DIU d'Hypertension Artérielle, risque vasculaire et rénal
Toulouse, 24 mai 2013

L'HTA en Afrique et chez les patients afro-américains.

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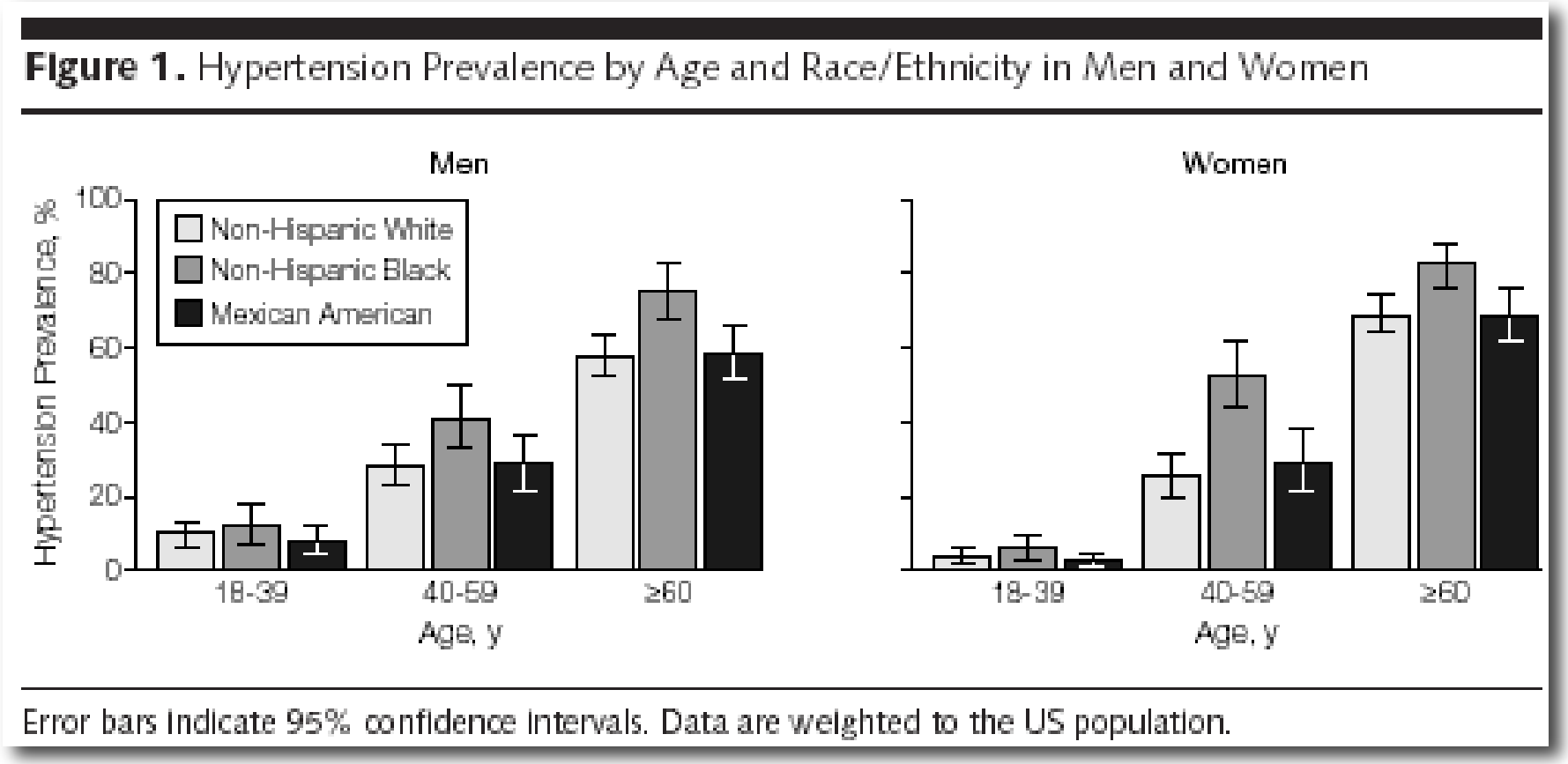
Plan

- **Epidémiologie**
- Physiopathologie
- Génétique
- Profil de risque
- Spécificités thérapeutiques

HTA prevalence USA 1988-2000



(Hajjar, JAMA 2003, 290, 199-206)

Epidémiologie comparée

- Prévalence selon l'âge (PA > 140/90 mmHg):

	Afrique*		Antilles#	USA$		France¤
	Hommes	Femmes	Noirs	Noirs	Blancs	
15-24 ans	11.4%	8.3%				
25-34 ans	17.8%	16.1%				
18-34 ans						8%
35-44 ans	23.8%	23.4%				
35-49 ans						28%
40-49 ans			33.4%			
45-54 ans	40.2%	50.0%				
50-59 ans			52.5%			
50-64 ans						57%
55-64 ans	60.8%	66.7%				
60-69 ans			67.6%			
≥ 65 ans	68.0%	81.8%				76%
70-79 ans			73.5%			
Total	23.6%	21.5%		32.4%	23.3%	41%

(*Astagneau, J Hypertension 1992, 10:1095; # Hennis, J Hypertens 2002, 20:2363;
$ Burt, Hypertension 1995, 25:305; ¤ Chamontin, AJH 1998, 11: 759)

Hypertension
JOURNAL OF THE AMERICAN HEART ASSOCIATION
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Why Do Black Americans Have Higher Prevalence of Hypertension?: An Enigma Still Unsolved

Flávio D. Fuchs

Hypertension 2011;57;379-380; originally published online Feb 7, 2011;
DOI: 10.1161/HYPERTENSIONAHA.110.163196
Hypertension is published by the American Heart Association. 7272 Greenville Avenue, Dallas, TX 72514
 Print ISSN: 0194-911X. Online ISSN: 1524-4563

«Although population-level adoption of healthy behaviors may contribute to reduction of the societal burden of cardiovascular disease in general, these findings suggest that racial/ethnic differences in some health behaviors do not explain the disparities in hypertension prevalence and control.»

Health Behaviors and Racial Disparity in Blood Pressure Control in the National Health and Nutrition Examination Survey

Race/Ethnicity	Sample Size, n	Prevalence of Poor BP Control (%±SE)†	Odds Ratio (95% CI)	
			Model 1 (Base Model)*	Model 2 (Model 1+Health Behaviors)†
Total	21 489	18.2±0.8		
Non-Hispanic white	13 170	17.0±0.8	Reference	Reference
Non-Hispanic black	4535	27.4±1.1	1.90 (1.57 to 2.31)	1.88 (1.53 to 2.32)
Mexican American	3784	20.2±1.5	1.08 (0.80 to 1.45)	1.14 (0.85 to 1.53)

«None of the self-reported health behaviors, includingphysical activity, mitigated the disparity in BP control in our study.»

Redmond N, Baer HJ, Hicks LS. Health Behaviors and Racial Disparity in Blood Pressure Control in the National Health and Nutrition Examination Survey. Hypertension. 2011 Feb 16;57(3):383–9.

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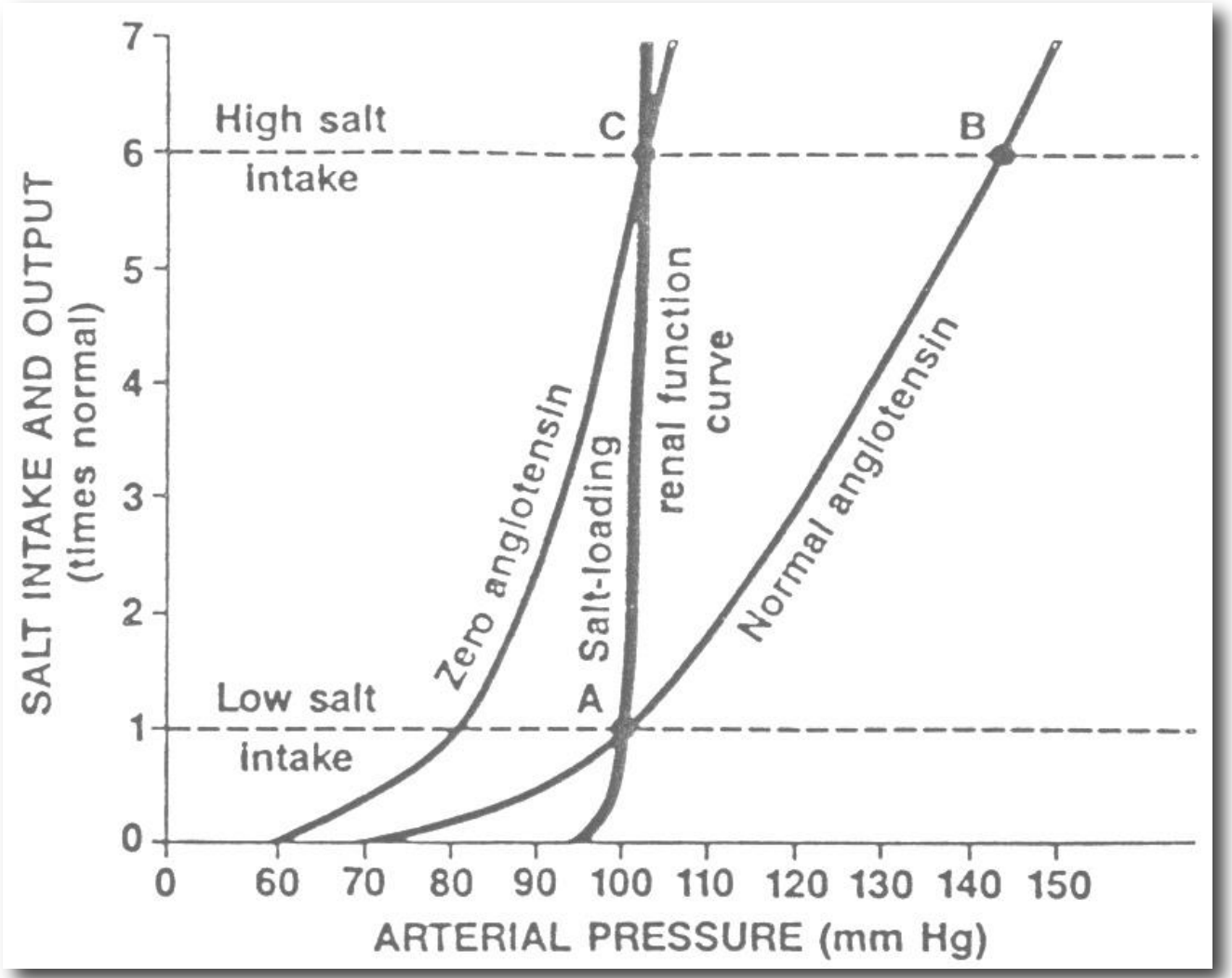
Glomérule & sensibilité au sel



(d'après Kimura, 1993)

Réponse à la charge sodée

	Sel-résistants		Sel-sensibles	
Race	9 B + 6 N		11 N	
Age (ans)	52		47	
Poids (kg)	83		89	
Apport Na (mmol/j)	20	200	20	200
PAM (mmHg)	102	98	100	114*
DFG (ml/min)	97	100	102	107
FPR (ml/min)	482	531	538	464*
FF (%)	20.8	19.1	19.2	22.8*

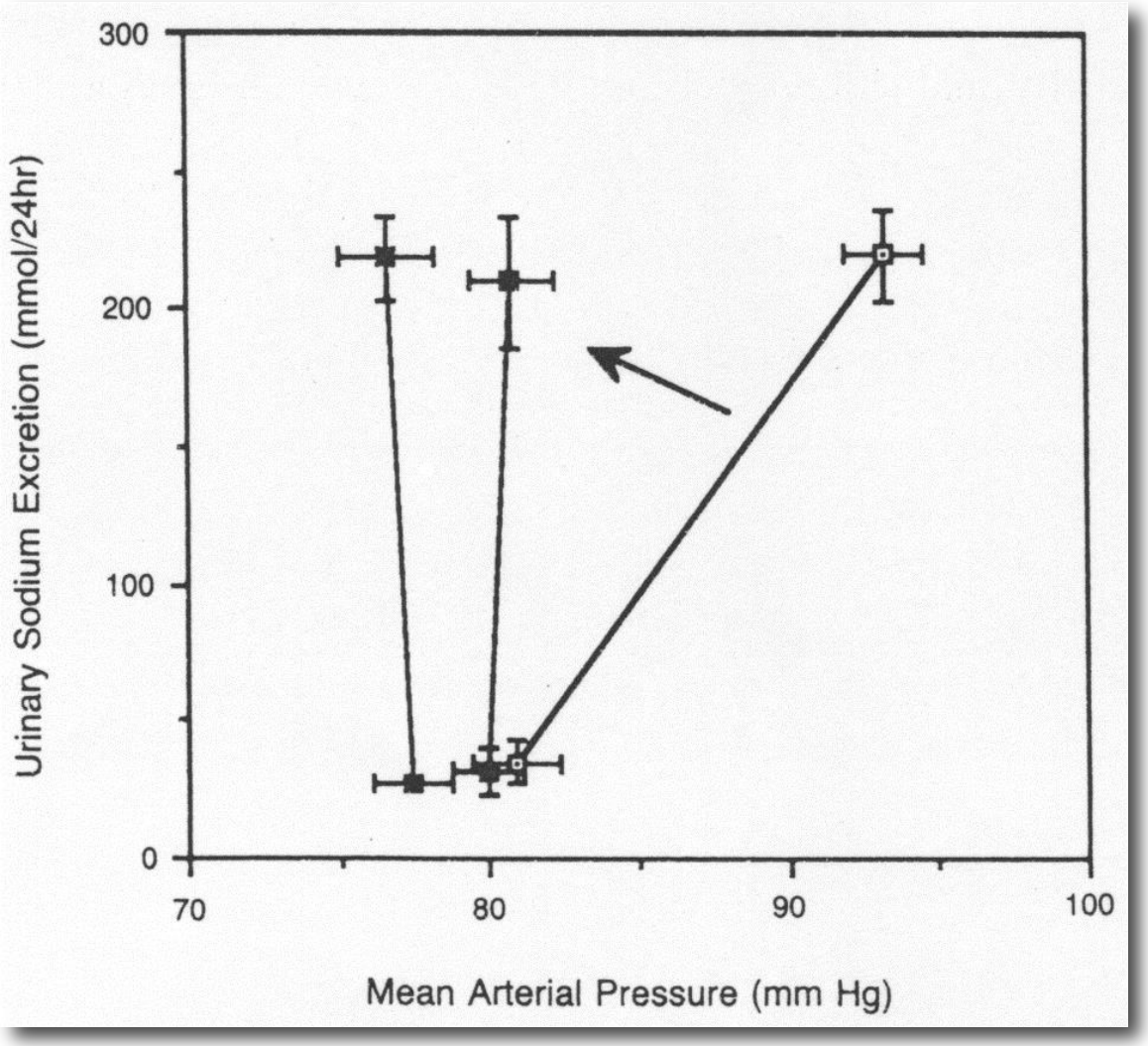
Sodium & potassium (observations)

- Rapport sodium / potassium urinaire chez 325 migrants urbains (Nairobi) et 267 ruraux de la même tribu (Luo):

Suivi	Hommes			Femmes		
(mois)	Ruraux	Urbains	P	Ruraux	Urbains	P
0	2.5	4.3	.001	2.8	4.0	.001
6	2.3	4.8		2.5	3.8	
12	2.3	4.5		2.6	3.9	
24	2.8	4.3		2.7	3.9	

- (Poulter, BMJ 1990, 300:967-72)

Excès pondéral (interventions)



(Rocchini, NEJM 1989)

Cohortes de sujets normotendus

- Analyse longitudinale du risque d'HTA chez l'adulte
- (TOHP-1): 140 Noirs et 237 Blancs suivis 7 ans.
-
-

	Blancs	Noirs
Hommes (%)	27.9	73.4
Age (ans)	43.6	44.1
IMC (kg/m2)	27.5	27.0
UNa/K	3.85	3.79
PAS (mmHg)	121.9	122.8
PAD (mmHg)	83.1	83.3
HT suivi: n (%)	36 (25.7)	60 (25.3)

Glomérule & sensibilité au sel

↗ Résistances préglomérulaires

↘ Coefficient d'UF

↗ Réabsorption tubulaire Na

Déplacement à droite de la relation pression/natriurèse

Altération de la relation pression/natriurèse

Hypertension résistante au sel

Hypertension sensible au sel

Pression glomérulaire normale/basse

Pression glomérulaire élevée

(d'après Kimura, 1993)

Hémodynamique rénale

- Réponse du FPR au captopril chez 144 adultes normotendus Noirs (32) ou Caucasiens (82).

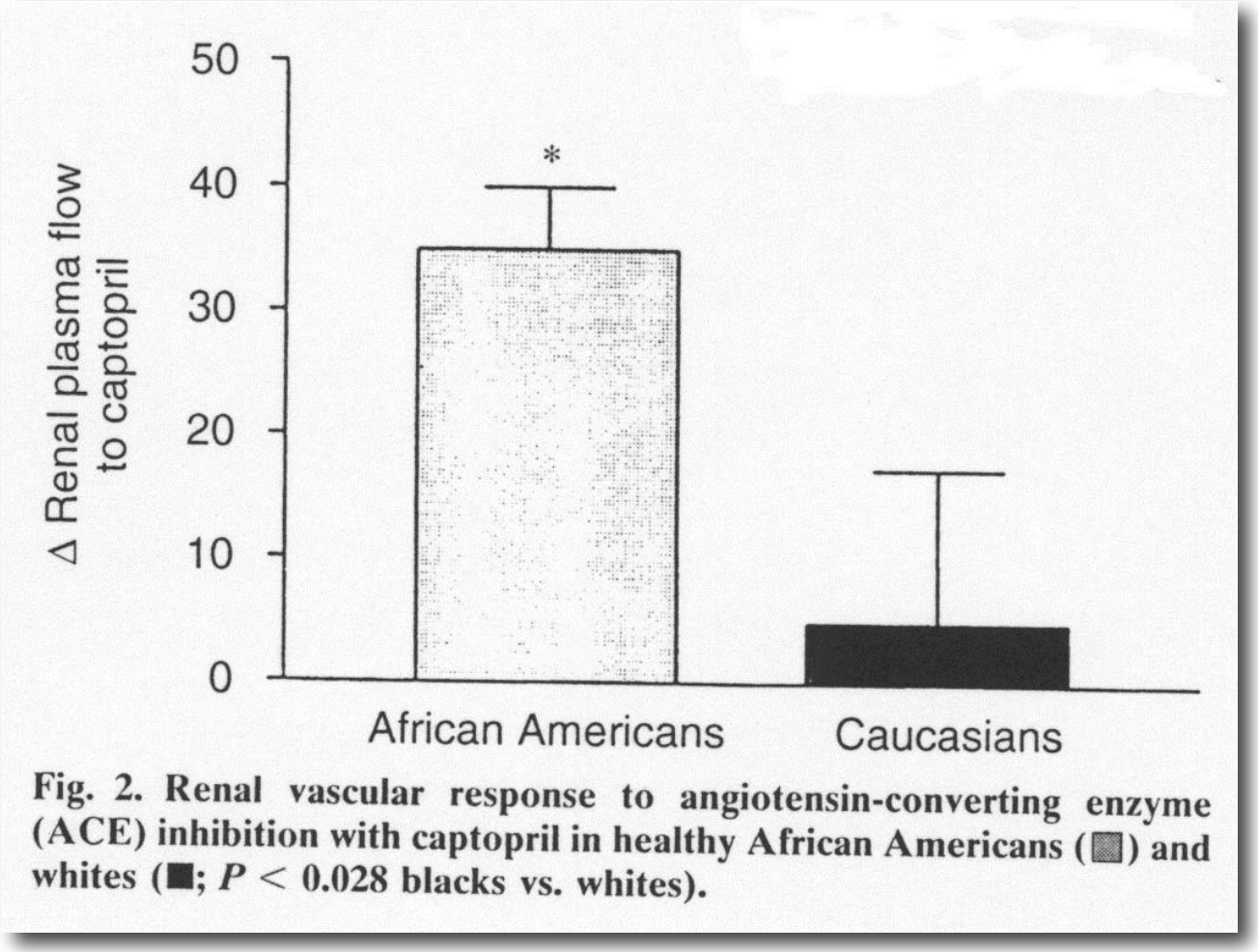


Fig. 2. Renal vascular response to angiotensin-converting enzyme (ACE) inhibition with captopril in healthy African Americans (▩) and whites (■; $P < 0.028$ blacks vs. whites).

- (Price, Kidney Int 2001, 59:1037-43)

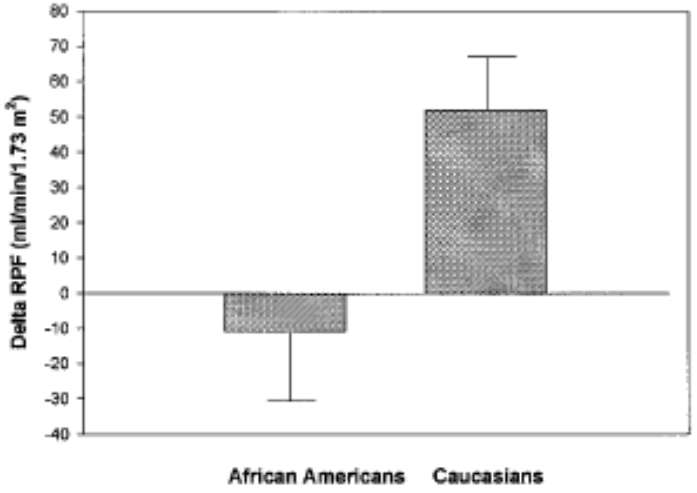
Hémodynamique rénale

Réponse au sel chez des hypertendus Noirs (19) ou Blancs (22).

Subjects	Blacks	Whites
Low salt		
RPF, mL/min/1.73m²	586±21	585±16
GFR, mL/min/1.73m²	108±5.6	112±3.6
FF, %	18.4±0.7	18.5±0.8
High salt		
RPF, mL/min/1.73m²	575±24*	636±20†
GFR, mL/min/1.73m²	117±5.9	124±5.4
FF, %	19.6±0.8	18.4±0.6

RPF indicates renal plasma flow; GFR, glomerular filtration rate; FF, filtration fraction.

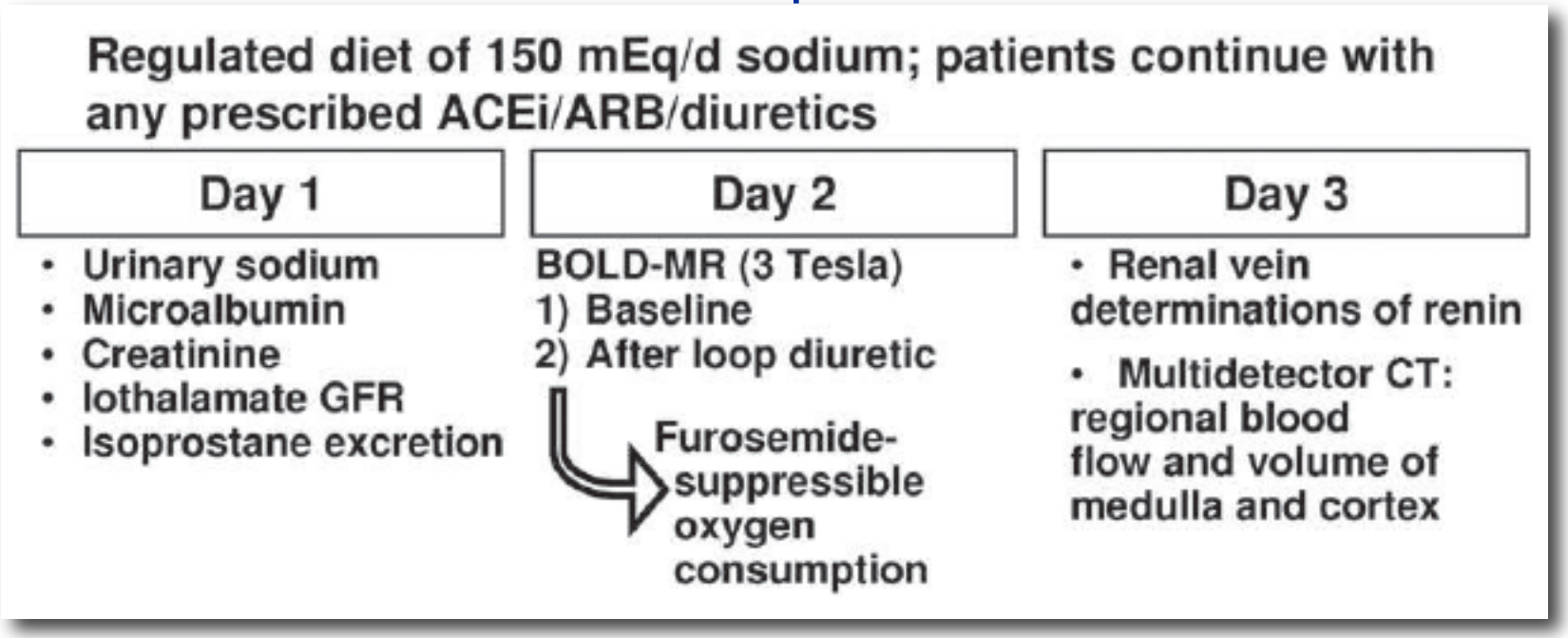
*P=0.033 vs whites on high salt; †P=0.05 vs whites on low salt.



(Price, Hypertension 2002, 40:186-189)

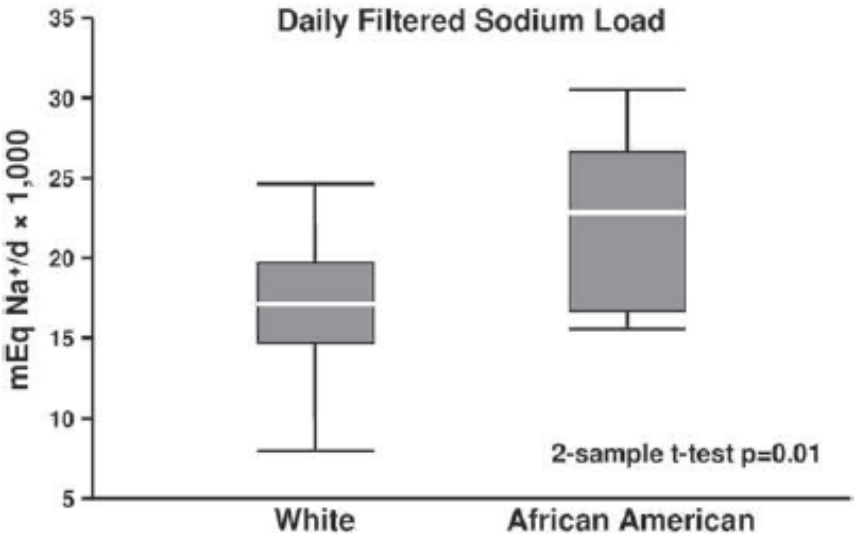
Hypoxie médullaire

«We hypothesized that increased glomerular filtration would beassociated with increased metabolic work of solute transport, potentially magnifying medullary hypoxia due to oxygen consumption.»



Textor SC, Gloviczki ML, Flessner MF, Calhoun DA, Glockner J, Grande JP, et al. Association of filtered sodium load with medullary volumes and medullary hypoxia in hypertensive African Americans as compared with whites. Am J Kidney Dis. 2012 Feb;59(2):229–37.

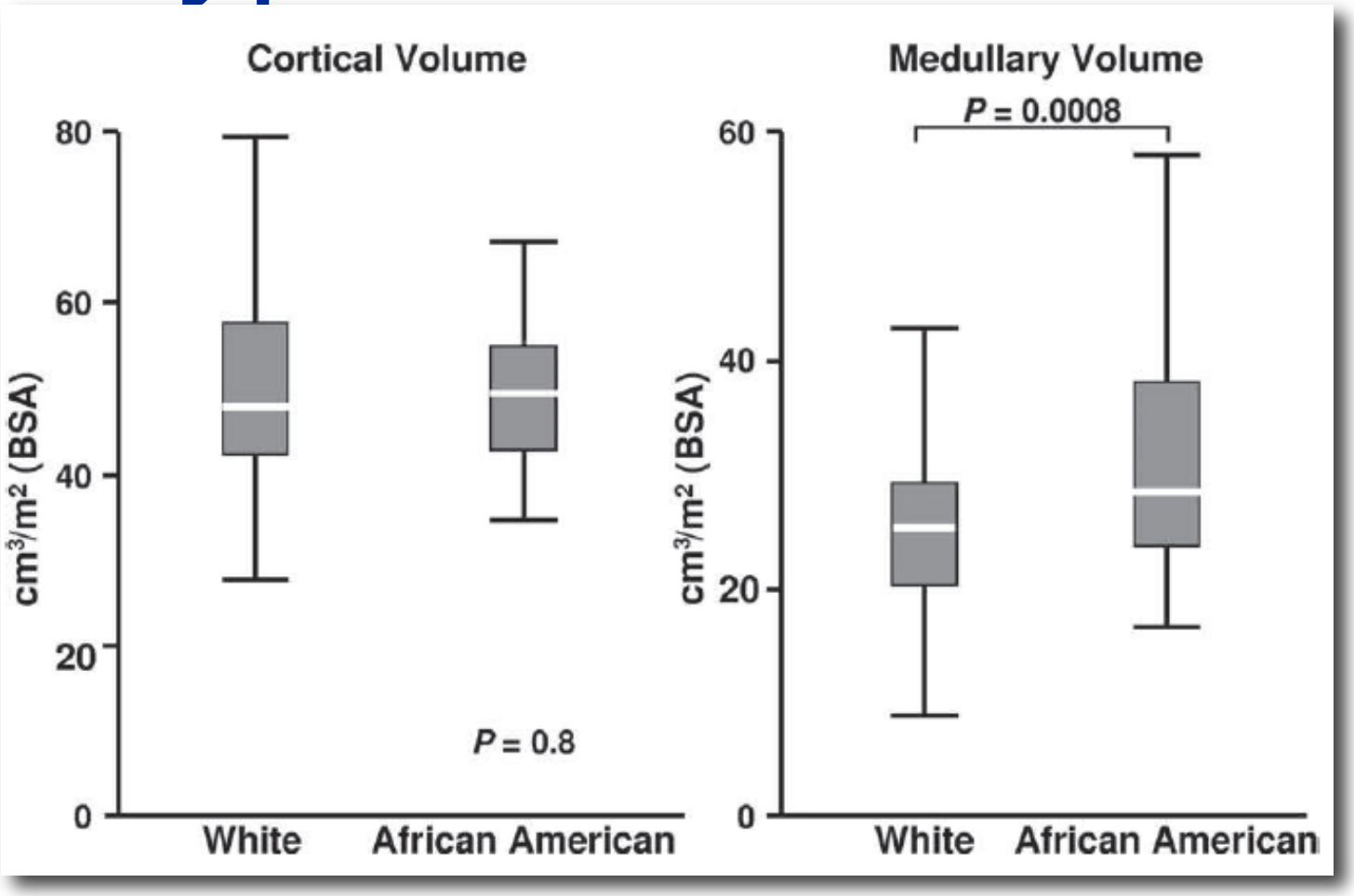
Hypoxie médullaire



	Whites (n = 29; 58 kidneys)	African Americans (n = 20; 40 kidneys)	ANOVA *P*
Kidney volume			
Total volume (cm^3/m^2)	75 ± 15	83 ± 18	0.02
Cortex (cm^3/m^2)	49.8 ± 11.9	50.6 ± 10.0	0.7
Medulla (cm^3/m^2)	25.1 ± 7.4	32.3 ± 11.2	<0.001
Tissue perfusion			
Cortex (mL/min/cm^3 tissue)	3.48 ± 1.04	3.97 ± 0.83	0.02
Medulla (mL/min/cm^3 tissue)	1.33 ± 0.49	1.41 ± 0.28	0.4
Blood flow[a]			
Total renal (mL/min/m^2)	207 ± 78	248 ± 73	0.02
Cortex (mL/min/m^2)	174 ± 72	204 ± 66	0.05
Medulla (mL/min/m^2)	32 ± 23	45 ± 17	<0.001
Venous Po_2 (mm Hg)	60 ± 9	64 ± 11	0.08

Textor SC, Gloviczki ML, Flessner MF, Calhoun DA, Glockner J, Grande JP, et al. Association of filtered sodium load with medullary volumes and medullary hypoxia in hypertensive African Americans as compared with whites. Am J Kidney Dis. 2012 Feb;59(2):229–37.

Hypoxie médullaire



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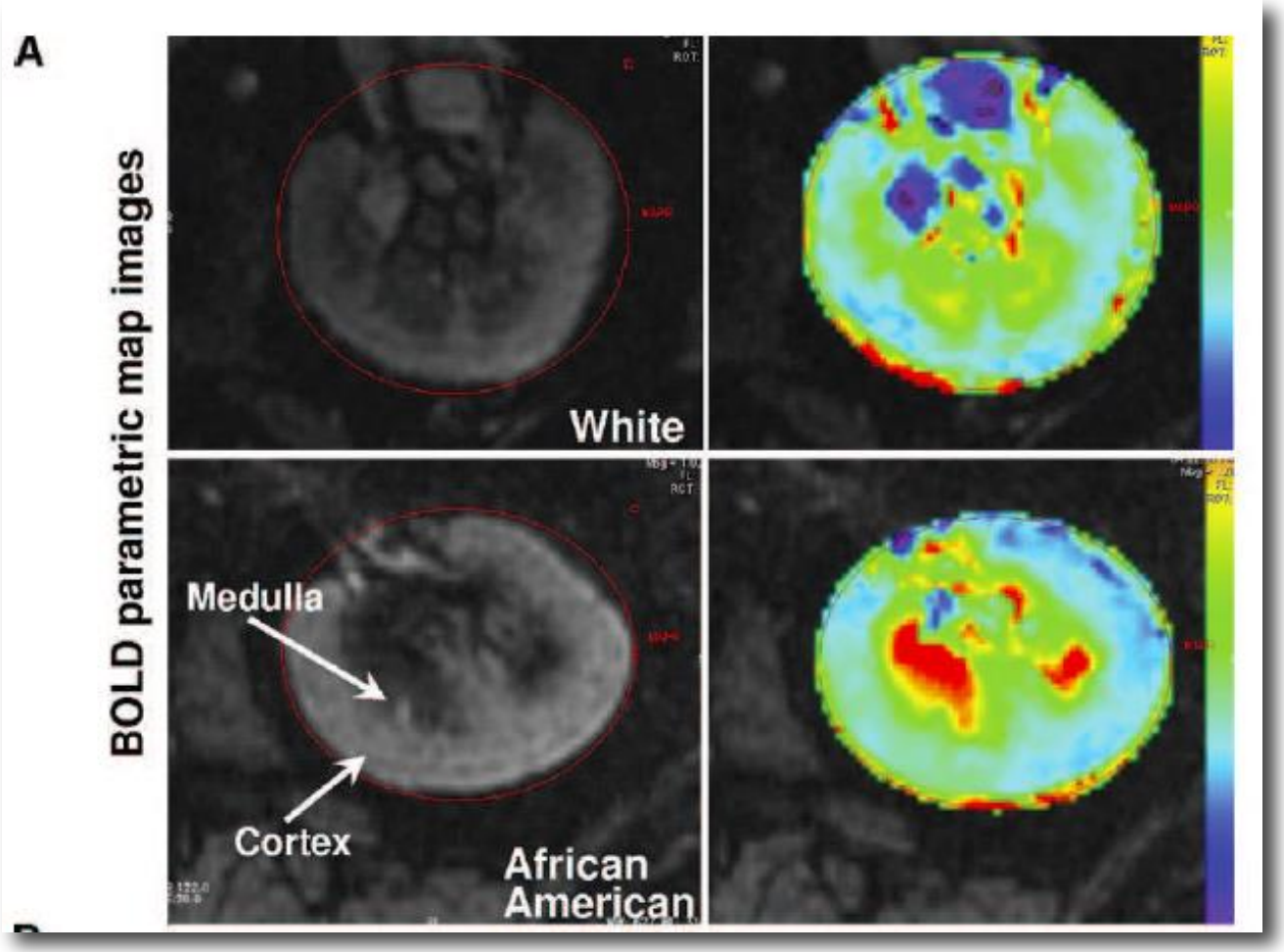

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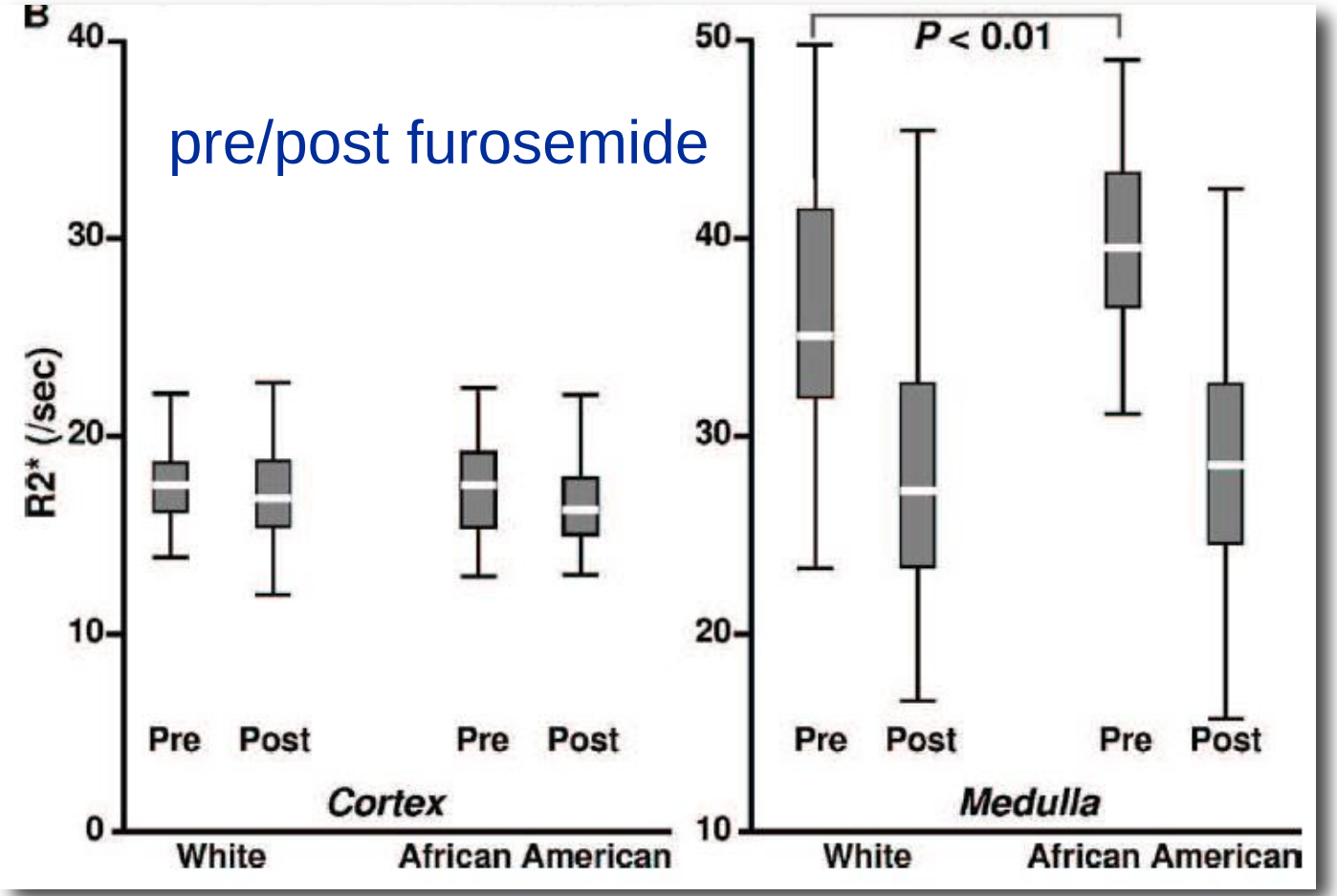

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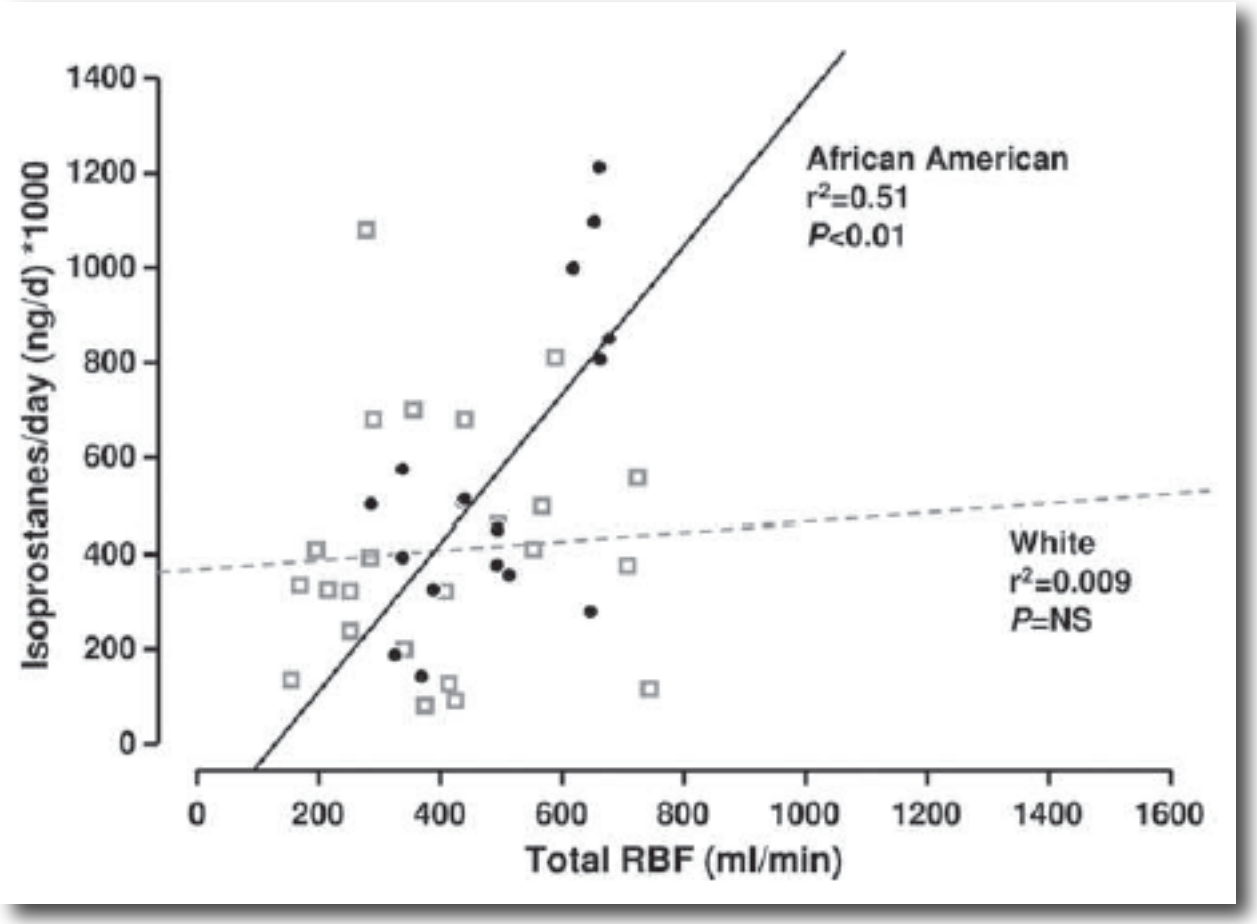

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Et la vitamine D ?

Variable	Race/ethnicity			p-value†
	Black (N = 331)	Hispanic (N = 362)	White (N = 421)	
Mean* 25(OH)D ± SD, (ng/mL‡)	25.0 ± 14.7 [a,b]	32.9 ± 13.9 [b]	37.4 ± 14.0	< 0.001
25(OH)D Quartiles (ng/mL)				< 0.001
Q1: 25(OH)D ≤ 20.8	44.4 [a,b]	23.1 [b]	11.4	
Q2: 20.8 < 25(OH)D ≤ 31.3	25.6	24.5	27.2	
Q3: 31.3 < 25(OH)D ≤ 42.7	18.2	30.8	28.5	
Q4: 25(OH)D > 42.7	11.7	21.6	32.9	

(Hannan MT, et al, JCEM, 2008, 93:40-46)

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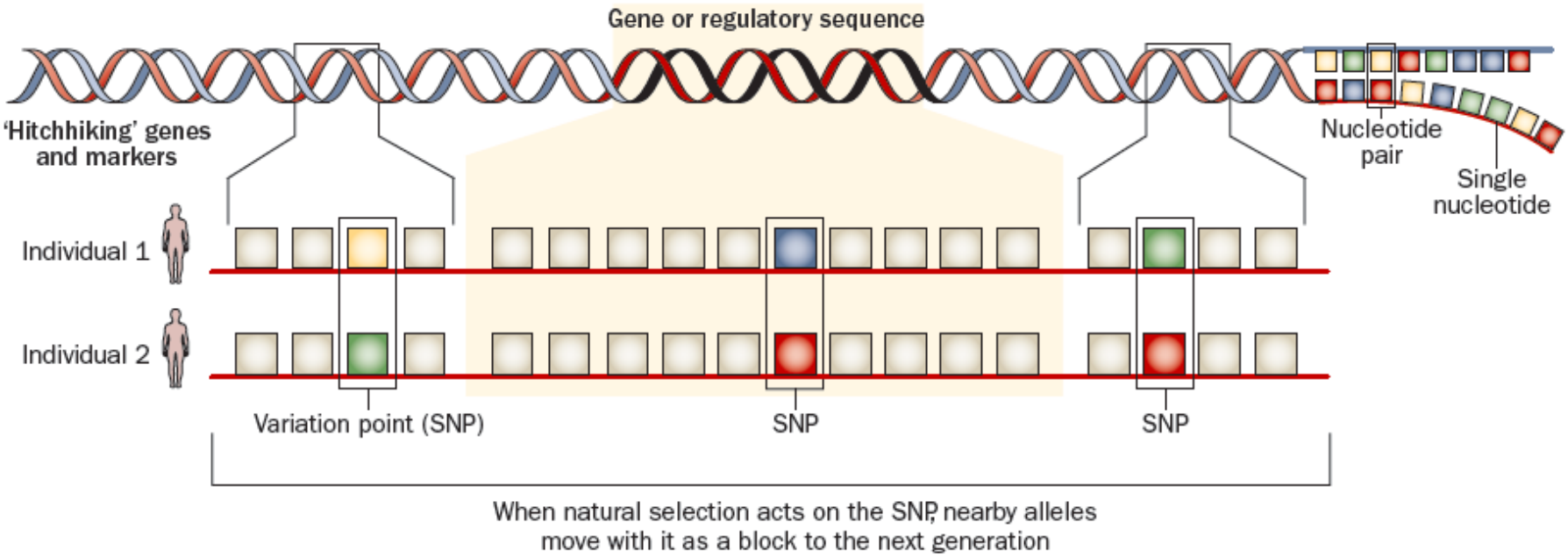
The population genetics of chronic kidney disease: insights from the *MYH9–APOL1* locus

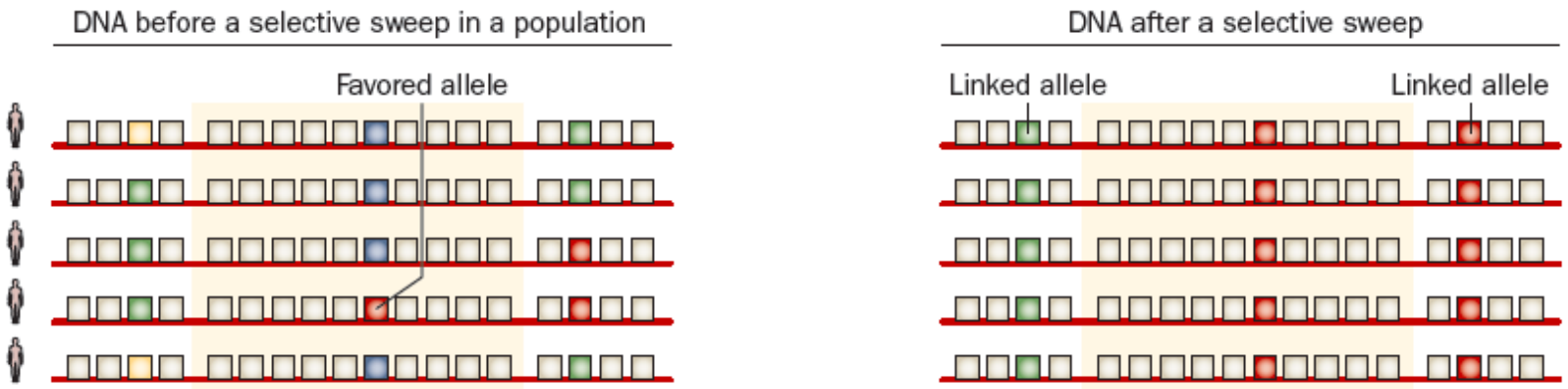
Saharon Rosset, Shay Tzur, Doron M. Behar, Walter G. Wasser and Karl Skorecki

Abstract | Many rare kidney disorders exhibit a monogenic, Mendelian pattern of inheritance. Population-based genetic studies have identified many genetic variants associated with an increased risk of developing common kidney diseases. Strongly associated variants have potential clinical uses as predictive markers and may advance our understanding of disease pathogenesis. These principles are elegantly illustrated by a region within chromosome 22q12 that has a strong association with common forms of kidney disease. Researchers had identified DNA sequence variants in this locus that were highly associated with an increased prevalence of common chronic kidney diseases in people of African ancestry. Initial research concentrated on *MYH9* as the most likely candidate gene; however, population-based whole-genome analysis enabled two independent research teams to discover more strongly associated mutations in the neighboring *APOL1* gene. The powerful evolutionary selection pressure of an infectious pathogen in West Africa favored the spread of *APOL1* variants that protect against a lethal form of African sleeping sickness but are highly associated with an increased risk of kidney disease. We describe the data sources, process of discovery, and reasons for initial misidentification of the candidate gene, as well as the lessons that can be learned for future population genetics research.

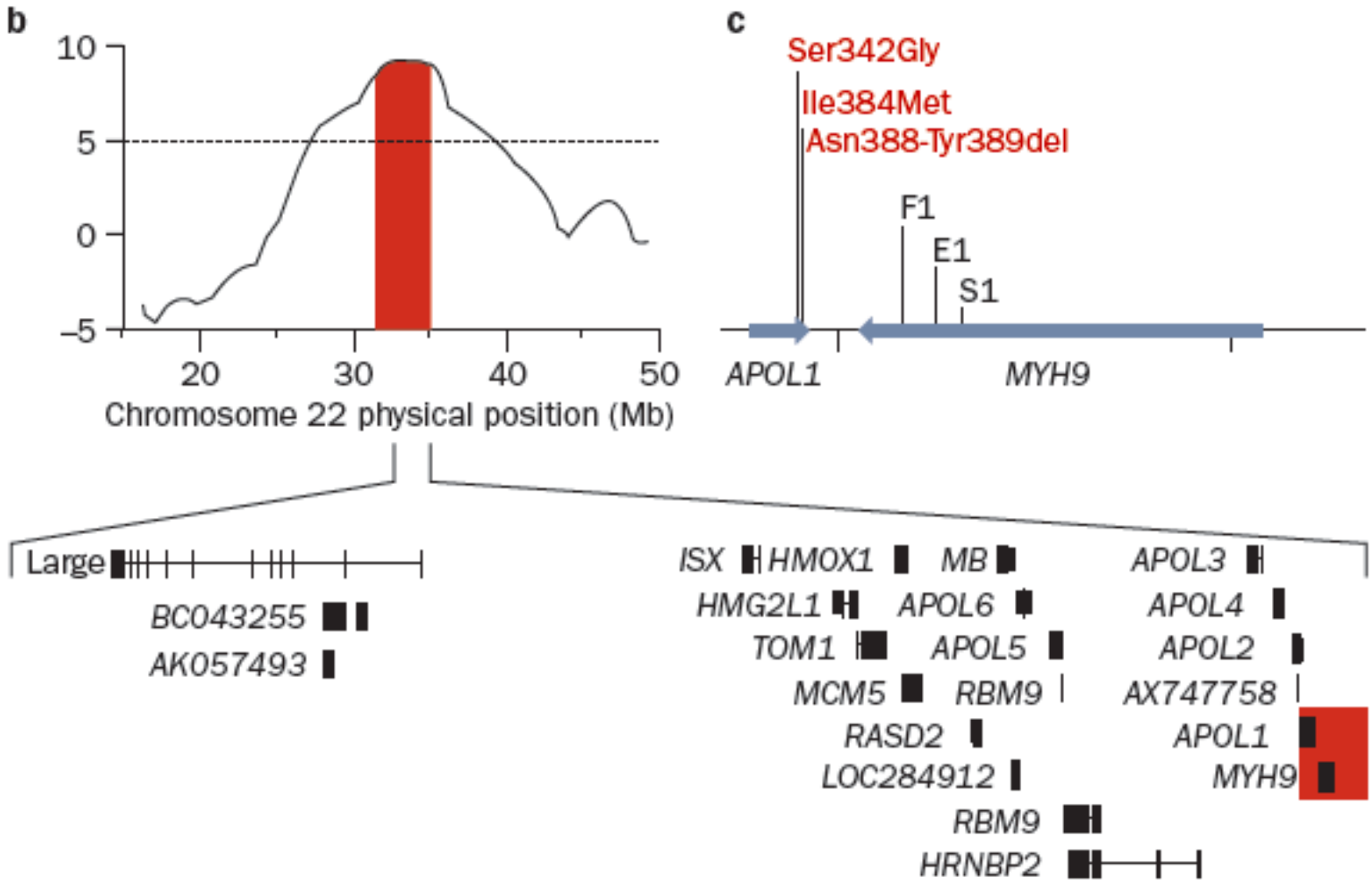
Rosset, S. *et al*. *Nat. Rev. Nephrol*. advance online publication 3 May 2011; doi:10.1038/nrneph.2011.52

Génétique


Gene or regulatory sequence
'Hitchhiking' genes and markers
Nucleotide pair
Single nucleotide
Individual 1
Individual 2
Variation point (SNP)
SNP
SNP
When natural selection acts on the SNP, nearby alleles move with it as a block to the next generation


DNA before a selective sweep in a population
Favored allele
DNA after a selective sweep
Linked allele
Linked allele

Génétique


b
10
5
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Chromosome 22 physical position (Mb)
c
Ser342Gly
Ile384Met
Asn388-Tyr389del
F1
E1
S1
APOL1
MYH9
Large
BC043255
AK057493
ISX
HMOX1
MB
HMG2L1
APOL6
TOM1
APOL5
MCM5
RBM9
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Génétique

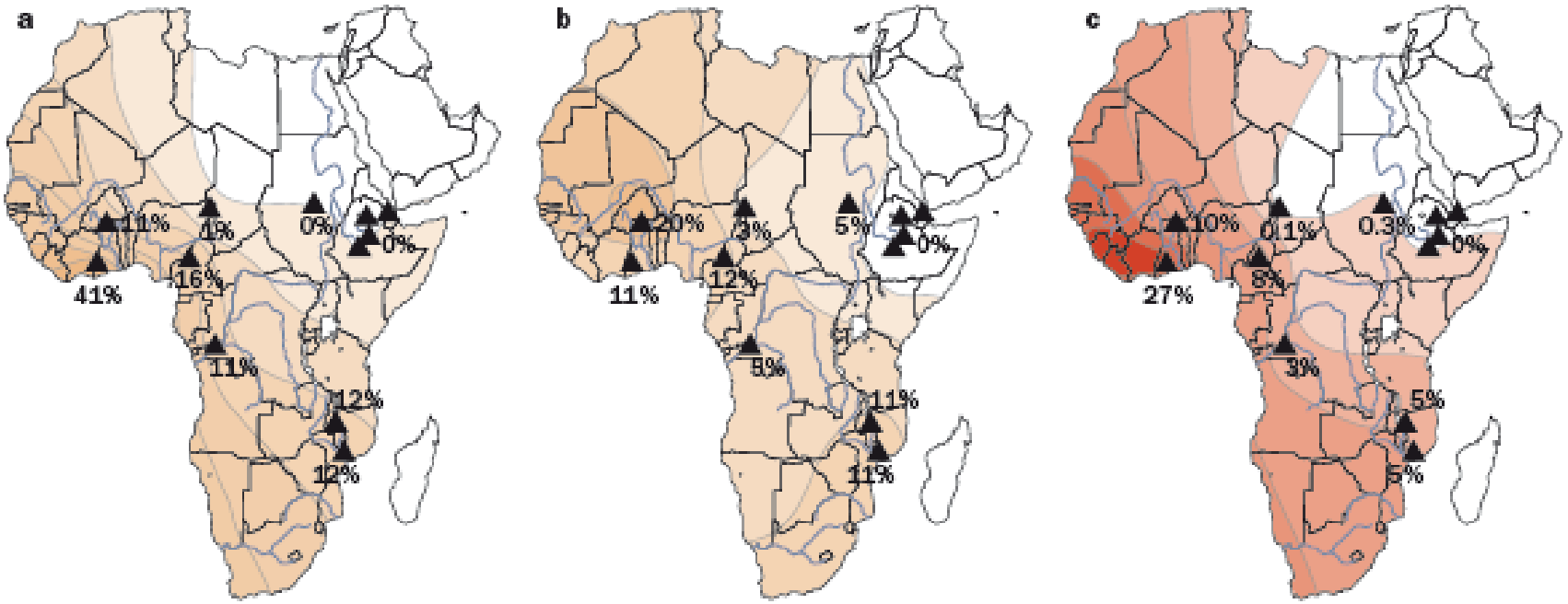
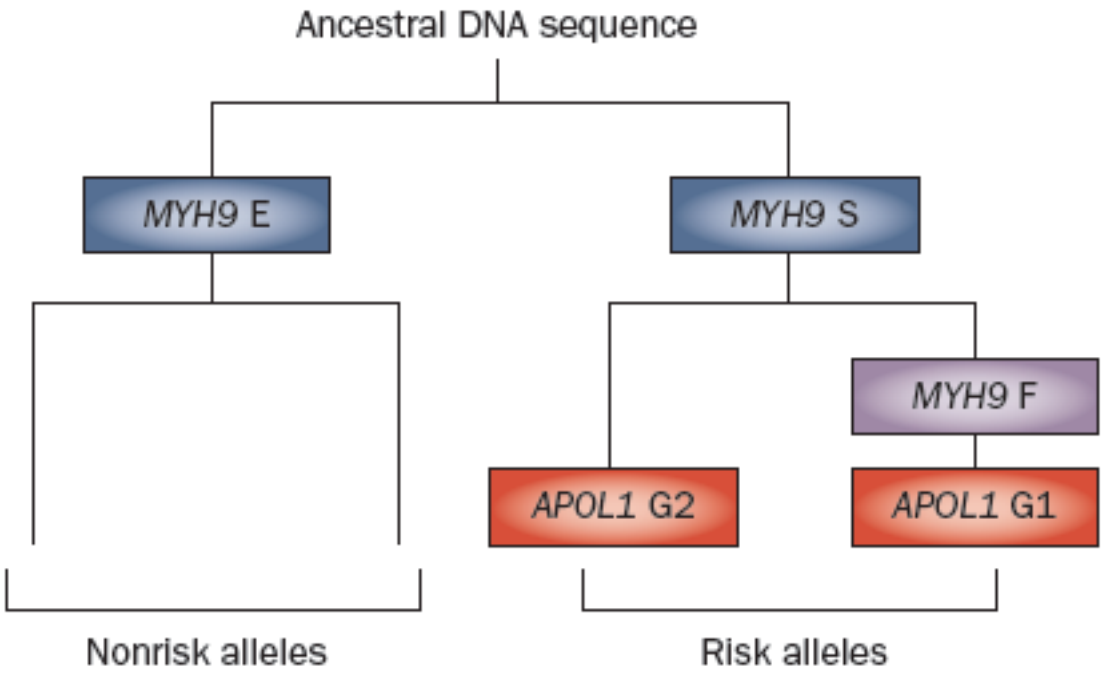


Figure 4 | Contour maps of allele frequency distributions of identified *APOL1* risk variants in a number of African countries. **a** | Distribution map for the *APOL1* Ser342Gly missense mutation (rs73885319), also termed G1. **b** | Distribution map for the *APOL1* 6 bp deletion (rs71785313), also termed G2. **c** | Contour map showing the frequency of the three risk genotypes G1G1, G2G2, and G1G2 combined. Maps were based on the genotypes of 12 African populations ($n=676$), whose locations are marked by triangles. Data were extrapolated for regions that were not sampled. Colors denote allele frequencies. Notably, in Ethiopians ($n=304$), risk allele frequencies for both G1 and G2 are zero. This distribution is consistent with the observed absence of HIV-associated nephropathy among Ethiopians.[105]

Rosset S, Tzur S, Behar DM, Wasser WG, Skorecki K. The population genetics of chronic kidney disease: insights from the MYH9-APOL1 locus. Nat Rev Nephrol. 2011 May 3.

Génétique


Ancestral DNA sequence
MYH9 E
MYH9 S
MYH9 F
APOL1 G2
APOL1 G1
Nonrisk alleles
Risk alleles

see commentary on page 6

Apolipoprotein L1 gene variants associate with hypertension-attributed nephropathy and the rate of kidney function decline in African Americans

Michael S. Lipkowitz[1,16], Barry I. Freedman[2,16], Carl D. Langefeld[3], Mary E. Comeau[3], Donald W. Bowden[4], W.H. Linda Kao[5], Brad C. Astor[6], Erwin P. Bottinger[7], Sudha K. Iyengar[8], Paul E. Klotman[9], Richard G. Freedman[2], Weijia Zhang[10], Rulan S. Parekh[11,12], Michael J. Choi[13], George W. Nelson[14], Cheryl A. Winkler[14,16], Jeffrey B. Kopp[15,16] and the AASK Investigators

«In recessive models, APOL1 risk variants were significantly associated with kidney disease in all cases compared to controls with an odds ratio of 2.57. In AASK cases with more advanced disease, such as a baseline urine protein to creatinine ratio over 0.6 g/g or a serum creatinine over 3mg/dl during follow-up, the association was strengthened with odds ratios of 6.29 and 4.61, respectively. APOL1 risk variants were consistently associated with renal disease progression across medication classes and blood pressure targets. Thus, kidney disease in AASK participants was strongly associated with APOL1 renal risk variants.»

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Causes de décès

Autopsies systématiques de sujets décédés de cause cardiovasculaire (NYC, 1991): n=587.

	Blancs (n=273)	Noirs (314)
Age de décès (ans)		
- hommes	51.2	51.7
- femmes	61.5	54.7*
Cause de décès		
Athérome coronaire (%)	64	38
Hypertension (%) (HVG, ICC, AVC, NHT,…)	23	42

(Onwuanyi, Hypertension 1998, 31: 1070-76)

Risque coronarien

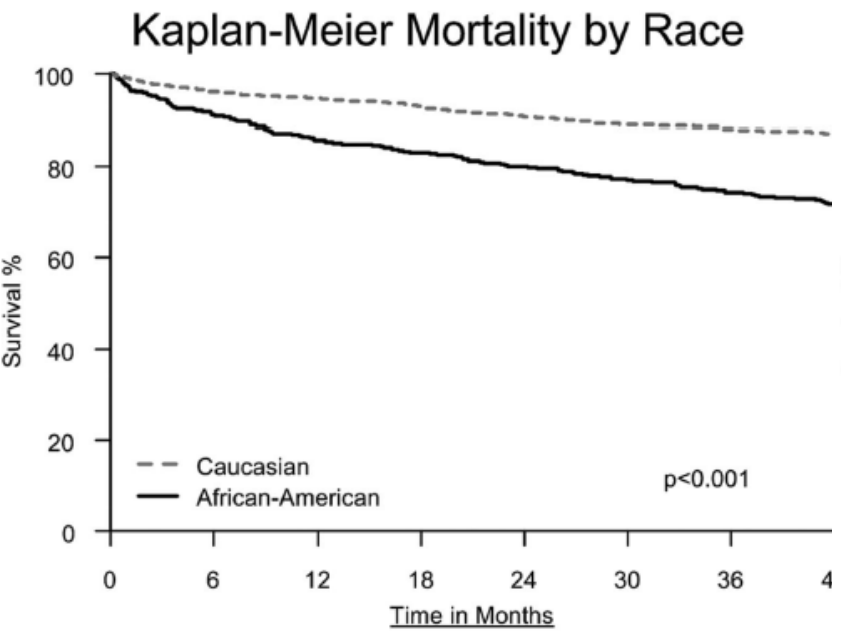
- Atherosclerosis Risk in Communities Study
- (ARIC, 1987-97): 15792 sujets, 45-64 ans

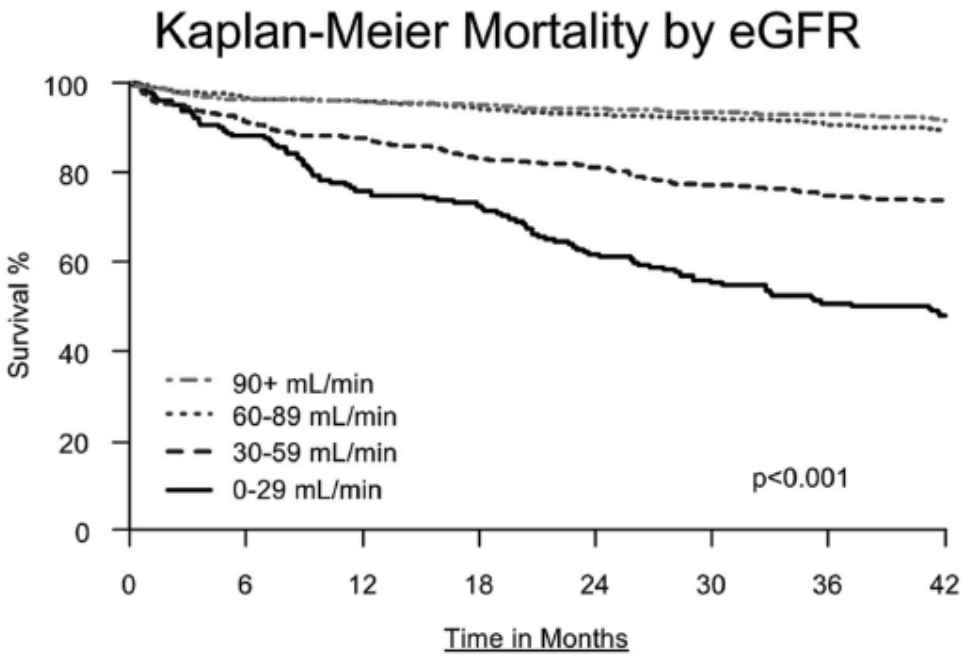
- Evènements coronariens: IdM, revascularisation, ou décès.

	Femmes		Hommes	
	Noirs	Blancs	Noirs	Blancs
Effectif	2298	5686	1396	4682
Ev. Coro.	5.1%	4.0%	10.6%	12.5%
Rapport de risque (ajusté sur l'âge) lié à:				
- Hypertension	5.3	2.7	2.0	1.8
- LDL chol.	1.3	1.4	1.2	1.4
- Diabète	2.3	5.1	1.7	2.4
- Tabac	2.6	2.9	1.7	1.8
- BMI (+ 1 SD)	1.2	1.2	1.0	1.1

- (Jones, Arch Intern Med 2002, 162: 2565-2571)

Risque coronarien





Wetmore et al. Association of decreased glomerular filtration rate with racial differences in survival after acute myocardial infarction. Clinical journal of the American Society of Nephrology : CJASN (2011) vol. 6 (4) pp. 733-40

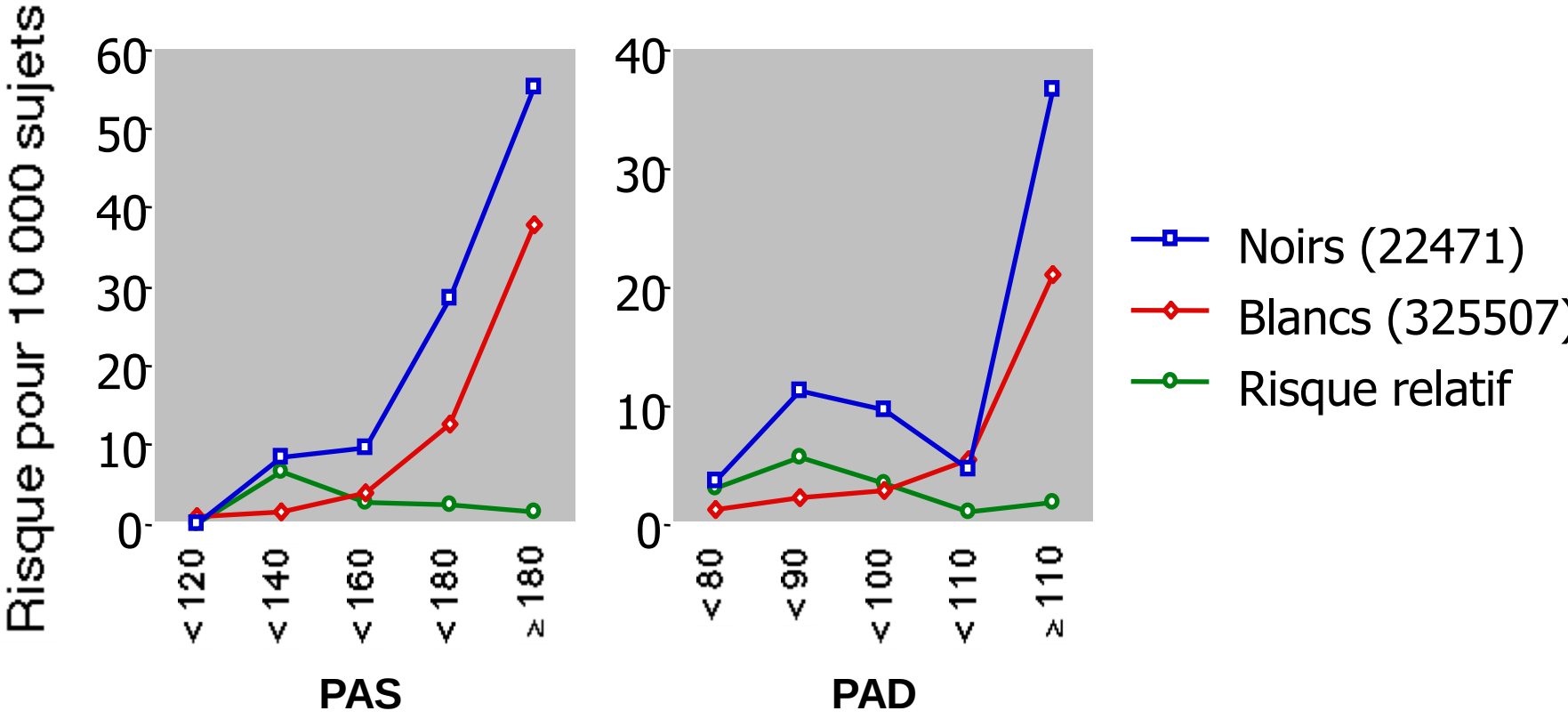
Risque vasculaire cérébral

	Blancs	Noirs
NHANES I[1]		
Population	7814	1298
RR ajusté sur âge, PA, diabète:		
- hommes	1	1.1
- femmes	1	1.4
Cincinatti[2]		
Population	1086462	171718
Premier AVC hémorragique	221	45
RR hémorr. méningée	1	2.1*
RR hémorr. intracérébrale	1	1.4

(1. Kittner, JAMA 1990, 264:1267-70;
2. Broderick, NEJM 1992, 326:733-6)

Risque de NHT: pression

MRFIT : mortalité de cause rénale chez 347978 hommes, suivis 12 ans.



(Flack JM et al., Am J Kidney Dis 1993, 4 (suppl. 1), 31-40)

Risque de NHT: cohortes

Evolution sous traitement:
11912 vétérans (≈ 50 ans) traités, suivis 15 ans.

Fréquence de l'insuffisance rénale terminale :

	PAS ≤ 136	PAS > 136	Total	
			Noirs	Blancs
Effectif	4864	4780	5730	6182
IRC - T	67	132	163	82*
	(1,38%)	(2,76%)	(2,84%)	(1,33%)

(Perry HM et al., Hypertension 1995, 25, 587-594)

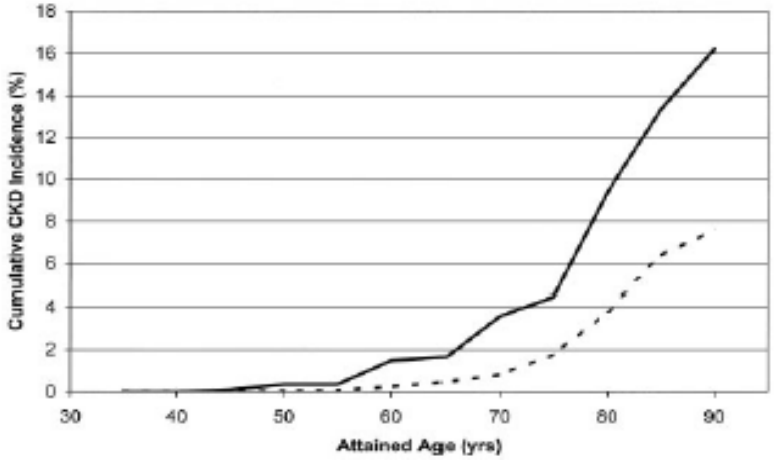
Epidémiologie de l'IRC: NHANES II (1976-80)

- Adultes, 30-74 ans, suivi moyen 14 ans:

	Noirs		(RR)	Blancs	
	N	N/$10^{5(*)}$		N	N/$10^{5(*)}$
Effectif	894			7664	
IRCT traitée	12	97	(8.9)	25	11
IRC (diabète, HTA)	13	83	(12)	28	7
IRC (toutes causes)	33	222	(2.7)	139	84

- (*) ajusté sur l'âge

Epidémiologie et risque d'IRCT



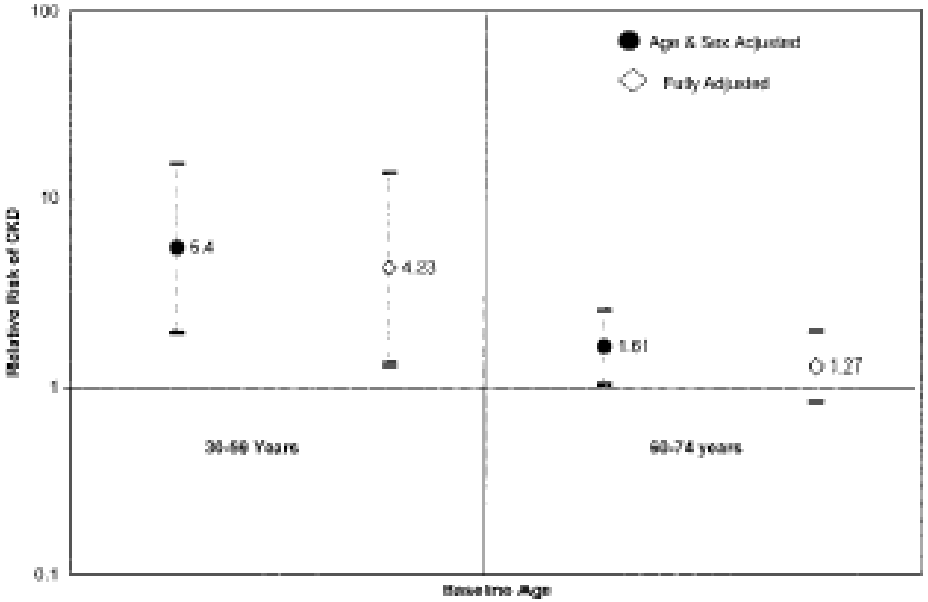


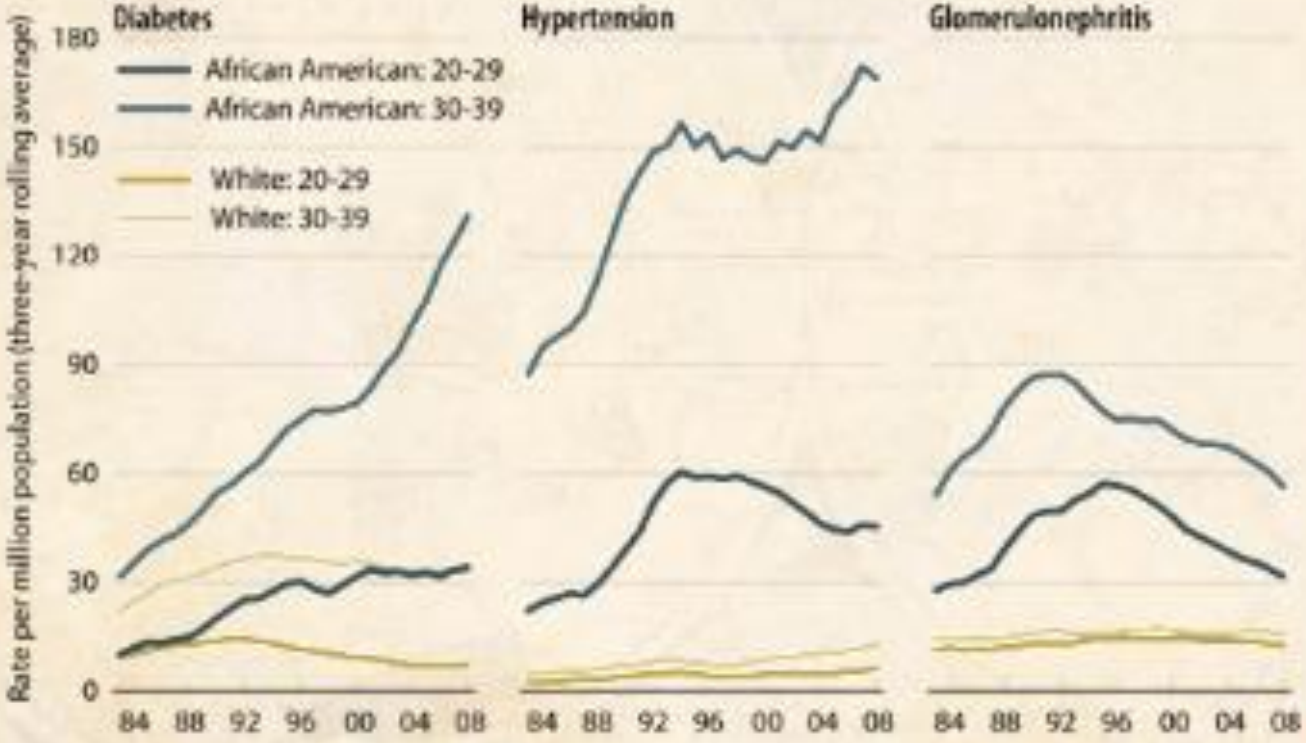
Table 3. Excess risk of CKD among African Americans *versus* whites in relation to potentially modifial risk factors[a]

Adjusted for	RR for African Americans (*versus* Whites)	Excess Risk Explained (%)[b]
Age and gender only	2.69 (1.50 to 4.82)	
Sociodemographic factors[c]	2.49 (1.33 to 4.67)	11.8
Lifestyle factors[d]	2.29 (1.31 to 4.01)	23.7
Clinical factors[e]	2.15 (1.18 to 3.92)	32.0
All risk groups[f]	1.95 (1.05 to 3.63)	43.8

(Tarver-Carr ME,
JASN 2002, 13: 2363-70)

Adjusted incident rates of ESRD, by age, cause of ESRD, & race

Figure 2.1 (Volume 2)

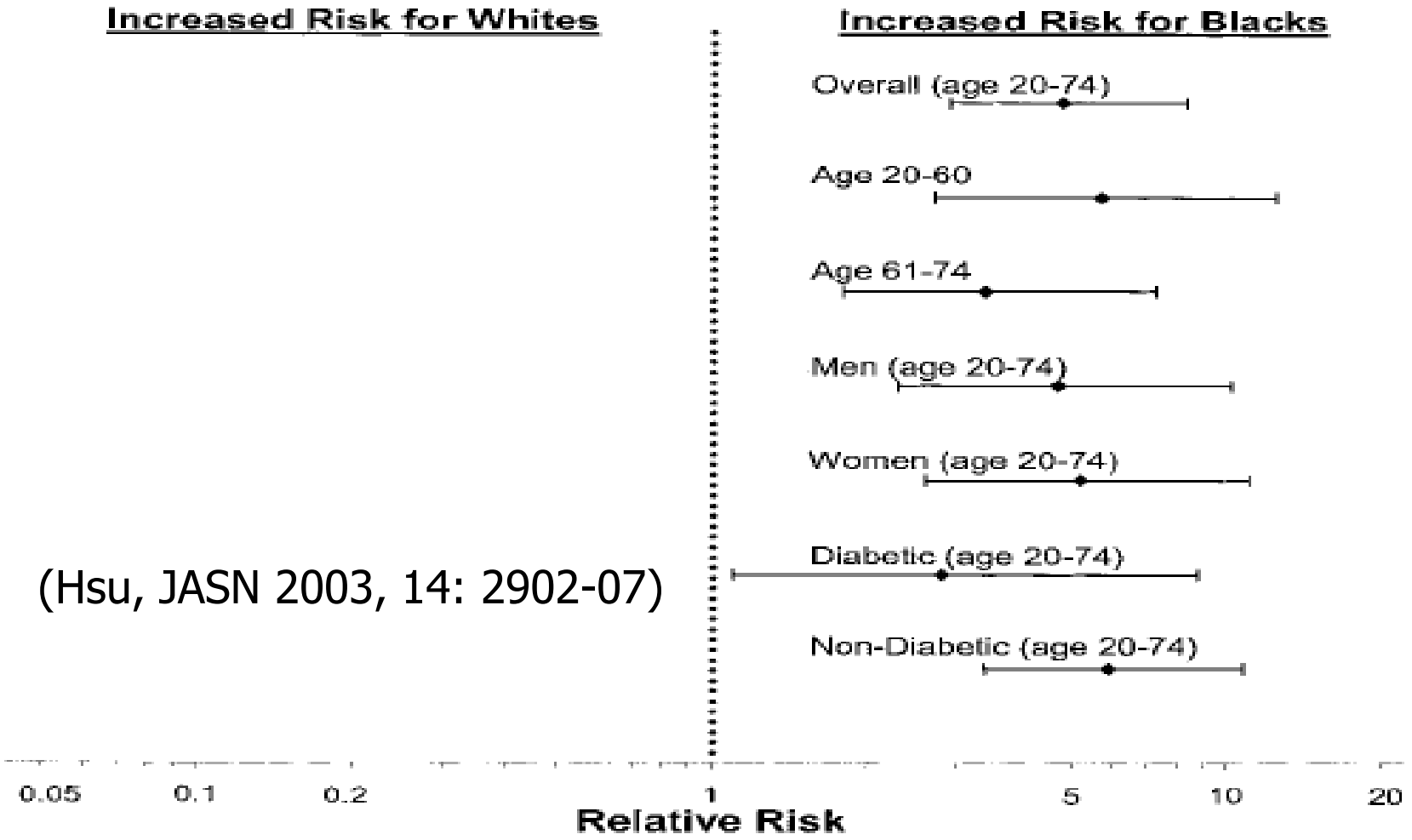


Three-year rolling average; adjusted for gender.

USRDS 2010 ADR

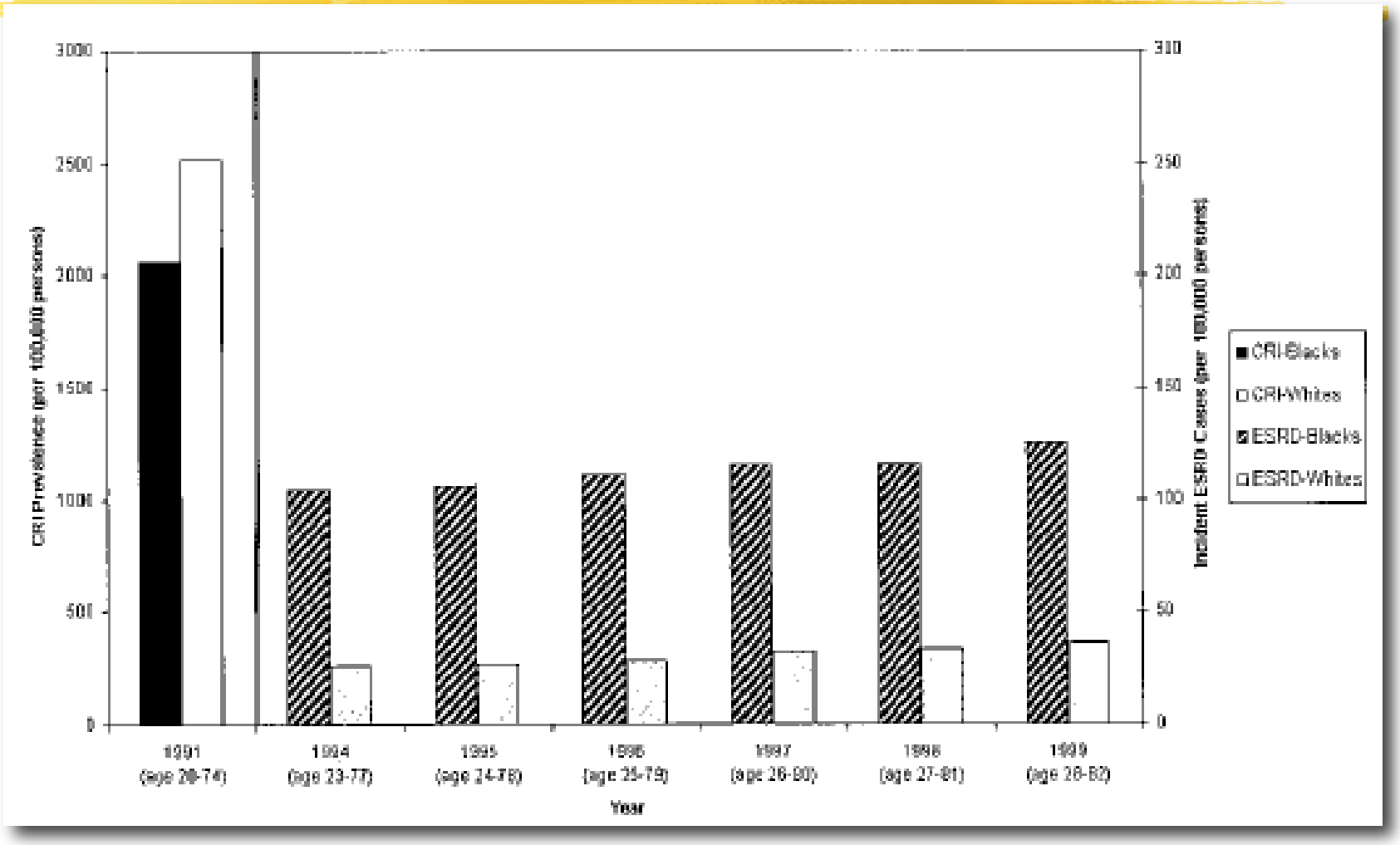
USRDS

Risque d'IRCT: NHANES III



(Hsu, JASN 2003, 14: 2902-07)

Risque d'IRCT vs IR: NHANES III



(Hsu, JASN 2003, 14: 2902-07)

Fonction rénale: HTA récente

- DFG et FPR chez des femmes hypertendues appariées sur l'âge (moyenne=46 ans):

Origine	Antilles	Métropole
	(n=21)	(n=21)
Cl. inuline (ml/min/1.73)	107.4	115.5
Cl. PAH (ml/min/1.73)	489.0	542.4*
Fraction filtrée (%)	22	21
Microalbumine (mg/j)	35.5	26.2

- (Mpio, AMCV 1999, 92:957-60)

Estimation du DFG

Table 3. Performance of the MDRD study equation according to ethnicity in the CKD-EPI group (33) (bias is median difference between estimated GFR and measured GFR)

	Estimated GFR <60 ml/min per 1.73 m^2			Estimated GFR >60 ml/min per 1.73 m^2		
	Sample	Median Difference (%)	P30	Sample	Median Difference (%)	P30
Overall	2874	−3	82	2630	−8.7	84
Caucasian and other	1668	−2.3	82	1799	−11.7	83
African American	1085	−3.4	82	643	−0.2	88

P30, percentage of subjects with an estimated GFR within 30% of measured GFR.

Delanaye et al. Are the creatinine-based equations accurate to estimate glomerular filtration rate in African American populations?. Clinical journal of the American Society of Nephrology : CJASN (2011) vol. 6 (4) pp. 906-12

Critères de diagnostic de NHT

Facteurs ethniques : diagnostic rétrospectif de NHT chez les dialysés aux USA :

Histoire clinique similaire	Noirs	Blancs
Nombre de dossiers	99	98
Nombre de diagnostics de néphropathie hypertensive	39 (39,4%)	27 (27,5%)

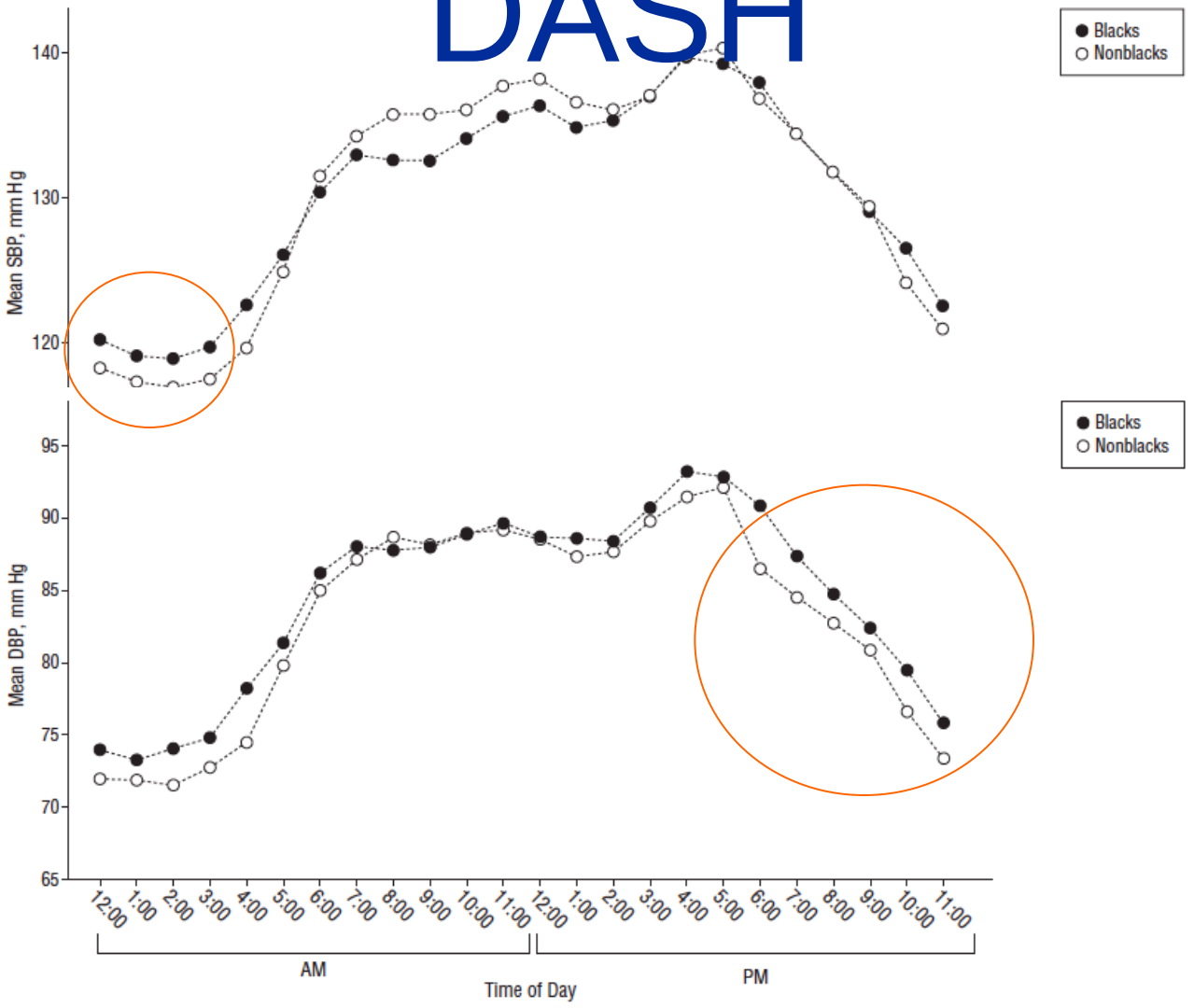
RR de diagnostic de NHT cause de l'IR terminale : 1,4

d'après Pernegger TV, Am J Epidemiol 1995, 141, 10-15.

Plan

- Epidémiologie
- Physiopathologie
- Génétique
- Profil de risque
- **Spécificités thérapeutiques**

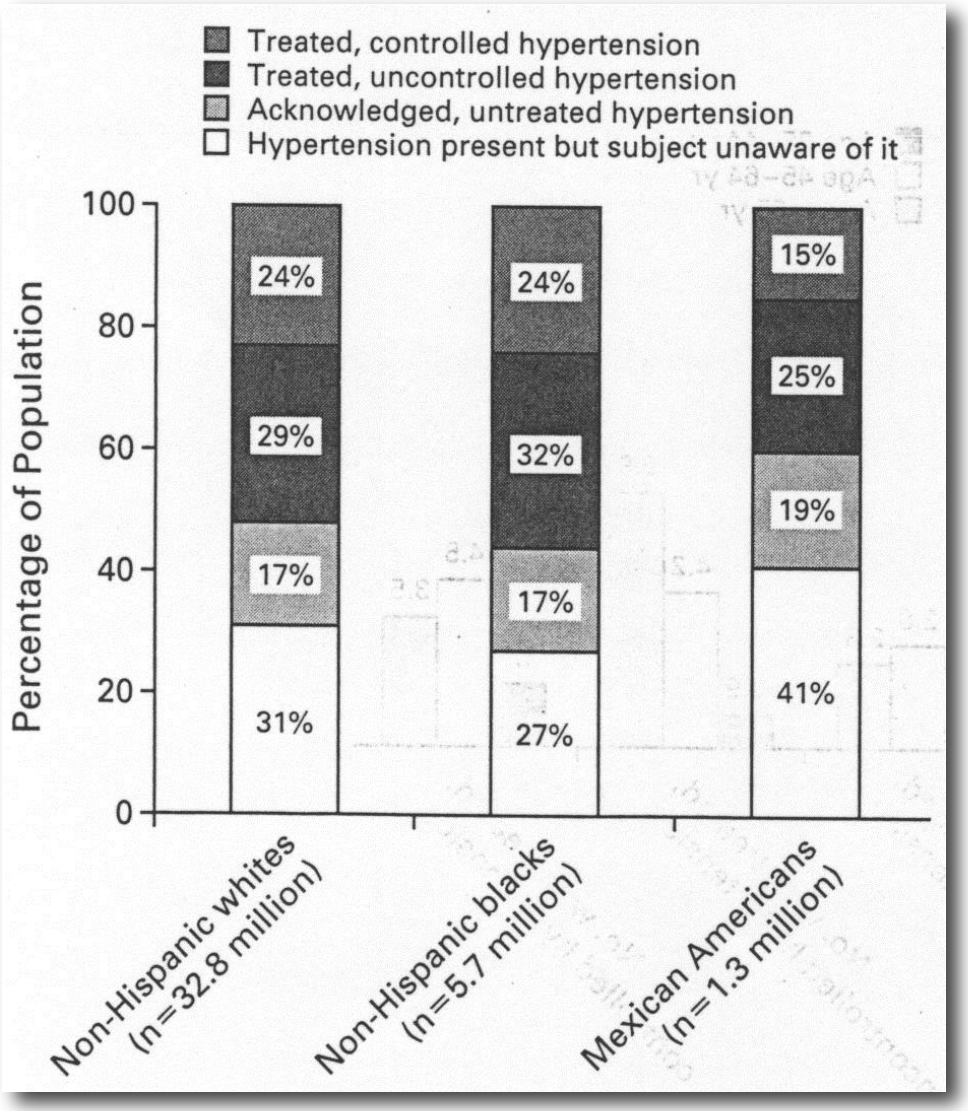
MAPA run-in étude DASH



(Jehn ML et al, Arch Int Med, 2008, 168:996-1002

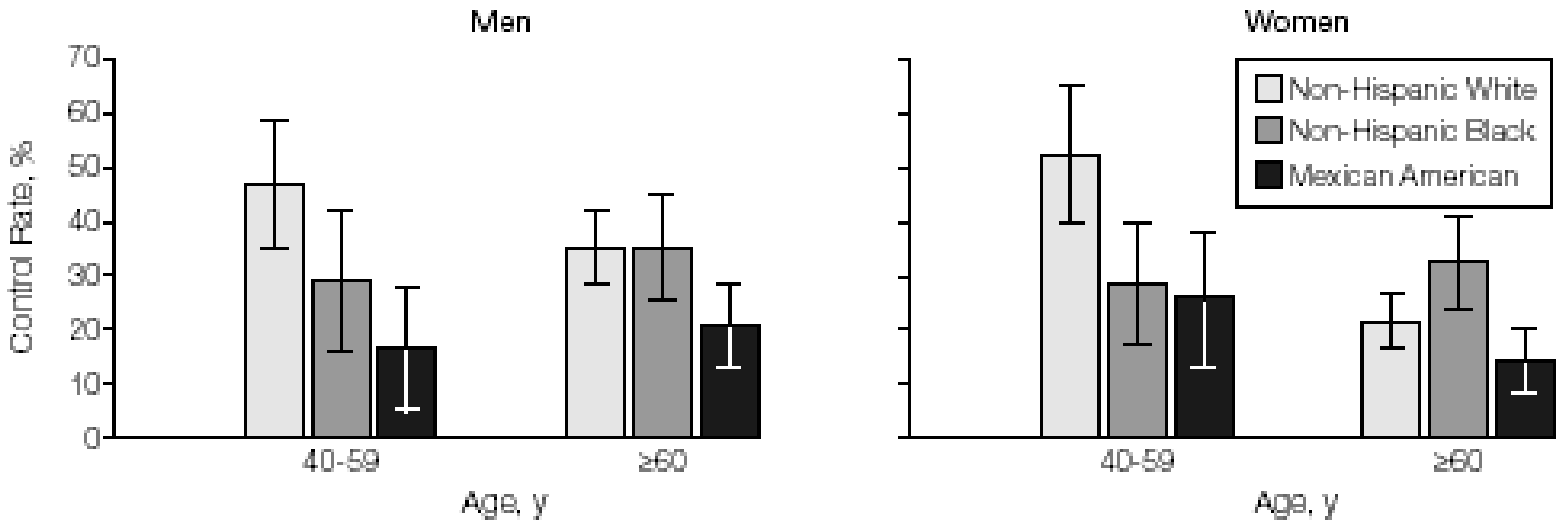
BP awareness and control

- NHANES III.
- 16095 subjects.
- BP > 140/90 mmHg.
- HTN is more frequent but BP control is similar
- (Hyman, NEJM 2001; 345: 479-86)



Epidémiologie USA 1988-2000

Figure 2. Overall Hypertension Control Rates in 1999-2000 by Age and Race/Ethnicity in Men and Women



Error bars indicate 95% confidence intervals. Data are weighted to the US population. For comparisons between racial/ethnic groups (with non-Hispanic whites as the referent), *P* values are as follows: for Mexican Americans, men aged 40 to 59 years, $P<.001$, men aged at least 60 years, $P=.003$, women aged 40 to 59 years, $P=.002$, and women aged at least 60 years, $P=.04$; for non-Hispanic blacks, men aged 40 to 59 years, $P=.02$, men aged at least 60 years, $P=.51$, women aged 40 to 59 years, $P=.003$, and women aged at least 60 years, $P=.98$.

(Hajjar, JAMA 2003, 290, 199-206)

Potassium (interventions): DASH

	Blancs (n=156)	Noirs (n=275)
Age (ans)	46	44
Femmes (%)	33	59
PAS (mmHg)	130.9	131.8
PAD (mmHg)	84.5	84.8
Hypertendus (%)	26	32
Obèses (%)		
- hommes	46	57
- femmes	54	66
Δ PAS (mmHg)	-3.3	-6.9
Δ PAD (mmHg)	-2.4	-3.7

(Svetkey, Arch Intern Med 1999, 159:285-93)

Sodium (intervention): DASH-2

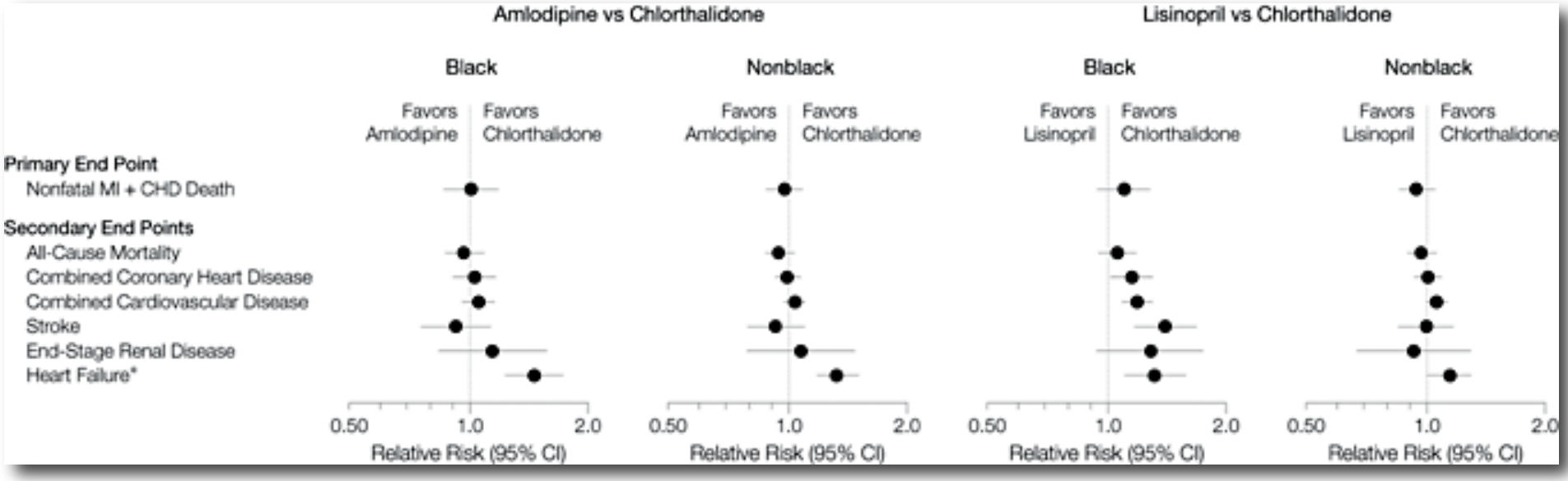
- Comparison high-salt (8 g/d) vs low-salt (3 g/d),
- 56% Black subjects

	Δ SBP (mmHg)
HTN	11.5
- Blacks	12.6*
- others	9.5
NTN	7.1
- Blacks	7.2
- Others	6.9

* p=0,007

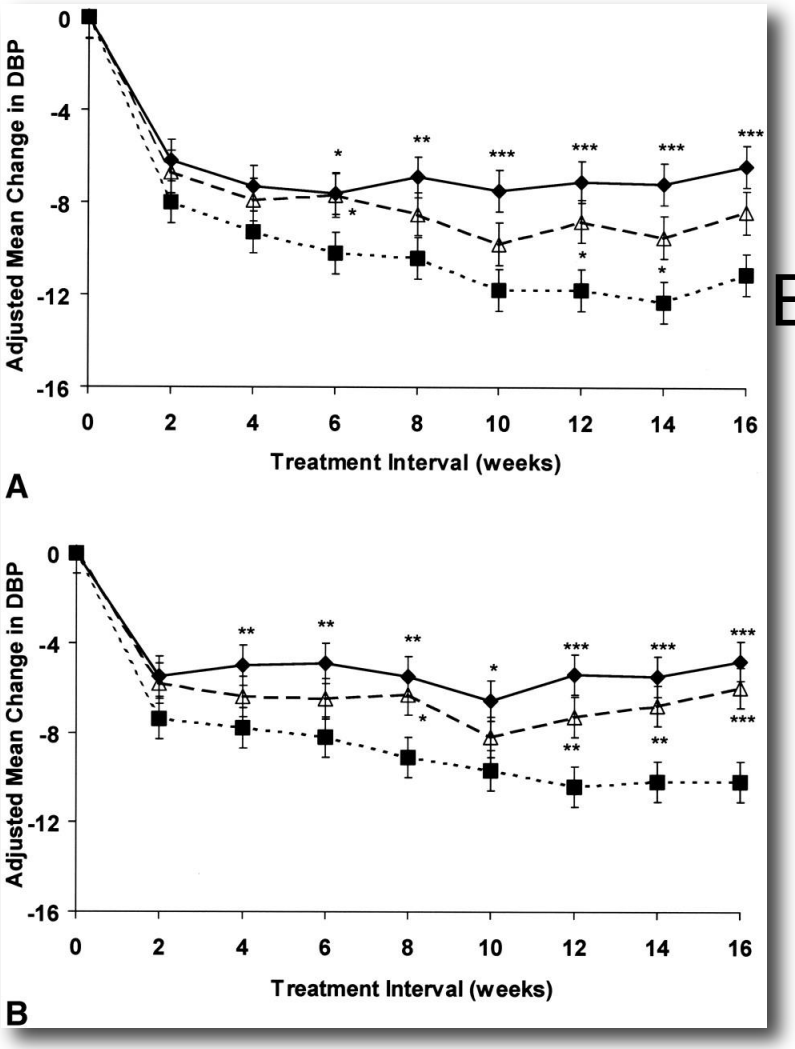
(Sacks, NEJM 2001)

ALLHAT



- Relative Risks for Comparisons of Amlodipine vs Chlorthalidone and Lisinopril vs Chlorthalidone in Blacks and Nonblacks

Mean change from baseline for diastolic blood pressure (DBP) in (A) black patients and (B) white patients

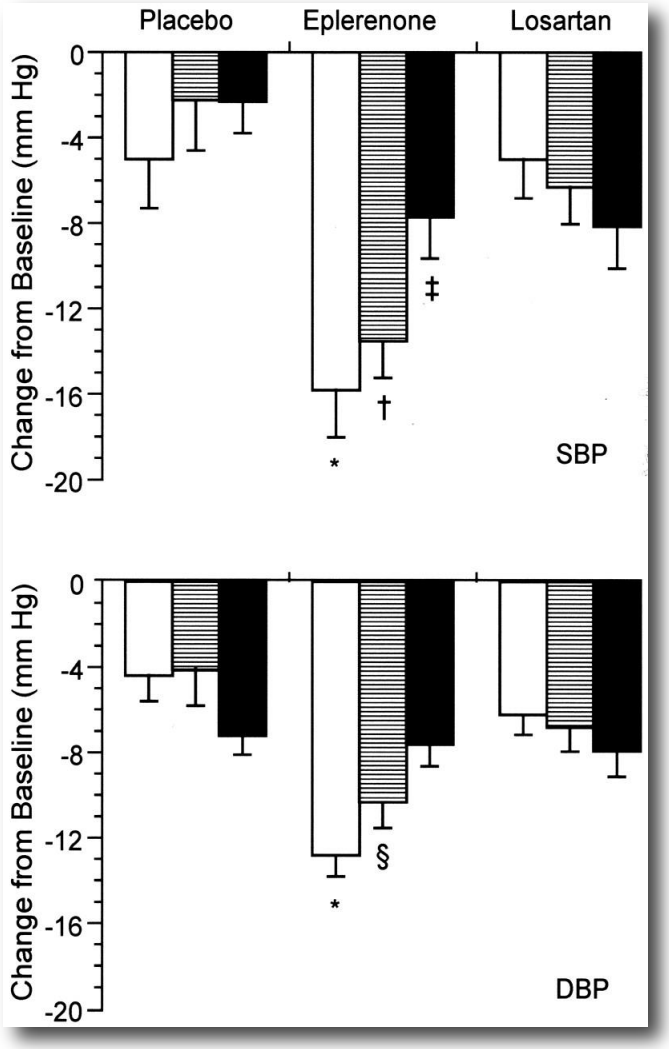


Placebo
Losartan
Eplerenone

Flack, J. M. et al. J Am Coll Cardiol 2003;41:1148-1155

JACC Journal of the American College of Cardiology

Changes SBP and DBP associated with treatment according to baseline level of active renin



White bars = <8.2 mU/l;
lined bars = 8.2 and <13.4 mU/l;
black bars = 13.4 mU/l.

Flack, J. M. et al. J Am Coll Cardiol 2003;41:1148-1155

Protection rénale: AASK

- African American Study of Kidney Disease and Hypertension
- Questions:
  - Un contrôle PA agressif ralentit-il la décroissance de la fonction rénale?
  - L'antihypertenseur initial modifie-t-il le pronostic rénal?
- Patients:
  - Noirs américains non diabétiques
  - Age 18-70 ans
  - PAD ≥ 95 mmHg
  - DFG 20-65 ml/min
- Chronologie de l'étude:
  - Pilote 1992-1994
  - Complète 1995-2001
- Interventions
  - Suivi 3,8 ans

Cible PA	Metoprolol 50-200 mg	Amlodipine 5-10 mg	Ramipril 2,5-10 mg
PA 140/90 (141/85)	20% (217)	10% (108)	20% (215)
PA 125/75 (128/78)	20% (224)	10% (109)	20% (221)
Total	441	217	436

(Wright, JAMA 2002, 288: 2421-2431)

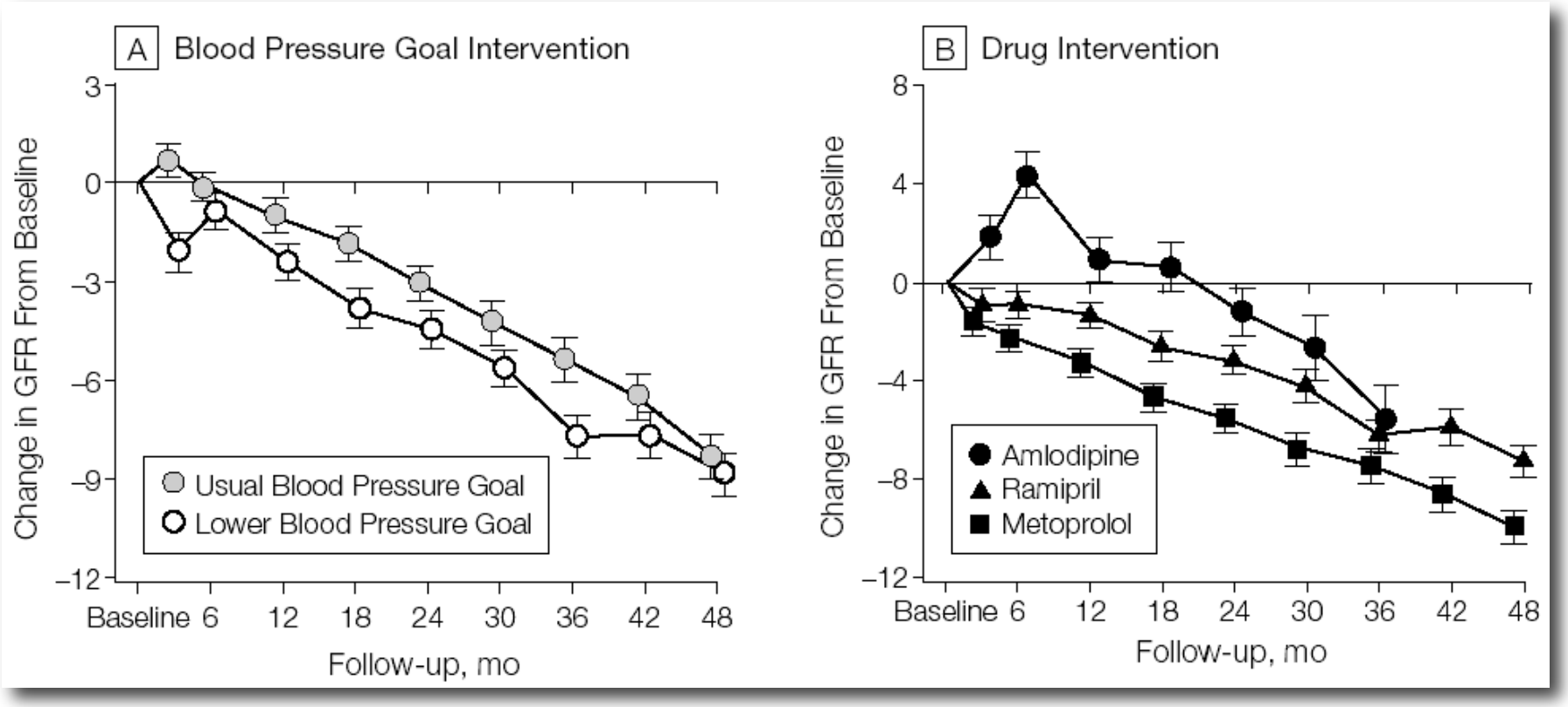
Protection rénale: AASK

- Caractéristiques initiales:

	Metoprolol	Ramipril	Amlodipine
Age	54.4±12.8	54.5±10.7	54.9±10.4
% Femmes	38.5	39.6	38.6
PAS (mmHg)	151±23	150±25	150±24
PAD (mmHg)	96±15	96±14	95±14
DFG (ml/min)	45.4±12.8	45.8±12.8	45.9±13.4

- Traitements associés: diurétiques (62%), IECA (38%), β-bloquants (28%), ICa (63%)

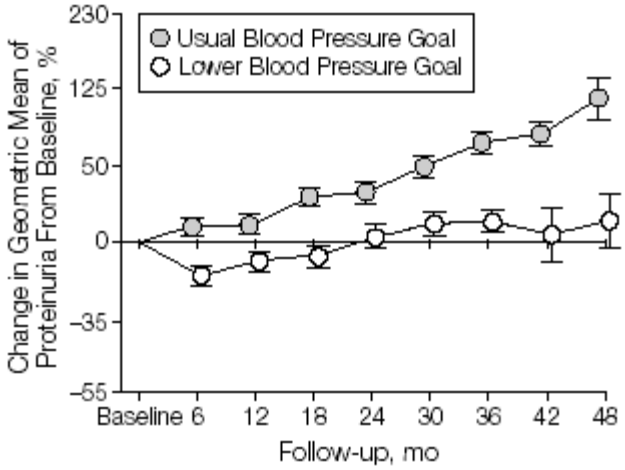
Renal outcomes: AASK

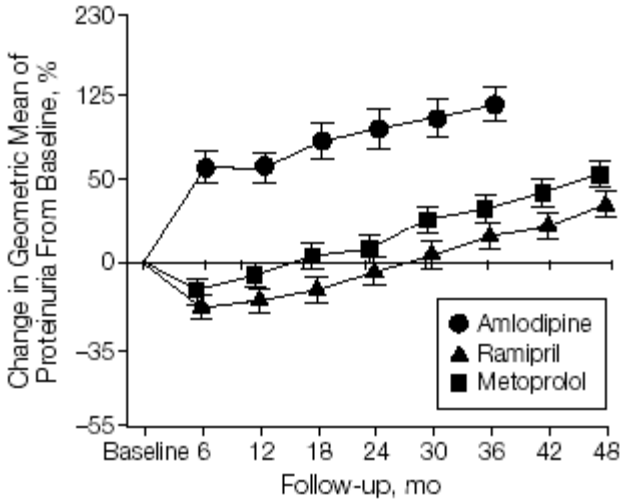


Protection rénale: AASK

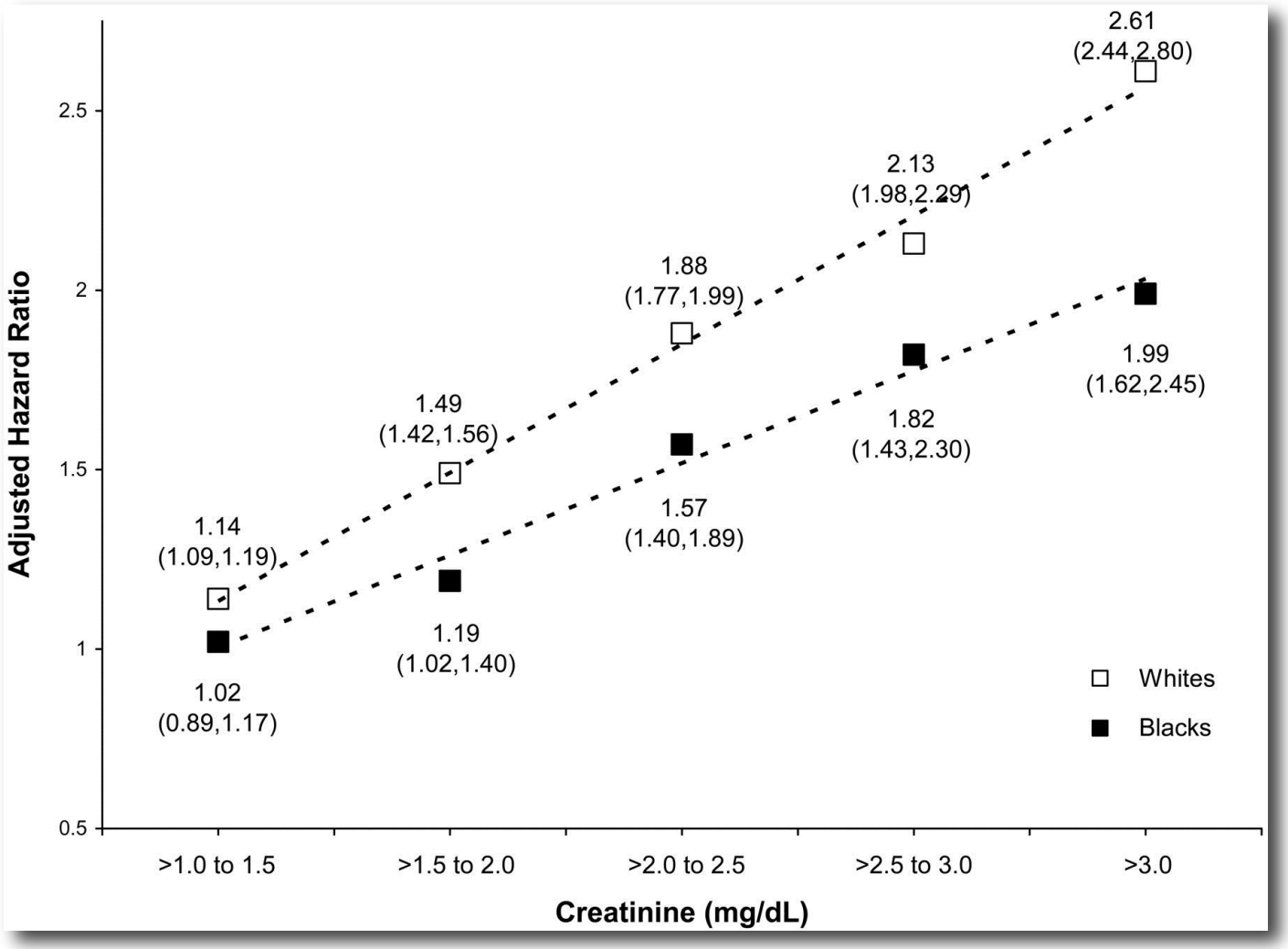
Résultats (%RRR)

	↘ DFG*,IRCT, décès	P
Low vs		
Usual BP	2	0.85
Ramipril vs		
Metoprolol	22	0.04
Metoprolol vs		
Amlodipine	20	0.17
Ramipril vs		





Adjusted hazard ratios for mortality by creatinine and race



Circulation

Smith, G. L. et al. Circulation 2005;111:1270-1277

Conclusions

- Epidémiologie
  - L'HTA est plus fréquente et très dépendante de l'environnement
- Physiopathologie
  - La sensibilité au sel est en partie génétique, en partie liée à l'excès pondéral surtout chez les femmes
  - La réactivité vasculaire aux stimuli vasoconstricteurs est augmentée
  - Le flux sanguin rénal est diminué avec un profil d'hyperfiltration
  - La microalbuminurie n'est pas significativement plus fréquente
- Risques
  - Le risque coronarien est très dépendant de l'HTA
  - Le risque d'HVG est lié à l'absence de baisse tensionnelle nocturne
  - Le risque d'AVC est accru
  - Le risque d'IRC est accru surtout pour les hypertensions modérées
- Traitement
  - La normalisation du poids et de l'apport sodé est essentielle
  - Un contrôle tensionnel satisfaisant peut être obtenu
  - Les IECA assurent une protection rénale significative