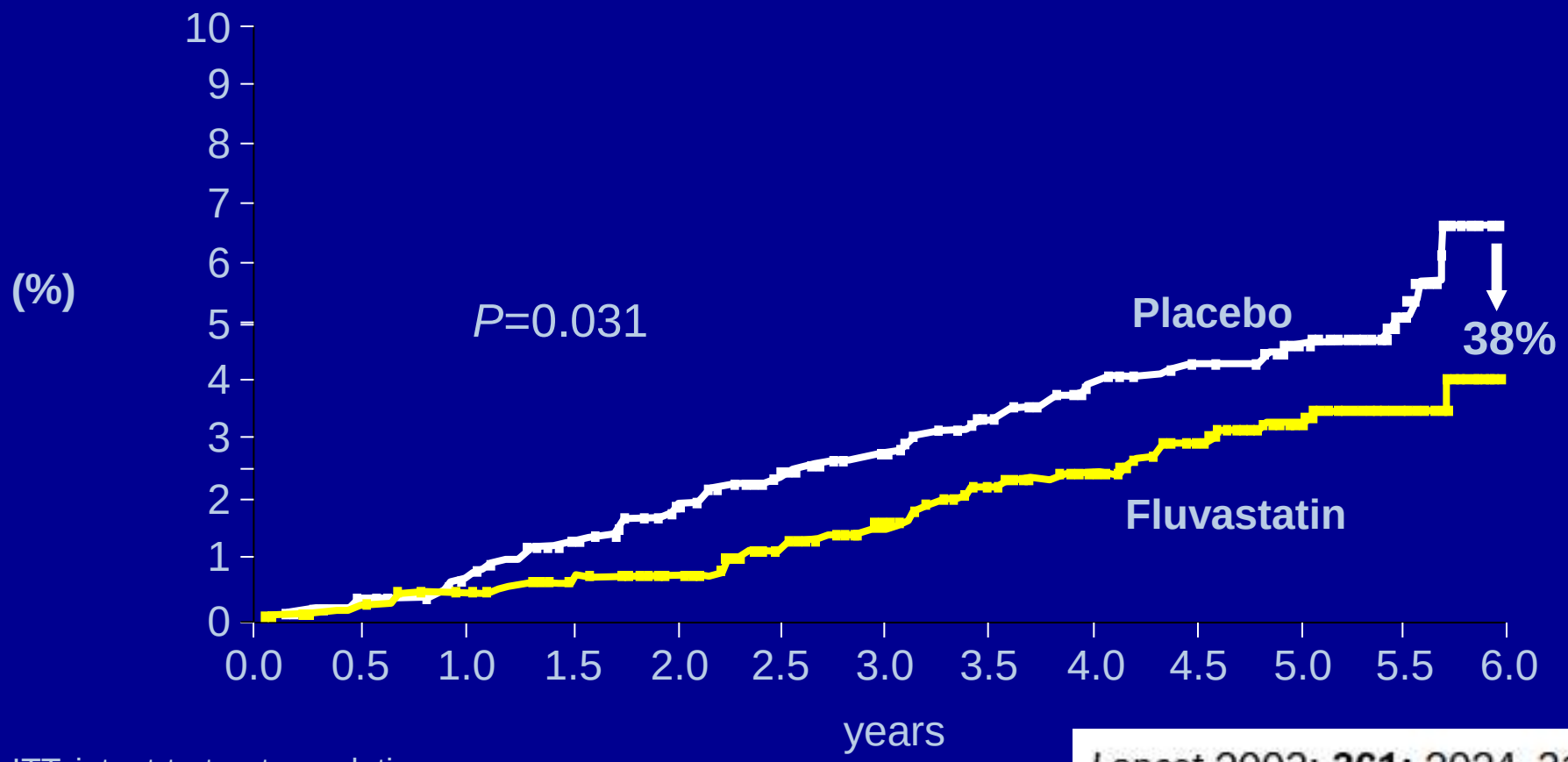
Kasiske BL et al. *J Am Soc Nephrol.* 2000;65:1735-1743.
Wilson PWF et al. *Circulation.* 1998;97:1837-1847.

ALERT: Assessment of Lescol in Renal Transplantation

- Randomized, double blind, placebo controlled multicentric study, 2102 Tx patients
- Fluvastatin (40 mg/d - 80 mg/d) or placebo
- Outcome: cardiac mortality, AMI, coronary intervention

Effect of fluvastatin on cardiac outcomes in renal transplant recipients: a multicentre, randomised, placebo-controlled trial

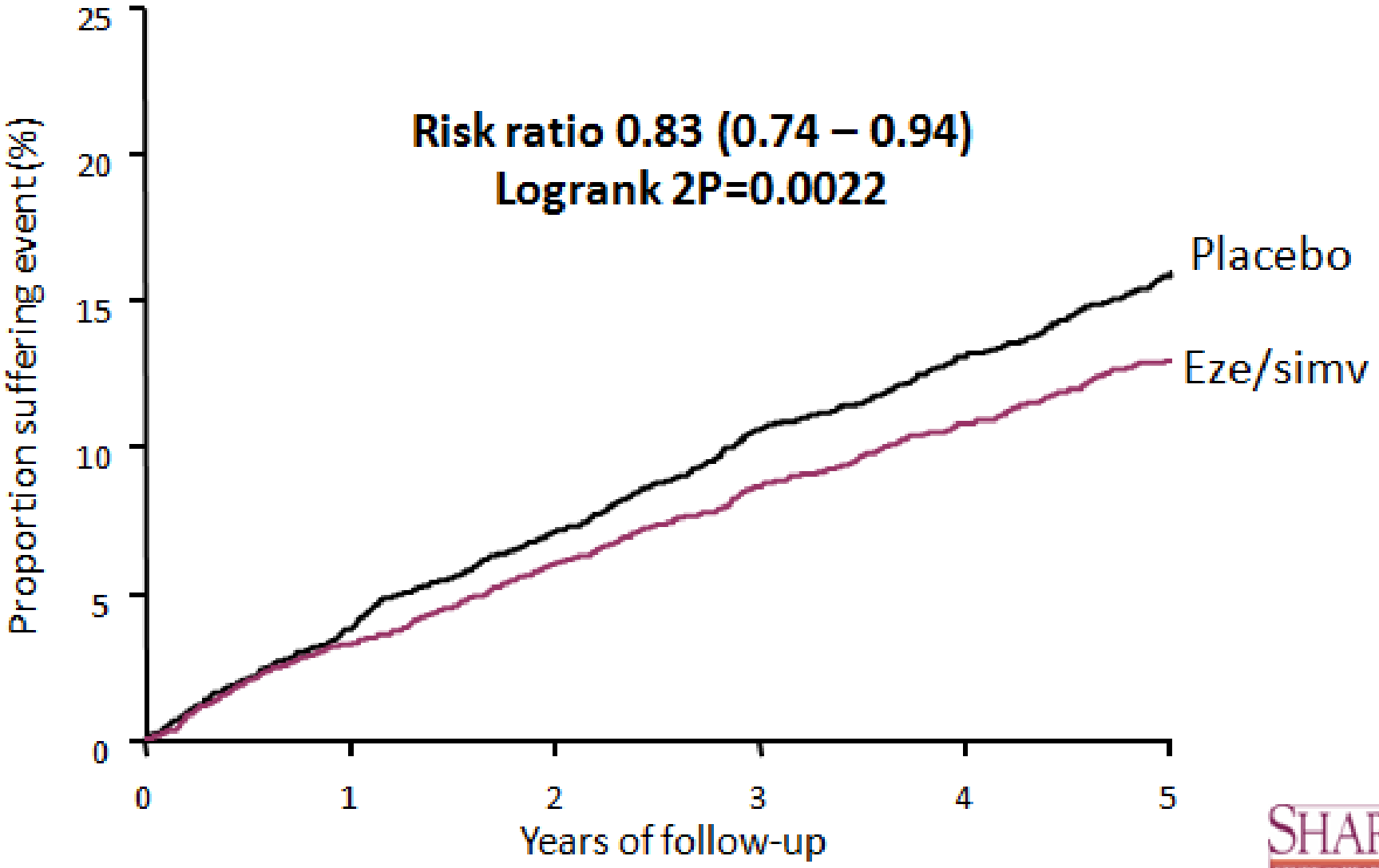
*Hallvard Holdaas, Bengt Fellström, Alan G Jardine, Ingar Holme, Gudrun Nyberg, Per Fauchald, Carola Grönhagen-Riska, Søren Madsen, Hans-Hellmut Neumayer, Edward Cole, Bart Maes, Patrice Ambühl, Anders G Olsson, Anders Hartmann, Dag O Solbu, Terje R Pedersen, on behalf of the Assessment of LEscol in Renal Transplantation (ALERT) Study Investigators**



ITT, intent-to-treat population.

Lancet 2003; **361:** 2024–31.

SHARP: Major Atherosclerotic Events



SHARP
STUDY OF HEART AND RENAL PROTECTION

Endorsement of the Kidney Disease Improving Global Outcomes (KDIGO) guidelines on kidney transplantation: a European Renal Best Practice (ERBP) position statement

Uwe Heemann[1], Daniel Abramowicz[2], Goce Spasovski[3] and Raymond Vanholder[4]
for the European Renal Best Practice (ERBP) Work Group on kidney transplantation

16.2.1: Measure a complete lipid profile in all adult (≥18 years old) and adolescent (puberty to 18 years old) KTRs (based on KDOQI Dyslipidemia Recommendation 1):

- 2–3 months after transplantation;
- 2–3 months after a change in treatment or other conditions known to cause dyslipidaemias;
- at least annually, thereafter.

Nephrol Dial Transplant (2011) 26: 2099–2106
doi: 10.1093/ndt/gfr169
Advance Access publication 9 May 2011

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Uwe Heemann[1], Daniel Abramowicz[2], Goce Spasovski[3] and Raymond Vanholder[4]
for the European Renal Best Practice (ERBP) Work Group on kidney transplantation

16.2.2.1: For KTRs with fasting triglycerides ≥500 mg/dL (≥5.65 mmol/L) that cannot be corrected by removing an underlying cause, treat with:

- Adults: therapeutic lifestyle changes and a triglyceride-lowering agent (based on KDOQI Recommendation 4.1);
- Adolescents: therapeutic lifestyle changes (based on KDOQI Recommendation 5.1).

Nephrol Dial Transplant (2011) 26: 2099–2106
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- Adults: If low density lipoprotein cholesterol (LDL)-C ≥100 mg/dL (≥2.59 mmol/L), treat to reduce LDL-C to <100 mg/dL (<2.59 mmol/L) (based on KDOQI Guideline 4.2);
- Adolescents: If LDL-C ≥130 mg/dL (≥3.36 mmol/L), treat to reduce LDL-C to <130 mg/dL (<3.36 mmol/L) (based on KDOQI Guideline 5.2).

Nephrol Dial Transplant (2011) 26: 2099–2106
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Obesity

Obesity is BAD!

- 1. Obesity is associated with increased morbidity and mortality, esp. Metabolic Syndrome and Diabetes mellitus, in the general population.
- 2. Obesity is a risk factor for the development of CKD and ESRD.
- 3. Obesity is a risk factor for CVD, CAD and CHF.
- 4. Obesity is associated with increased pro-inflammatory cytokines & oxidative stress.

BMI = Weight (kg) / height (m)^2

<20: Lean (<18.5: Malnourished?)

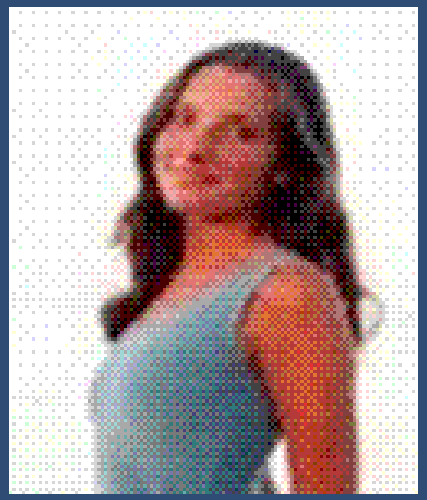
20-25: Healthy ?

25-30: Overweight

30-35: Obese

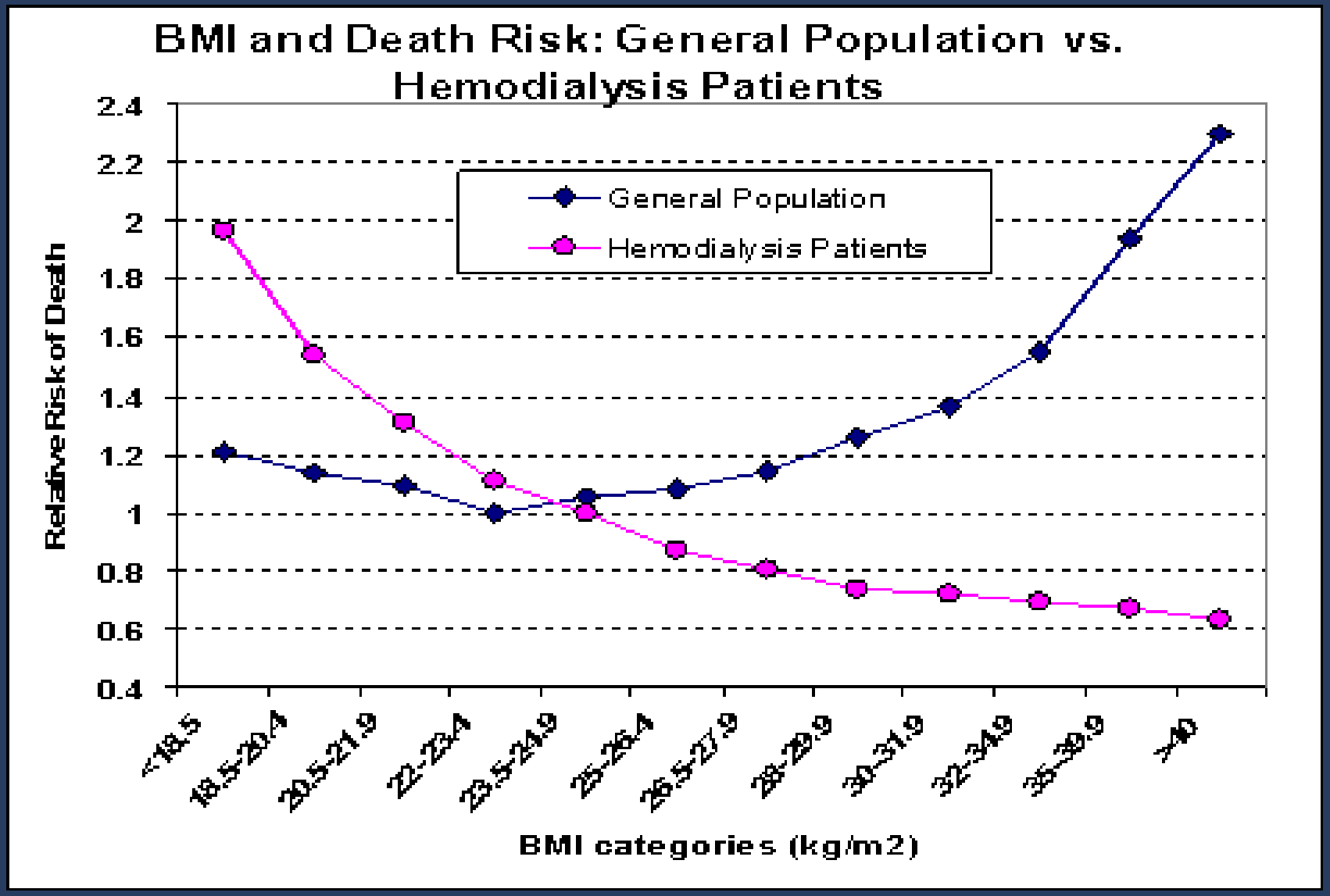
>35: Morbidly obese (>40 if no other risk)

Who will survive longer on dialysis?



Female: 28 y/o
weight 123 lbs
BMI 21 kg/m^2
BP 110/65
Cholesterol 141 mg/dL

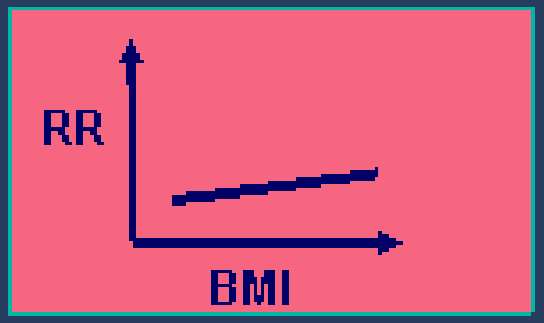
Female: 26 y/o
weight 241 lbs
BMI 43 kg/m^2
BP 165/105
Cholesterol 220 mg/dL


BMI and Death Risk: General Population vs. Hemodialysis Patients
General Population
Hemodialysis Patients
Relative Risk of Death
2.4
2.2
2
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
<18.5
18.5-20.4
20.5-21.9
22-23.4
23.5-24.9
25-26.4
26.5-27.9
28-29.9
30-31.9
32-34.9
35-39.9
>40
BMI categories (kg/m2)

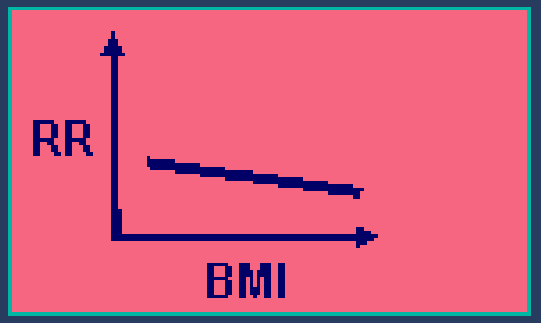
Why is there a reverse epidemiology in the dialysis population?

Are there other populations with similar epidemiology?

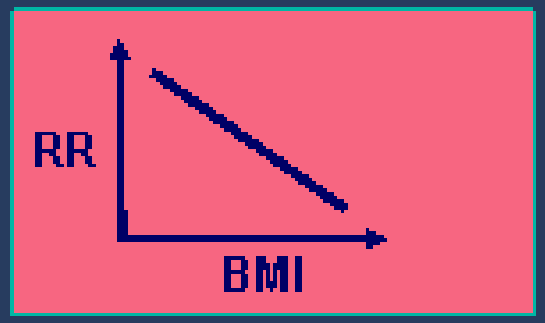
Aging → Risk Factor Reversal



> 65 years



65-75 years



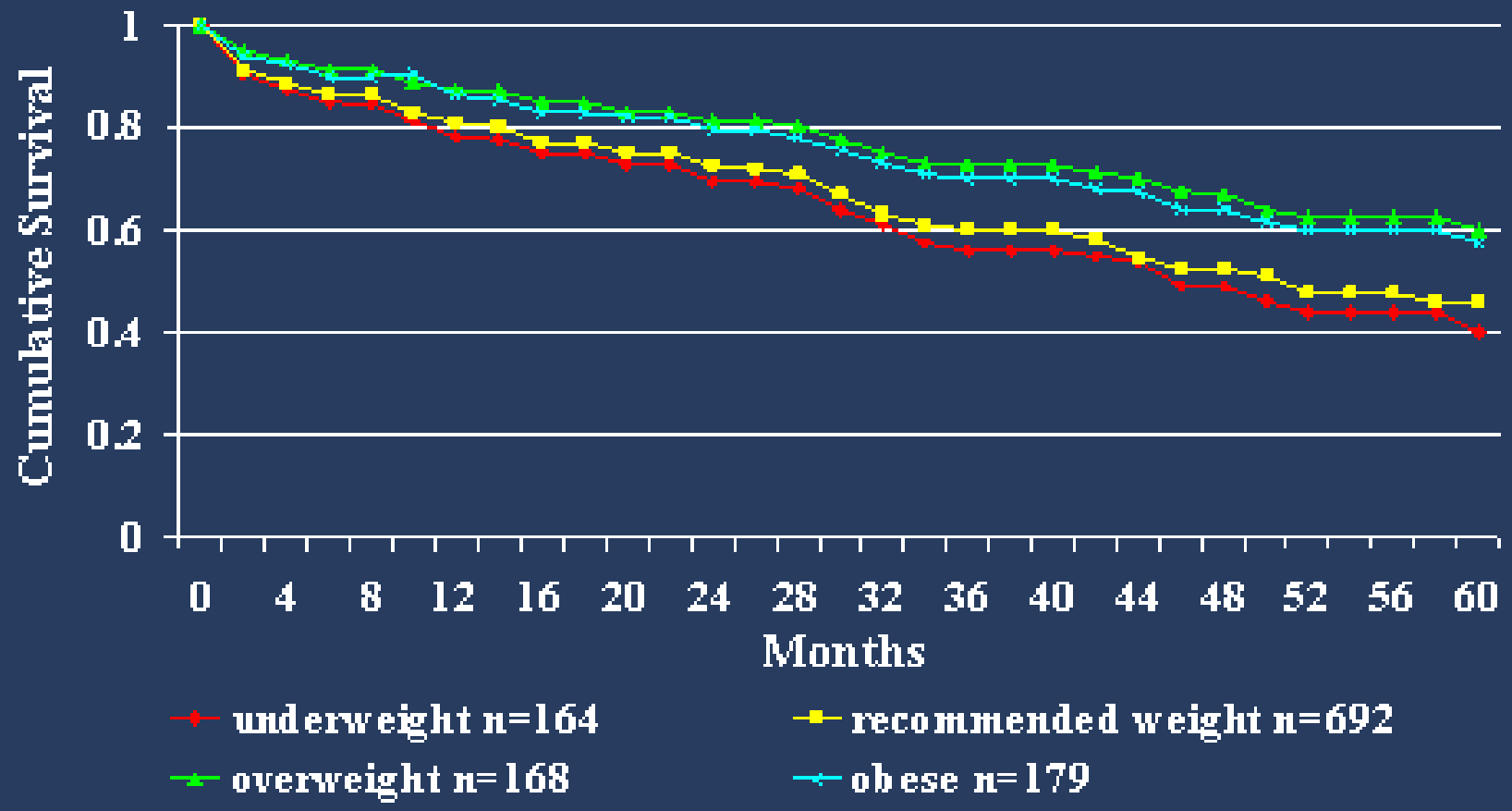
>75 years

Obesity-related excess mortality declines with age at all levels of obesity!

Stevens et al, NEJM, 1998
Bender et al. JAMA, 1999
Landi et al, Arch Int Med, 2000

Risk-adjusted five year survival in CHF patients for the BMI categories

Horwich et al, J Am Coll Cardiol 2001;38:789-795



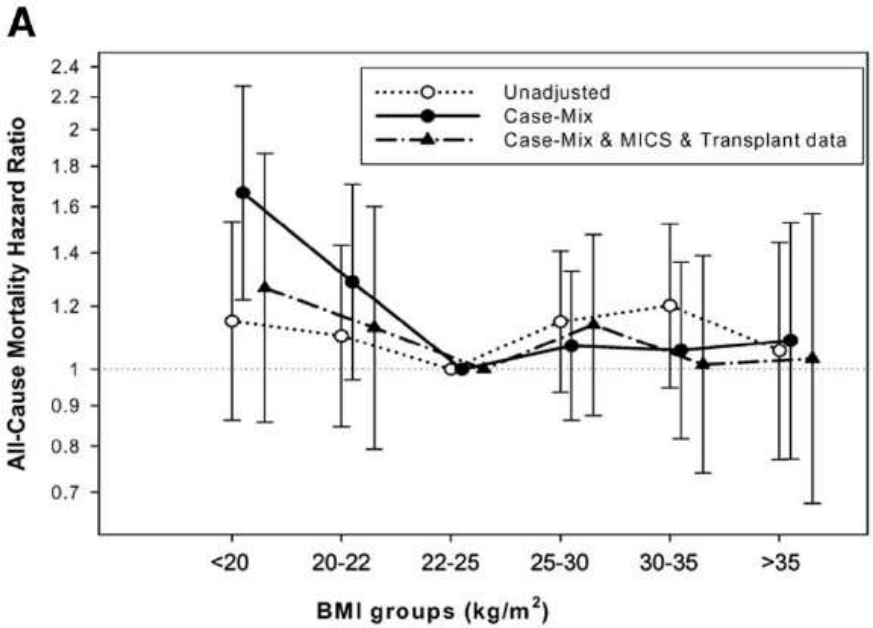
Arnold's BMI: 37 kg/m2 (1995)

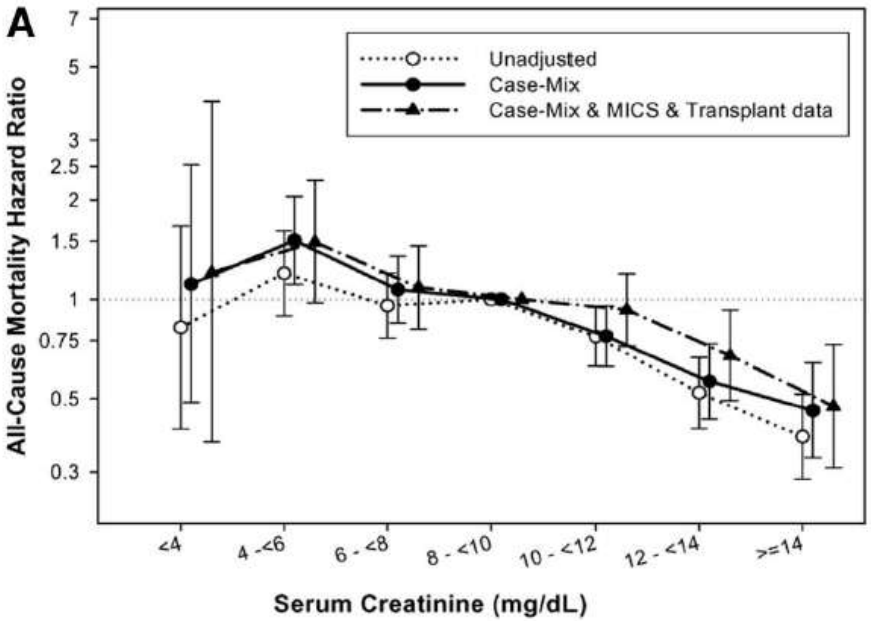
Actor or Athlete	Height	Weight in lbs. *(in 2003)*	BMI
Sylvester Stallone	5'9"	228	34
Arnold Schwarzenegger	6'2"	257	33
Sammy Sosa	6'0"	220	30
Harrison Ford	6'1"	218	29
George Clooney	5'11"	211	29
Bruce Willis	6'0"	211	29
Mike Piazza	6'3"	215	27
Brad Pitt	6'0"	203	27
Michael Jordan	6'6"	216	25

From: "Celebrity Height Weight Chart"

Associations of Pretransplant Weight and Muscle Mass with Mortality in Renal Transplant Recipients

Elani Streja,† Miklos Z. Molnar,*‡ Csaba P. Kovesdy§‖ Suphamai Bunnapradist¶ Jennie Jing,* Allen R. Nissenson,¶** Istvan Mucsi,†† Gabriel M. Danovitch,¶ and Kamyar Kalantar-Zadeh*†¶*





***CJASN ePress*. Published on March 17, 2011**

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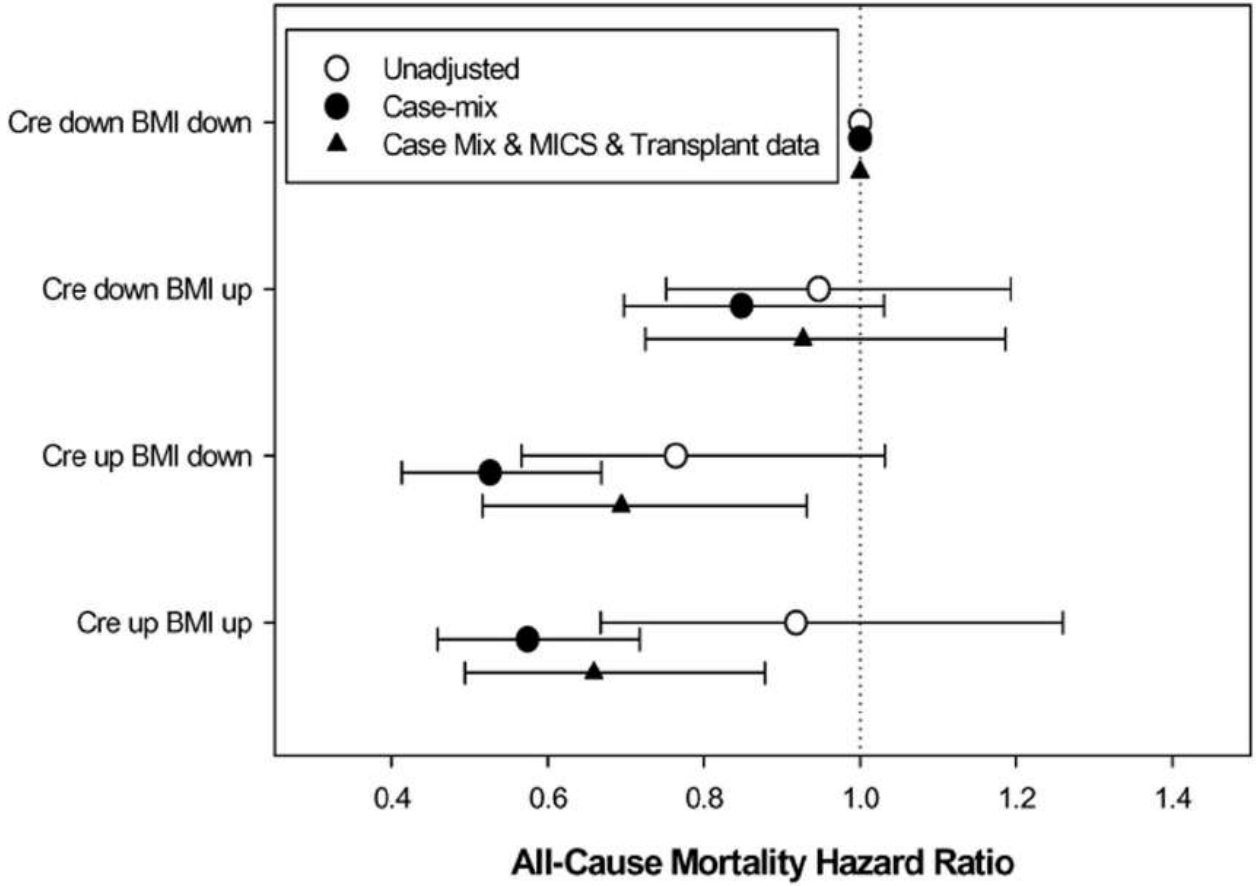
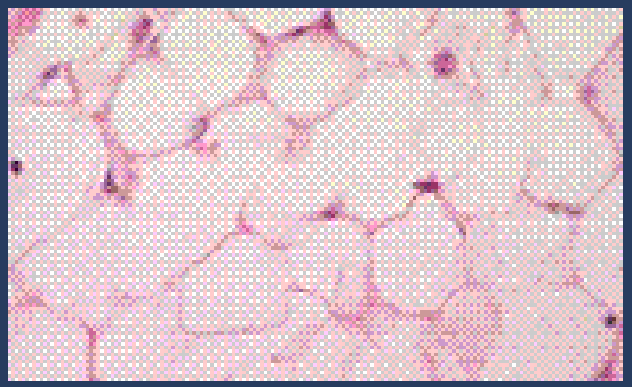


***CJASN ePress*. Published on March 17, 2011**

Adipocytokines → *modulation of inflammation*



Bad Cytokines

TNF-α

IL-6

Leptin

Resistin

+

Inflammation

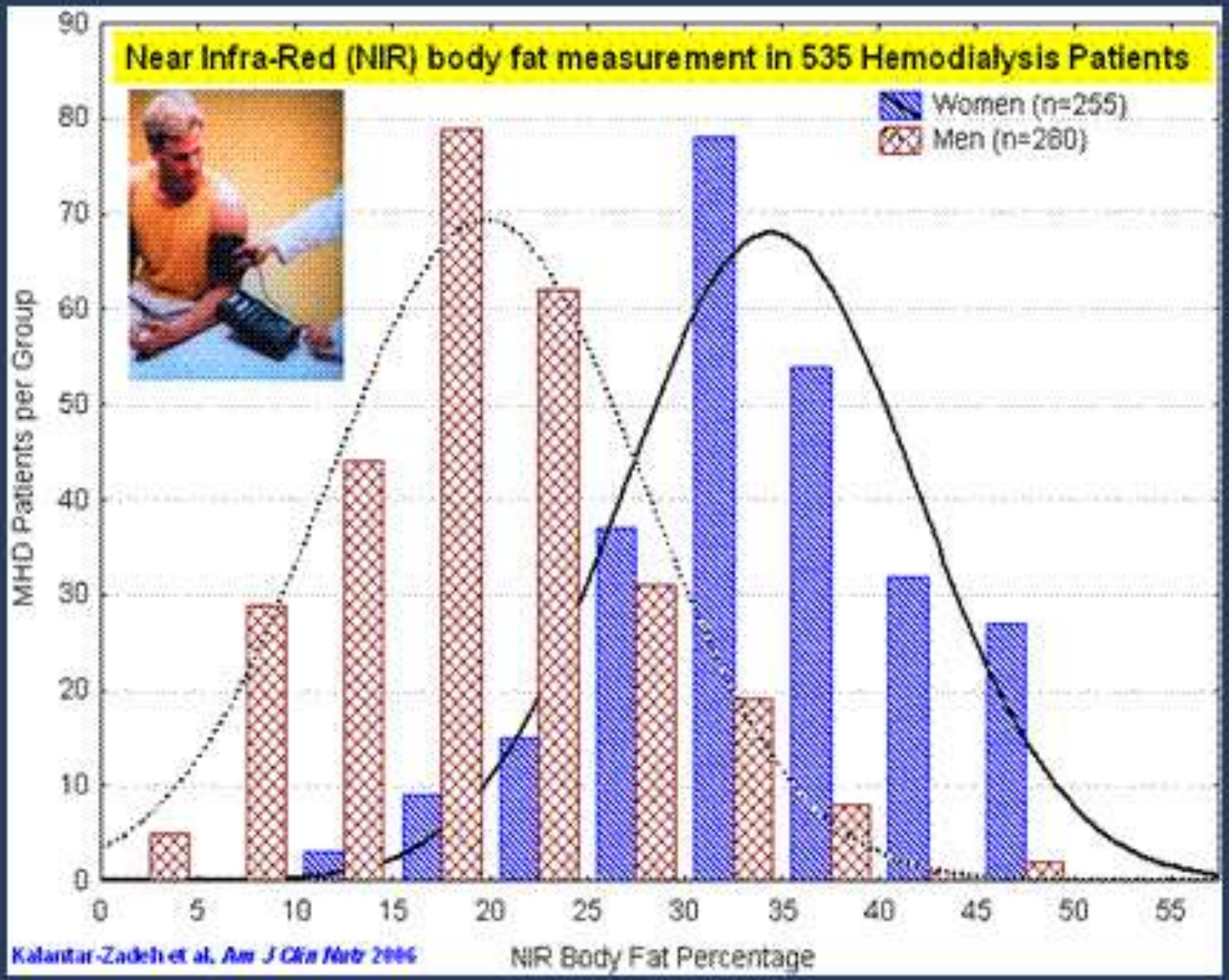
Good Cytokines

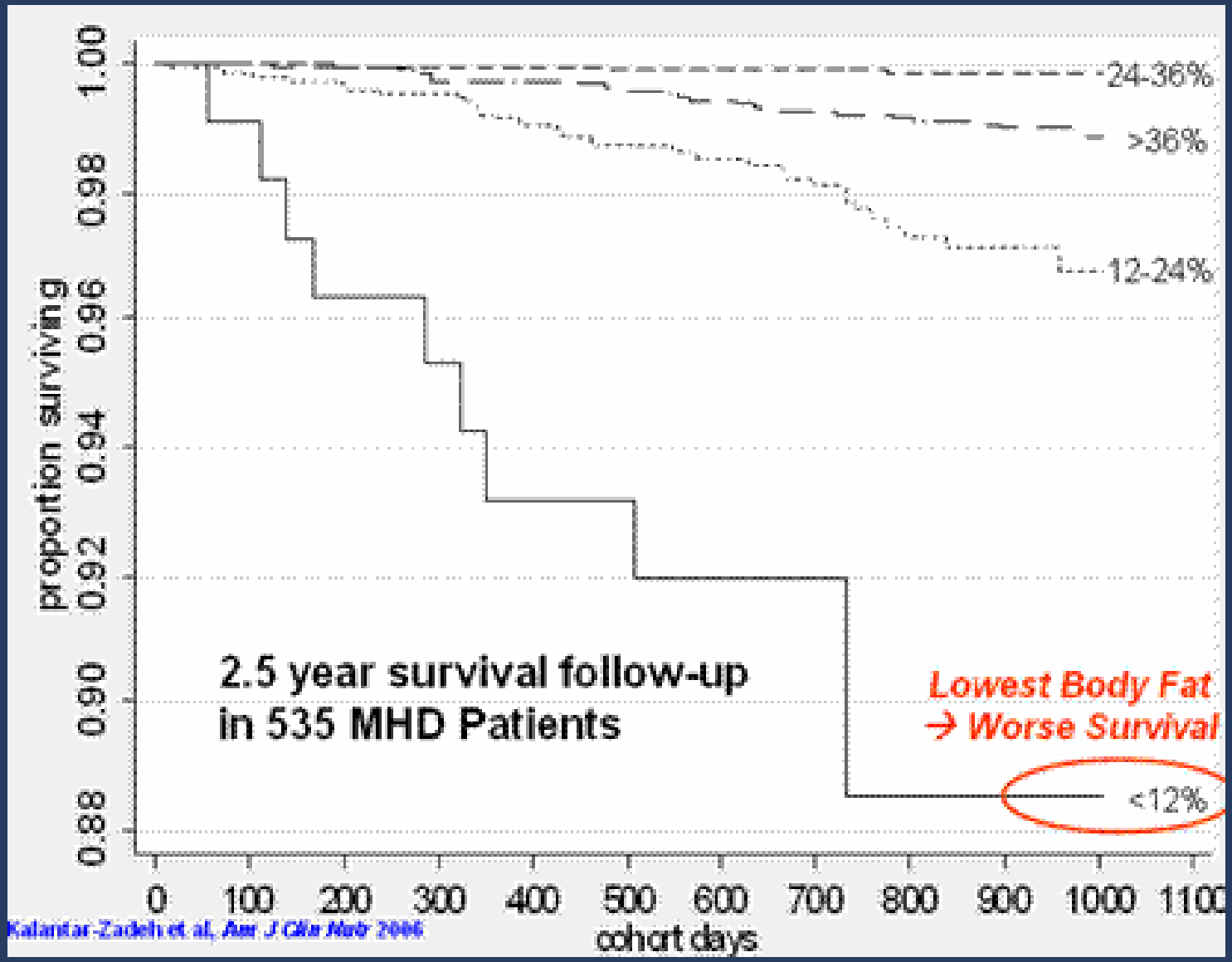
Adiponectin

?

IL-10

-


Near Infra-Red (NIR) body fat measurement in 535 Hemodialysis Patients
Women (n=255)
Men (n=280)
MHD Patients per Group
NIR Body Fat Percentage
0
10
20
30
40
50
60
70
80
90
0
5
10
15
20
25
30
35
40
45
50
55
Kalantar-Zadeh et al. Am J Clin Nutr 2006


proportion surviving
1.00
0.98
0.96
0.94
0.92
0.90
0.88
24-36%
>36%
12-24%
<12%
2.5 year survival follow-up
in 535 MHD Patients
Lowest Body Fat
→ Worse Survival
0
100
200
300
400
500
600
700
800
900
1000
1100
cohort days
Kalantar-Zadeh et al, Am J Clin Nutr 2006

Body Mass Index, Waist Circumference and Mortality in Kidney Transplant Recipients

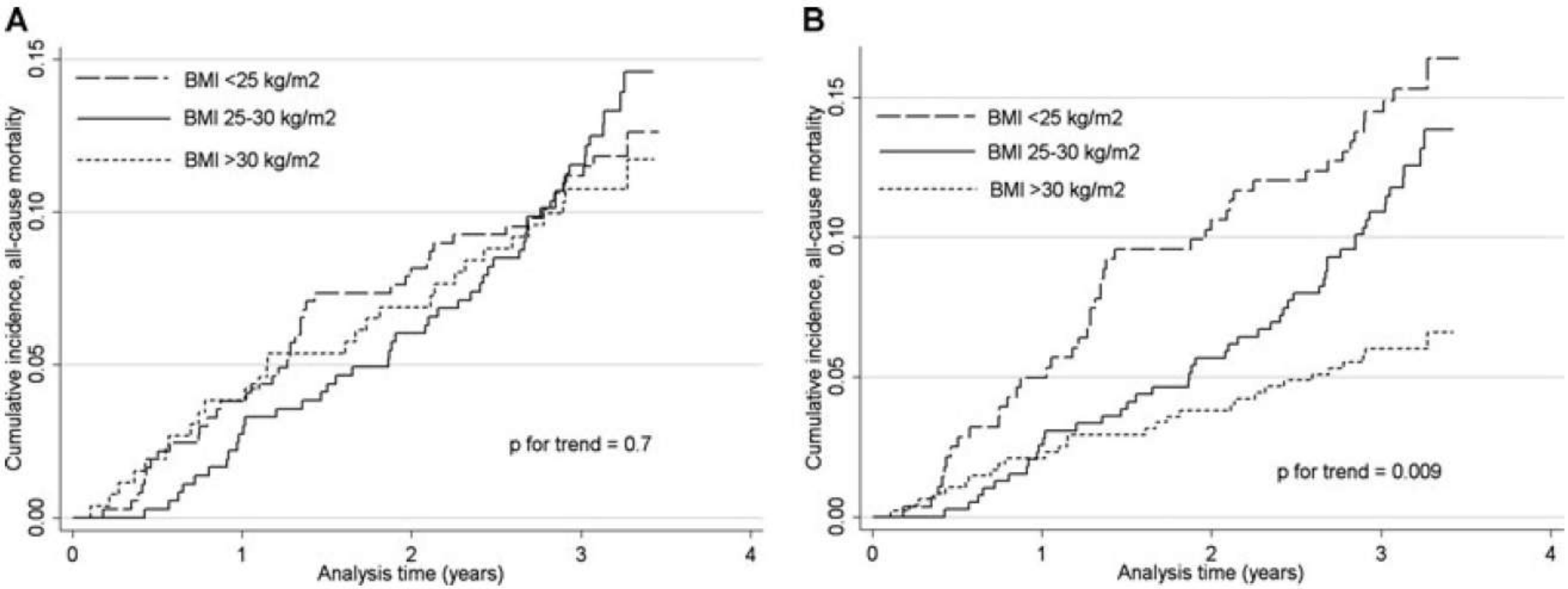


Figure 1: Kaplan–Meier curves of unadjusted (A) and waist circumference-adjusted (B) cumulative incidence of all-cause mortality in kidney transplant recipients grouped according to their body mass index.

C. P. Kovesdy[a,b,*], M. E. Czira[c], A. Rudas[c], A. Ujszaszi[c], L. Rosivall[d], M. Novak[c,e], K. Kalantar-Zadeh[f], M. Z. Molnar[c,d,f] and I. Mucsi[c,d,g]

American Journal of Transplantation 2010; 10: 2644–2651

Body Mass Index, Waist Circumference and Mortality in Kidney Transplant Recipients

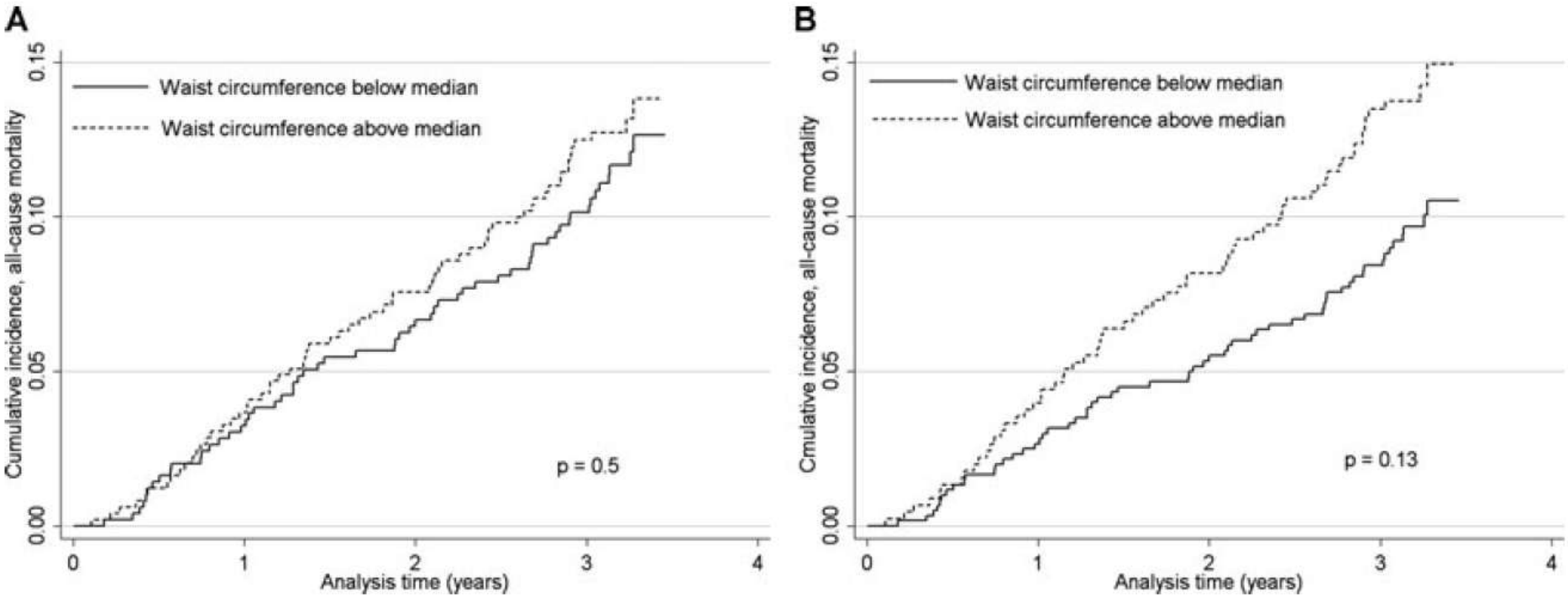


Figure 2: Kaplan–Meier curves of unadjusted (A) and body mass index-adjusted (B) cumulative incidence of all-cause mortality in kidney transplant recipients grouped according to their waist circumference. Median waist circumference was 103 cm in males and 93 cm in females.

C. P. Kovesdy[a,b,*], M. E. Czira[c], A. Rudas[c], A. Ujszaszi[c], L. Rosivall[d], M. Novak[c,e], K. Kalantar-Zadeh[f], M. Z. Molnar[c,d,f] and I. Mucsi[c,d,g]

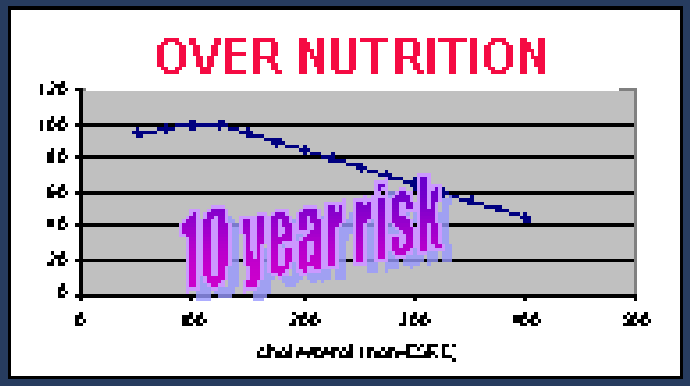
American Journal of Transplantation 2010; 10: 2644–2651

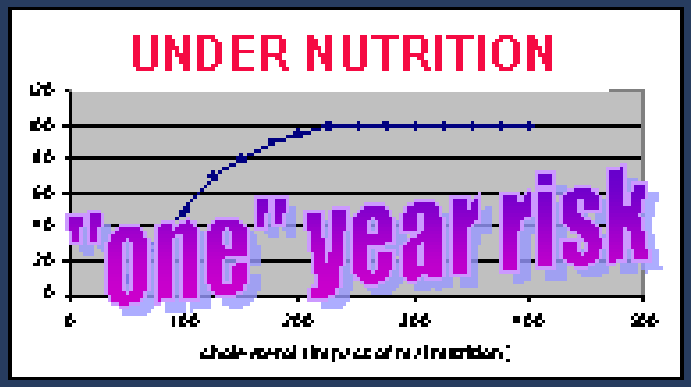
Why is there an Obesity Paradox?

Kidney Disease Wasting (Malnutrition-inflammation-complex syndrome)
Time discrepancy between competitive risk factors: overnutrition vs. undernutrition
Unusual genetic constellation due to survival selection during CKD progression
Sequestration/storage of uremic toxins in fat tissue
Anti-inflammatory cytokines related to body mass, including adiponectins
Tumor necrosis factor alpha receptors
Endotoxin-lipoprotein hypothesis
Stability of hemodynamic status in obese patients
Neurohormonal alterations in obesity
Alteration of conventional risk factors in uremic milieu ("beyond Framingham")
Reverse causation
Survival bias
Advantages of obesity in the history of man kind (the Ultimate Hypothesis)

Kalantar-Zadeh & Kopple, *Contrib Nephrol* 2006

Reverse Epidemiology of ESRD: Competitive Risk Factors with Time Discrepancy





KDIGO clinical practice guideline for the care of kidney transplant recipients

16.4: OBESITY

16.4.1: Assess obesity at each visit. *(Not Graded)*

- **Measure height and weight at each visit, in adults and children.**
- **Calculate BMI at each visit.**
- **Measure waist circumference when weight and physical appearance suggest obesity, but BMI is <35 kg/m^2.**

16.4.2: Offer a weight-reduction program to all obese KTRs. *(Not Graded)*

American Journal of Transplantation 2009; 9 (Suppl 3): S1–S157

Malnutrition-inflammation/ Protein-energy wasting

Malnutrition Inflammation Complex Syndrome (MICS)
Protein-Energy Wasting
Kidney Disease Wasting
Cachexia-in-Slow-Motion

1. Evidence of Protein-Energy Malnutrition (PEM):
   - Wasting syndrome (cachexia), ↓ BMI
   - ↓ lean body mass
   - ↓ appetite, ↓ food intake, ↓ nPNA (nPCR)
   - ↓ cholesterol, ↓ albumin, ↓ transferrin

2. Evidence of Inflammation:
   - ↑ CRP
   - ↑ pro-inflammatory cytokines (IL-6, TNF-α, IL-1β)
   - ↑ EPO resistance
3. High cardiovascular disease, high mortality

Malnutrition-inflammation complex syndrome (MICS)

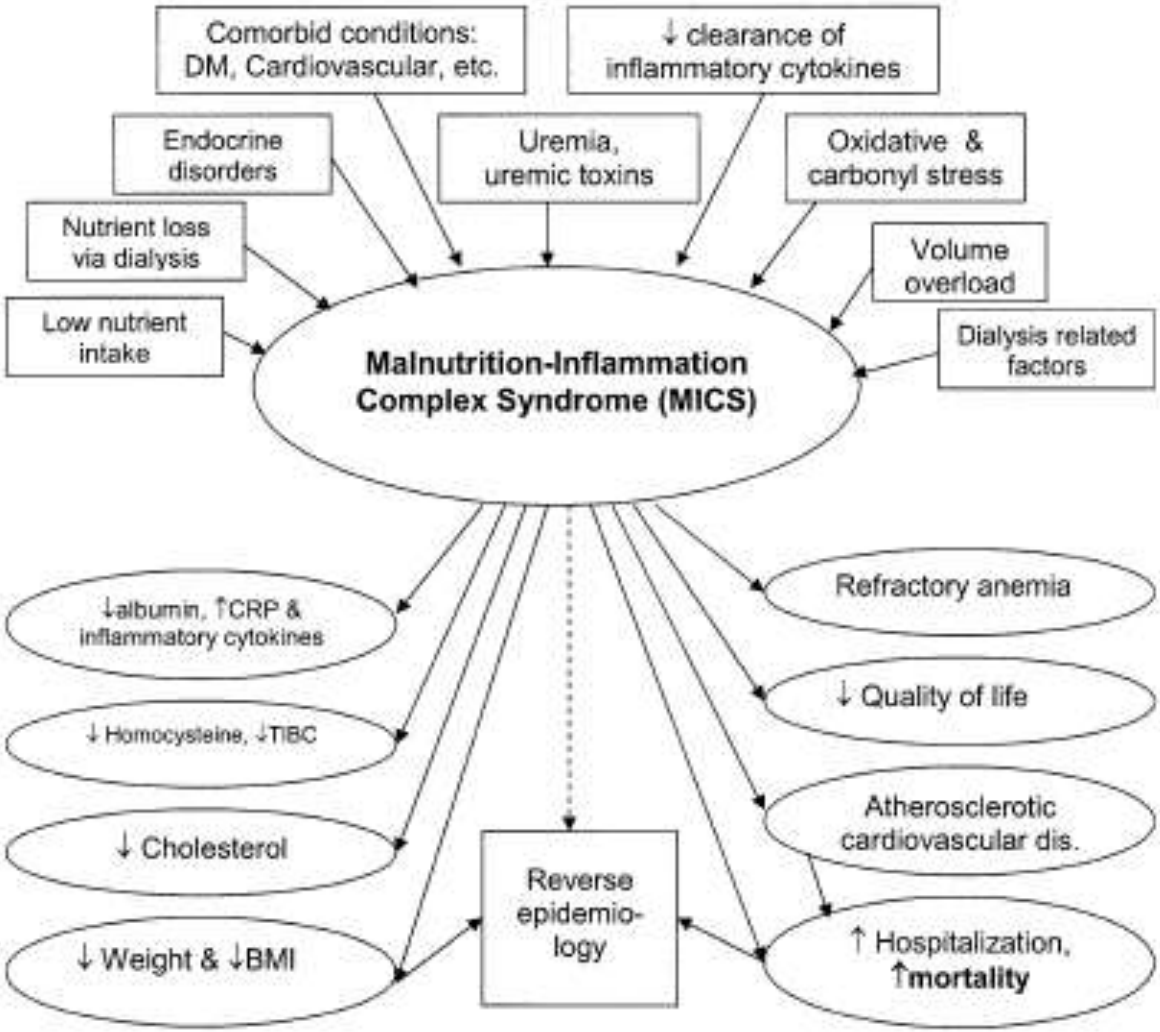
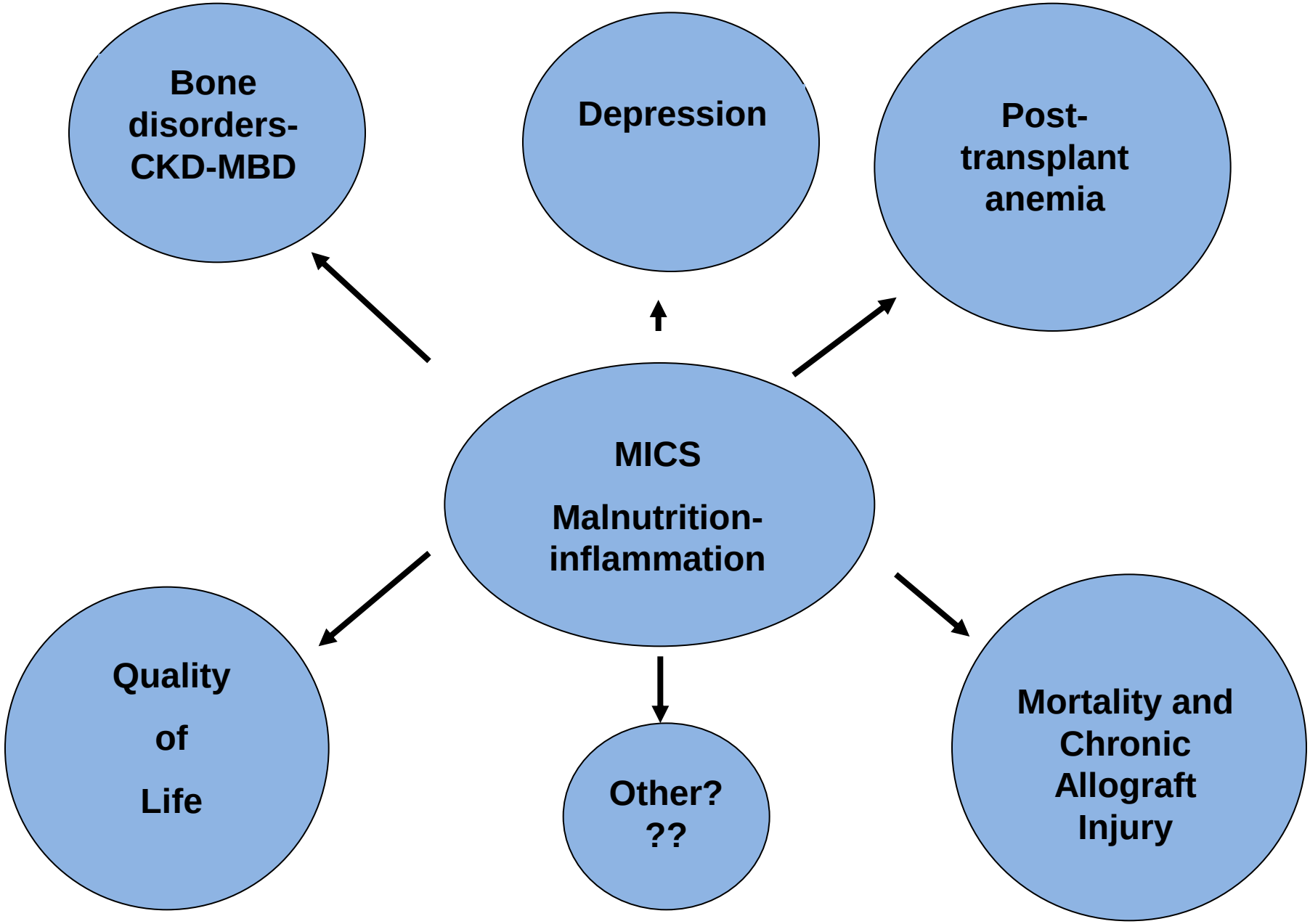


Fig 2. Schematic representation of the causes and consequences of MICS. Abbreviation: DM, diabetes mellitus.

Kalantar-Zadeh; AJKD; 2003

MICS in kidney transplanted patients

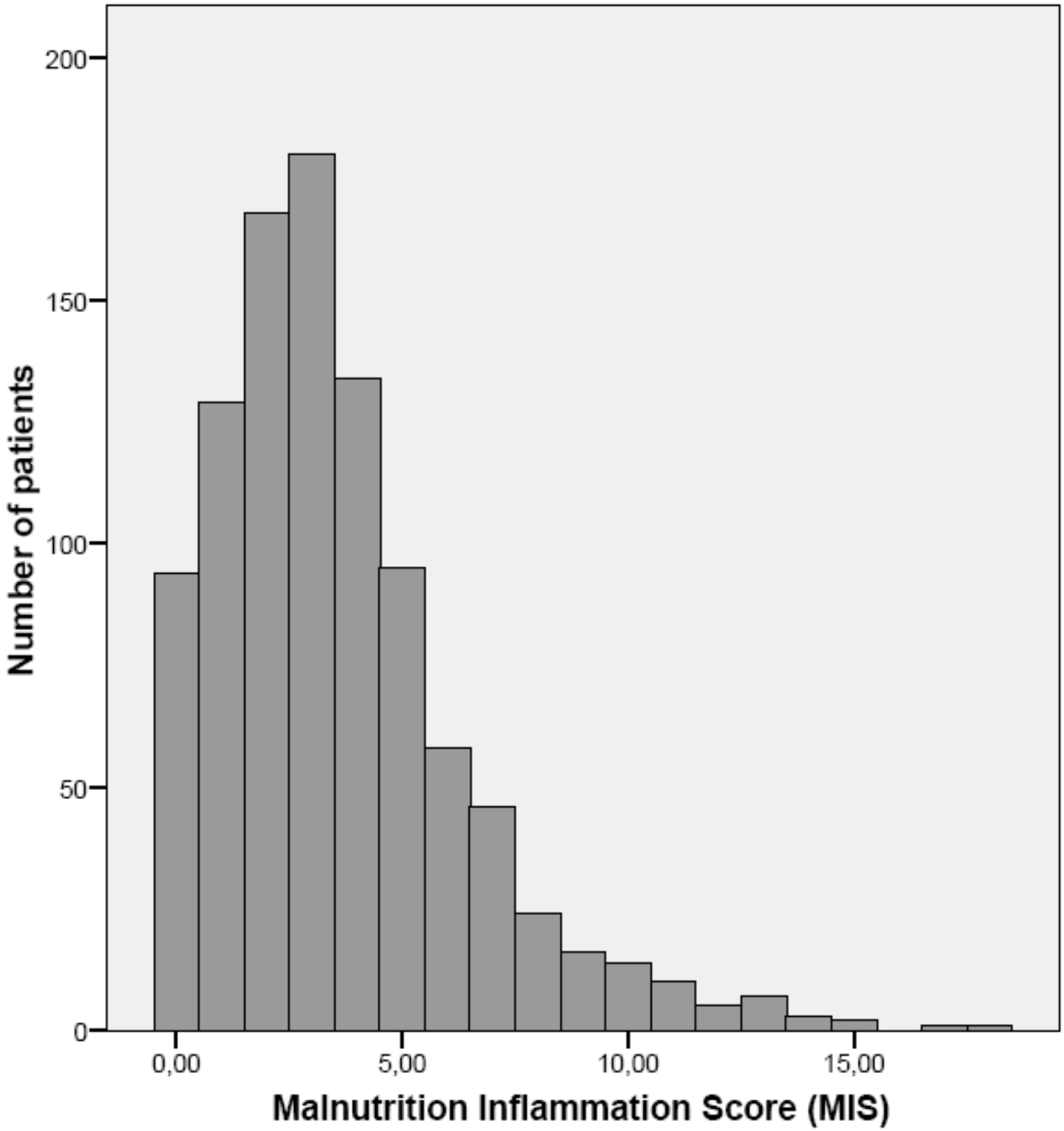


Malnutrition and Inflammation Score (MIS)

(Kalantar-Zadeh et al.)

A. Medical history
   A. Change in weight over past 3-6 months
   B. Dietary intake
   C. Gastrointestinal symptoms
   D. Functional capacity
   E. Comorbidity
B. Physical exam (according to SGA criteria)
   A. Decreased fat stores or loss of subcutaneous fat
   B. Signs of muscle wasting
C. Body mass index (BMI)
D. Laboratory results
   A. Serum albumin
   B. Serum transferrin

- MICS was assessed by the MIS score developed by Kalantar-Zadeh.



Distribution of MIS in our KTx patients

Association between the malnutrition-inflammation score and post-transplant anaemia

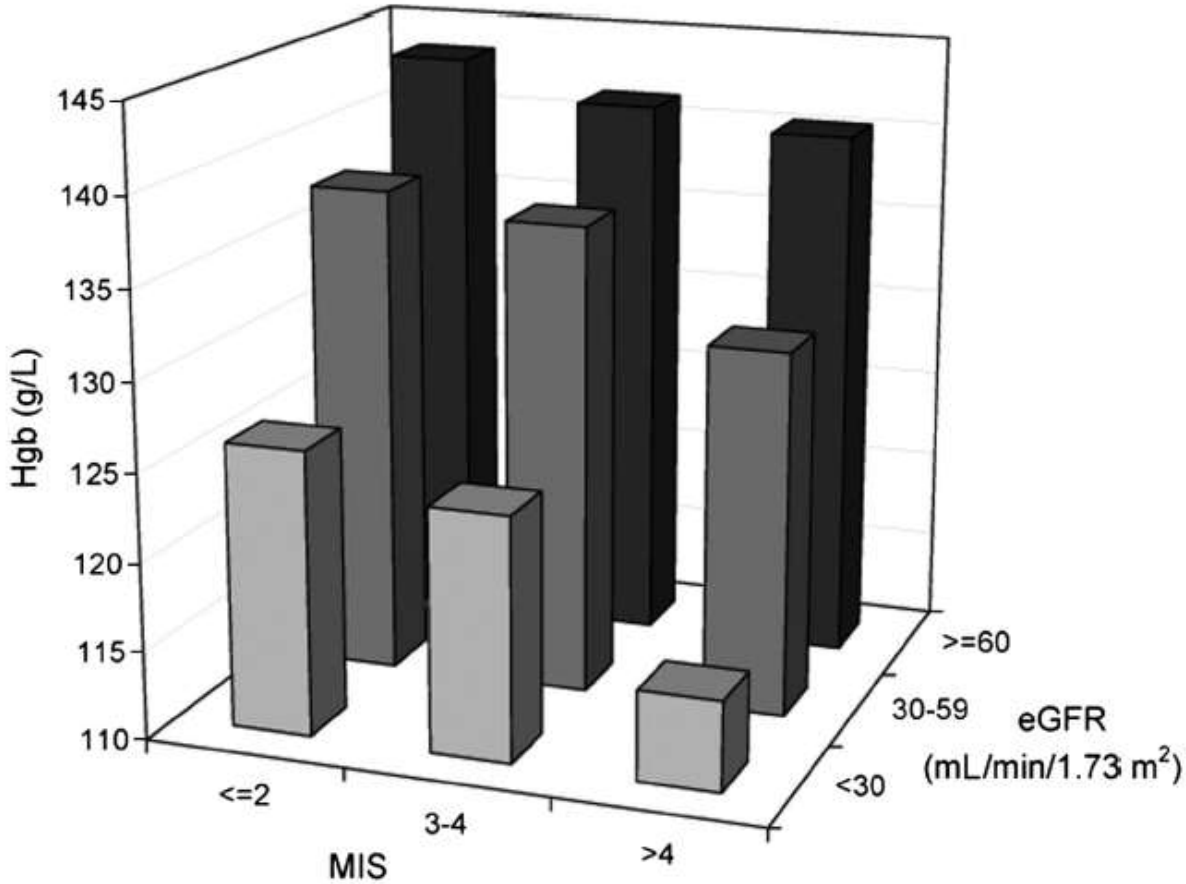
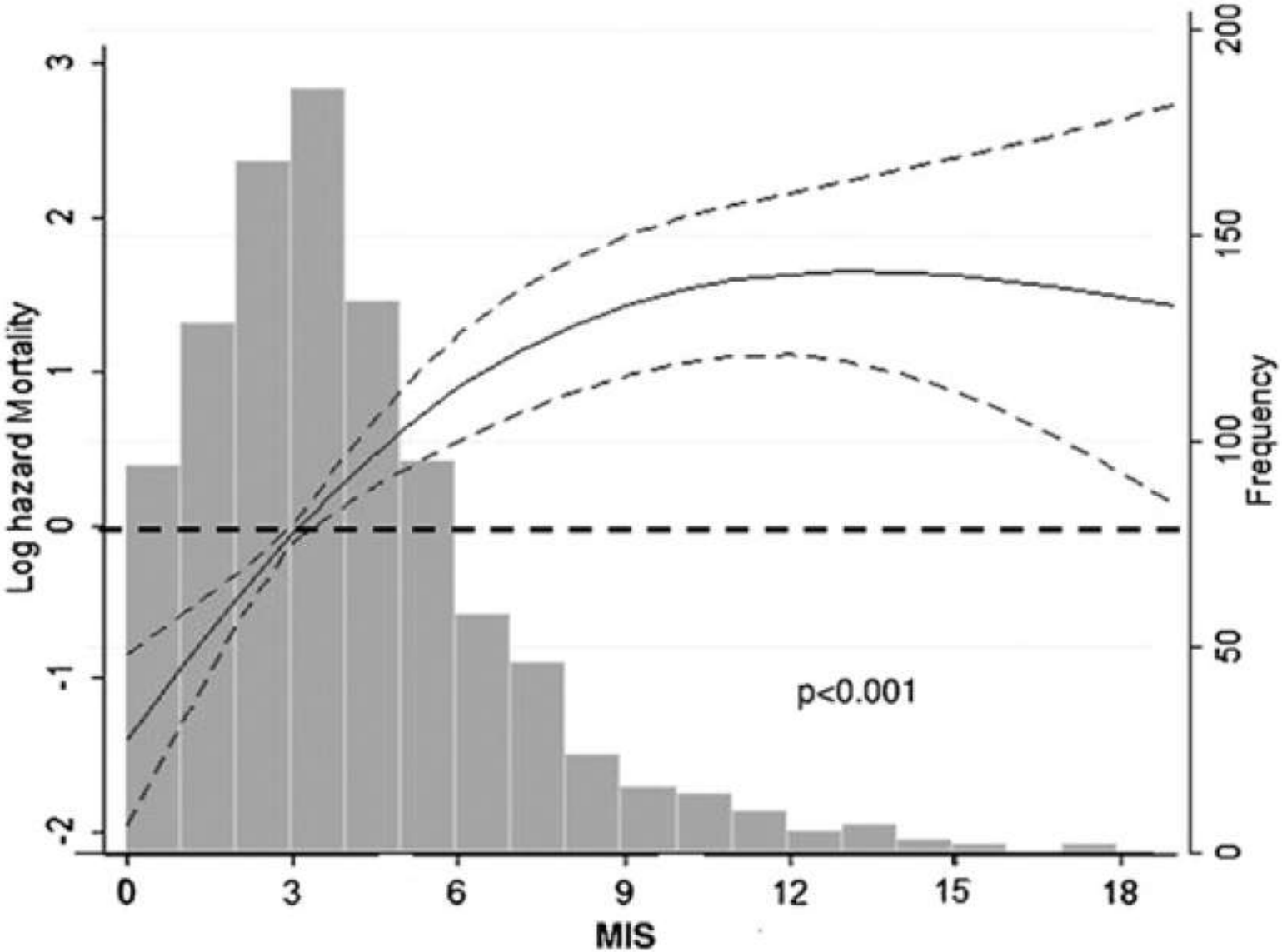


Fig. 2. Predicted Hb levels from the final, fully adjusted multivariate linear regression model (Model 2) in subgroups defined by MIS and eGFR.

MZ Molnar et al.:

Nephrol Dial Transplant (2011) 26: 2000–2006

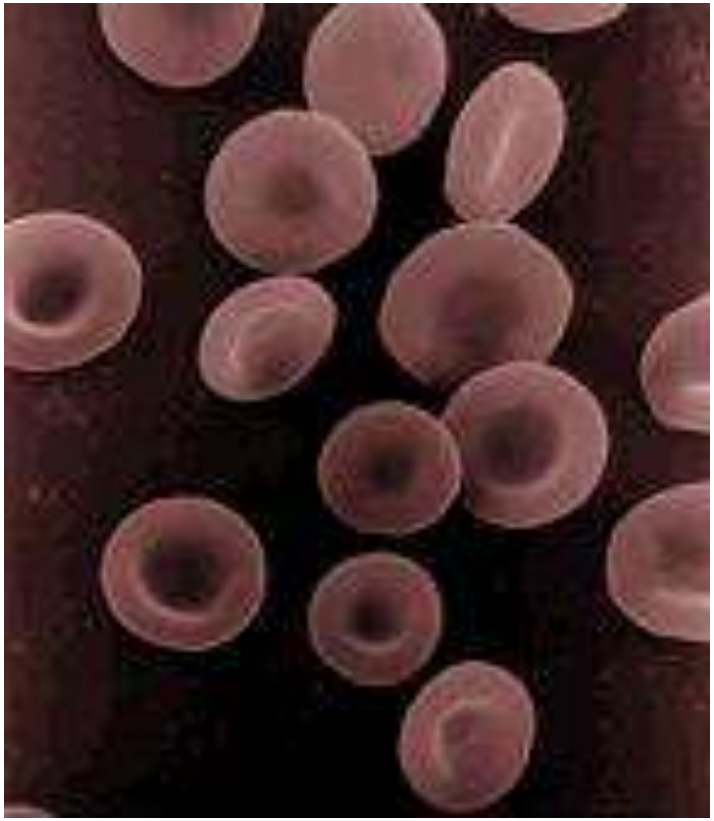
Association of the Malnutrition-Inflammation Score With Clinical Outcomes in Kidney Transplant Recipients



MZ Molnar et al.: Am J Kidney Dis. 58(1):101-108. © 2011

Red Cell Distribution Width?

- RDW = the variation in red blood cell volume (anisocytosis)
- Elevated in variety of diseases
  - Iron deficiency
  - Malnutrition
  - Chronic kidney disease
- Calculated automatically on every CBC



Red cell distribution width in heart failure: Prediction of clinical events and relationship with markers of ineffective erythropoiesis, inflammation, renal function, and nutritional state

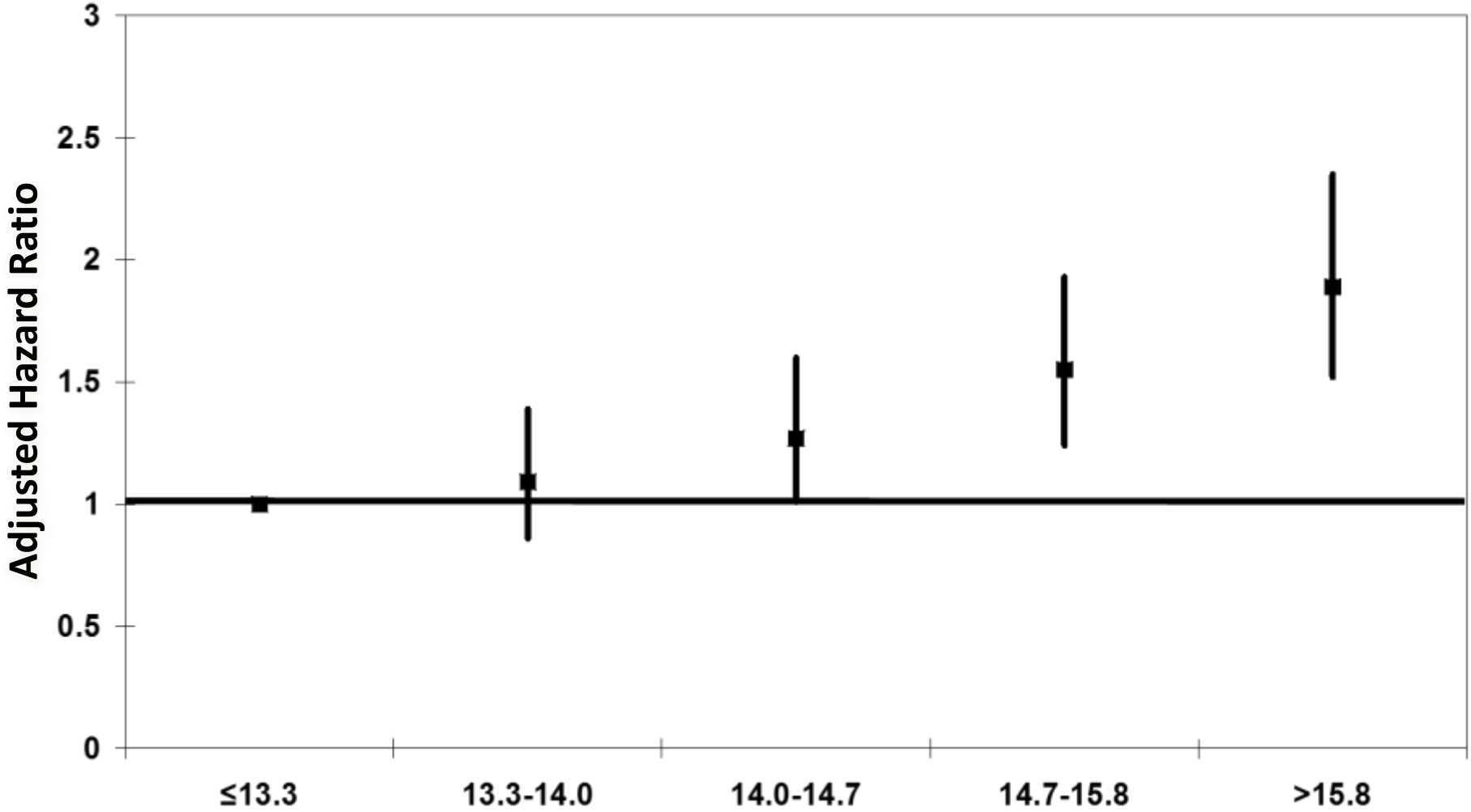
Table IV. Baseline characteristics of the cohort stratified by RDW values

	RDW ≤13.9%		RDW 13.9%-15.2%		RDW ≥15.2%		
	Median	Interquartile range	Median	Interquartile range	Median	Interquartile range	P*
Markers of ineffective erythropoiesis							
Iron (μmol/L)	15.4	10.9-19.4	13	9-16.9	10	6.64-13.7	<.0001
Ferritin (μg/L)	140.7	94.6-259.4	135.8	66.5-220.7	98.8	57.6-157.9	.01
Transferrin (g/L)	2.7	2.4-3.0	2.7	2.4-3.1	2.8	2.4-3.2	.53
Transferrin sat. (%)	23	18-28	18.5	13.5-24	16	10-20	<.0001
Soluble transferrin receptor (nmol/L)	2.3	2.7-4.3	4.8	5.9-7.8	5.9	4.7-7.2	<.0001
EPO (U/mL)	8.9	4.9-15.2	12.6	7.4-18.9	14.1	7.9-25.7	.002
Markers of inflammation							
IL-6 (pg/mL)	6.62	3.88-12.35	10.89	6.89-14.18	14.59	8.52-25.32	.0001
TNF-α (pg/mL)	2.03	1.07-3.62	2.37	1.68-3.47	2.78	1.73-4.75	.077
TNF-RI (ng/mL)	4.61	3.42-6.69	6.64	3.81-10.37	6.93	4.22-10.68	.0007
TNF-RII (ng/mL)	3.40	2.34-4.57	4.42	3.40-5.78	5.07	3.75-6.68	<.0001
Prealbumin (g/L)	0.26	0.21-0.30	0.21	0.17-0.27	0.18	0.14-0.24	<.0001
CRP (mg/L)	4.2	1.7-10.7	8.36	3.6-15.3	6.7	3.9-15.9	.01
Markers of damaged renal function							
GFR (mL/1.73 m^2 per min)	75	59-95	65.5	45-81	55	38-77.5	<.0001
Creatinine (μmol/L)	91	75-105	97.5	78-142	111.5	84.5-165.5	.0045
Markers of nutritional deficiency							
Albumin (g/L)	43	40.5-45	41	39-44	39	36-42	<.0001
Total protein (g/L)	72	67-76	73	69-78	70	62.76	.052
Total cholesterol (mmol/L)	4.21	3.81-5.31	4.48	3.89-5.11	3.62	2.95-4.14	<.0001

Z Forhecz et al.: (Am Heart J 2009;158:659-66.)

CHARM Adjusted HR by quintile of RDW for

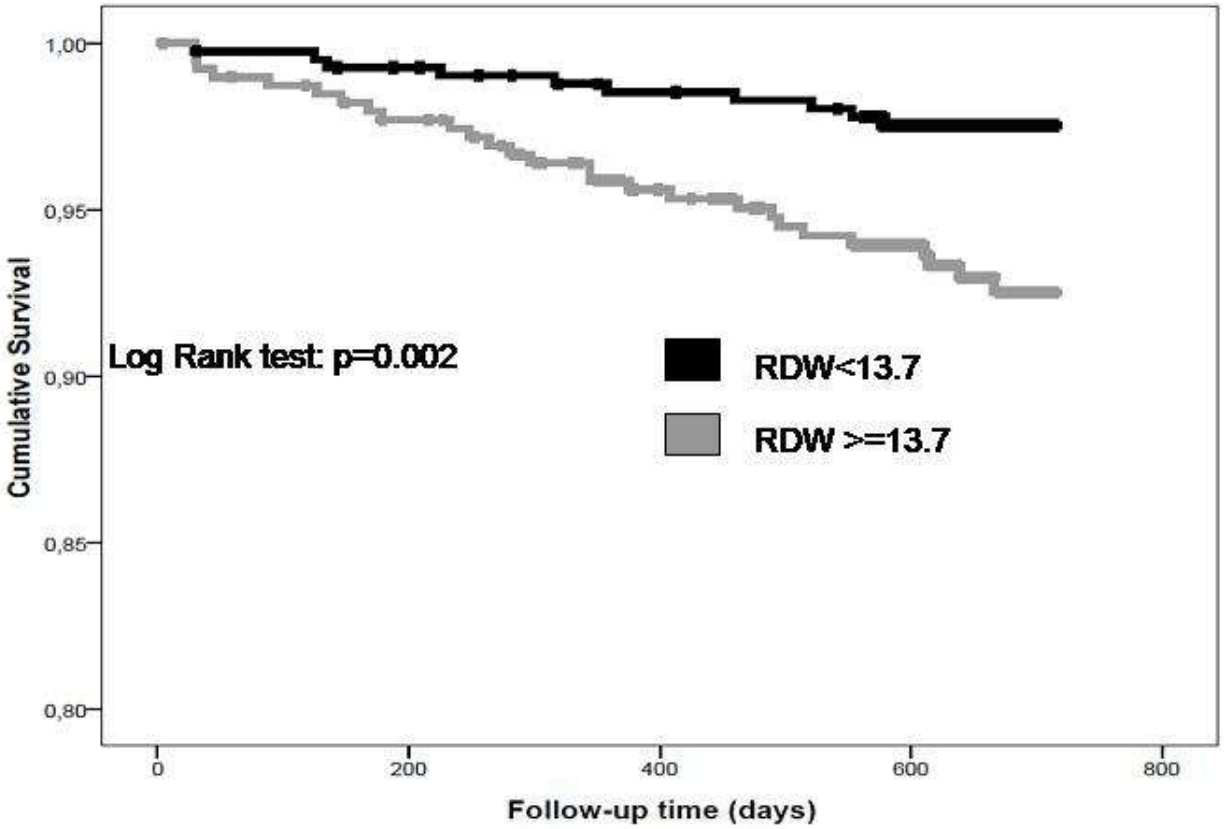
CV Death or HF Hospitalization



Events = 952

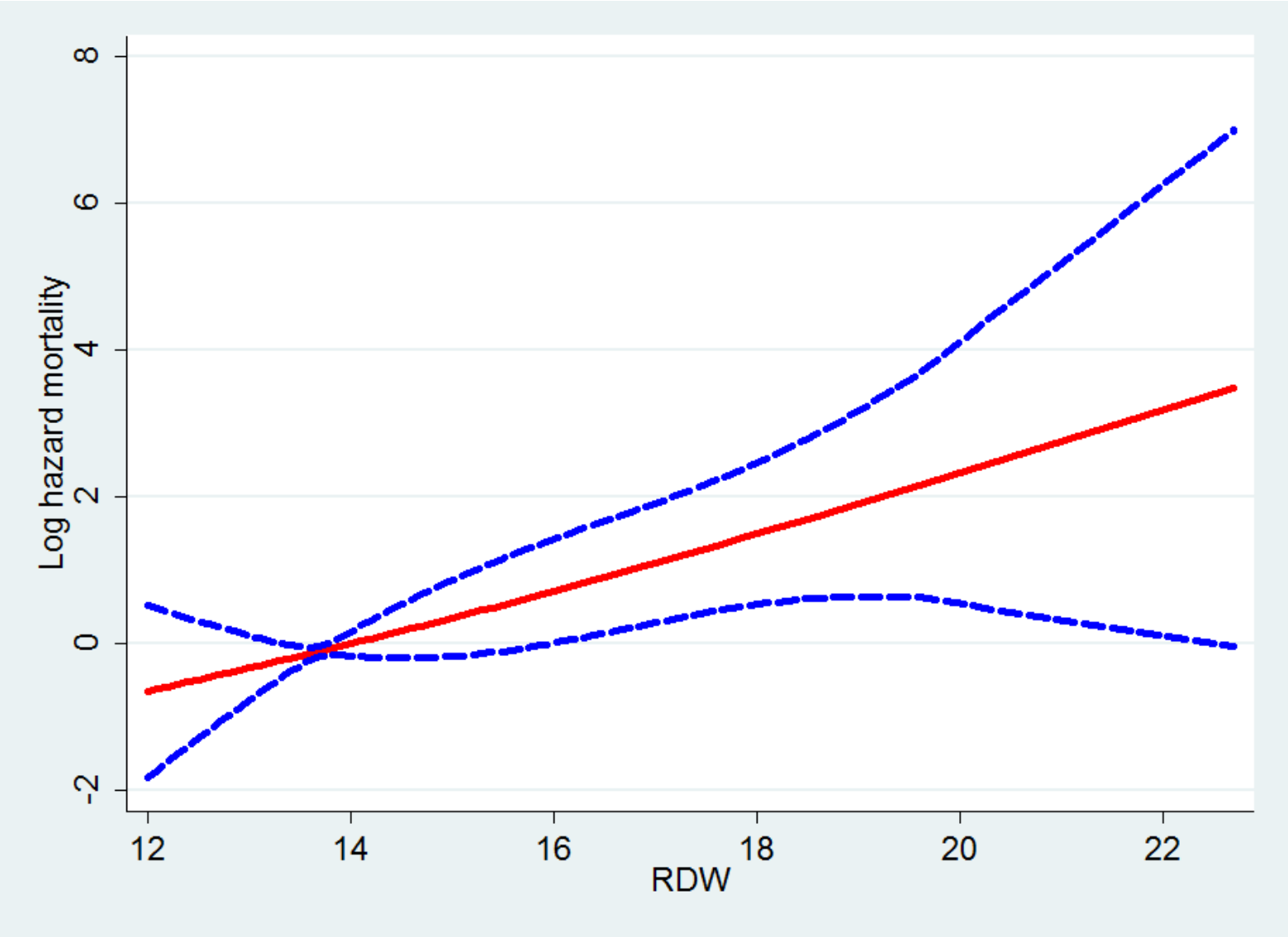
RDW Quintiles

Survival of KTx patients with RDW below vs above median



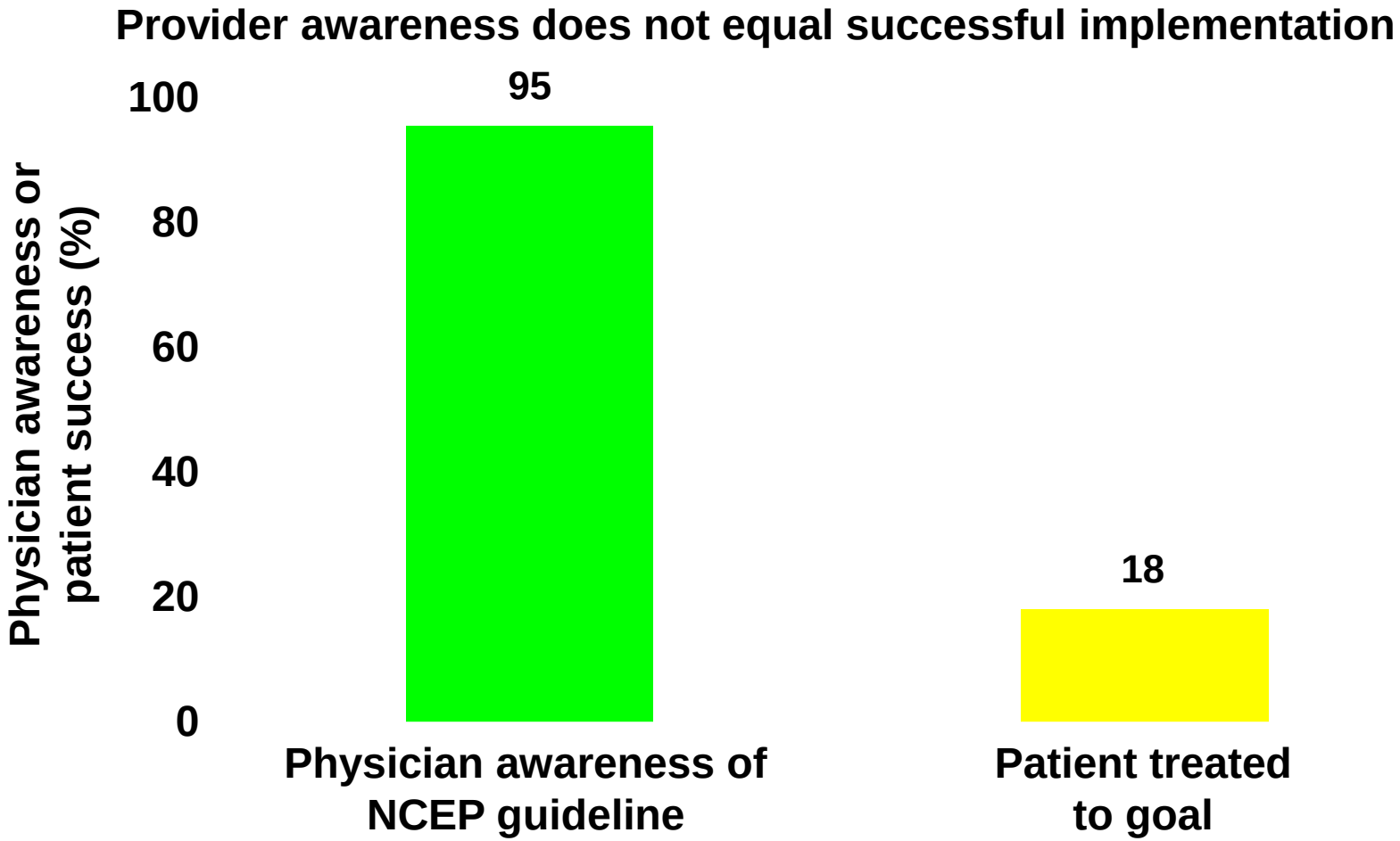
Number of remaining cases					
	411	405	397	348	RDW<13.7
	393	379	351	314	RDW >=13.7

Hazard ratio (95% confidence intervals) of mortality versus RDW using adjusted Cox regression analyses



Multidisciplinary care

CAD treatment gap in the community



NCEP = National Cholesterol Education Program

Pearson TA, et al. Arch Intern Med 2000;160:459–67

Steno 2: Intensive Therapy

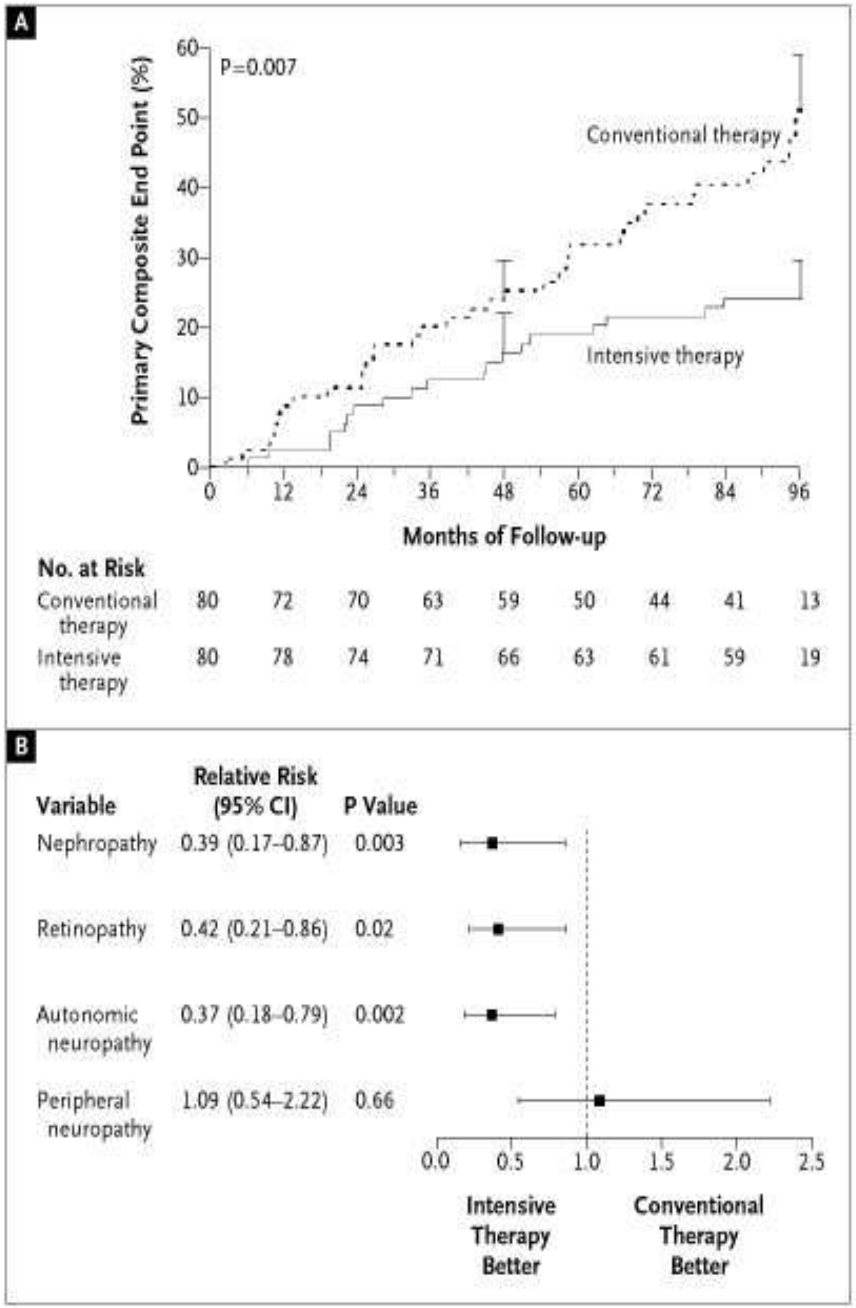
NB: combined cardio/renal protection

- Multidisciplinary team (MD, nurse, dietician)
- Diet
- Exercise 30 minutes 3 – 5x/wk
- Smoking cessation courses
- ACEI/ARB independent of BP
- Vitamin – mineral supplement
- ASA
- Glycemic control
- BP control
- Lipid control

Gaede P et al. NEJM 2003; 348: 383-393

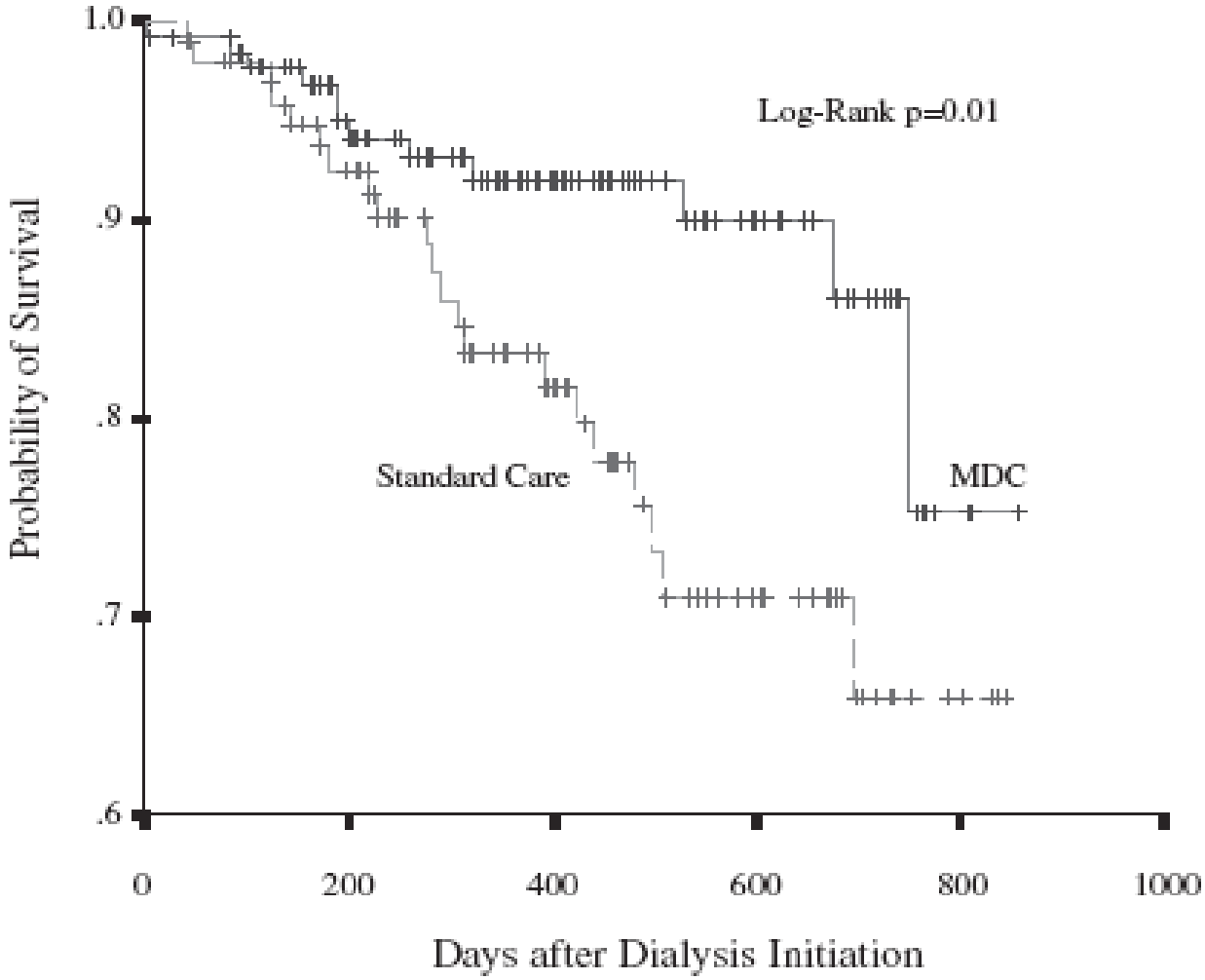
Steno 2: Outcomes

- Hazard ratio = 0.47 in favor of intensive group (.24 - .73, p=0.008)
- Absolute RR = 20%
- NNT 5 patients to prevent one CV event in 7.8 years



Gaede P et al. NEJM 2003; 348: 383-393

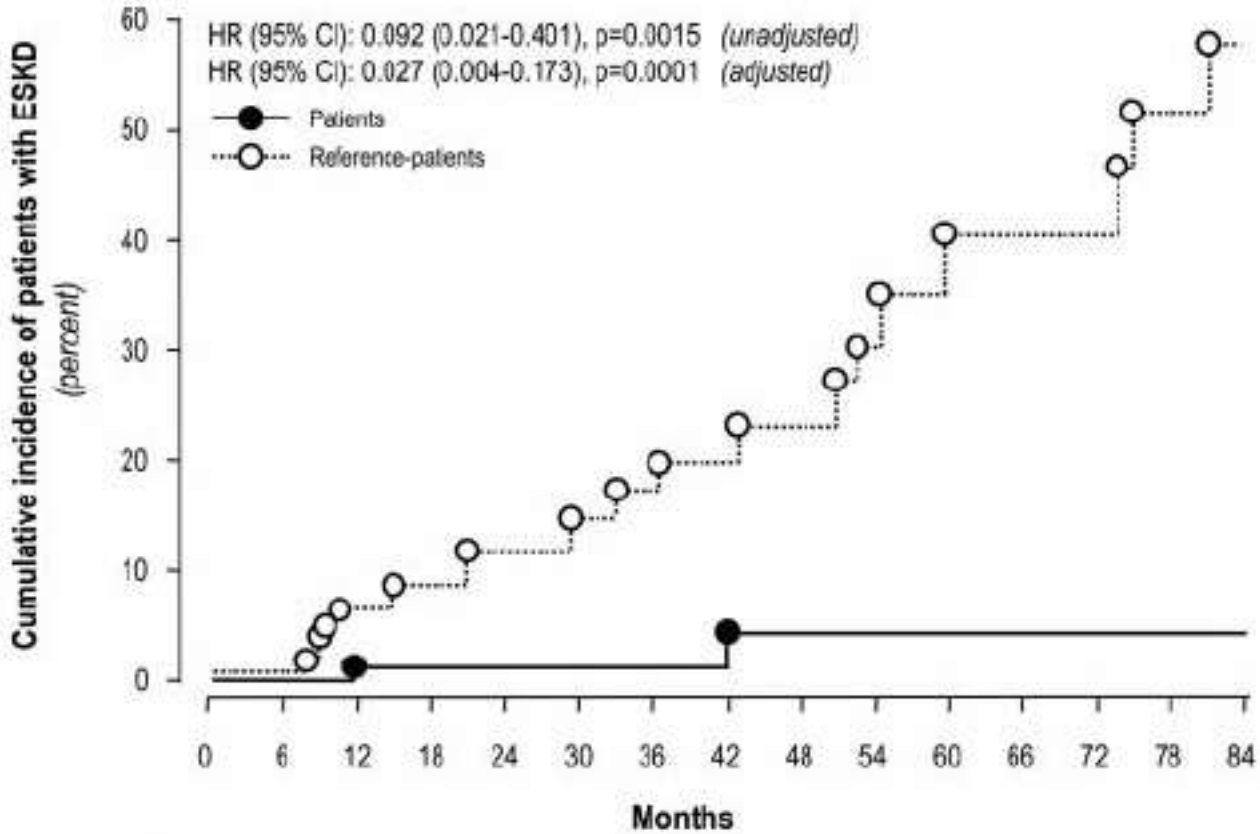
The short- and long-term impact of multi-disciplinary clinics in addition to standard nephrology care on patient outcomes



Nephrol Dial Transplant (2005) 20: 147–154

Role of Remission Clinics in the Longitudinal Treatment of CKD

Piero Ruggenenti,*† Elena Perticucci,† Paolo Cravedi,*† Vincenzo Gambara,†
Marco Costantini,* Sanjib Kumar Sharma,*‡ Annalisa Perna,* and Giuseppe Remuzzi*†



Subjects at risk

Patients	56	56	55	55	53	51	46	37	36	32	30	22	16	7	6
Reference-patients	56	54	48	44	12	40	29	26	22	16	12	10	10	6	4

J Am Soc Nephrol 19: 1213–1224, 2008.

Multidisciplinary care

- Education program
- Protocollized clinic f/u
- Protocollized lab
- Regular audits/CQI

- Nephrologist
- Nurse practitioner
- Social worker/psychologist
- Dietician
- Pharmacist
- Physiotherapist

Summary and conclusion

- Multiple metabolic derangements are prevalent in KTx patients and they are associated with increased mortality/CV events
- Screening for impaired glucose metabolism using OGT is necessary
- Treatment of impaired glucose metabolism by implementing life style modifications and using appropriate pharmacotherapy is indicated
- Regular screening for dyslipidemia is recommended
- Lifestyle modifications and statins are likely to improve outcomes
- Protein-energy wasting/inflammation is prevalent in KTx patients and is associated with worse clinical outcomes
- Multidisciplinary “risk management clinics” may be necessary to target all these metabolic problems among kidney tarnsplant recipients to improve patient outcomes