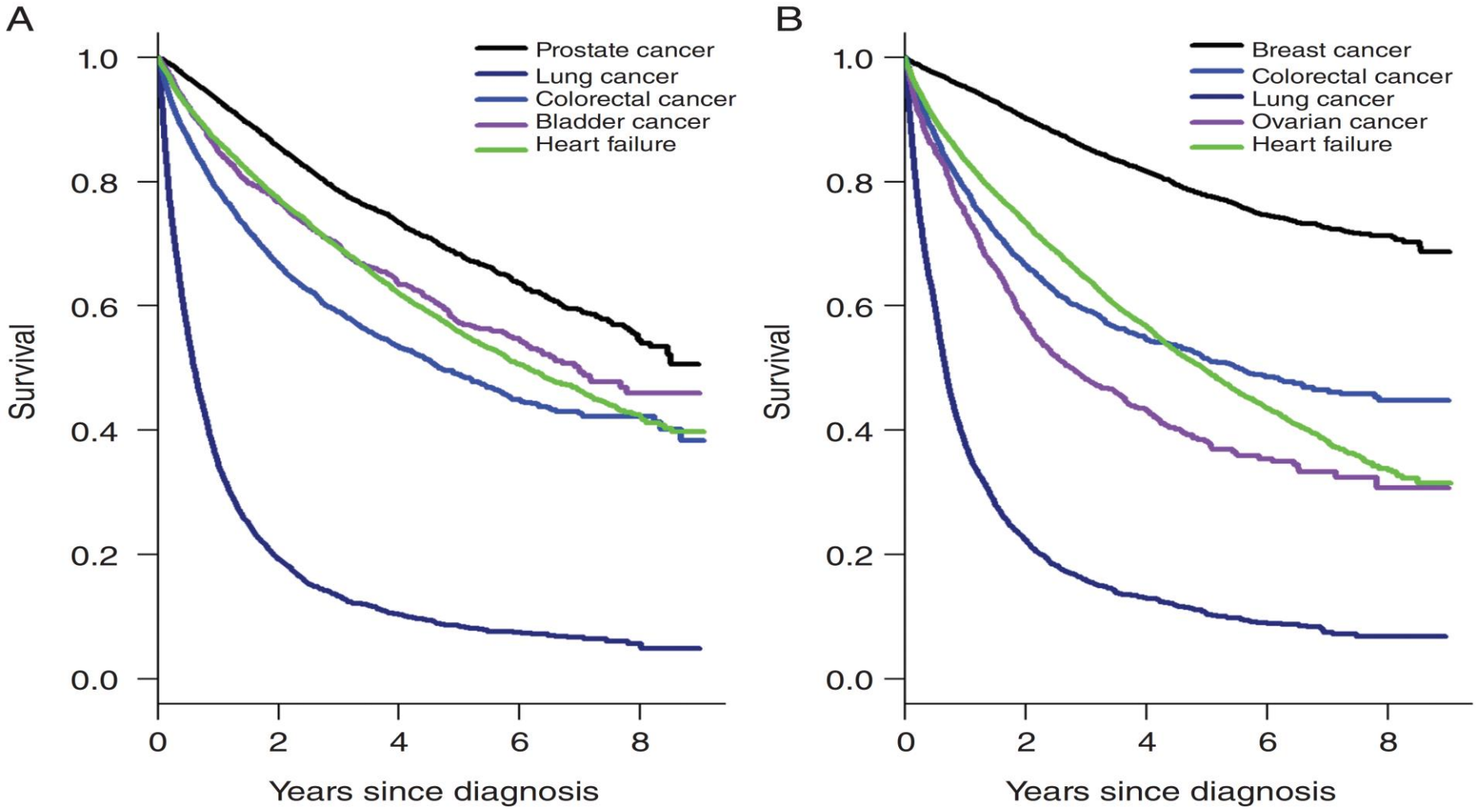
Giovambattista Desideri
UO Geriatria
Università dell'Aquila

Iperkaliema e sideropenia nello scompenso cardiaco

Kaplan–Meier curves for overall survival in (A) men or (B) women with diffrent cancero or hrart failure



Mamas MA et al. European Journal of Heart Failure (2017) 19, 1095–1104

Quando l'insufficienza renale complica lo scompenso cardiaco: carenza marziale ed iperpotassiemia

Stampa

Richiesta: **04047794** del **04/04/2018**
Provenienza: A432 A-Geriatria

Sig. [redacted]

Data Nascita: 01/06/1925
Identificativo Paziente: 00001721
Sesso M Età: 92 Anni

Data di accettazione: 04/04/2018
Data e ora di presa in carico: 04/04/2018 09:41
Data e ora di stampa: 05/04/2018 11:45

ESAMI IN ROUTINE

Esame	*Esito*		*U.M.*	*Valori di Riferimento*	*Metodica*	*Validatore*
Biochimica Clinica						
S-GLUCOSIO	147	*	mg/dl	60 - 110	(Colorimetrica)	T03
S-UREA	100	*	mg/dl	10 - 50	(Colorimetrico)	T03
S-CREATININA	1.28	*	mg/dl	0.70 - 1.20	(Colorimetrica 2 punti	T03
Filtrato Glomerulare Medio	53		ml/min/1,73 mq	> 60		
	Valore calcolato secondo l'equazione MDRD, valido per persone tra 18 - 70 anni caucasiche; moltiplicare per 1.21 se di etnia afro-americana. Valori di eGFR < 60 ml/min/1.73 mq vanno valutati nell'ambito del contesto clinico generale.					
S-SODIO	140		mEq/L	135 - 150	(Ioni selettivi)	T03
S-POTASSIO	6.4	*	mEq/L	3.5 - 5.5	(Ioni selettivi)	T03
S-CALCIO TOTALE	9.2		mg/dl	8.5 - 11.0	(Colorimetrica)	T03
S-COLESTEROLO TOTALE	145		mg/dl	Valore ottimale: 150 - 200 Border line: 200 - 240 Alto: >240	(Colorimetrica)	T03
S-TRIGLICERIDI	51	*	mg/dl	60 - 170	(Colorimetrico)	T03
S-URATO	8.9	*	mg/dl	3.5 - 7.0	(Colorimetrica)	T03
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S-BILIRUBINA TOTALE (BT)	0.75		mg/dl	0.20 - 1.20	(Colorimetrica)	T03
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Coagulazione						
P-Tempo di Protrombina (PT)						T03

Continua nella pagina seguente

Il laboratorio effettua controllo di qualità interno con sistema UNITY della ditta BIO-RAD, e partecipa al controllo esterno di qualità della Regione ABRUZZO
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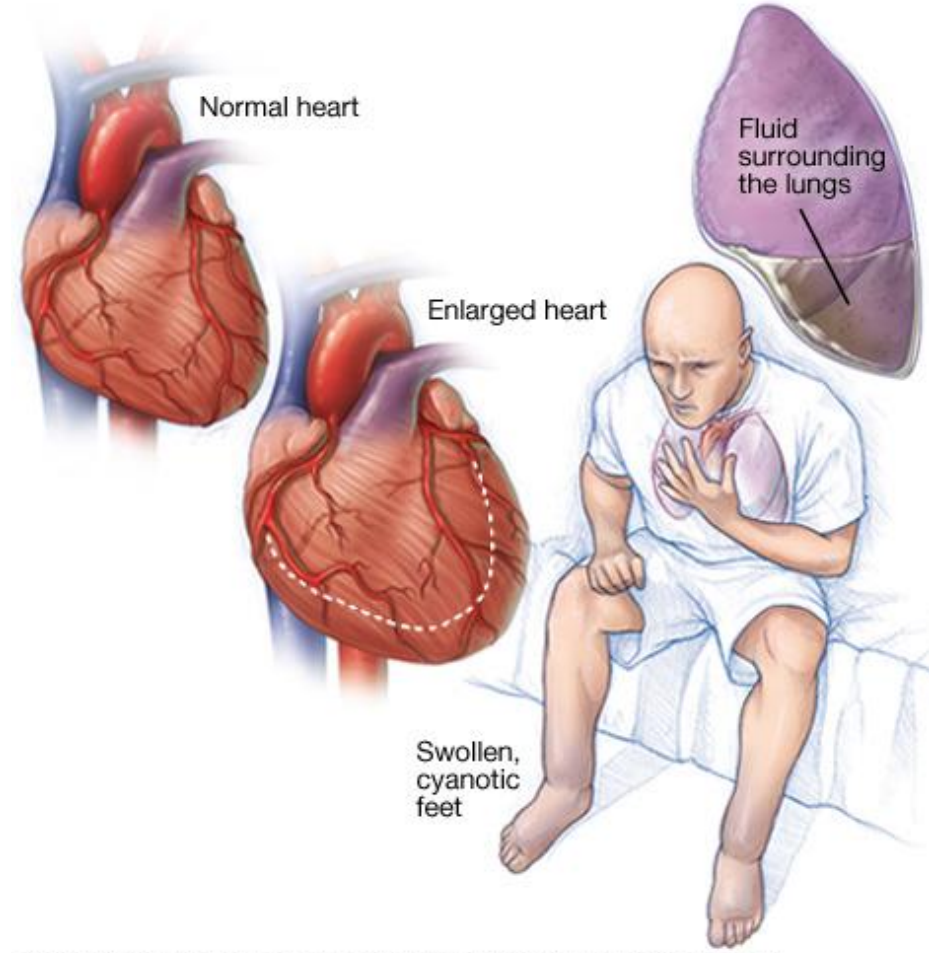
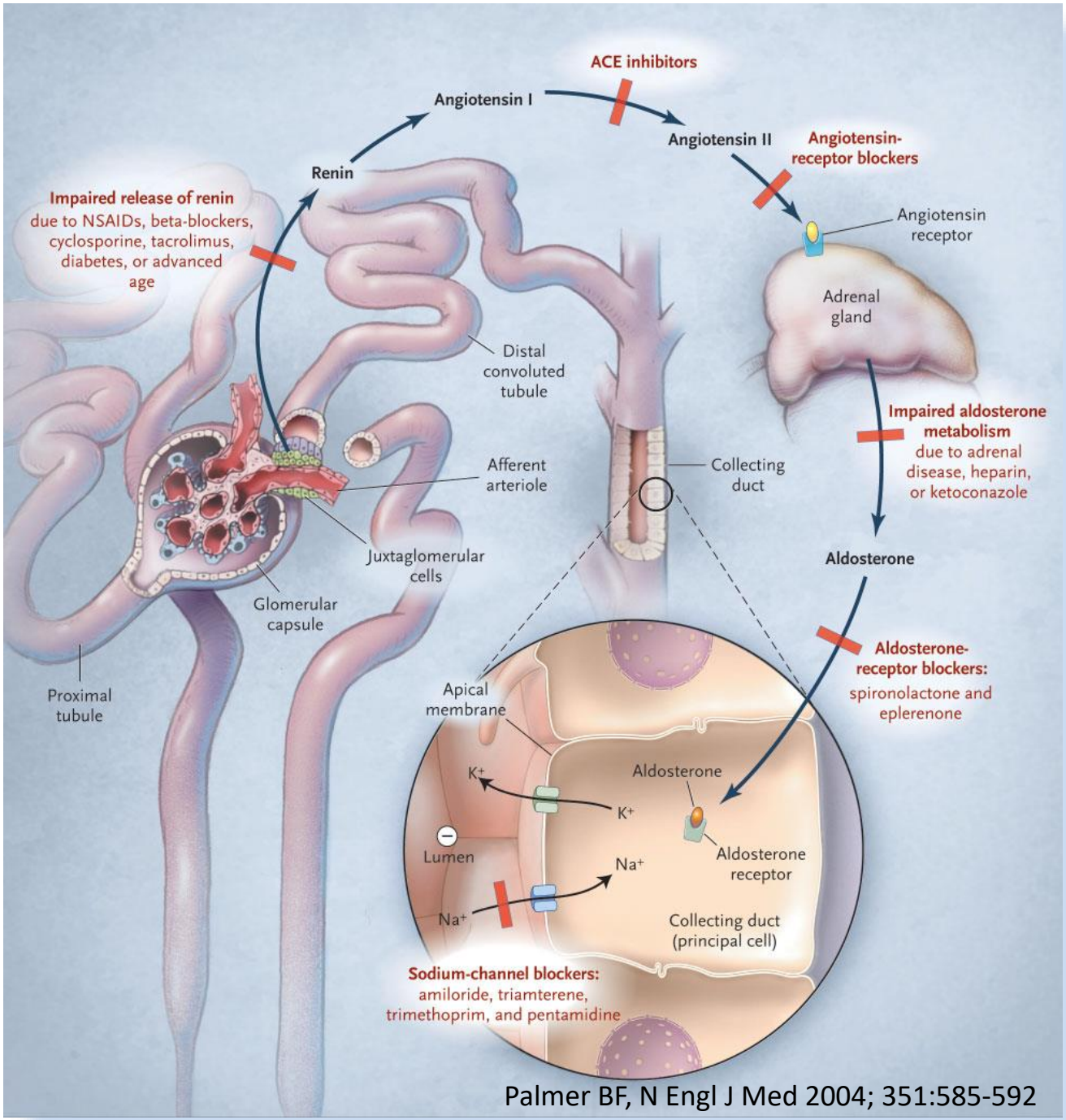
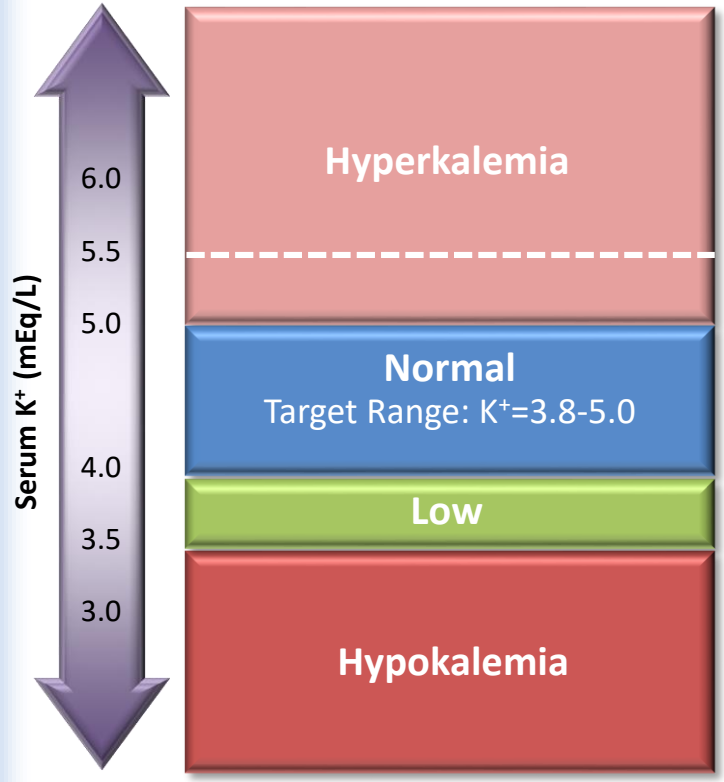


The RAAS and Regulation of Potassium Excretion in the Kidney



Palmer BF, N Engl J Med 2004; 351:585-592



Guidelines for the diagnosis and treatment of acute and chronic heart failure

2016 ESC Guidelines

An **ACE-I** is recommended, in addition to a beta-blocker, for symptomatic patients with HFrEF to reduce the risk of HF hospitalization and death.	I	A
A beta-blocker is recommended, in addition an **ACE-I**, for patients with stable, symptomatic HFrEF to reduce the risk of HF hospitalization and death	I	A
An **MRA** is recommended for patients with HFrEF, who remain symptomatic despite treatment with an **ACE-I** and a beta-blocker, to reduce the risk of HF hospitalization and death	I	A

2017 ACC/AHA/HFSA Focused Update

The clinical strategy of inhibition of the reninangiotensin system with **ACE inhibitors** (Level of Evidence: A), OR **ARBs** (Level of Evidence: A), OR **ARNI** (Level of Evidence: B-R) in conjunction with evidencebased beta blockers, and aldosterone antagonists in selected patients, is recommended for patients with chronic HFrEF to reduce morbidity and mortality.

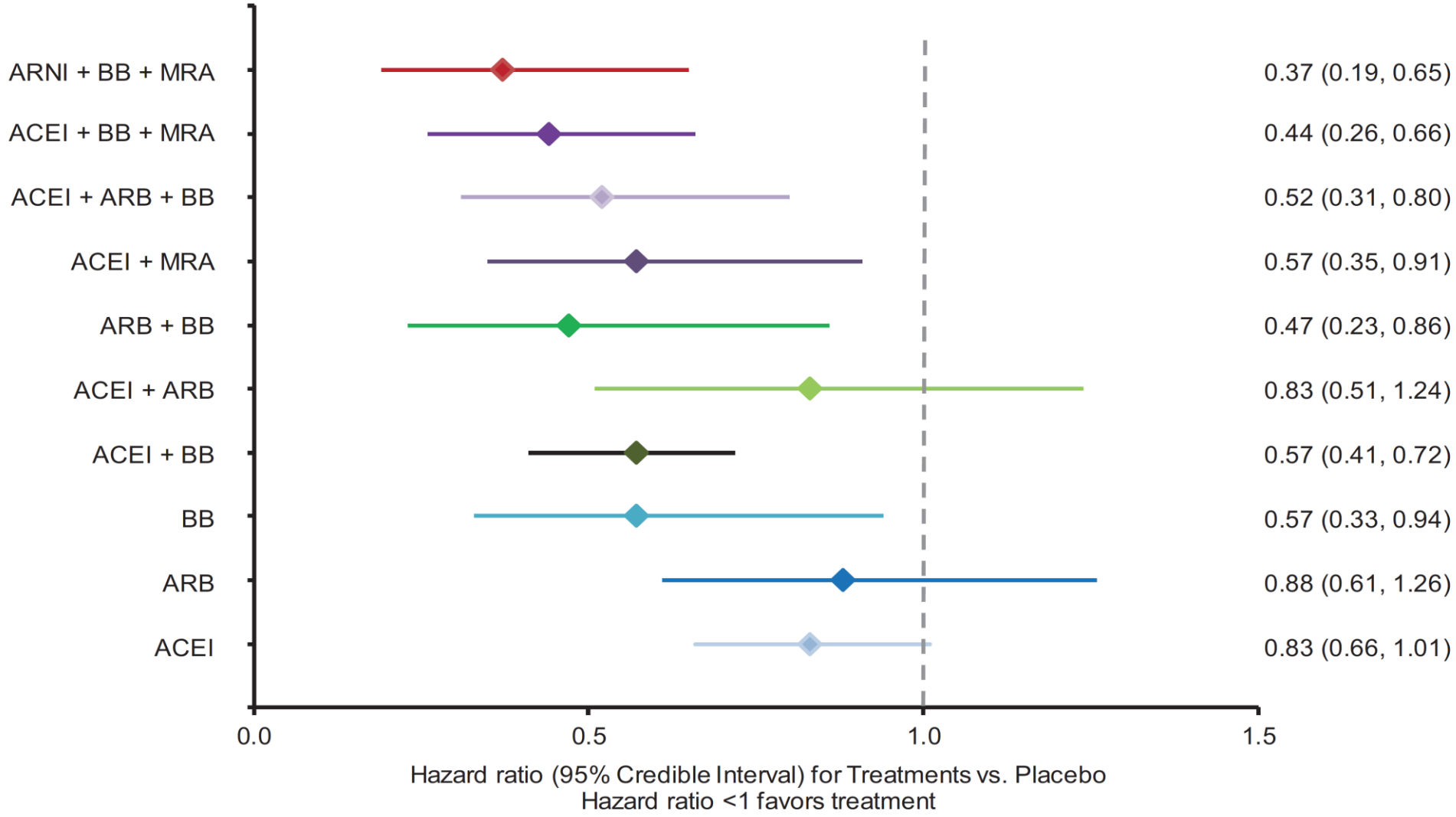
Ponikowski P ey al. Eur Heart J 2016

Yancy CW et al. Circulation 2017

Thirty Years of Evidence on the Efficacy of Drug Treatments for Chronic Heart Failure With Reduced Ejection Fraction

A Network Meta-Analysis

All-cause mortality



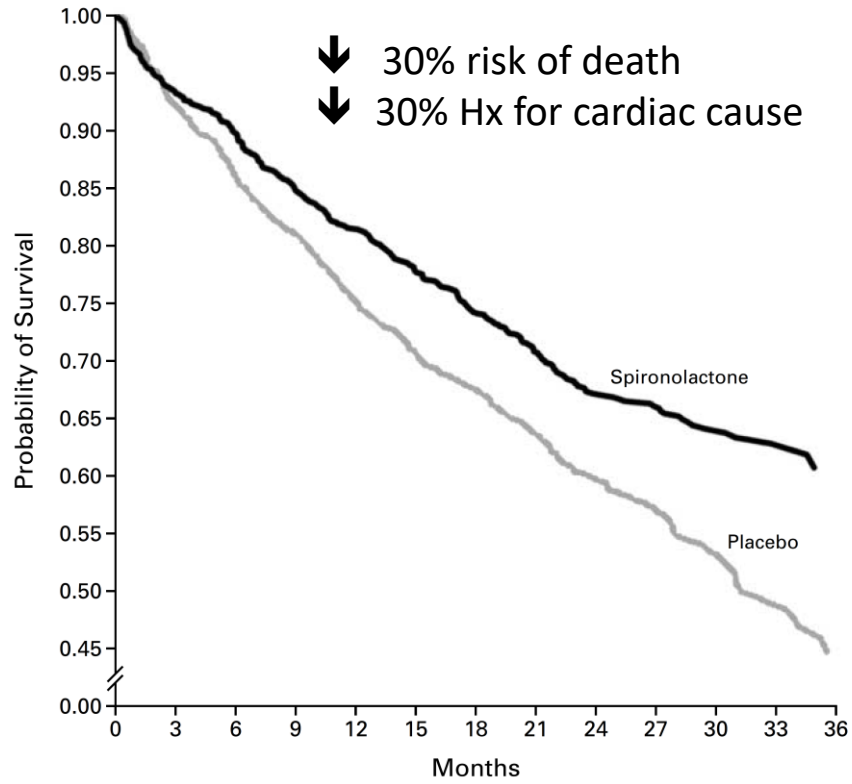
Burnett H et al. Circ Heart Fail. 2017;10:e003529.

The New England
Journal of Medicine

VOLUME 341 SEPTEMBER 2, 1999 NUMBER 10

THE EFFECT OF SPIRONOLACTONE ON MORBIDITY AND MORTALITY IN PATIENTS WITH SEVERE HEART FAILURE

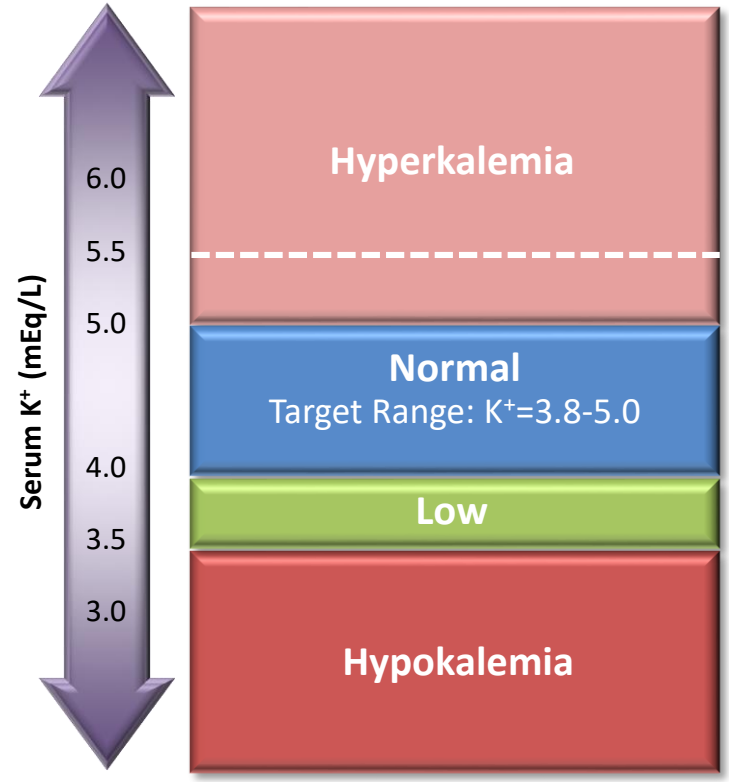
BERTRAM PITT, M.D., FAIEZ ZANNAD, M.D., WILLEM J. REMME, M.D., ROBERT CODY, M.D., ALAIN CASTAIGNE, M.D., ALFONSO PEREZ, M.D., JOLIE PALENSKY, M.S., AND JANET WITTES, PH.D., FOR THE RANDOMIZED ALDACTONE EVALUATION STUDY INVESTIGATORS*

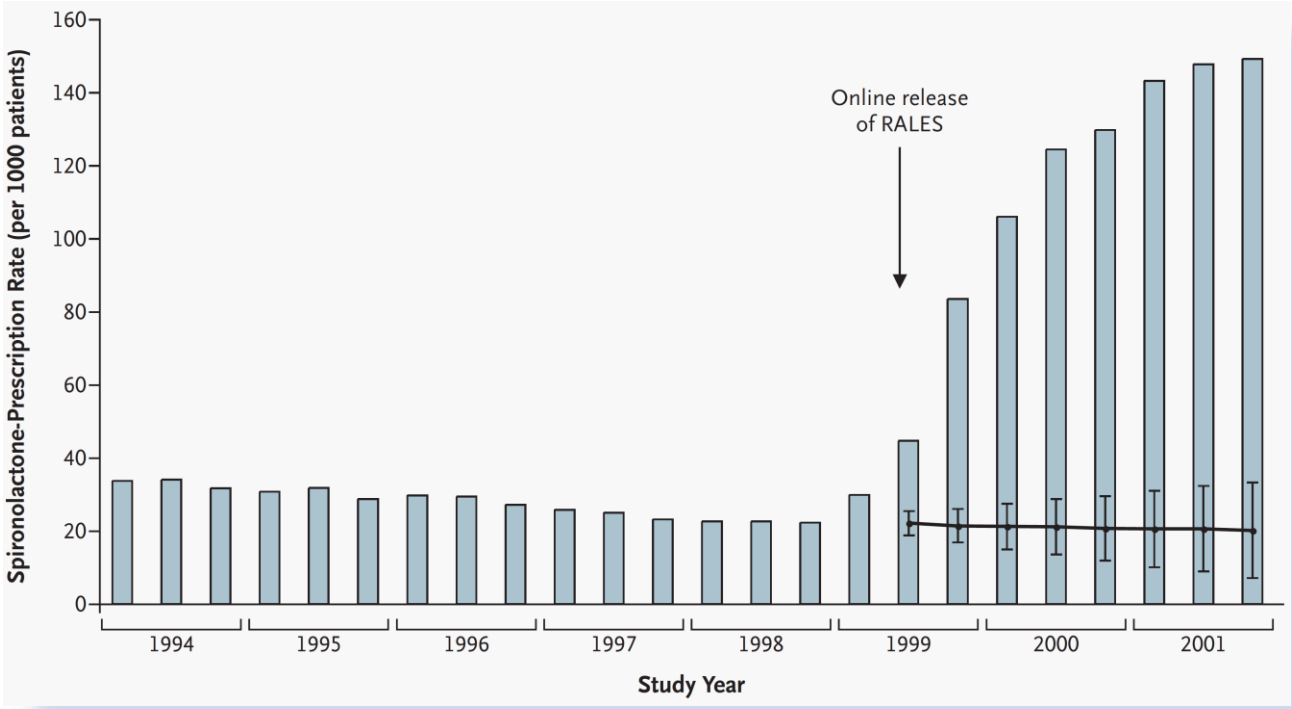


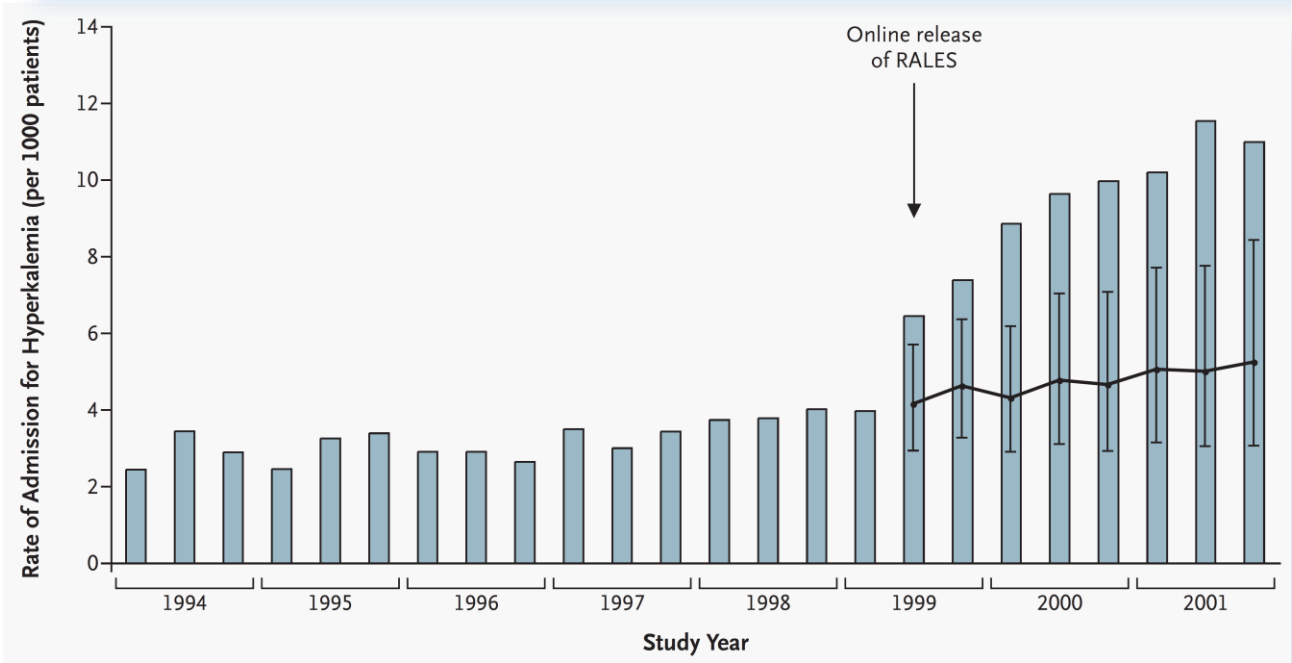
NO. AT RISK

Placebo	841	775	723	678	628	592	565	483	379	280	179	92	36
Spironolactone	822	766	739	698	669	639	608	526	419	316	193	122	43

Pitt B et al. N Eng J Med 1999

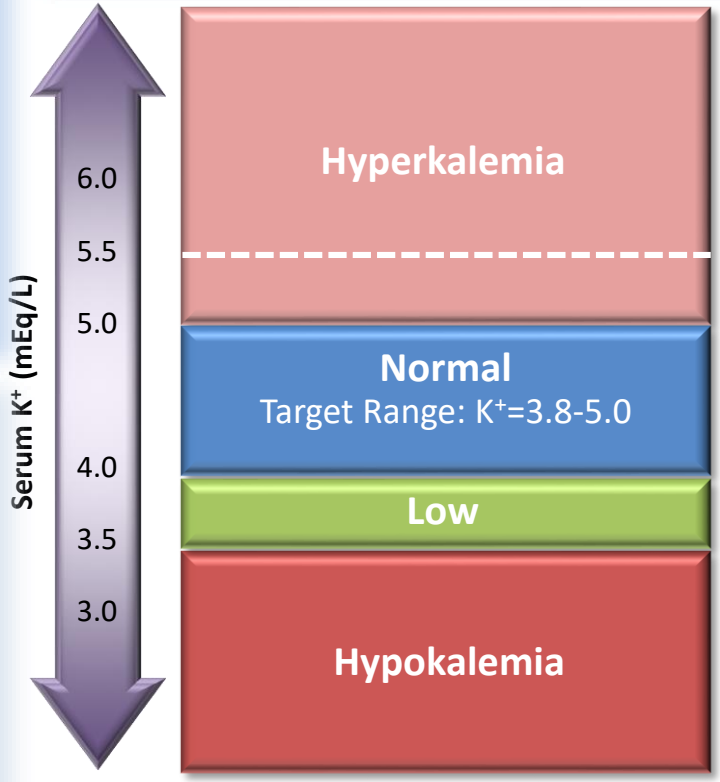






Rate of hospital admission for hyperkalemia among patients recently hospitalized for HF who were receiving ACE-I

(Juurlink DN et al. N Engl J Med 2004;351:543-51)

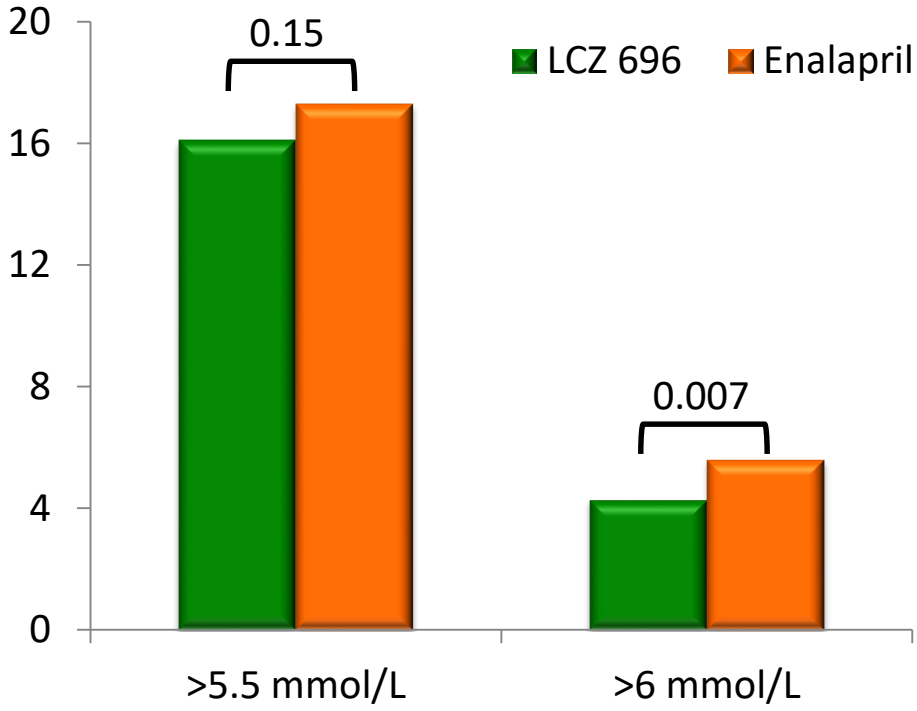


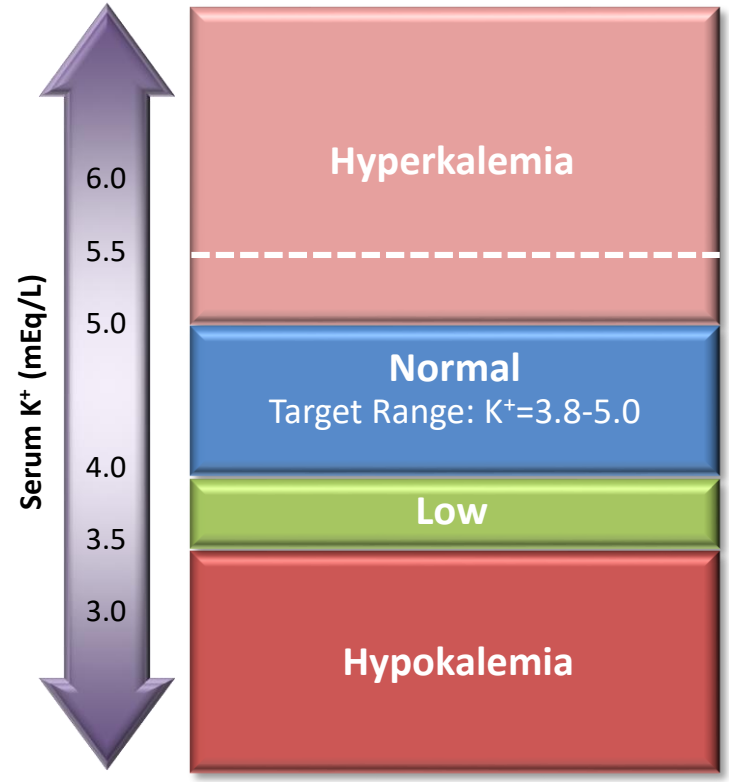
The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 SEPTEMBER 11, 2014 VOL. 371 NO. 11

Angiotensin–Neprilysin Inhibition versus Enalapril in Heart Failure

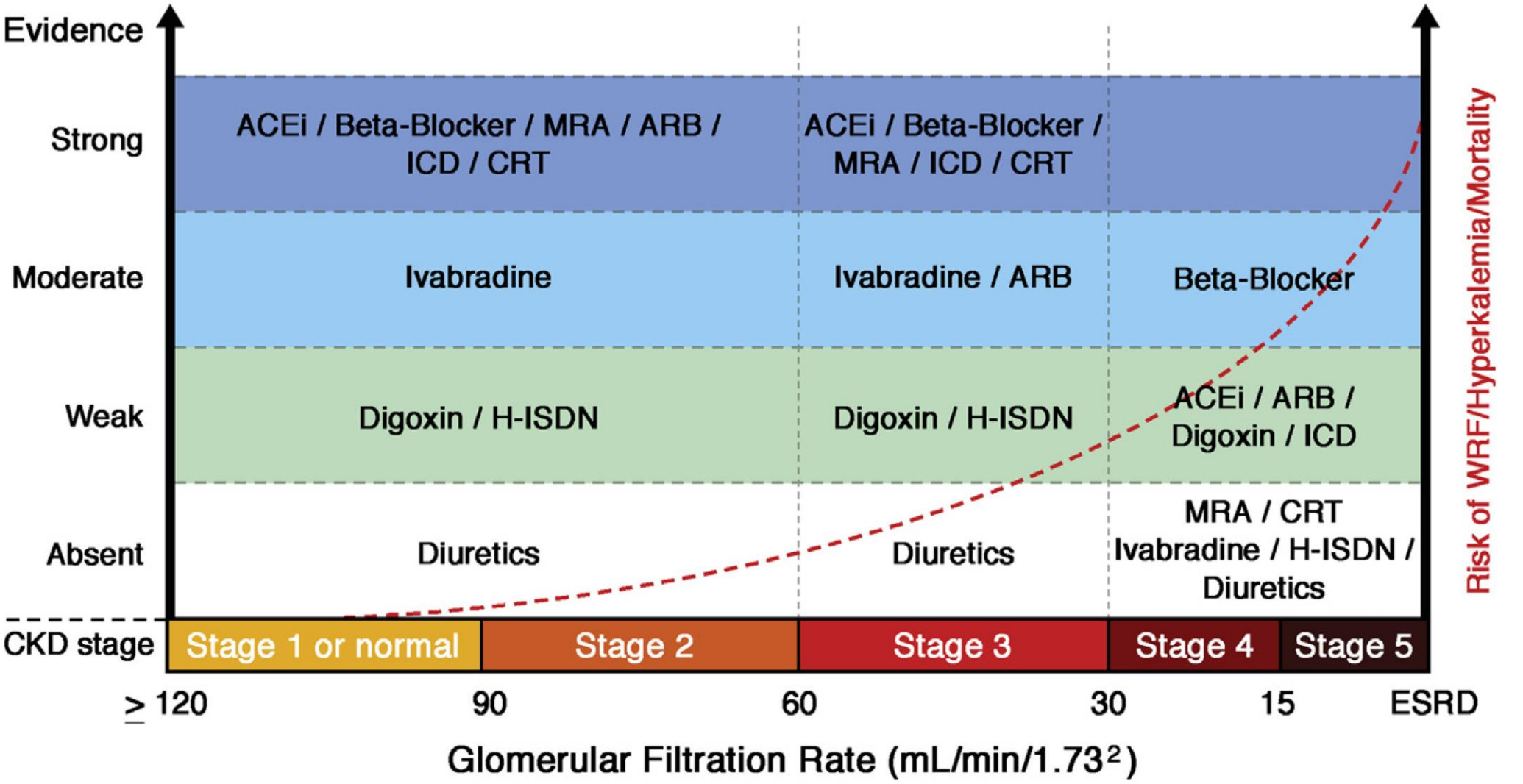
John J.V. McMurray, M.D., Milton Packer, M.D., Akshay S. Desai, M.D., M.P.H., Jianjian Gong, Ph.D., Martin P. Lefkowitz, M.D., Adel R. Rizkala, Pharm.D., Jean L. Rouleau, M.D., Victor C. Shi, M.D., Scott D. Solomon, M.D., Karl Swedberg, M.D., Ph.D., and Michael R. Zile, M.D., for the PARADIGM-HF Investigators and Committees*





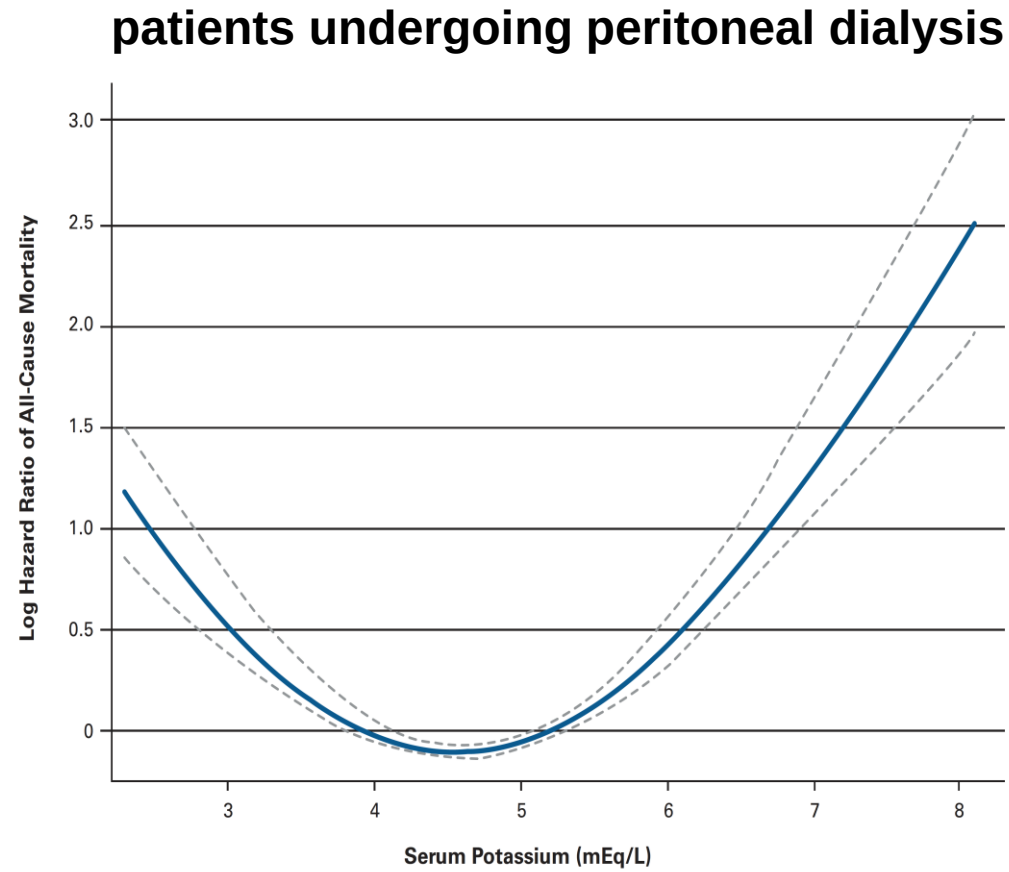
McMurray JJV et al. N Engl J Med 2014;371:993-1004.

Current Evidence on Treatment of Patients With Chronic Systolic Heart Failure and Renal Insufficiency



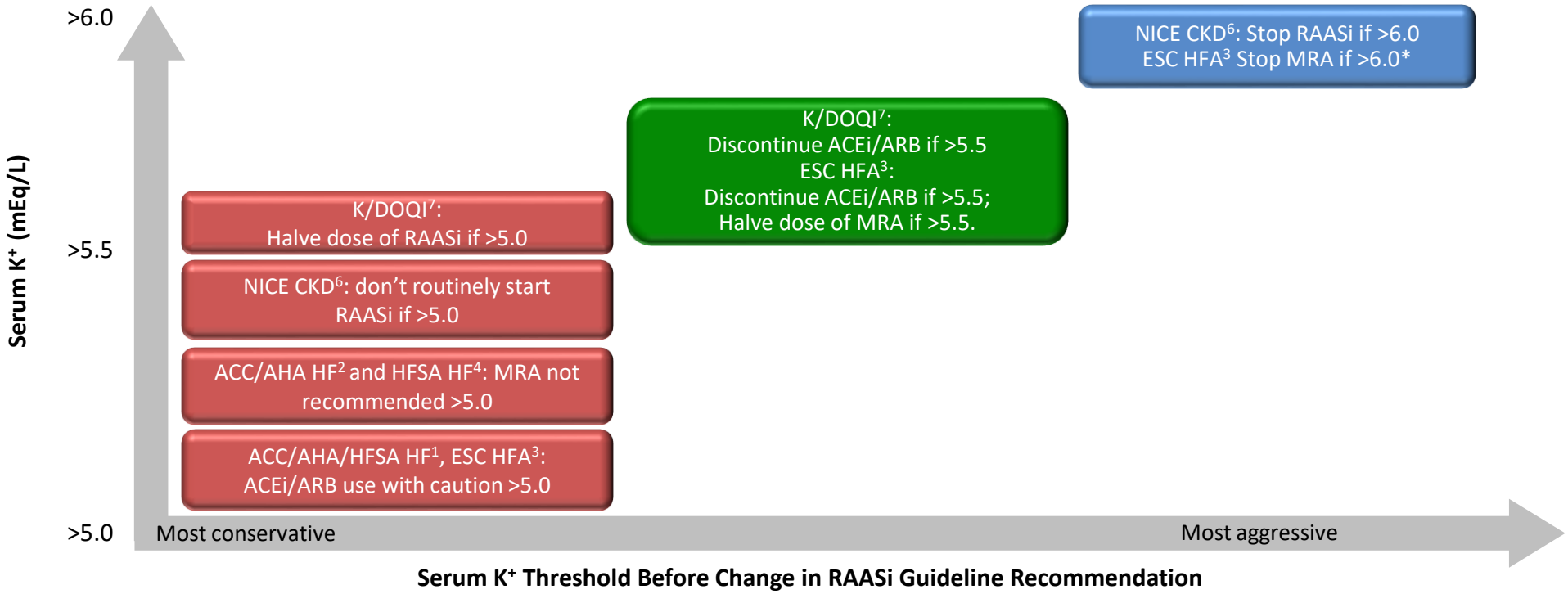
Damman K et al. J Am Coll Cardiol 2014;63:853–71

All-Cause mortality associated with serum potassium levels in non-dialysis dependent patients with chronic kidney disease and in patients undergoing peritoneal dialysis



Dunn JD et al. Am J Manag Care 2015;21(15 suppl):e307-e315

Guidelines Recommend RAASi Dose Modifications With Increasing Serum K+



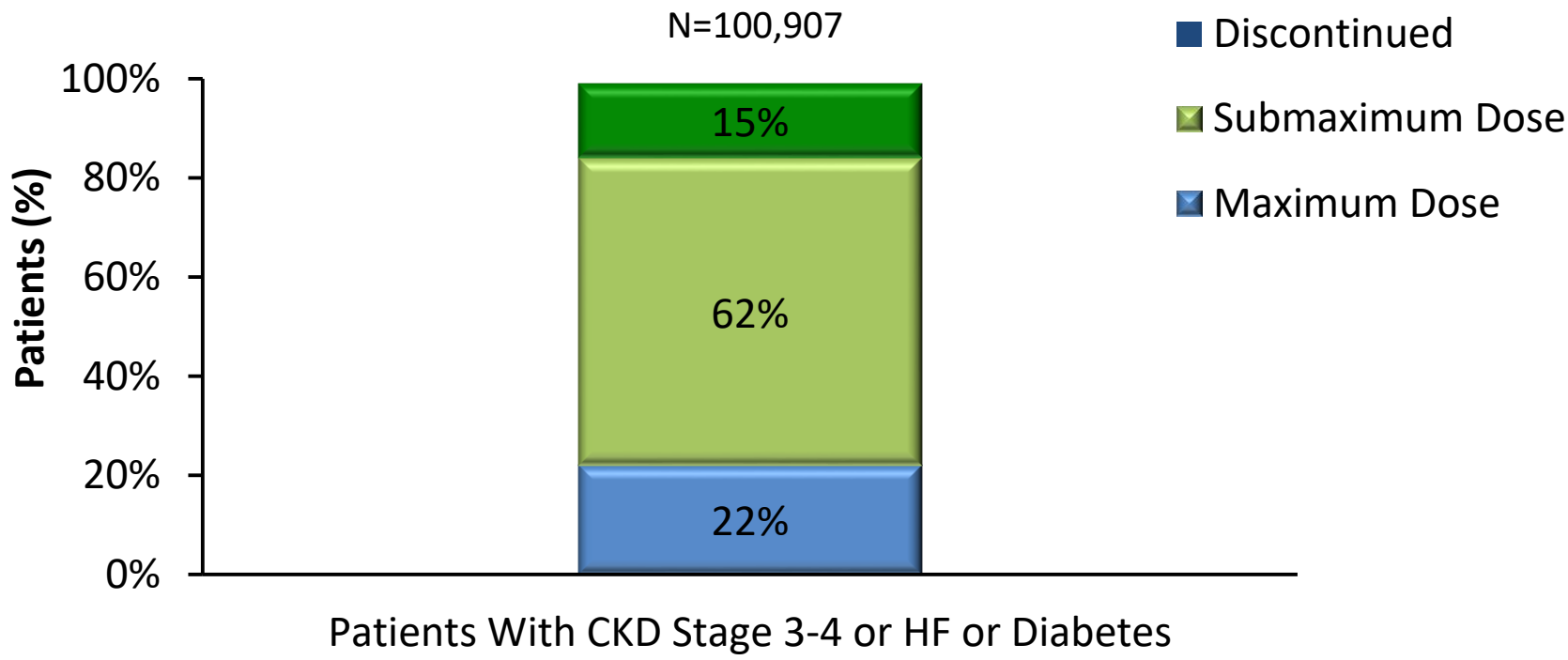
KDIGO Guidelines do not provide recommendations[5]

**ESC HFA: Management of acute hyperkalemia (>6.0) may require a short-term cessation of K+-retaining agents and RAASi, but this should be minimized and RAASi should be carefully reintroduced as soon as possible while monitoring K+ levels.[3]*

1. *Yancy CW, et al. Circulation. 2016;134:[Epub ahead of print].* ***2.*** *Yancy CW, et al. Circulation. 2013;128:1810-1852.* ***3.*** *Ponikowski P, et al. European Heart Journal. 2016 May 20. pii: ehw128. [Epub ahead of print].* ***4.*** *Heart Failure Society of America, Lindenfeld J, et al. J Card Fail. 2010;16(6):475-539.*
5. *KDIGO Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int Suppl. 2013;3(1).* ***6.*** *National Institute for Health and Clinical Excellence (NICE) [UK]. Chronic kidney disease (partial update): Early identification and management of chronic kidney disease in adults in primary and secondary care. 2014. https://www.nice.org.uk/guidance/cg182/evidence/update-full-guideline-191905165.* ***7.*** *National Kidney Foundation. Guideline 11. http://www2.kidney.org/professionals/kdoqi/guidelines_bp/guide_11.htm. Accessed February 17, 2015.*

Few Patients Are on Maximum RAASi

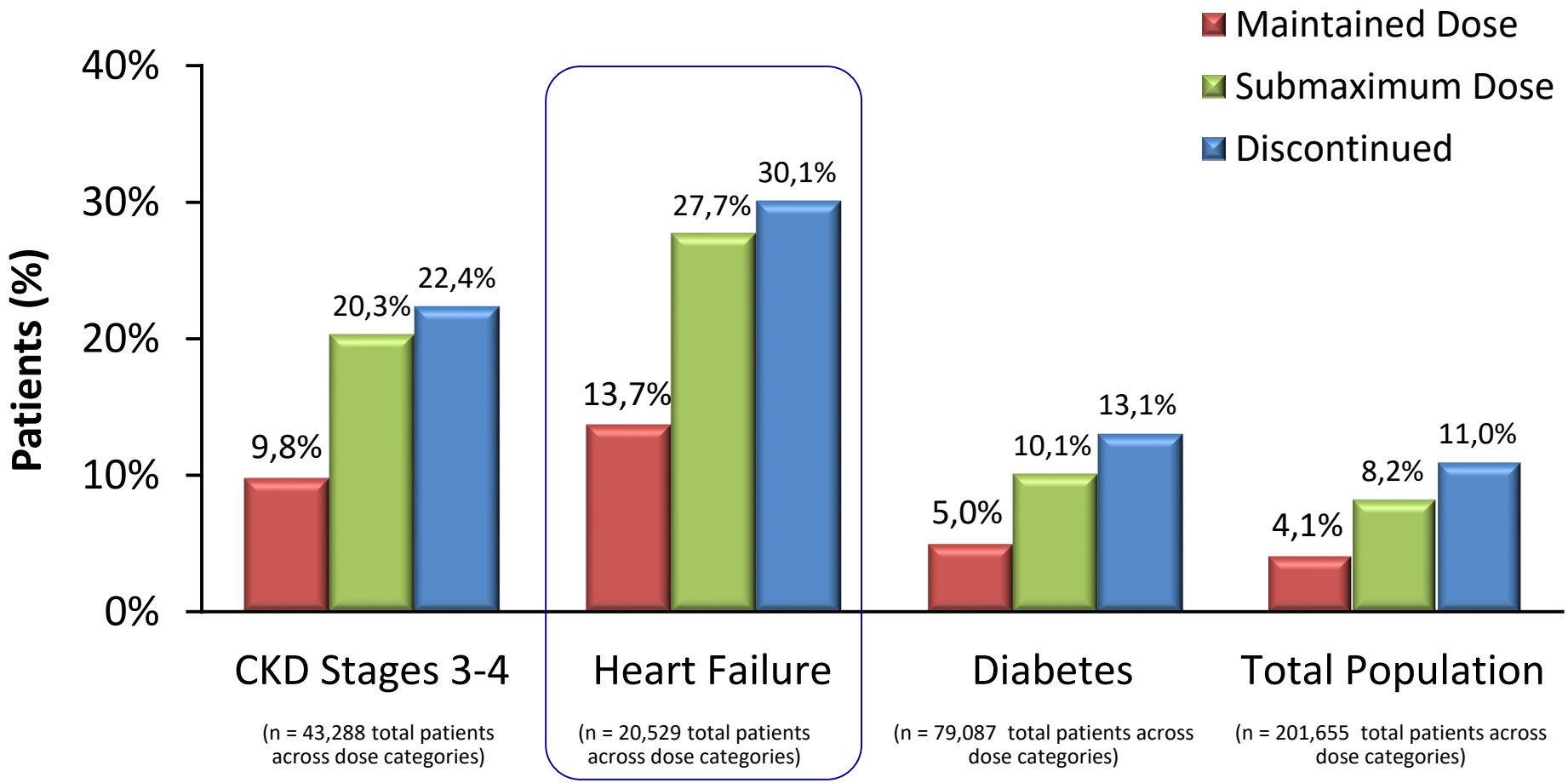
- Distribution of RAASi Dose Levels in Patients With CKD Stage 3-4 or Heart Failure or Diabetes



- RAASi prescriptions were classified by dose level using the following dose categories:
  - **"Maximum,"** defined as the labeled dose
  - **"Submaximum,"** defined as any RAASi dose lower than the labeled dose
  - **"Discontinued,"** defined as the absence of RAASi prescriptions for >390 days subsequent to prior prescription

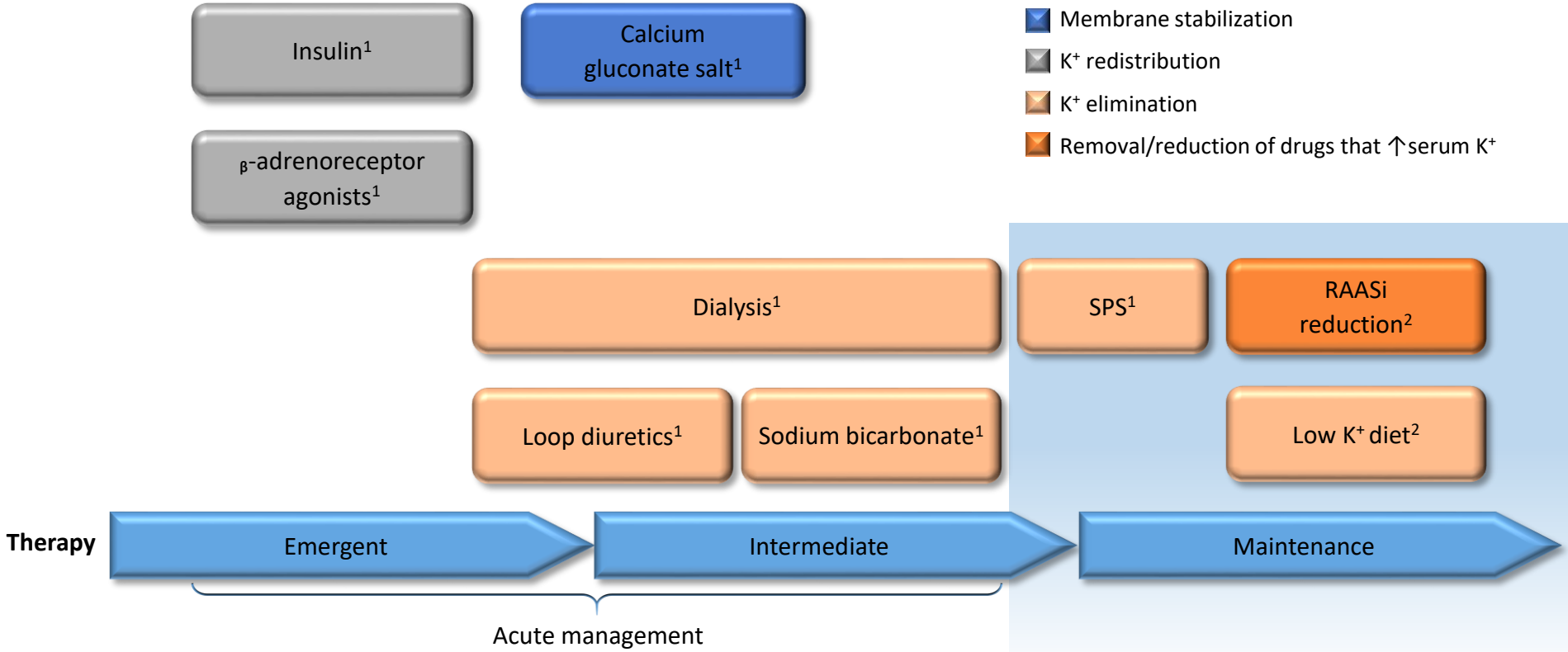
Epstein M, et al. *Am J Manag Care.* 2015;21:S212-S220.

Percent Mortality by Prior RAASi Dose



Epstein M, et al. *Am J Manag Care.* 2015;21:S212-S220.

Traditional Treatment Options for Hyperkalemia

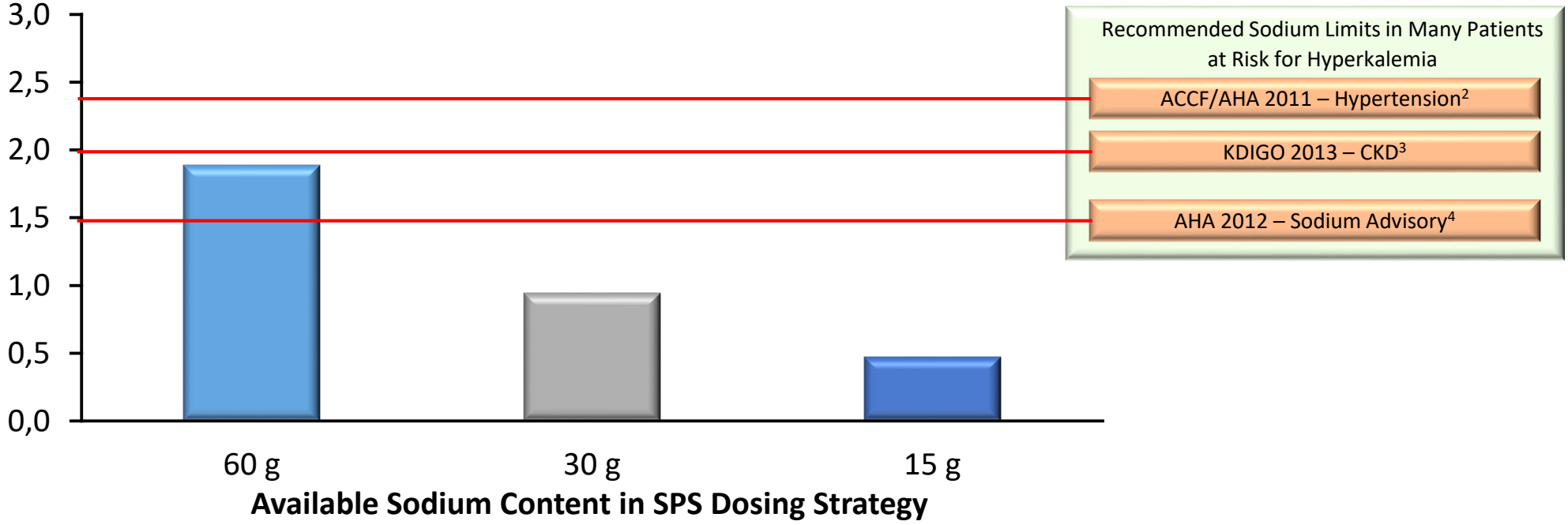


RAASi: renin-angiotensin-aldosterone system inhibitor, SPS: sodium polystyrene sulfonate

1. Weisberg L. *Crit Care Med*. 2008;36(12):3246-3251. 2. Palmer BF, et al. *N Engl J Med.* 2004;351(6):585-592.

Sodium Polystyrene Sulfonate and Sodium Content

SPS Contains 9.4% Sodium by Weight (4.1 mEq/g)[1]



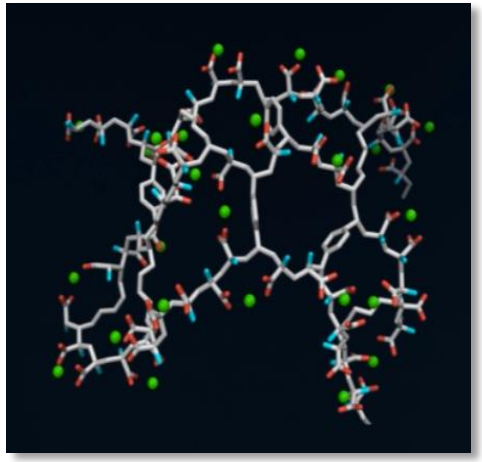
Since the in vivo efficiency of SPS is about 33%, approximately 1/3 of the resin's actual sodium content is being delivered to the body[1]

SPS dosing may use up a sizable portion of a sodium-restricted patient's daily sodium allowance

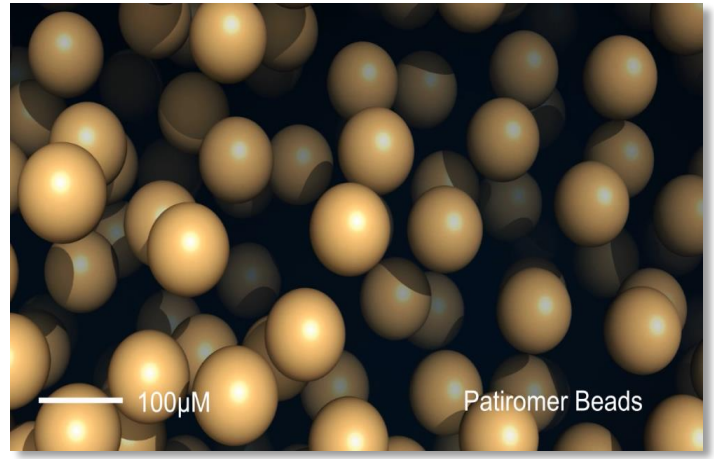
1. Kayexelate [package insert]. Bridgewater, NJ: Sanofi-Aventis; 2010.
2. Whelton P, et al. *Circulation*. 2012;126:2880-2889.
3. KDIGO Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. *Kidney Int Suppl*. 2013;3(1).
4. Aronow WS, et al. *Circulation.* 2011;123:2434 -2506.

Patiromer Is a Novel, Sperical, Non-absorbed K^+ Binder

- High-capacity K^+ binder
- Average bead size (100 µM) is too large for patiromer to be absorbed from the gastrointestinal tract, enabling patiromer to be passed through the entire GI tract and absorb more K^+
- Uniform spherical shape, size, and low-swelling beads ratio



High-capacity polymer



Uniform, spherical patiromer beads

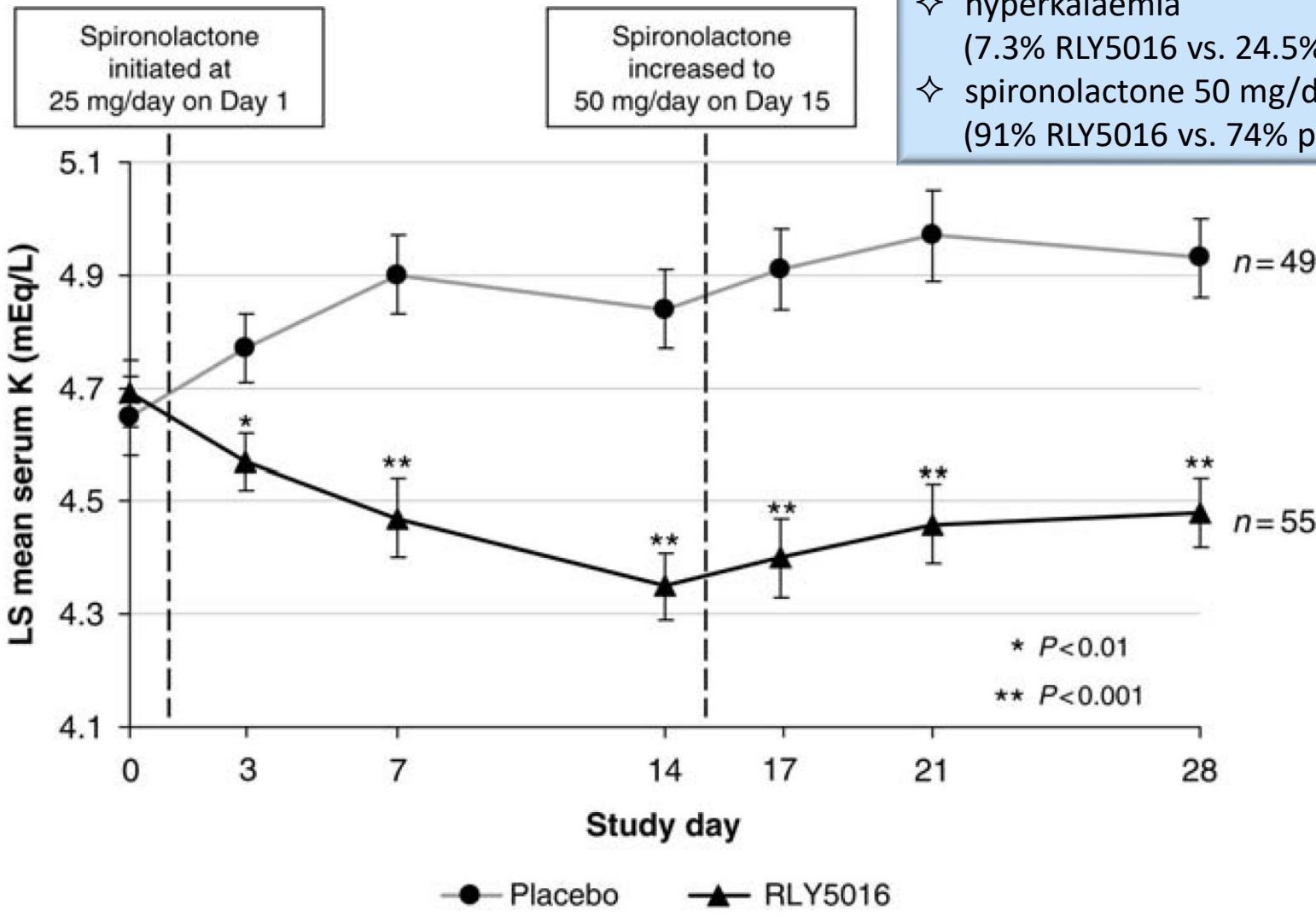
Patiromer travels through the gastointestinal tract over 24-72 hours

Patiromer is fully ionized at the physiologic pH of the colon for optimal ion exchange. Carboxylate groups of patiromer bind to K^+, which is primarily in the colon due to upregulation of BK channels in colonic epithelial cells. Patiromer beads are excreted, leading to removal of excess K^+ and reduction of serum K^+ levels. Ca^{2+} is exchanged for K^+

Li L, et al. *J Cardiovasc Pharmacol Ther.* 2016;doi:10.1177/1074284166295549.

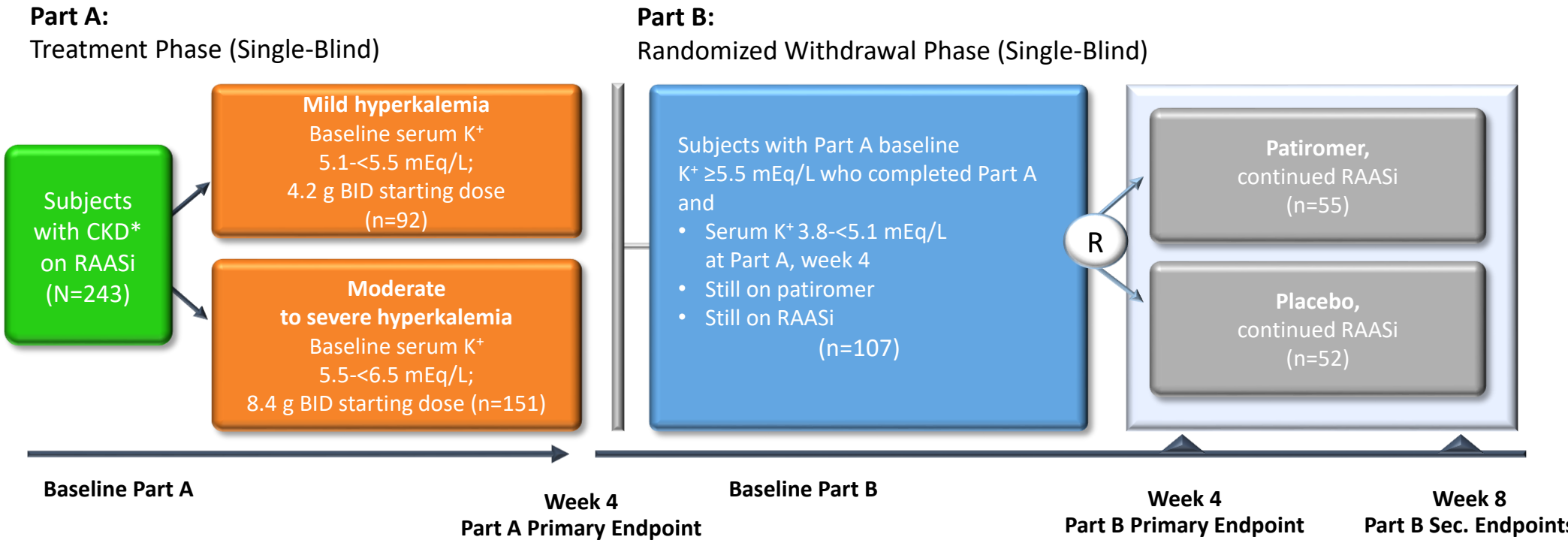
Evaluation of the efficacy and safety of RLY5016, a polymeric potassium binder, in a double-blind, placebo-controlled study in patients with chronic heart failure (the PEARL-HF) trial

- hyperkalaemia (7.3% RLY5016 vs. 24.5% placebo, P . 0.015)
- spironolactone 50 mg/day (91% RLY5016 vs. 74% placebo, P . 0.019).



Pitt B et al. European Heart Journal (2011) 32, 820–828

Patiromer in Patients with Kidney Disease and Hyperkalemia Receiving RAAS Inhibitors: OPAL-HK

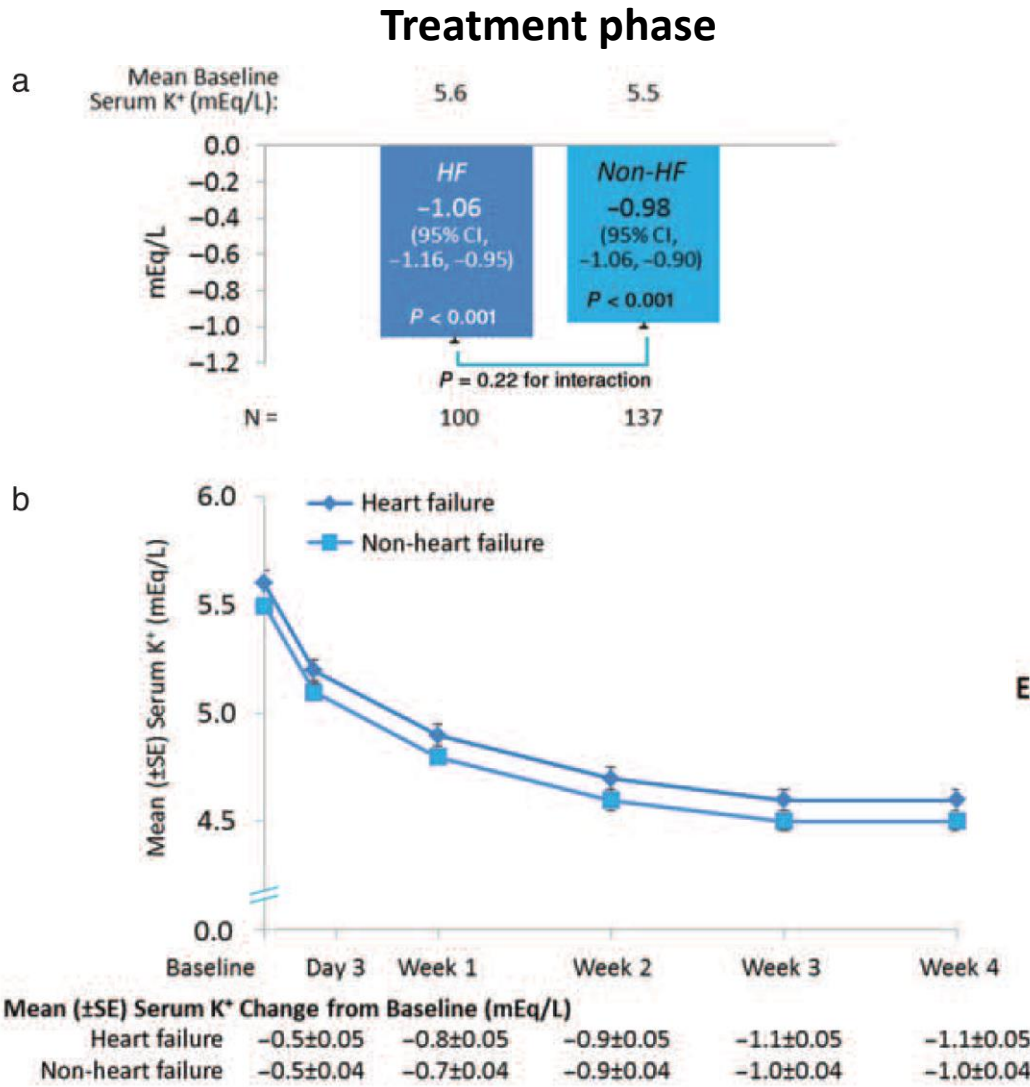


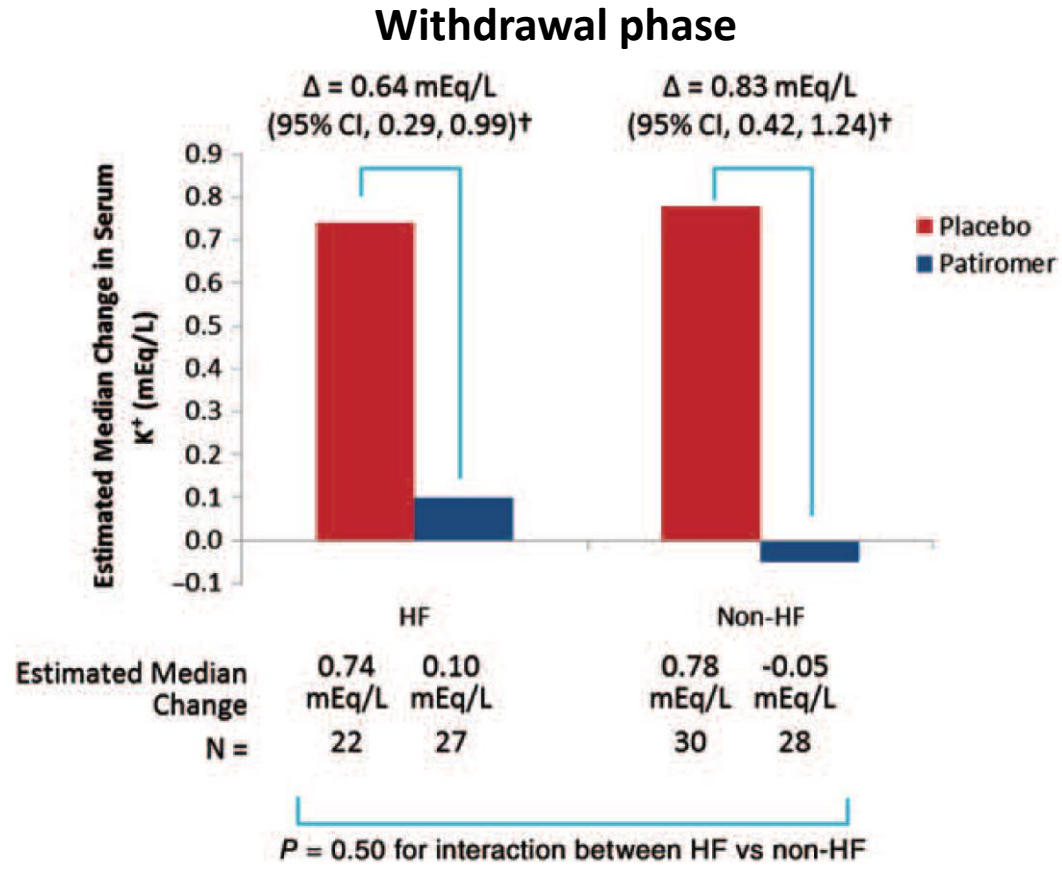
*eGFR 15 to <60 mL/min/1.73 m2.

eGFR: estimated glomerular filtration rate; R : randomization.

Protocol for Weir MR, et al. *N Engl J Med*. 2015;372(3):211-221; Pitt B, et al. Presented at: 18th Annual Scientific Meeting of the Heart Failure Society of America; Las Vegas, NV; Sept 14-17, 2014.

Effect of patiromer on reducing serum K+ and preventing recurrent hyperkalaemia in patients with HF and CKD on RAAS inhibitors: OPAL-HK





Pitt B et al. European Journal of Heart Failure (2015) 17, 1057–1065

Quando l'insufficienza renale complica lo scompenso cardiaco: carenza marziale ed iperpotassiemia

Stampa

Richiesta: **04047794** del **04/04/2018**
Provenienza: A432 A-Geriatria

Sig. [redacted]

Data Nascita: 01/06/1925
Identificativo Paziente: 00001721
Sesso M Età: 92 Anni

Data di accettazione: 04/04/2018
Data e ora di presa in carico: 04/04/2018 09:41
Data e ora di stampa: 05/04/2018 11:45

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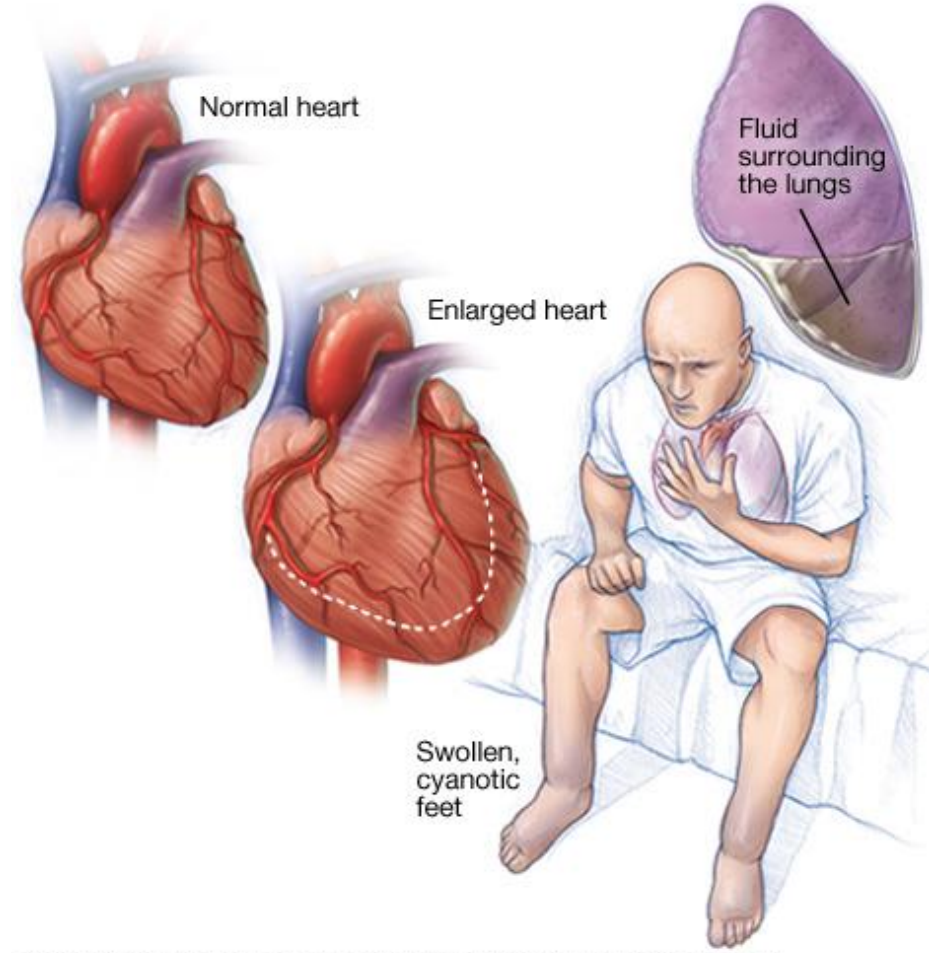
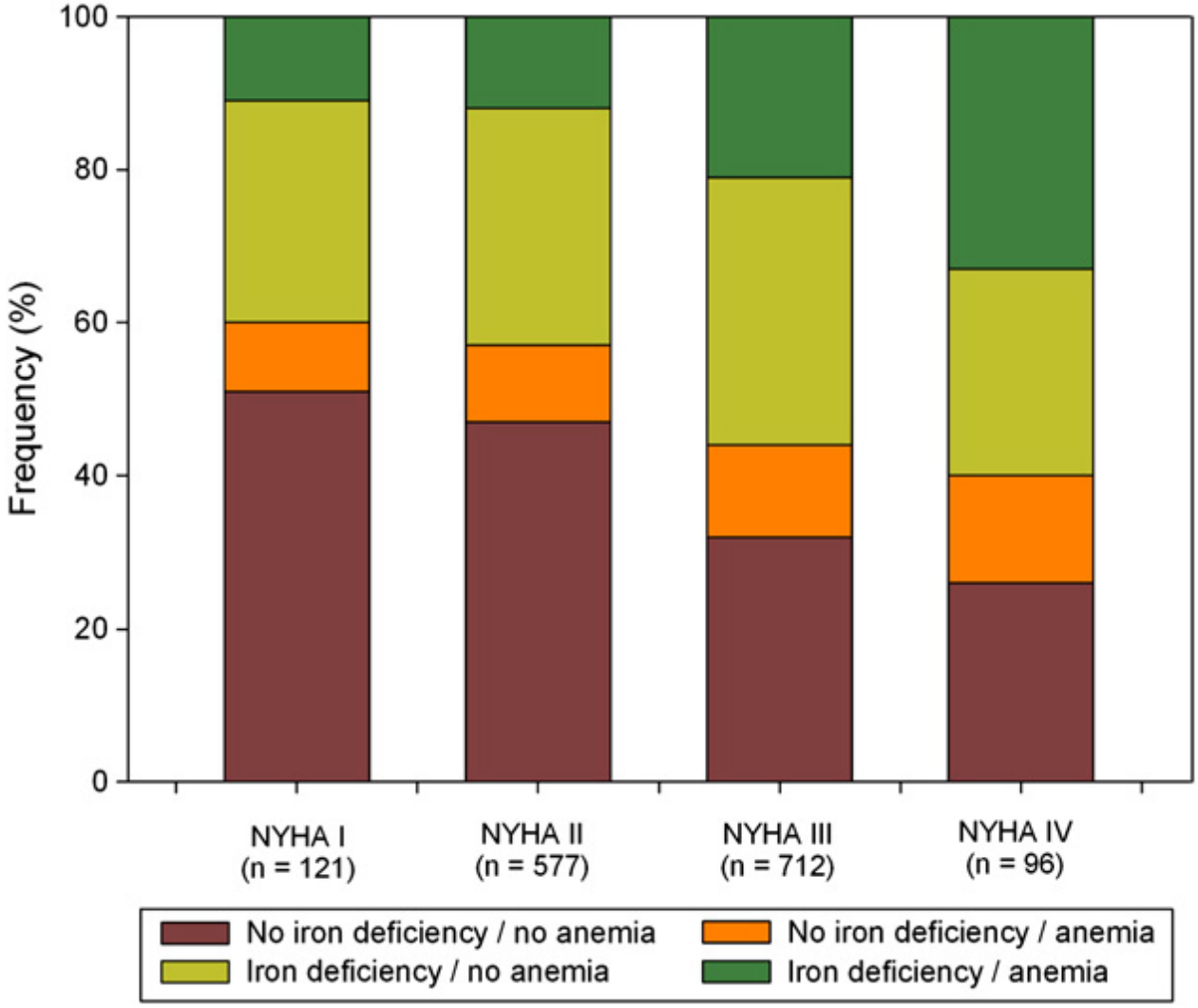


Iron deficiency and/or anemia stratified by NYHA functional class. Prevalence of ID and/or anemia per NYHA functional class.



ID *commonly* occurs even *without* anaemia in HF

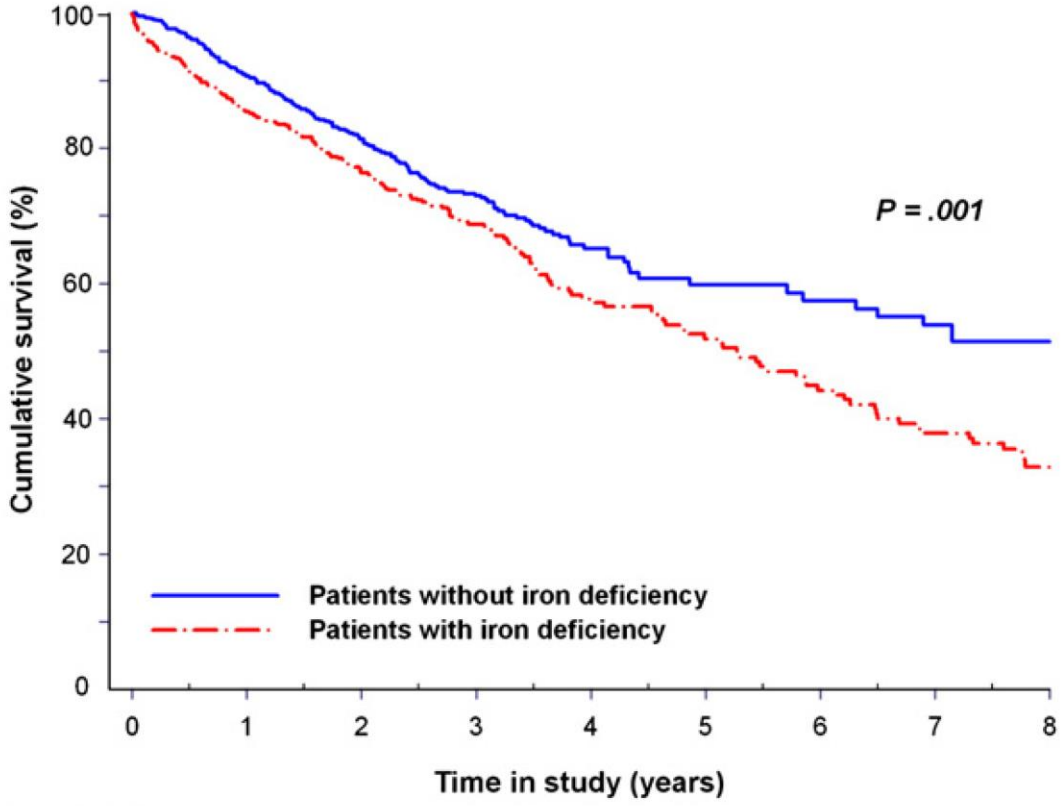
- ID (with or without anaemia) is present in 50% of HF patients

Iron deficiency definition used:

- Serum ferritin <100 µg/L or
- Serum ferritin <299 µg/L if TSAT <20%

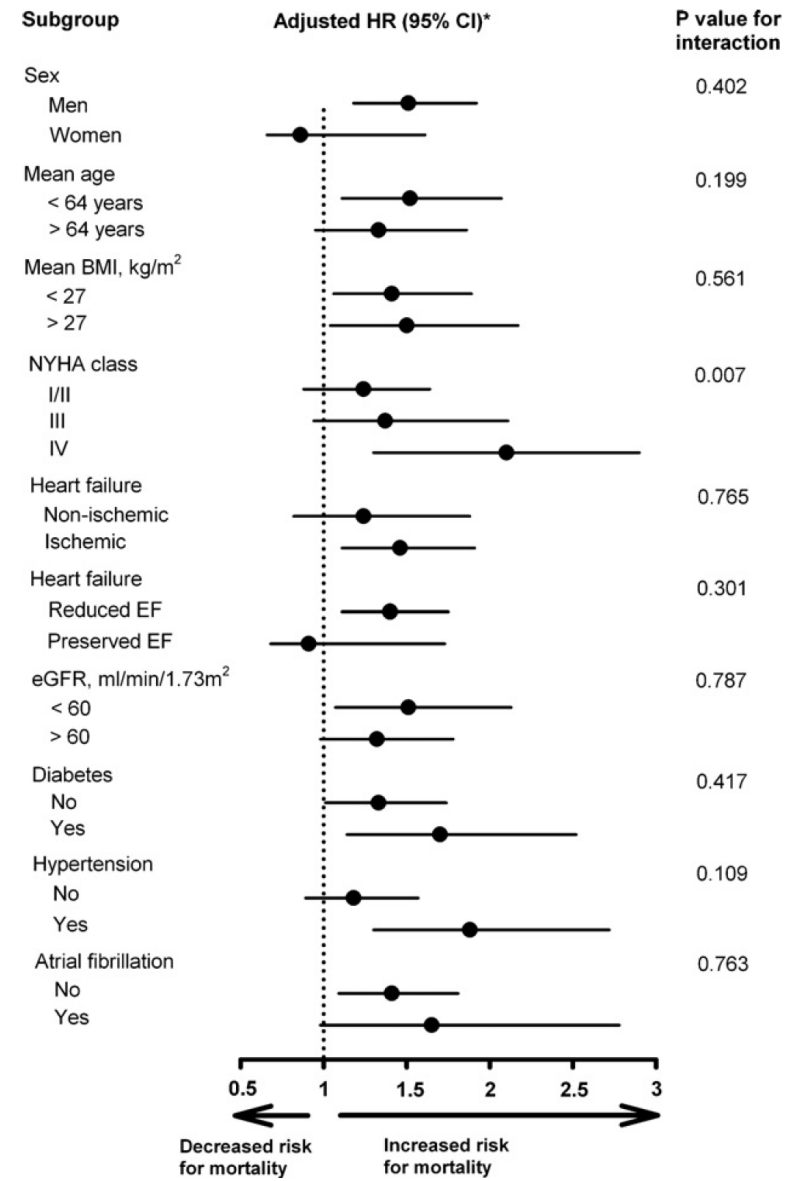
Klip IT et al. Am Heart J 2013;165:575-582

Difference in event-free survival rates between iron-deficient and non–iron-deficient patients with HF with or without anemia



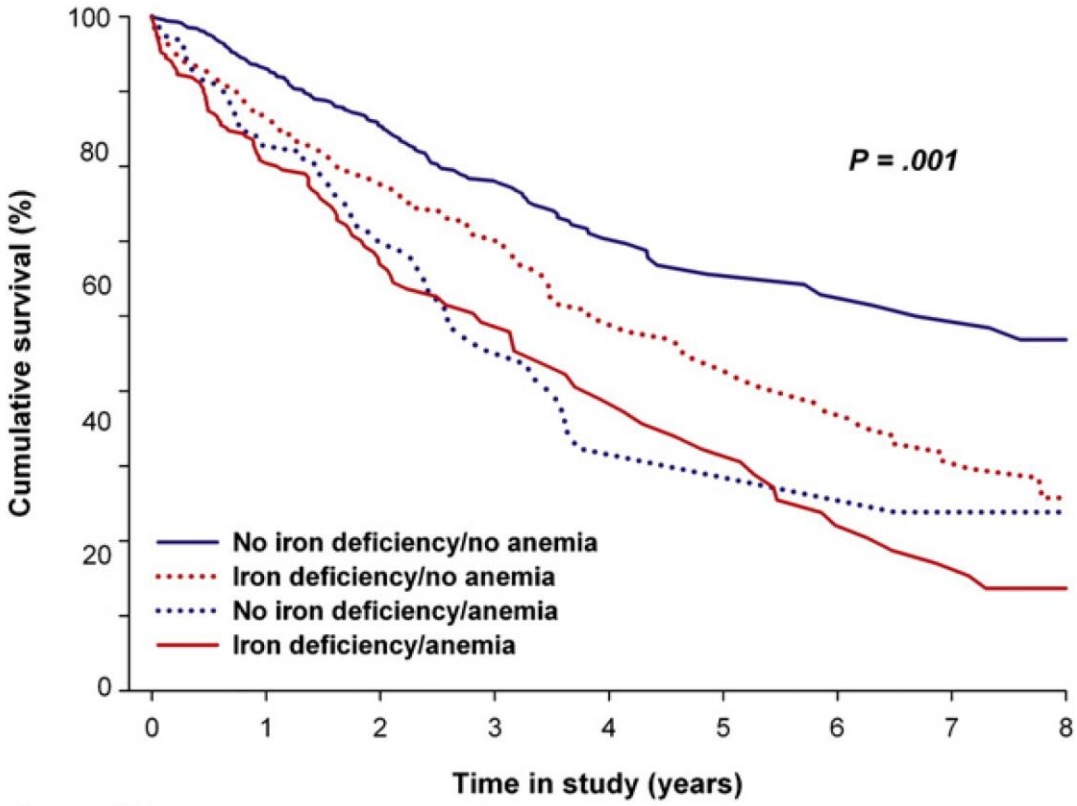
Numbers at risk:

ID absent	753	386	104	63	40
ID present	753	343	100	49	33



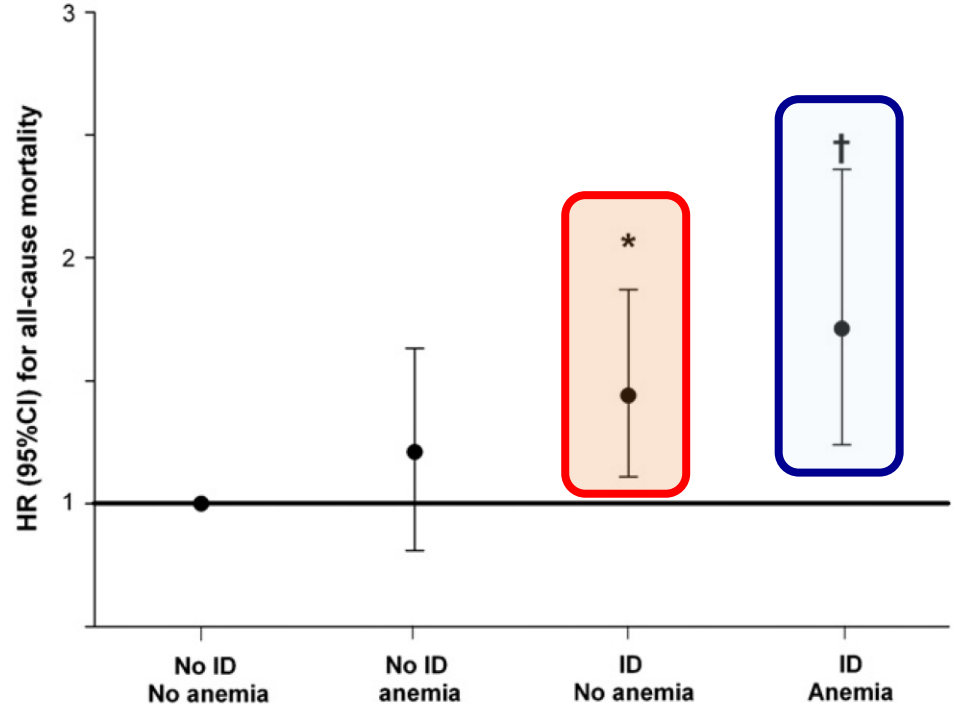
Klip IT et al. Am Heart J 2013;165:575-582

Difference in event-free survival rates between iron-deficient and non–iron-deficient patients with HF with or without anemia



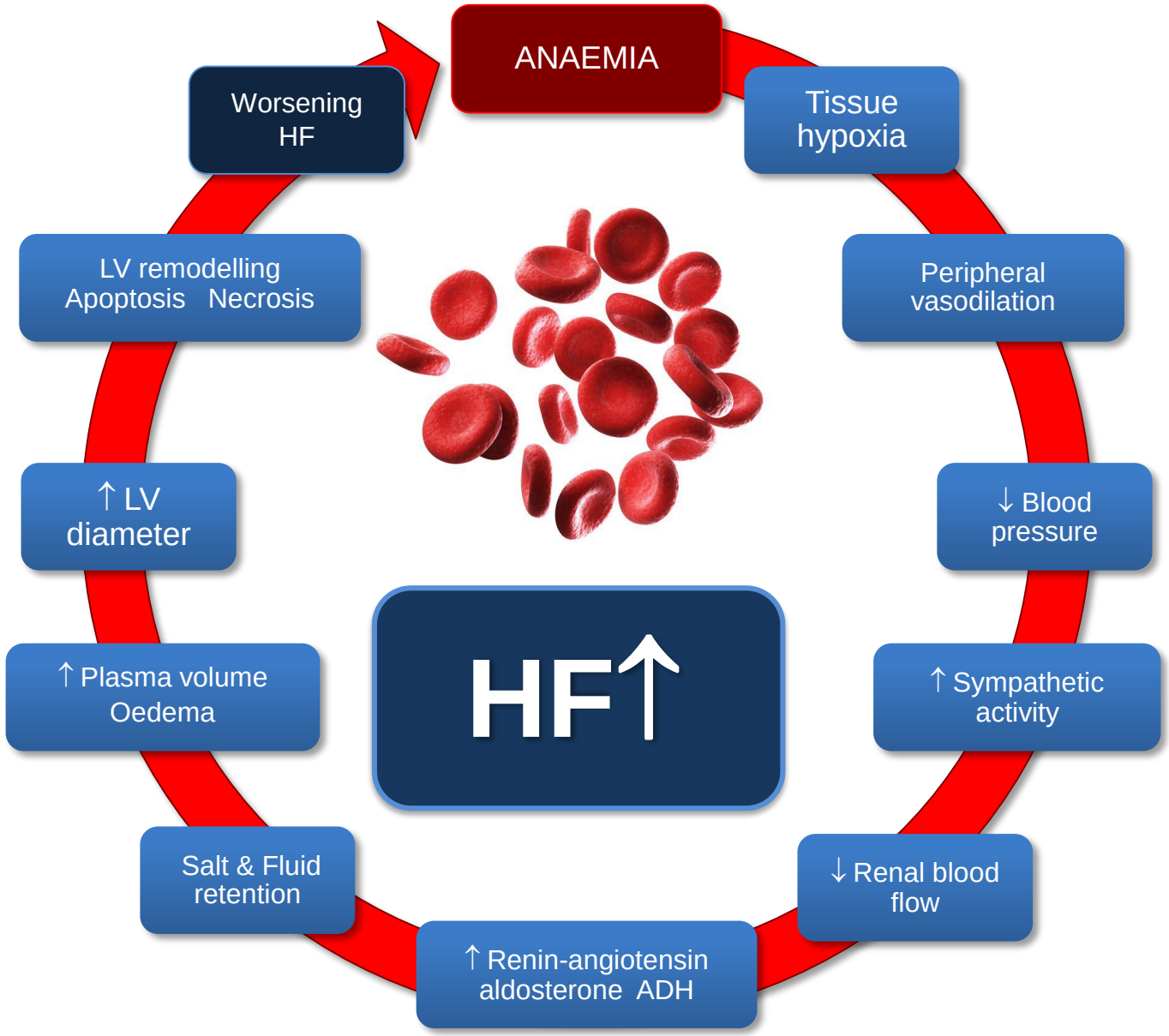
Numbers at risk:

No ID/no anemia	589	328	86	38	31
ID/no anemia	492	256	76	50	26
No ID/anemia	164	58	18	11	9
ID/anemia	261	87	24	13	7



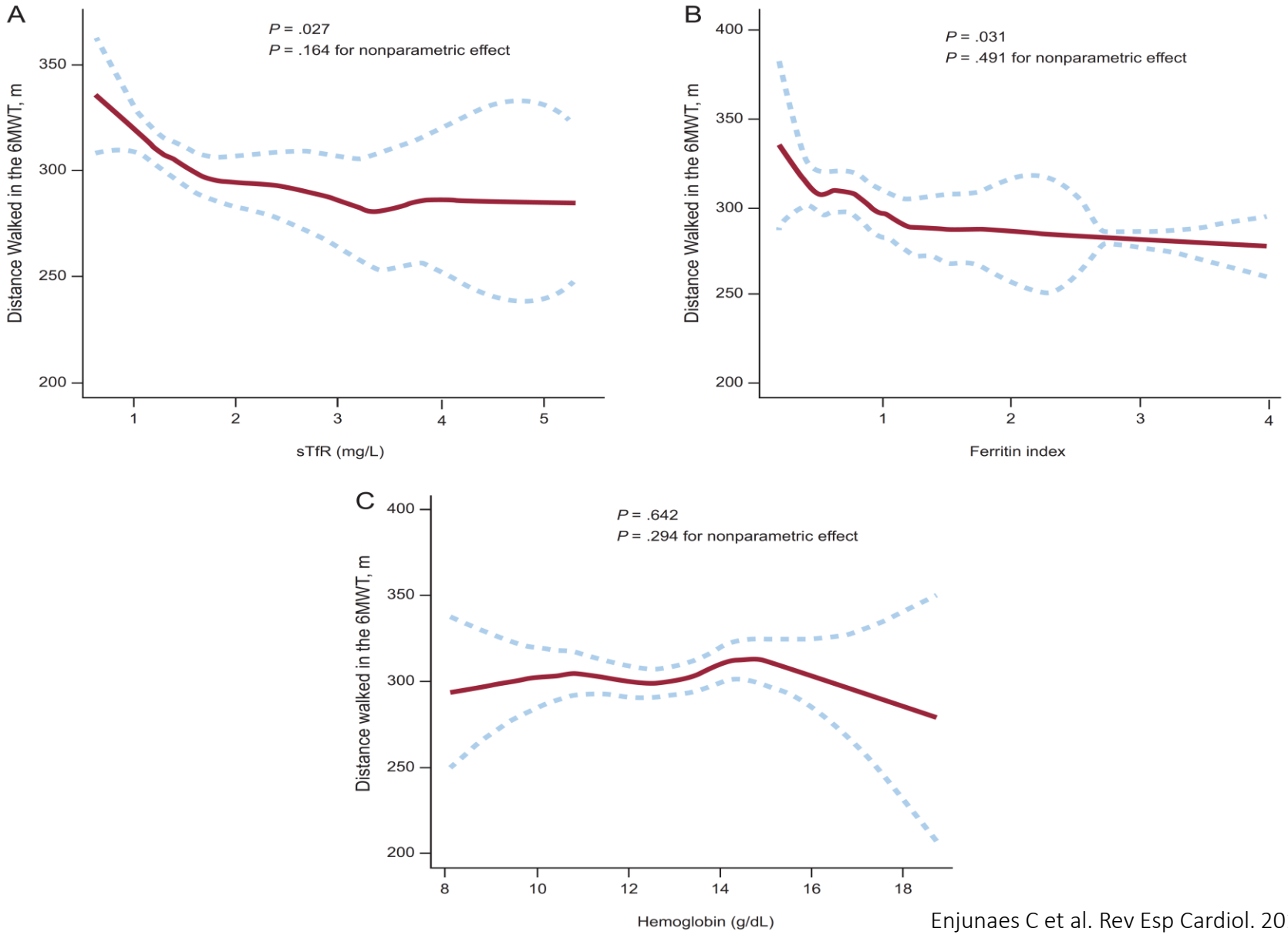
Klip IT et al. Am Heart J 2013;165:575-582

Vicious circle of Anemia and HF



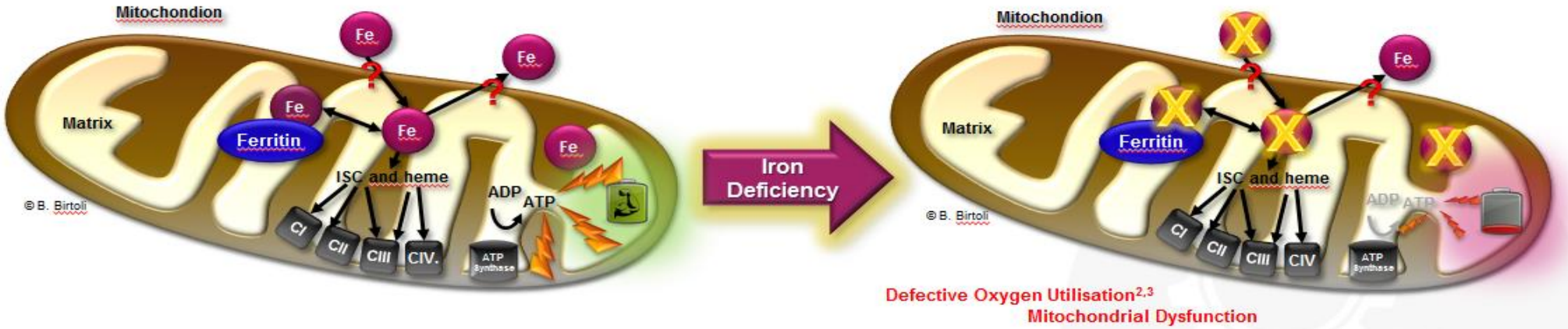
Anand IS et al. J Am Coll Cardiol 2008;52:501–11

Impact of iron status on exercise capacity (6-minute walk test) in patients with chronic heart failure



Enjunaes C et al. Rev Esp Cardiol. 2016;69(3):247–255

As a vigorously metabolically active tissue, the heart is a primary tissue target of iron delivery



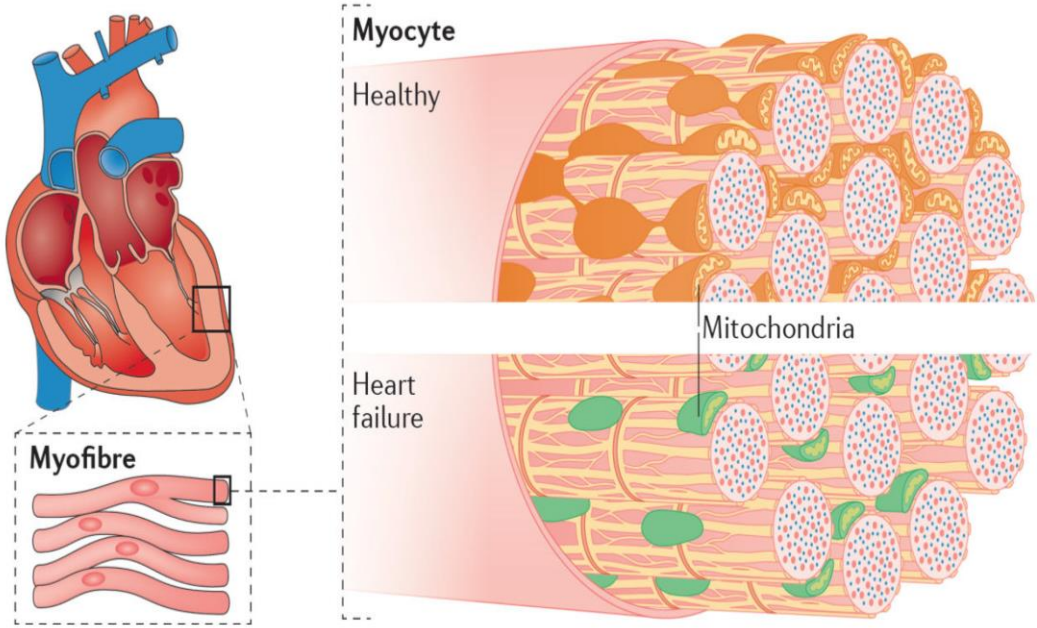
– The heart requires robust levels of ATP to sustain continuous contractions
  ⇨ **Cardiomyocytes are highly mitochondria-dense**
– Iron is required for iron/sulphur cluster protein and heme-containing cytochrome components of the electron transport chain complexes I-IV
  ⇨ **ATP synthase within the mitochondria**

ATP, Adenosine Tri-Phosphate; ADP, Adenosine Diphosphate; CHF, Chronic heart failure; Fe, iron

➢ **Iron Deficiency is associated with energy deficiency**
  ⇨ Reduced activity of respiratory complexes at the mitochondrial level

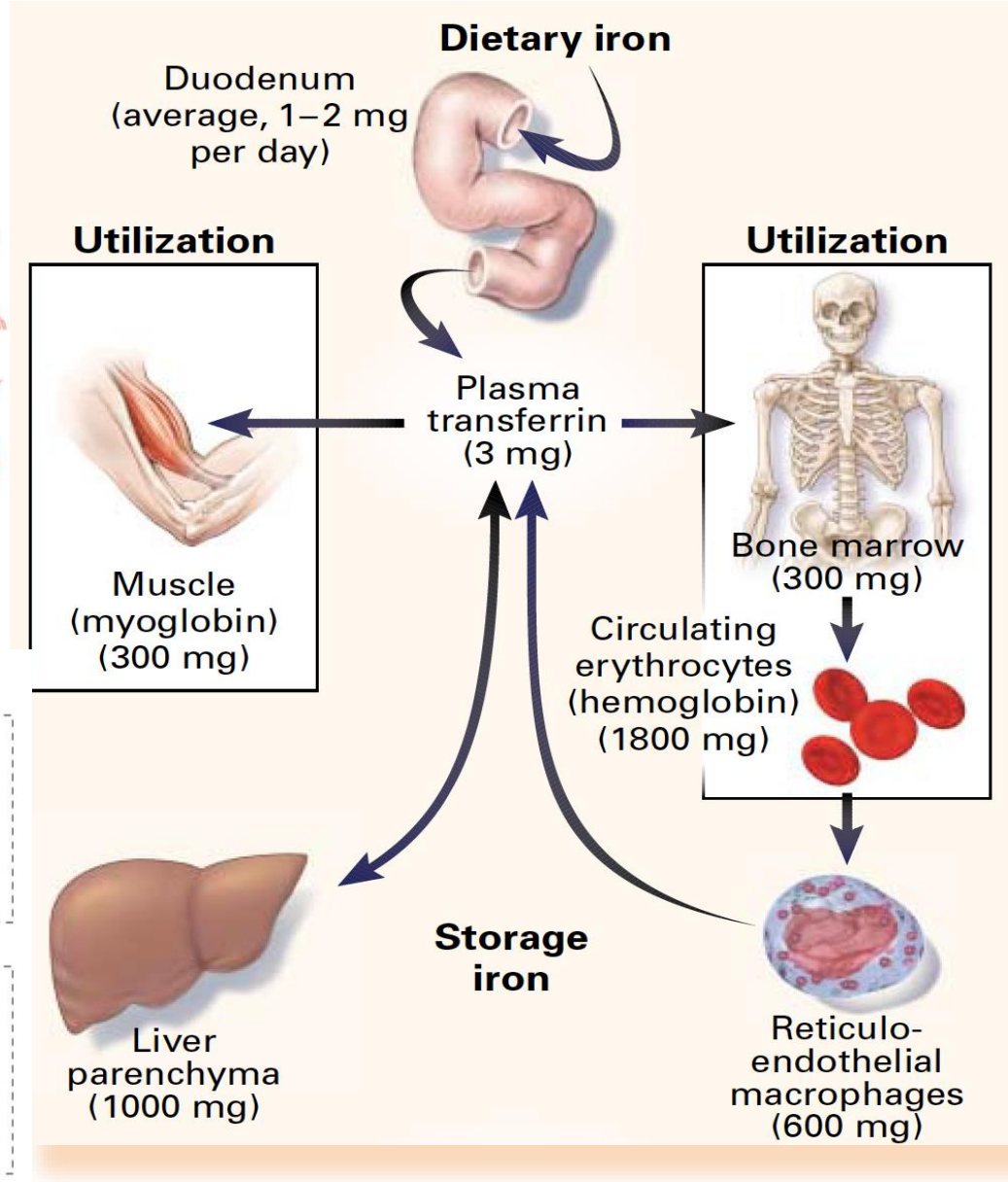
1. Rines AK and Ardehali H. *J Mol Cell Cardiol*. 2013; 55: 50–57
2. Cartier LJ *et al. J Biol Chem.* 1986 ;261:13827-32
3. Oexle H et al. *Biochim Biophys Acta*. 1999 ;1413:99-107

Distribution of iron in adults



The numbers of the heart:

- ✧ 100.800 beats/day
- ✧ 5-6 kg ATP/day
- ✧ mitochondria: 40% cell volume



Brown DA et al. Nat Rev Cardiol. 2017 April ; 14(4): 238–250.

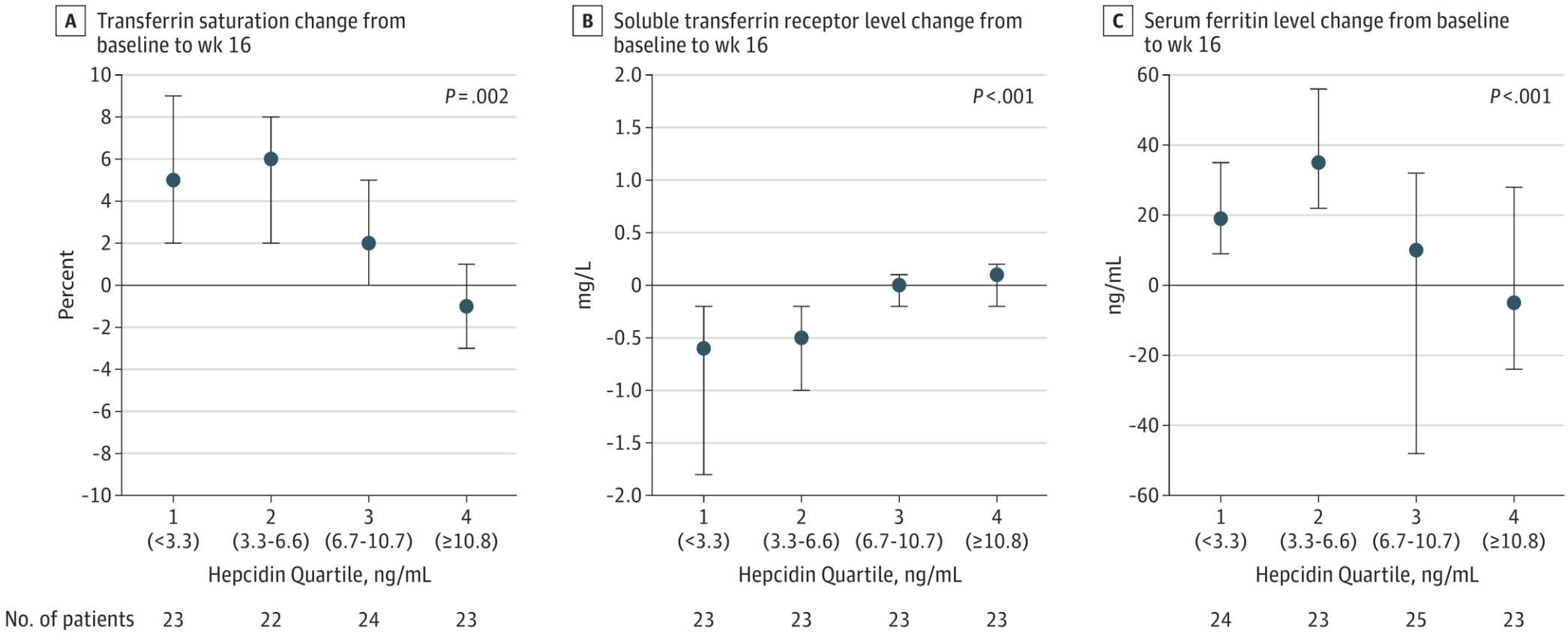
Andrews NC. N Engl J Med. 1999 Dec 23;341(26):1986-95.

Effect of Oral Iron Repletion on Exercise Capacity in Patients With Heart Failure With Reduced Ejection Fraction and Iron Deficiency. The IRONOUT HF Randomized Clinical Trial

	Median (IQR)					
	Week-16 Values[a]		Change From Baseline to Week 16		Difference in Change From Baseline (95% CI)	*P* Value
	Oral Iron	Placebo	Oral Iron	Placebo		
Primary End Point						
Peak $\dot{V}O_2$ at 16 wk, mL/min	1218 (892 to 1500)	1187 (902 to 1425)	23 (−84 to 142)	−2 (−110 to 104)	21 (−34 to 76)	.46
Ppeak $\dot{V}O_2$ at 16 wk, mL/kg/min	13.5 (11.7 to 16.3)	13.0 (10.2 to 15.9)	0.20 (−1.1 to 1.6)	0.01 (−1.1 to 0.9)	0.30 (−0.27 to 0.87)	.30
Secondary End Points						
6-Min walk distance at 8 wk, m	380 (322 to 467)	376 (286 to 448)	15 (−17 to 55)	21 (−24 to 56)	−1 (−24 to 23)	.95
6-Min walk distance at 16 wk, m	366 (315 to 456)	397 (299 to 472)	19 (−19 to 51)	32 (−12 to 66)	−13 (−32 to 6)	.19
Mean response time (O_2 uptake kinetics), s	52 (46 to 61)	47 (40 to 58)	2.5 (−7 to 9)	1 (−10 to 6)	3 (−2 to 8)	.19
Ventilatory efficiency (V_E/V_{CO2} slope)	34.8 (29.9 to 41.1)	33.5 (29.4 to 38.9)	−0.3 (−3.0 to 2.1)	−0.3 (−4.6 to 2.8)	0.8 (−0.3 to 2.6)	.35
NT-proBNP, pg/mL	889 (376 to 2373)	1085 (447 to 2582)	4 (−342 to 288)	−37 (−412 to 363)	159 (−280 to 599)	.48
KCCQ clinical summary score at 8 wk[b]	81.3 (70.8 to 91.7)	75.0 (58.9 to 87.5)	5.2 (−2.1 to 12.5)	1.0 (−7.3 to 8.3)	3.4 (−0.4 to 7.2)	.08
KCCQ clinical summary score at 16 wk[b]	80.7 (67.7 to 91.6)	77.1 (65.1 to 89.6)	3.1 (−4.2 to 13.5)	3.0 (−4.2 to 10.4)	1.0 (−2.4 to 4.4)	.57

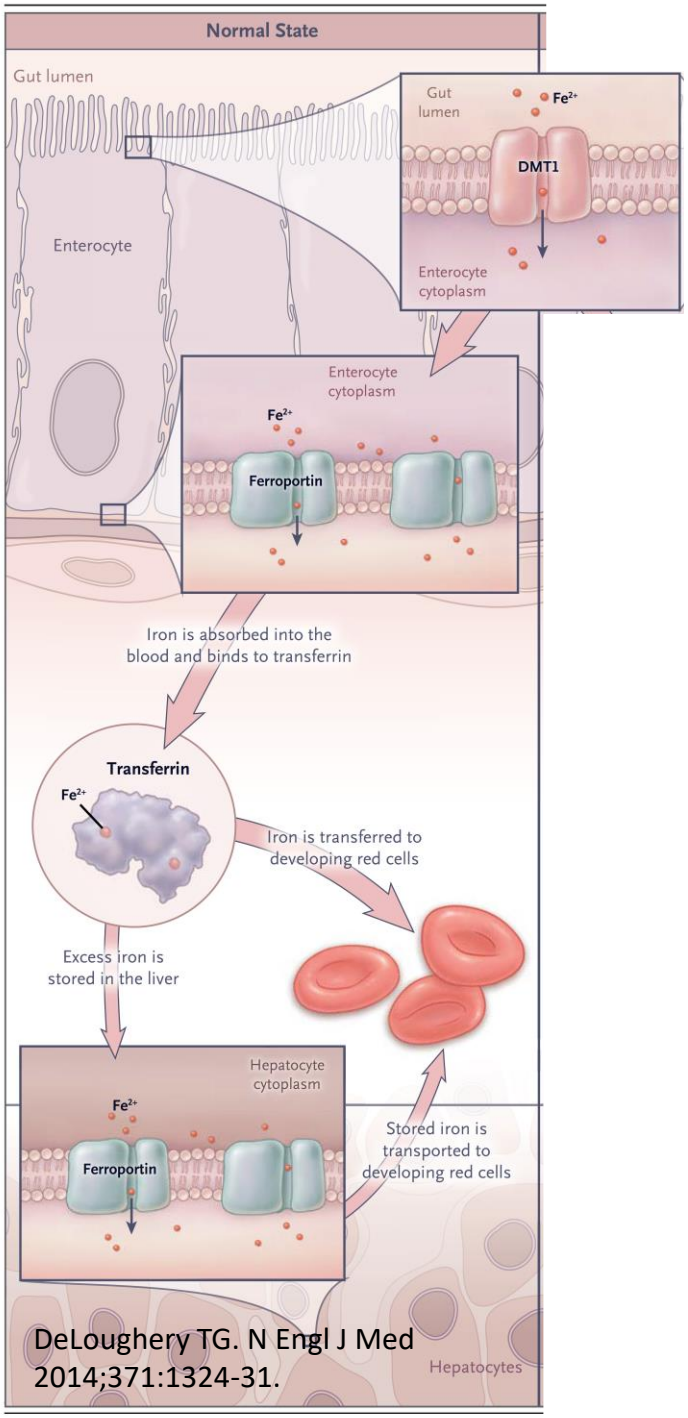
Lewis GD et al. JAMA. 2017;317(19):1958-1966.

Relationships Between Quartiles of Baseline Plasma Hepcidin Levels and Response in Participants Treated With Iron Polysaccharide The IRONOUT HF Randomized Clinical Trial

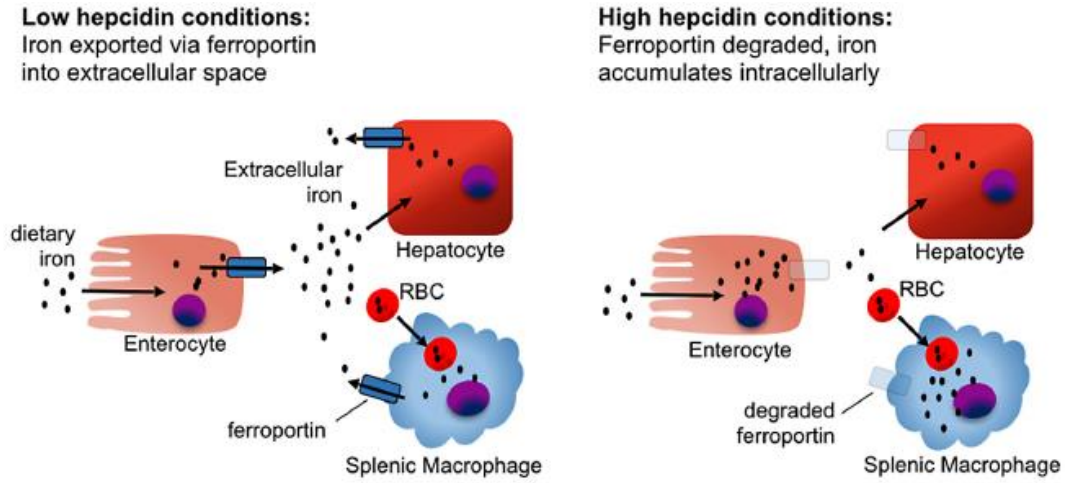


*…These findings do not support the use of **oral iron supplementation** in patients with heart failure and reduced left ventricular ejection fraction and iron deficiency.*

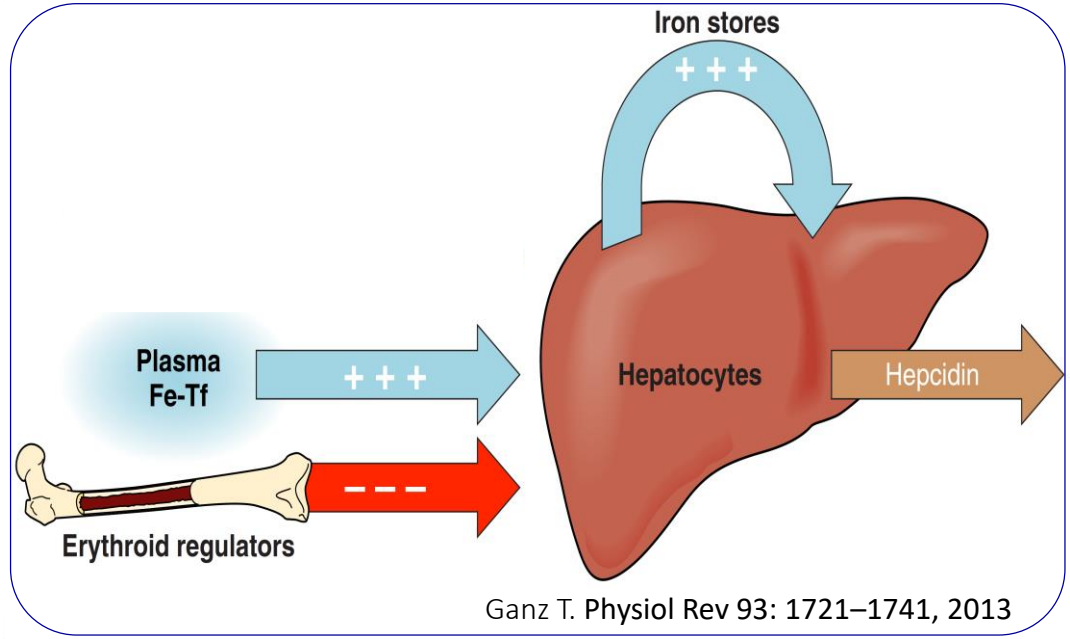
Lewis GD et al. JAMA. 2017;317(19):1958-1966.



DeLoughery TG. N Engl J Med 2014;371:1324-31.



Michels K et al. PLoS Pathog. 2015 Aug; 11(8): e1004998



Ganz T. Physiol Rev 93: 1721–1741, 2013

Recommendations for iron replacement in HF

2017 ACC/AHA/HFSA Focused Update of the 2013 ACCF/AHA Guideline for the Management of HF[1]	COR	LOE
In patients with NYHA class II and III HF and iron deficiency (ferritin <100 ng/mL or 100 to 300 ng/mL if transferrin saturation is <20%), **intravenous iron replacement** might be **reasonable to improve functional status and QoL.** *NEW: New evidence consistent with therapeutic benefit.*	IIb	B-R (3,4)

2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure[2]	Class	Level
Intravenous FCM should be considered in symptomatic patients (serum ferritin <100 µg/L, or ferritin between 100–299 µg/L and transferrin saturation <20%) in order to **alleviate HF symptoms, and improve exercise capacity and quality of life**.	IIa	A (3,4)

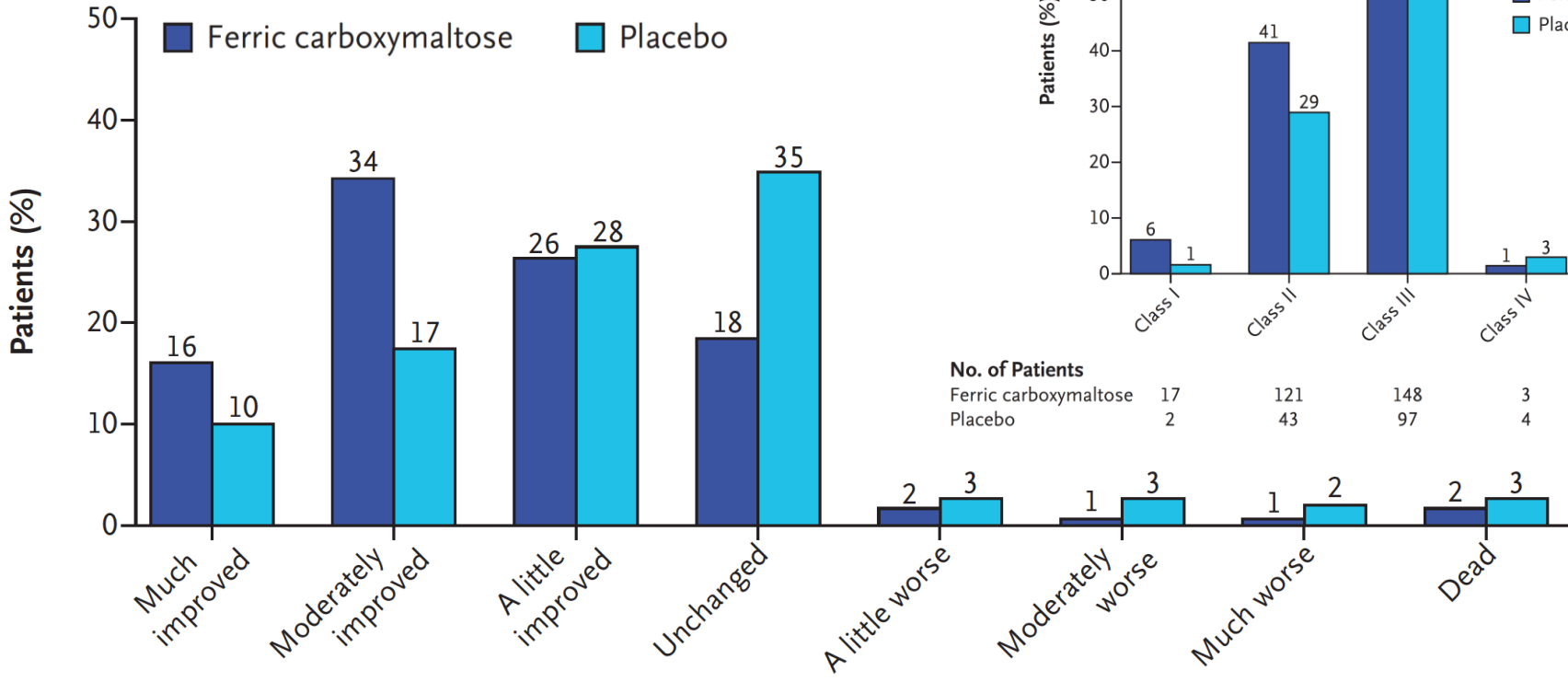
[3]Anker SD, et al. N Engl J Med 2009;361:2436–2448 ➔ FAIR-HF

[4]Ponikowski P, et al. Eur Heart J 2015;36:657–668 ➔ CONFIRM

[1]Yancy CW et al. Circulation. 2017 Aug 8;136(6):e137-e161

[2]Ponikowski P et al. European Heart Journal (2016) 37, 2129–2200

FAIR-HF: primary endpoint: Self-Reported Patient Global Assessment at Wk 24



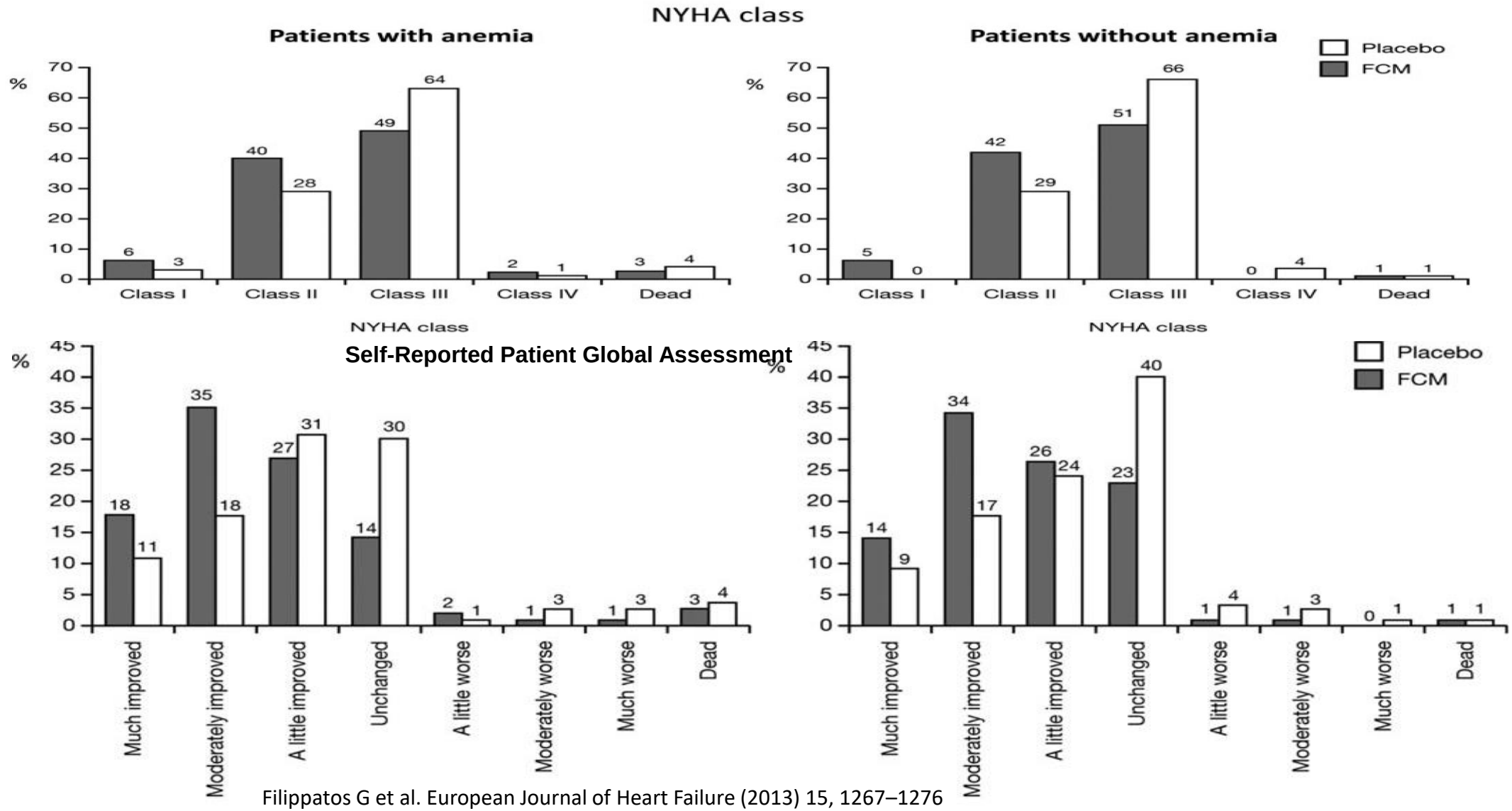
No. of Patients								
Ferric carboxymaltose	47	100	77	54	5	2	2	5
Placebo	15	26	41	52	4	4	3	4

FAIR-HF: primary endpoint: NYHA Functional Class at Wk 24

No. of Patients					
Ferric carboxymaltose	17	121	148	3	5
Placebo	2	43	97	4	4

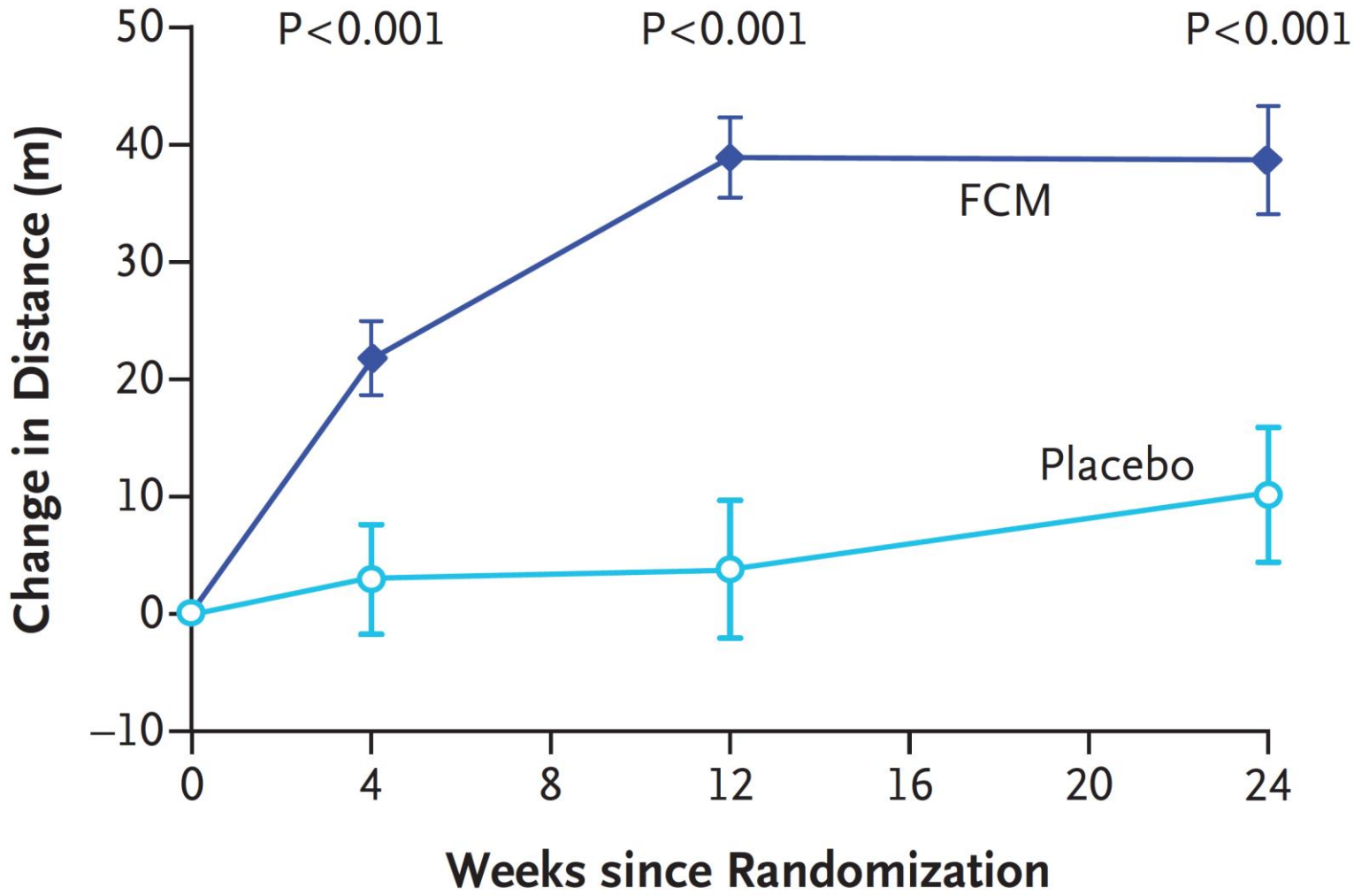
Anker SD et al. N Engl J Med 2009;361:2436-48.

FAIR-HF: primary endpoint: Self-Reported Patient Global Assessment and NYHA class at Wk 24 according to the presence or absence of anaemia at baseline



Filippatos G et al. European Journal of Heart Failure (2013) 15, 1267–1276

FAIR-HF: secondary endpoint: 6-Minute-Walk Test



Anker SD et al. N Engl J Med 2009;361:2436-48.

European Heart Journal (2015) **36**, 657–668
doi:10.1093/eurheartj/ehu385

Beneficial effects of long-term intravenous iron therapy with ferric carboxymaltose in patients with symptomatic heart failure and iron deficiency†

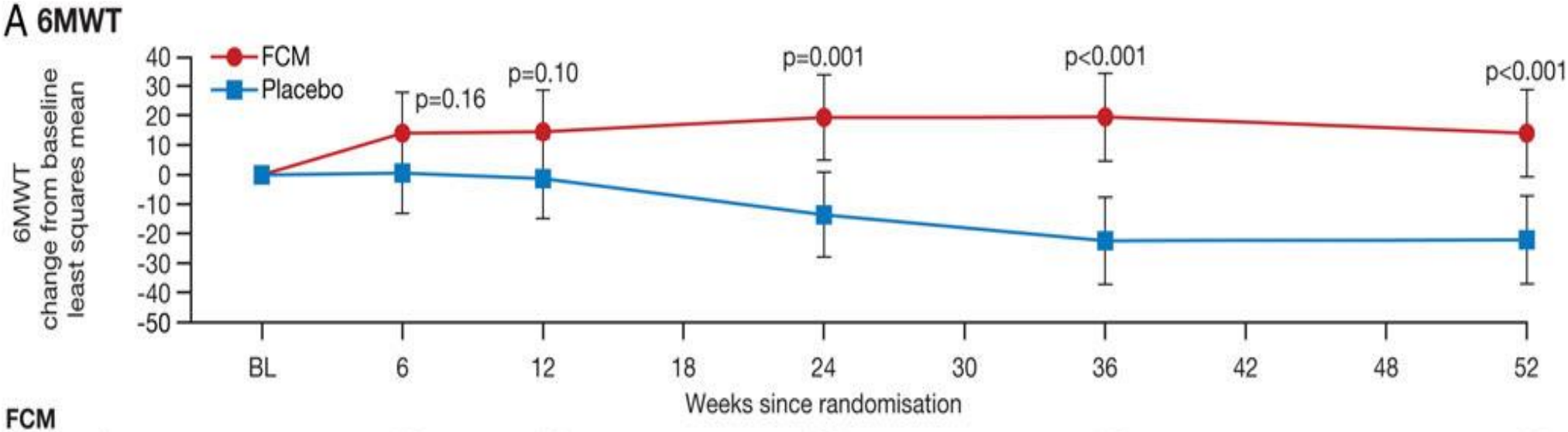
Piotr Ponikowski[1,2]*, Dirk J. van Veldhuisen[3], Josep Comin-Colet[4], Georg Ertl[5,6], Michel Komajda[7], Viacheslav Mareev[8], Theresa McDonagh[9], Alexander Parkhomenko[10], Luigi Tavazzi[11], Victoria Levesque[12], Claudio Mori[12], Bernard Roubert[12], Gerasimos Filippatos[13], Frank Ruschitzka[14], and Stefan D. Anker[15], for the CONFIRM-HF Investigators

Methods and results

CONFIRM-HF was a multi-centre, double-blind, placebo-controlled trial that enrolled 304 ambulatory symptomatic HF patients with left ventricular ejection fraction ≤45%, elevated natriuretic peptides, and iron deficiency (ferritin <100 ng/mL or 100–300 ng/mL if transferrin saturation <20%). Patients were randomized 1 : 1 to treatment with i.v. iron, as ferric carboxymaltose (FCM, $n = 152$) or placebo (saline, $n = 152$) for 52 weeks. The primary end-point was the change in 6-min-walk-test (6MWT) distance from baseline to Week 24. Secondary end-points included changes in New York Heart Association (NYHA) class, Patient Global Assessment (PGA), 6MWT distance, health-related quality of life (QoL), Fatigue Score at Weeks 6, 12, 24, 36, and 52 and the effect of FCM on the rate of hospitalization for worsening HF.

Ponikowsky P et al. European Heart Journal (2015) 36, 657–668

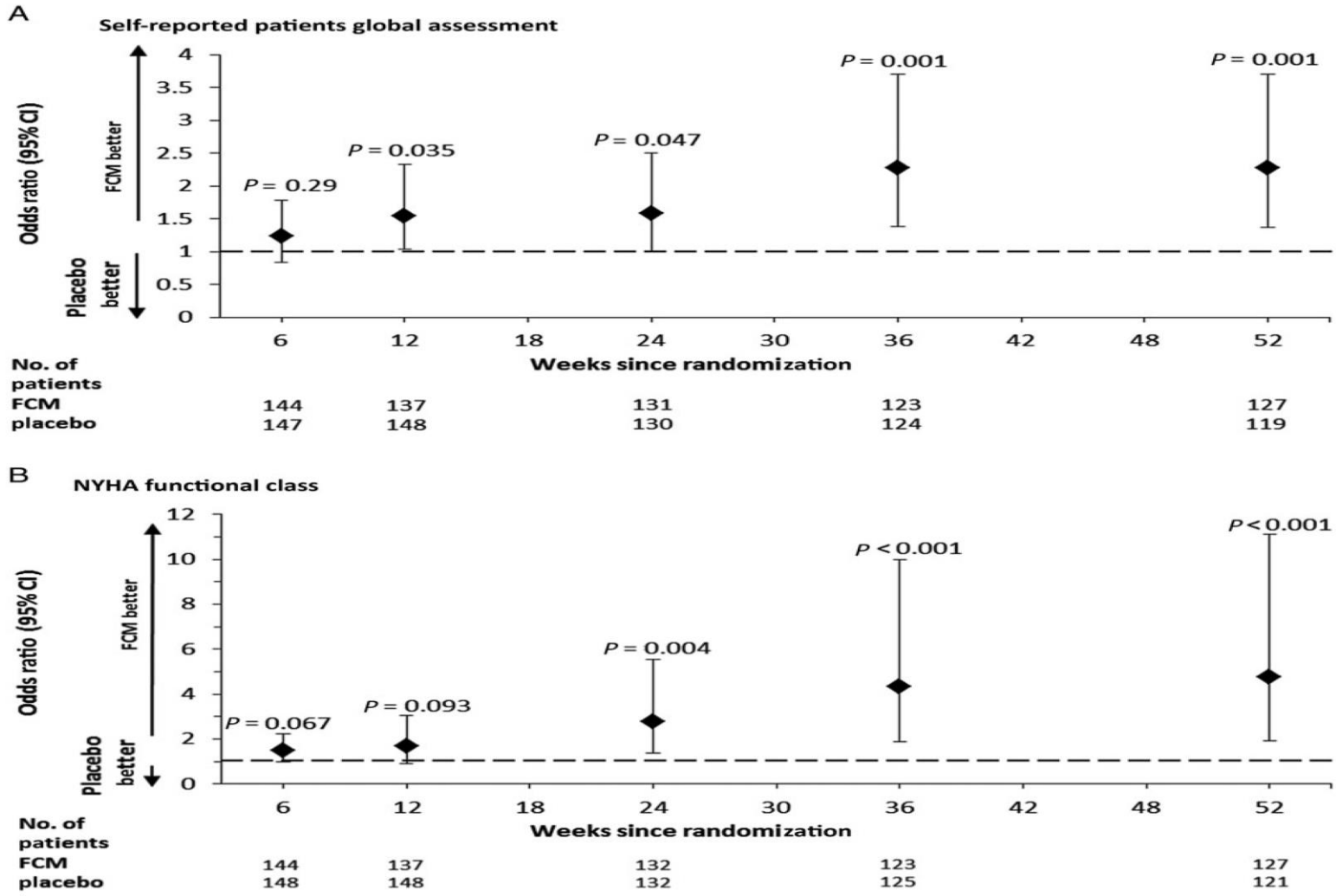
CONFIRM-HF: 6-Min Walk Test Over Time



FCM					
No of patients	143	137	130	122	125
LS mean (95% CI)	14 (0, 28)	15 (1, 29)	19 (5, 34)	20 (5, 34)	14 (-1, 29)
Placebo					
No of patients	148	146	131	123	121
LS mean (95% CI)	1 (-13, 14)	-1 (-15, 12)	-14 (-28, 1)	-22 (-37, -8)	-22 (-37, -7)
FCM vs Placebo	14 (-5, 33)	16 (-3, 35)	33 (13, 53)	42 (21, 62)	36 (16, 57)

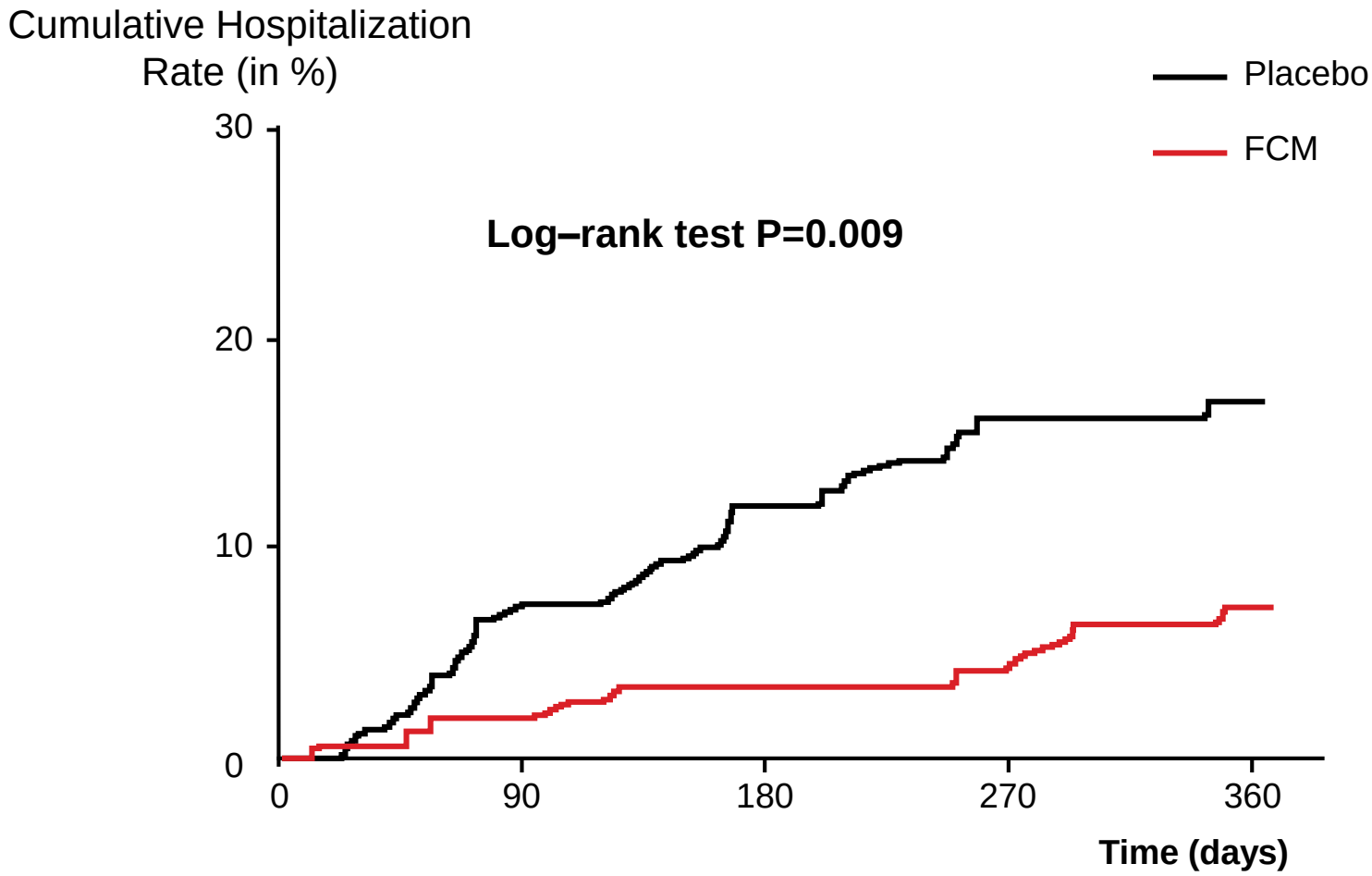
Ponikowsky P et al. European Heart Journal (2015) 36, 657–668

CONFIRM-HF: Patient Global Assessment and NYHA Functional Class over Time



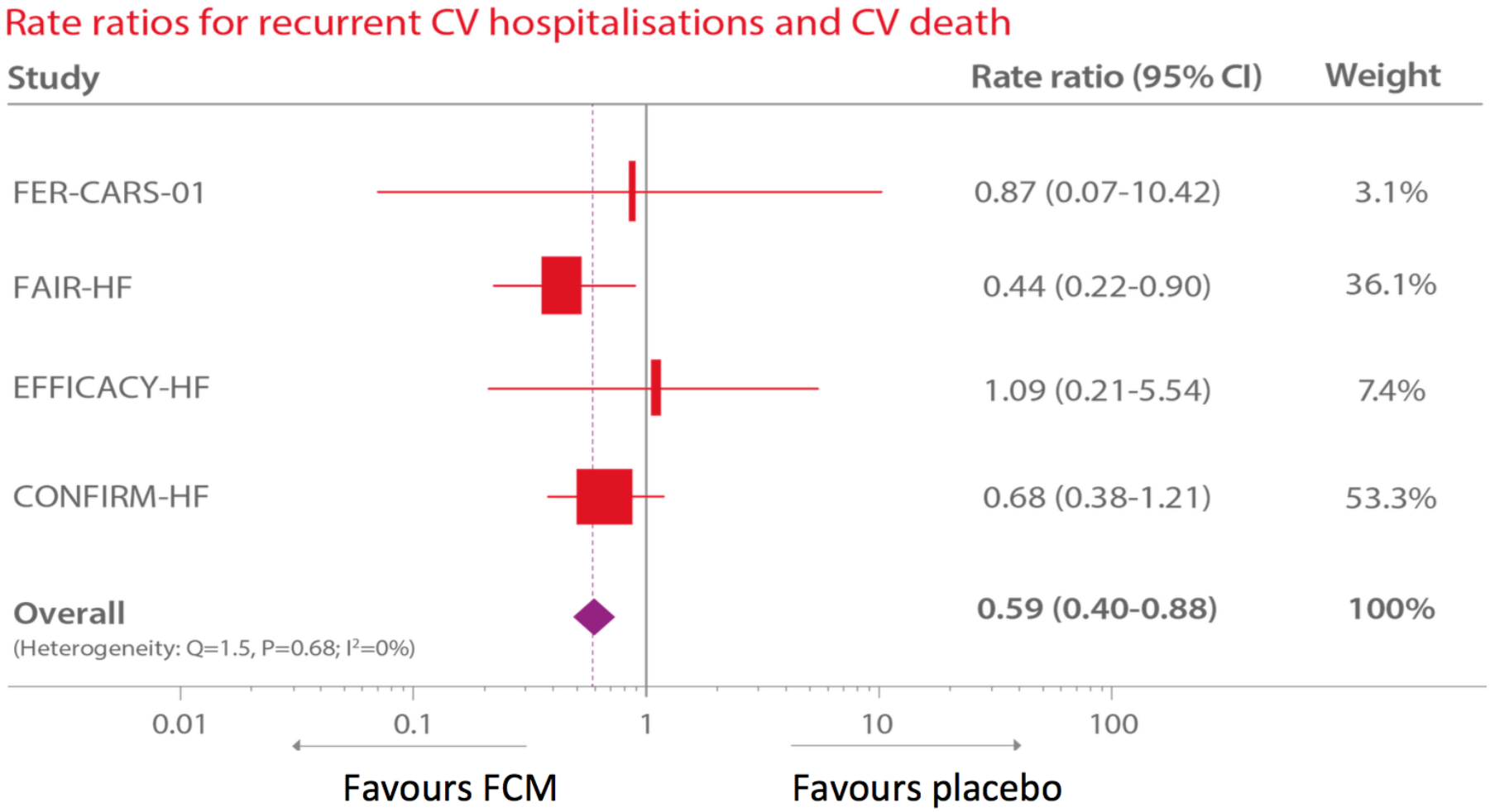
Ponikowsky P et al. European Heart Journal (2015) 36, 657–668

CONFIRM-HF: Time to first hospitalization due to worsening heart failure



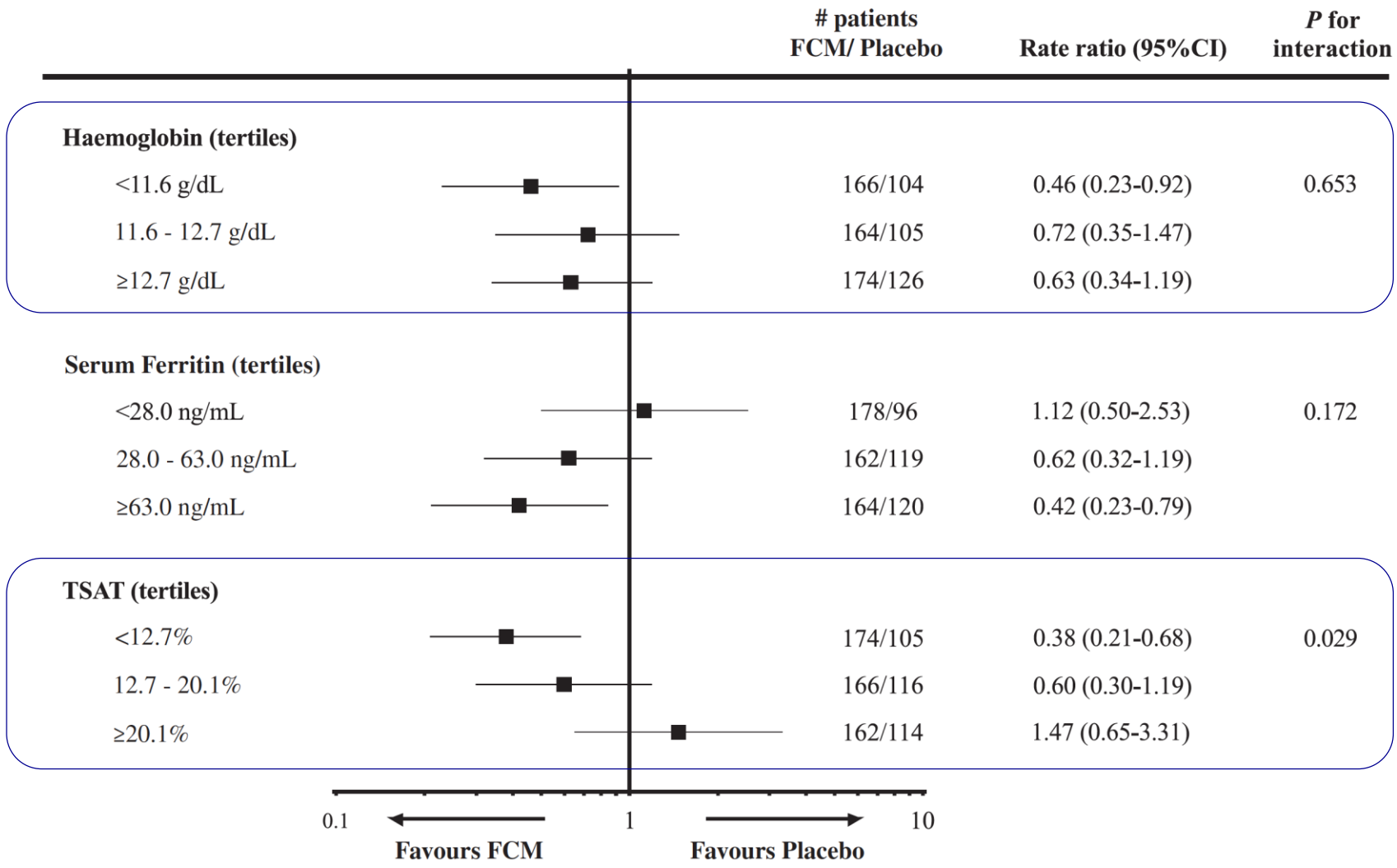
Ponikowsky P et al. European Heart Journal (2015) 36, 657–668

Effects of FCM on CV hospitalisations and mortality rates in iron-deficient HF patients: an individual patient data meta-analysis



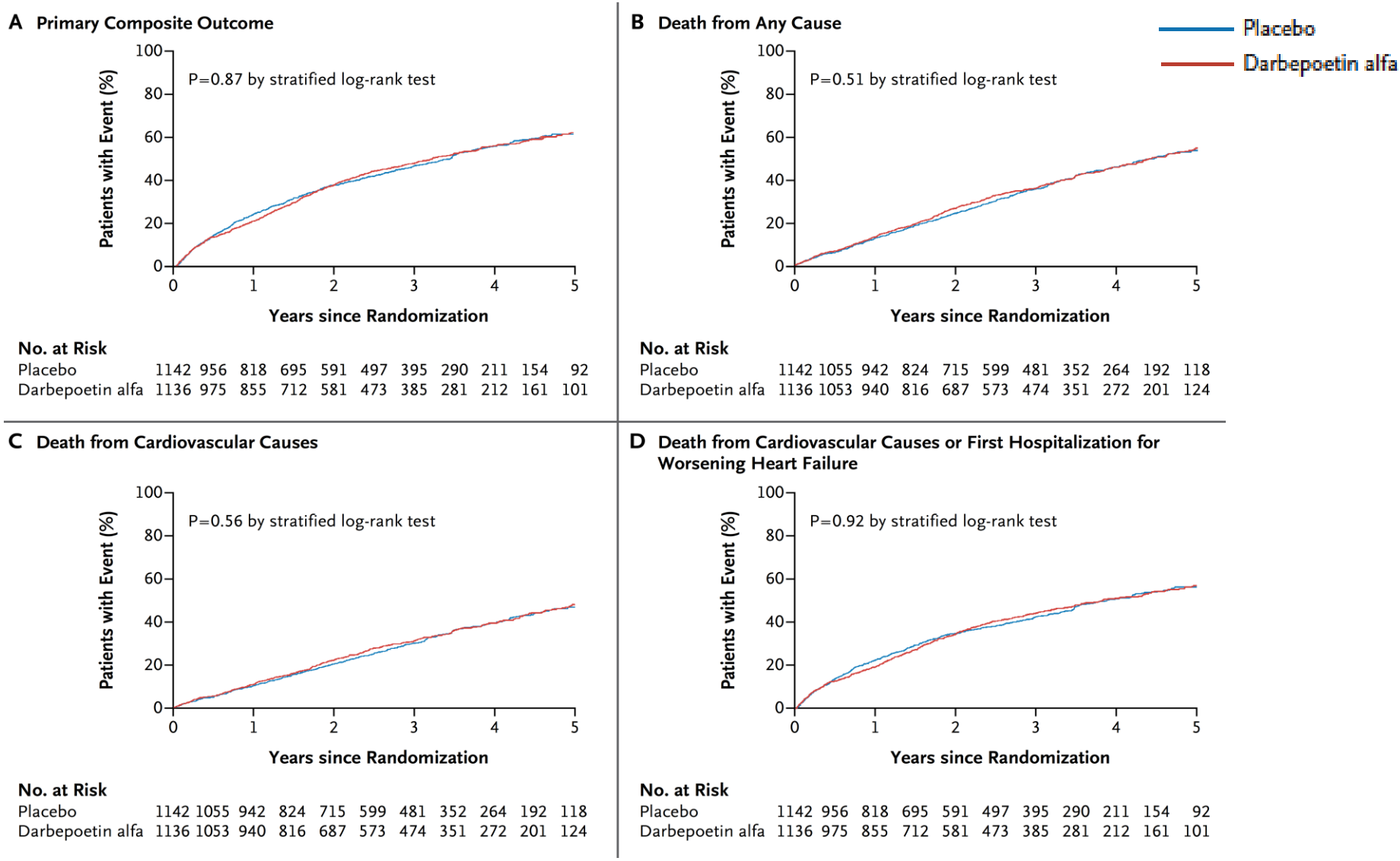
Anker SD et al. Eur J Heart Fail. 2018 Jan;20(1):125-133.

Effects of FCM on **CV hospitalisations and mortality rates** in iron-deficient HF patients: an individual patient data meta-analysis



Anker SD et al. Eur J Heart Fail. 2018 Jan;20(1):125-133.

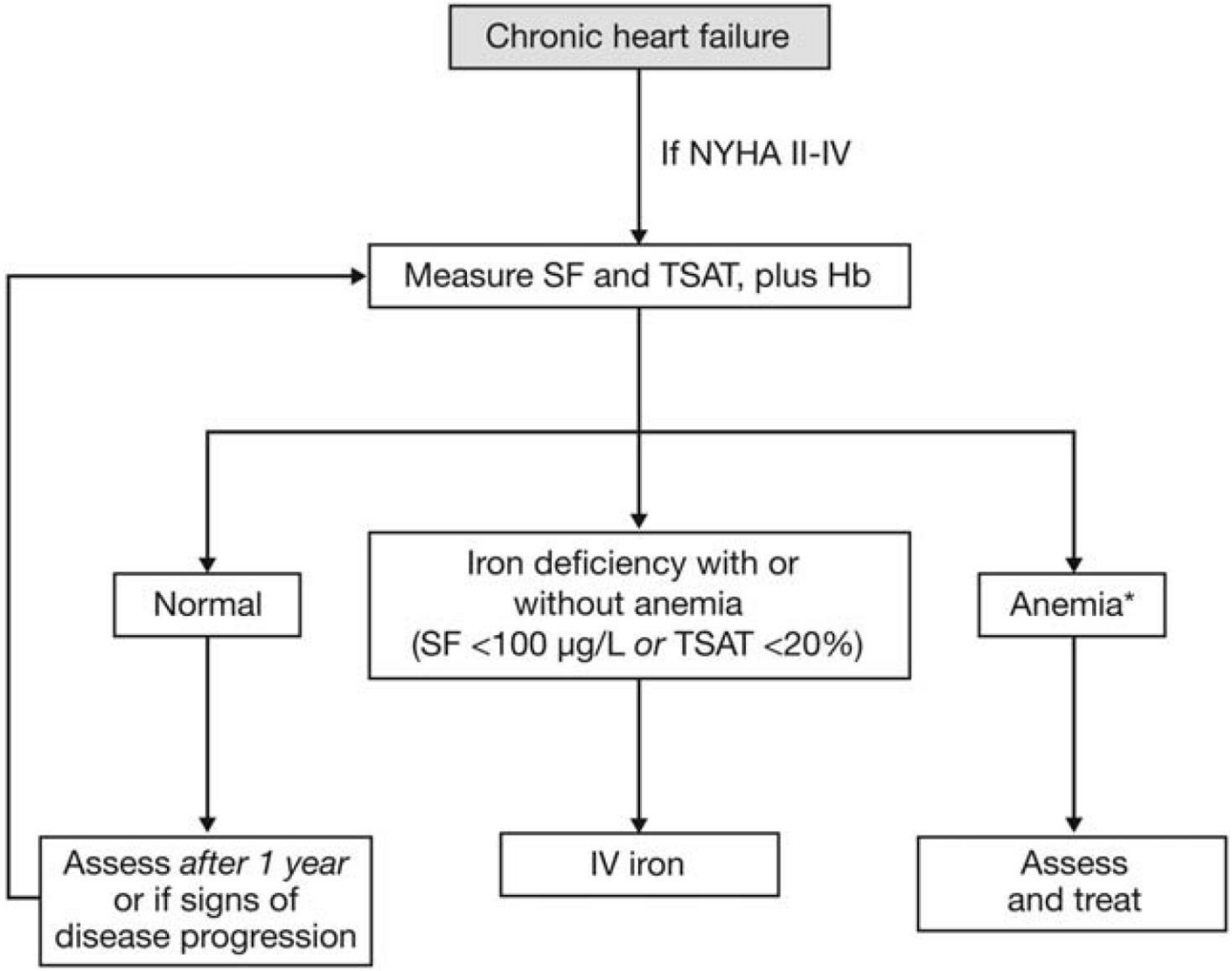
Treatment of Anemia with Darbepoetin Alfa in Systolic Heart Failure – RED HF study



Swedberg K et al. N Engl J Med 2013;368:1210-9.

Diagnostic algorithm: iron deficiency in chronic heart failure

International expert opinion on definition, diagnosis, and management



Maria Domenica Cappellini Am J Hematol. 2017 Jun 13. doi: 10.1002/ajh.24820

Concluding remarks (I)

- ✧ Hyperkalemia is a common and serious medical condition in patients with HF and CKD

- ✧ RAAS inhibitors are important therapies for these patients
  - These drugs offer cardioprotectiv and renoprotective benefits
  - The can also put patients at risk for hyperkalemia which should be carefully managed to ensure the obtaining of the maximal benefits form RAAS inhibitors

Concluding remarks (II)

- ✧ Iron deficiency is an important comorbidity and is prevalent in patients with heart failure but it is often neglected.

- ✧ Iron deficiency, **either absolute or functional**, is an independent predictor of outcomes and a major contributor to exercise intolerance, even in the absence of anaemia.

- ✧ Intravenous FCM should be considered in symptomatic patients with HF and iron deficiency