Παραδοσιακοί παράγοντες καρδιαγγειακού κινδύνου με επίδραση

στην εγκεφαλική λειτουργία στην ΧΝΝ

Ι. Βλάχου, MD, Msc

Καρδιολόγος Επιμελητής Β'

Γενικό Νοσοκομείο Πτολεμαίδος

ΠΑΡΑΓΟΝΤΕΣ ΚΑΡΔΙΑΓΓΕΙΑΚΟΥ ΚΙΝΔΥΝΟΥ

ΤΡΟΠΟΠΟΙΗΣΙΜΟΙ

- ΚΑΠΝΙΣΜΑ
- ΥΠΕΡΤΑΣΗ
- ΣΑΚΧΑΡΩΔΗΣ ΔΙΑΒΗΤΗΣ
- ΔΥΣΛΙΠΙΔΑΙΜΙΑ

ΜΗ ΤΡΟΠΟΠΟΙΗΣΙΜΟΙ

- ΗΛΙΚΙΑ
- ΦΥΛΟ
- ΚΛΗΡΟΝΟΜΙΚΟΤΗΤΑ

Table. Traditional and Nontraditional Risk Factors for CVD in CKD (Table view)

Risk factors for CVD in CKD	Specific aspects/treatment options compared with the non-CKD population	Ref.
Traditional		
Hypertension	Optimal target blood pressure has not yet been established	41
Dyslipidemia	Characteristic lipid pattern of hypertriglyceridemia and HDL cholesterol levels	42
Smoking	—	
Hyperglycemia	Intensive glucose control beneficial to avoid microvascular complications	43
Nontraditional		
Vascular calcifications	Treatment of electrolyte imbalances with magnesium	44, 45
	Vitamin K administration might be beneficial	46
Inflammation	Inhibition of proinflammatory effector molecule interleukin-1ß (IL-1ß) with canakinumab after myocardial infarction	47
Increased proteinuria	RAS blockade	48

CKD indicates chronic kidney disease; CVD, cardiovascular disease; HDL, high-density lipoprotein; and RAS, renin-angiotensin system.

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Circulation
Volume 143, Issue 11, 16 March 2021; Pages 1157-1172
https://doi.org/10.1161/CIRCULATIONAHA.120.050686

IN DEPTH

Cardiovascular Disease in Chronic Kidney Disease

Pathophysiological Insights and Therapeutic Options

Joachim Jankowski, PhD, Jürgen Floege, MD, Danilo Fliser, MD, Michael Böhm, MD, and Nikolaus Marx, MD

Abstract: Patients with chronic kidney disease (CKD) exhibit an elevated cardiovascular risk manifesting as coronary artery disease, heart failure, arrhythmias, and sudden cardiac death. Although the incidence and prevalence of cardiovascular events is already significantly higher in patients with early CKD stages (CKD stages 1–3) compared with the general population, patients with advanced CKD stages (CKD stages 4–5) exhibit a markedly elevated risk. Cardiovascular rather than end-stage kidney disease (CKD stage 5) is the leading cause of death in this high-risk population. CKD causes a systemic, chronic proinflammatory state contributing to vascular and myocardial remodeling processes resulting in atherosclerotic lesions, vascular calcification, and vascular senescence as well as myocardial fibrosis and calcification of cardiac valves. In this respect, CKD mimics an accelerated aging of the cardiovascular system. This overview article summarizes the current understanding and clinical consequences of cardiovascular disease in CKD.

Key Words: arrhythmias ■ cardiovascular disease ■ chronic kidney disease ■ clinical aspects ■ heart failure ■ sudden cardiac ■ death

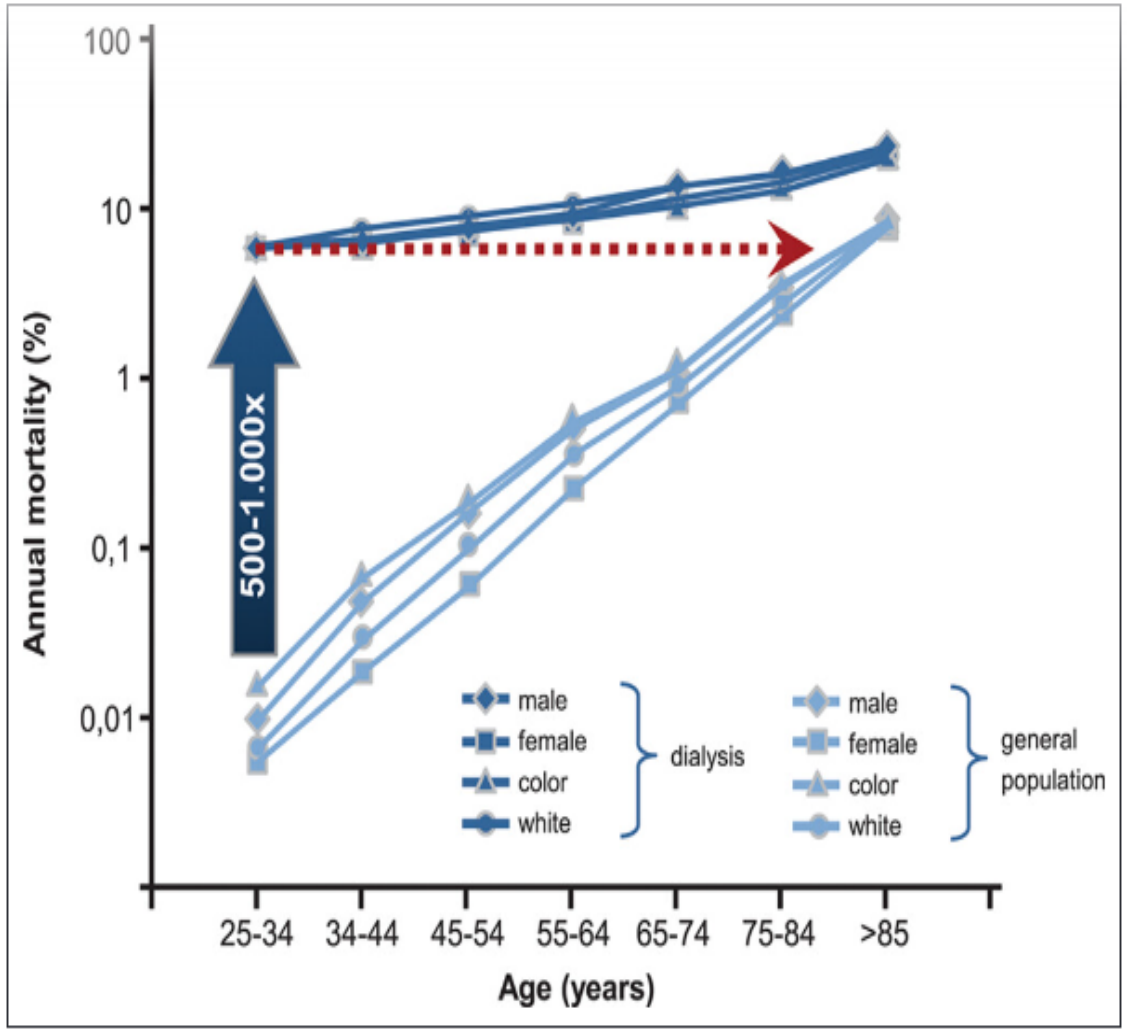


Figure 1. Cardiovascular mortality in the general population and in patients with end-stage kidney disease. In 25- to 34-year-old patients with end-stage kidney disease, annual mortality is increased 500- to 1000-fold and corresponds to that of the ≈85-year-old general population. Adapted from Foley et al.[5]

Circulation. 2021;143:1157–1172. DOI: 10.1161/CIRCULATIONAHA.120.050686

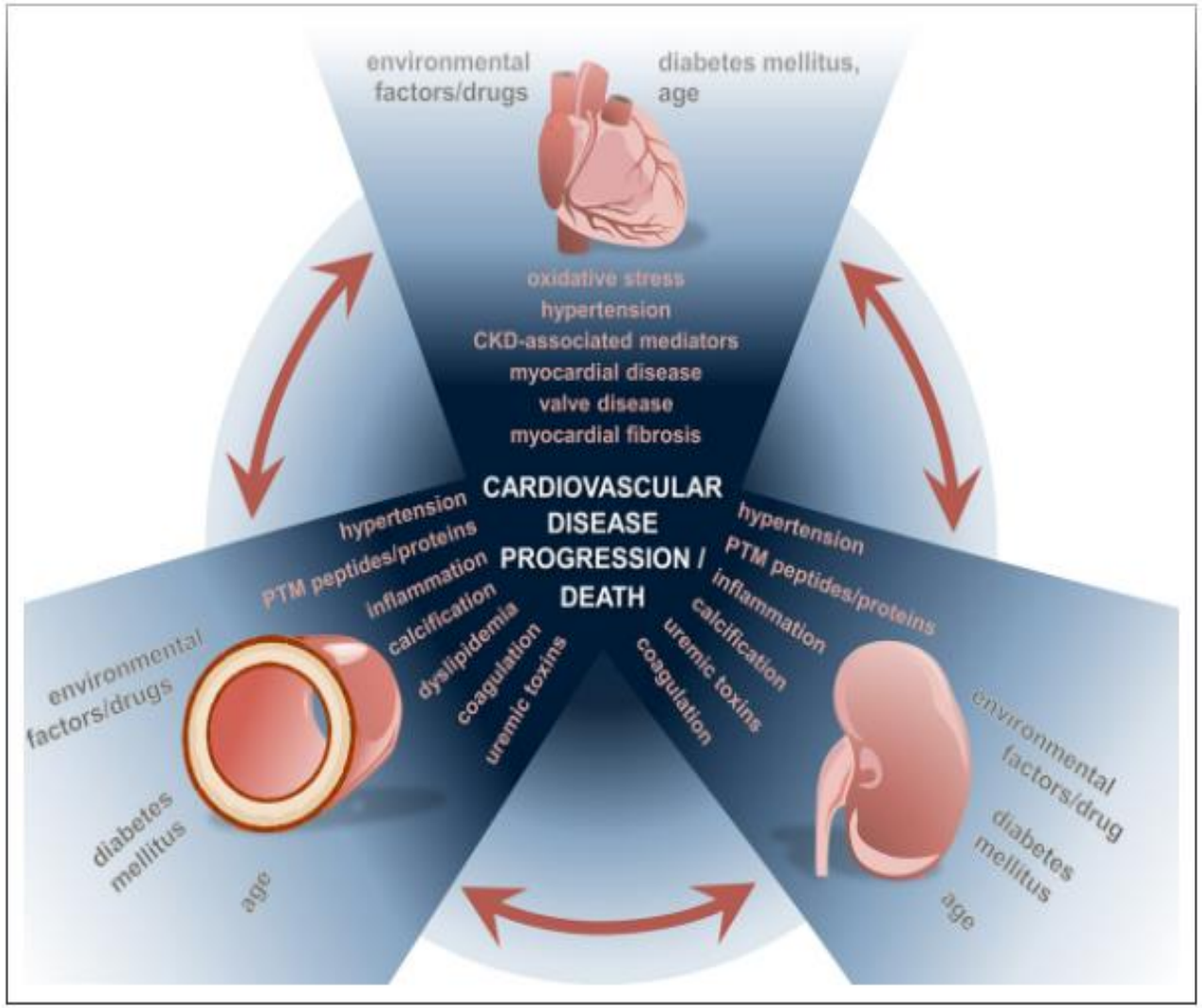


Figure 2. Interaction of cardiovascular disease (CVD) and chronic kidney disease (CKD). Various mediators and mechanisms in vascular disease, heart failure, and CKD contribute to the progression of CVD and influence the prognosis of patients. PTM indicates post-translational modification.

Circulation. 2021;143:1157–1172. DOI: 10.1161/CIRCULATIONAHA.120.050686

Γνωσιακή δυσλειτουργία και κάπνισμα

- Ένω υπάρχει σαφής συσχέτιση μεταξύ καπνίσματος και γνωσιακής δυσλειτουργίας δεν είναι απολύτως σαφές πως το κάπνισμα επιδρά στην γνωσιακή δυσλειτουργία. Υπάρχουν αρκετές θεωρίες οι κυριότερες των οποιών είναι οι παρακάτω.

- Επιτάχυνση της εγκεφαλικής γύρανσης.
- Άυξηση κινδύνου εμφάνισης Alzheimer μέσω αύξησης του οξειδωτικού στρες.
- Αγγειακή δυσλειτουργία (ΑΕΕ, Καρδιαγγειακή νόσος)
- Αυξημένα ποσοστά εμφάνισης υπνικής άπνοιας και διαταραχών ύπνου.

ONLINE FIRST

Impact of Smoking on Cognitive Decline in Early Old Age

The Whitehall II Cohort Study

Séverine Sabia, PhD; Alexis Elbaz, MD, PhD; Aline Dugravot, MSc; Jenny Head, MSc; Martin Shipley, MSc; Gareth Hagger-Johnson, PhD; Mika Kivimaki, PhD; Archana Singh-Manoux, PhD

Context: Smoking is a possible risk factor for dementia, although its impact may have been underestimated in elderly populations because of the shorter life span of smokers.

Objective: To examine the association between smoking history and cognitive decline in the transition from midlife to old age.

Design: Cohort study.

Setting: The Whitehall II study. The first cognitive assessment was in 1997 to 1999, repeated over 2002 to 2004 and 2007 to 2009.

Participants: Data are from 5099 men and 2137 women in the Whitehall II study, mean age 56 years (range, 44-69 years) at the first cognitive assessment.

Main Outcome Measures: The cognitive test battery was composed of tests of memory, vocabulary, executive function (composed of 1 reasoning and 2 fluency tests), and a global cognitive score summarizing performance across all 5 tests. Smoking status was assessed over the entire study period. Linear mixed models were used to assess the association between smoking history and 10-year cognitive decline, expressed as *z* scores.

Results: In men, 10-year cognitive decline in all tests except vocabulary among never smokers ranged from a quarter to a third of the baseline standard deviation. Faster cognitive decline was observed among current smokers compared with never smokers in men (mean difference in 10-year decline in global cognition=−0.09 [95% CI, −0.15 to −0.03] and executive function=−0.11 [95% CI, −0.17 to −0.05]). Recent ex-smokers had greater decline in executive function (−0.08 [95% CI, −0.14 to −0.02]), while the decline in long-term ex-smokers was similar to that among never smokers. In analyses that additionally took dropout and death into account, these differences were 1.2 to 1.5 times larger. In women, cognitive decline did not vary as a function of smoking status.

Conclusions: Compared with never smokers, middle-aged male smokers experienced faster cognitive decline in global cognition and executive function. In ex-smokers with at least a 10-year cessation, there were no adverse effects on cognitive decline.

Arch Gen Psychiatry. 2012;69(6):627-635.
Published online February 6, 2012.
doi:10.1001/archgenpsychiatry.2011.2016

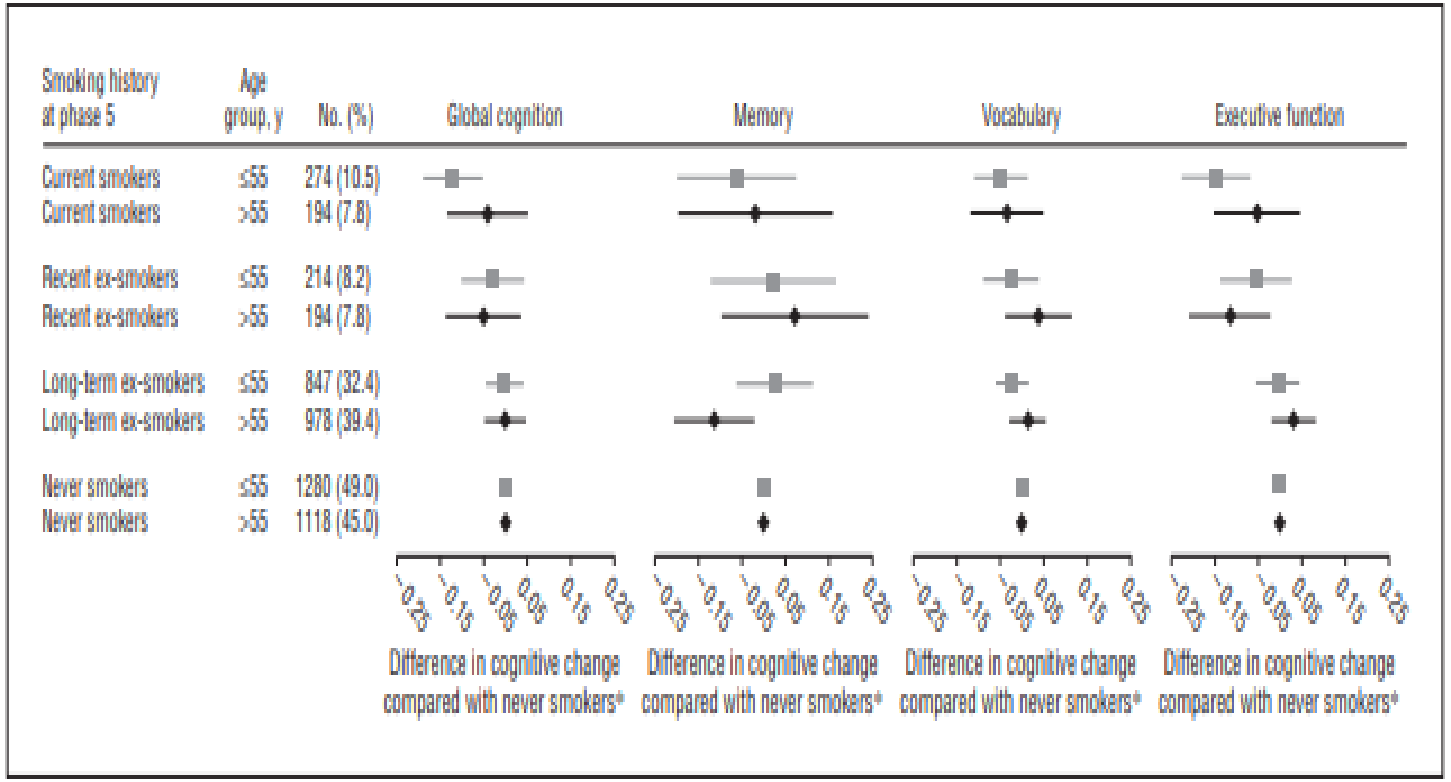
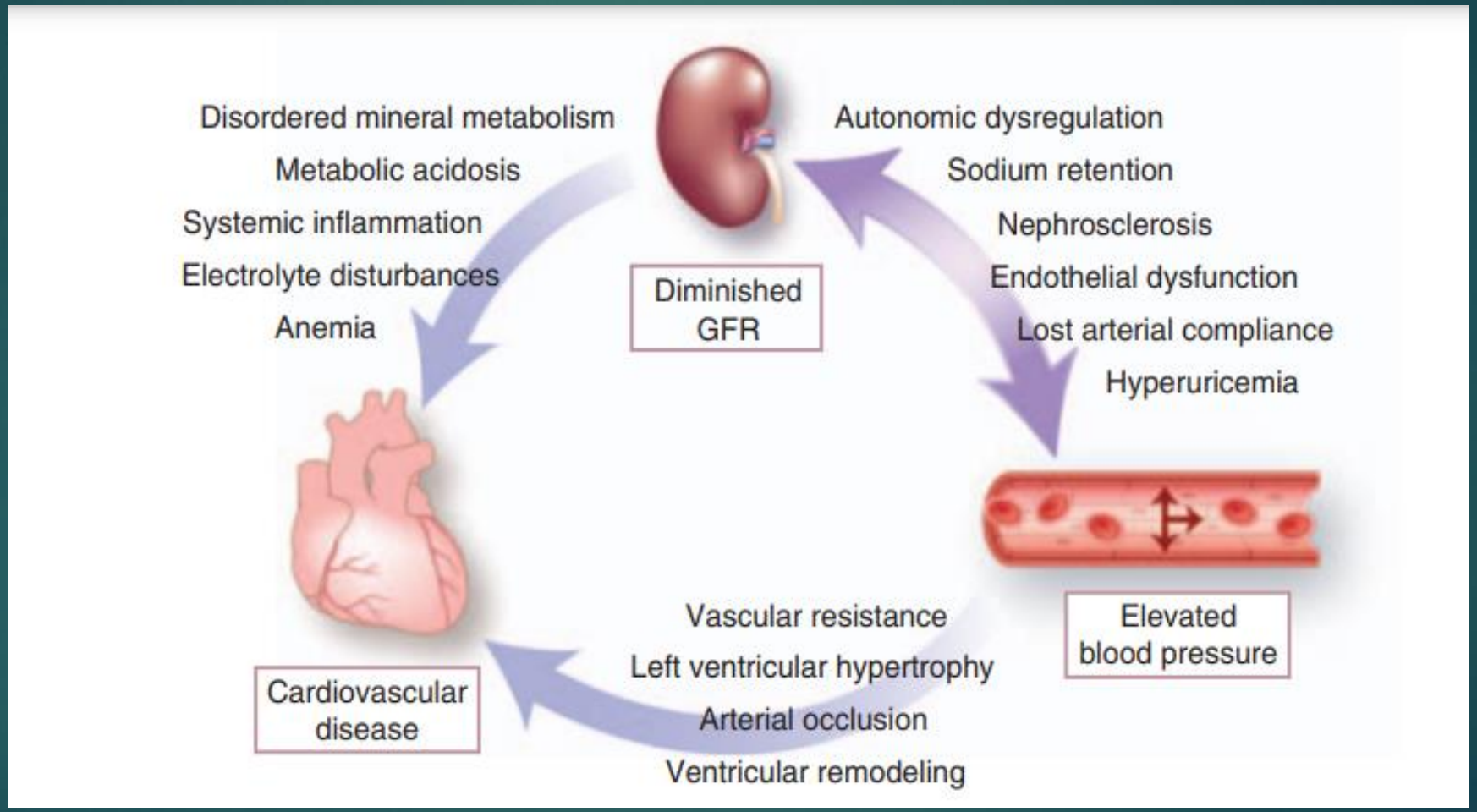


Figure 1. Association between smoking history at phase 5 and cognitive change over the subsequent 10 years in men as a function of age group (reference group: never smokers). *Estimates were obtained from model 1 (results in Table 2) but this time separately in men 55 years or younger (squares) and older than 55 years (diamonds). For example, current smokers 55 years or younger experienced an additional decline in global cognition of −0.12 (95% CI, −0.19 to −0.05) with respect to never smokers in the same age group. The corresponding figure for participants older than 55 years was −0.04 (95% CI, −0.13 to 0.05).

ΥΠΕΡΤΑΣΗ-ΧΝΝ ΚΑΙ ΚΑΡΔΙΑΓΓΕΙΑΚΟΣ ΚΙΝΔΥΝΟΣ



Kidney International (2010) **77,** 753–755. doi:10.1038/ki.2010.19

Blood Pressure as a Risk Factor for Cardiovascular Disease

The Framingham Study—30 Years of Follow-up

Joseph Stokes III, William B. Kannel, Philip A. Wolf,
Ralph B. D'Agostino, and L. Adrienne Cupples

Data from 30 years of follow-up of the original Framingham Study cohort of 5,070 men and women aged 30–62 years who were first examined during the period 1948–1952 and who were free of cardiovascular disease reveal that blood pressure is a strong and consistent predictor of the development of coronary heart disease, stroke, transient ischemic attack, and congestive heart failure. Other factors related to blood pressure like obesity, left ventricular hypertrophy as demonstrated on electrocardiograms, and heart enlargement as shown by x-ray radiography made several selective additional independent contributions to risk; heart enlargement by x-ray radiography was the best predictor of congestive heart failure. (*Hypertension* 1989;13(suppl I):I-13–I-18)

Table 23 **Office blood pressure treatment target range**

Age group	Office SBP treatment target ranges (mmHg)					Office DBP treatment target range (mmHg)
	Hypertension	+ Diabetes	+ CKD	+ CAD	+ Stroke[a]/TIA	
18 - 65 years	**Target to 130** *or lower if tolerated* Not <120	**Target to 130** *or lower if tolerated* Not <120	**Target to <140 to 130** *if tolerated*	**Target to 130** *or lower if tolerated* Not <120	**Target to 130** *or lower if tolerated* Not <120	70–79
65 - 79 years[b]	**Target to 130-139** *if tolerated*	**Target to 130-139** *if tolerated*	**Target to 130-139** *if tolerated*	**Target to 130-139** *if tolerated*	**Target to 130-139** *if tolerated*	70–79
≥80 years[b]	**Target to 130-139** *if tolerated*	**Target to 130-139** *if tolerated*	**Target to 130-139** *if tolerated*	**Target to 130-139** *if tolerated*	**Target to 130-139** *if tolerated*	70–79
Office DBP treatment target range (mmHg)	70–79	70–79	70–79	70–79	70–79	

CAD = coronary artery disease; CKD = chronic kidney disease (includes diabetic and non-diabetic CKD); DBP = diastolic blood pressure; SBP = systolic blood pressure; TIA = transient ischaemic attack.

[a]Refers to patients with previous stroke and does not refer to blood pressure targets immediately after acute stroke.

[b]Treatment decisions and blood pressure targets may need to be modified in older patients who are frail and independent.

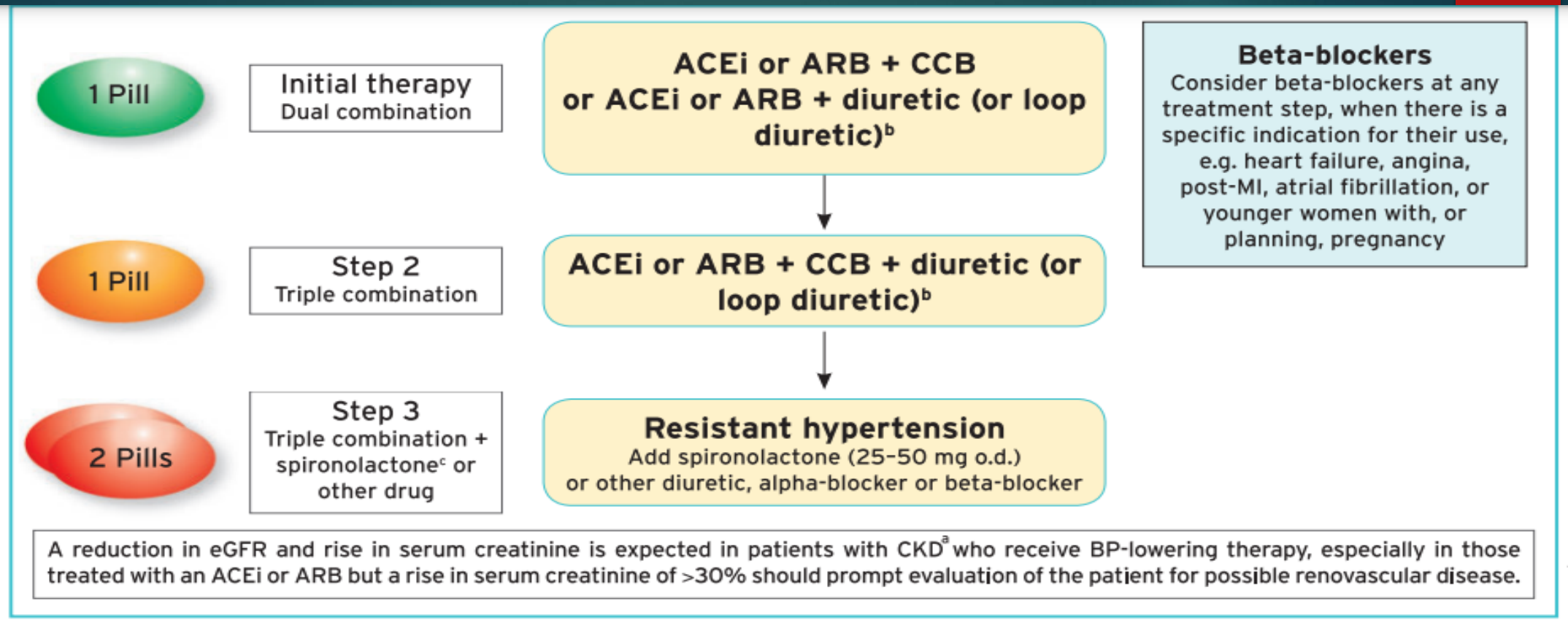


Figure 6 Drug treatment strategy for hypertension and chronic kidney disease. ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; BP = blood pressure; CCB = calcium channel blocker; CKD = chronic kidney disease; eGFR = estimated glomerular filtration rate; MI = myocardial infarction; o.d. = omni die (every day).

[a]CKD is defined as an eGFR <60 mL/min/1.72 m^2 with or without proteinuria.

[b]Use loop diuretics when eGFR is <30 mL/min/1.72 m^2, because thiazide/thiazide-like diuretics are much less effective/ineffective when eGFR is reduced to this level.

[c]Caution: risk of hyperkalaemia with spironolactone, especially when eGFR is <45 mL/min/1.72 m^2 or baseline K^+ ≥4.5 mmol/L.

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 NOVEMBER 26, 2015 VOL. 373 NO. 22

A Randomized Trial of Intensive versus Standard Blood-Pressure Control

The SPRINT Research Group*

ABSTRACT

BACKGROUND

The most appropriate targets for systolic blood pressure to reduce cardiovascular morbidity and mortality among persons without diabetes remain uncertain.

METHODS

We randomly assigned 9361 persons with a systolic blood pressure of 130 mm Hg or higher and an increased cardiovascular risk, but without diabetes, to a systolic blood-pressure target of less than 120 mm Hg (intensive treatment) or a target of less than 140 mm Hg (standard treatment). The primary composite outcome was myocardial infarction, other acute coronary syndromes, stroke, heart failure, or death from cardiovascular causes.

RESULTS

At 1 year, the mean systolic blood pressure was 121.4 mm Hg in the intensive-treatment group and 136.2 mm Hg in the standard-treatment group. The intervention was stopped early after a median follow-up of 3.26 years owing to a significantly lower rate of the primary composite outcome in the intensive-treatment group than in the standard-treatment group (1.65% per year vs. 2.19% per year; hazard ratio with intensive treatment, 0.75; 95% confidence interval [CI], 0.64 to 0.89; P<0.001). All-cause mortality was also significantly lower in the intensive-treatment group (hazard ratio, 0.73; 95% CI, 0.60 to 0.90; P=0.003). Rates of serious adverse events of hypotension, syncope, electrolyte abnormalities, and acute kidney injury or failure, but not of injurious falls, were higher in the intensive-treatment group than in the standard-treatment group.

CONCLUSIONS

Among patients at high risk for cardiovascular events but without diabetes, targeting a systolic blood pressure of less than 120 mm Hg, as compared with less than 140 mm Hg, resulted in lower rates of fatal and nonfatal major cardiovascular events and death from any cause, although significantly higher rates of some adverse events were observed in the intensive-treatment group. (Funded by the National Institutes of Health; ClinicalTrials.gov number, NCT01206062.)

The members of the writing committee (Jackson T. Wright, Jr., M.D., Ph.D., Jeff D. Williamson, M.D., M.H.S., Paul K. Whelton, M.D., Joni K. Snyder, R.N., B.S.N., M.A., Kaycee M. Sink, M.D., M.A.S., Michael V. Rocco, M.D., M.S.C.E., David M. Reboussin, Ph.D., Mahboob Rahman, M.D., Suzanne Oparil, M.D., Cora E. Lewis, M.D., M.S.P.H., Paul L. Kimmel, M.D., Karen C. Johnson, M.D., M.P.H., David C. Goff, Jr., M.D., Ph.D., Lawrence J. Fine, M.D., Dr.P.H., Jeffrey A. Cutler, M.D., M.P.H., William C. Cushman, M.D., Alfred K. Cheung, M.D., and Walter T. Ambrosius, Ph.D.) assume responsibility for the overall content and integrity of the article. The affiliations of the members of the writing group are listed in the Appendix. Address reprint requests to Dr. Wright at the Division of Nephrology and Hypertension, University Hospitals Case Medical Center, Case Western Reserve University, 1100 Euclid Ave. Cleveland, OH 44106-6053, or at jackson.wright@case.edu.

*A complete list of the members of the Systolic Blood Pressure Intervention Trial (SPRINT) Research Group is provided in the Supplementary Appendix, available at NEJM.org.

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DOI: 10.1056/NEJMoa1511939

ΥΠΕΡΤΑΣΗ ΚΑΙ ΓΝΩΣΙΑΚΗ ΔΥΣΛΕΙΤΟΥΡΓΙΑ

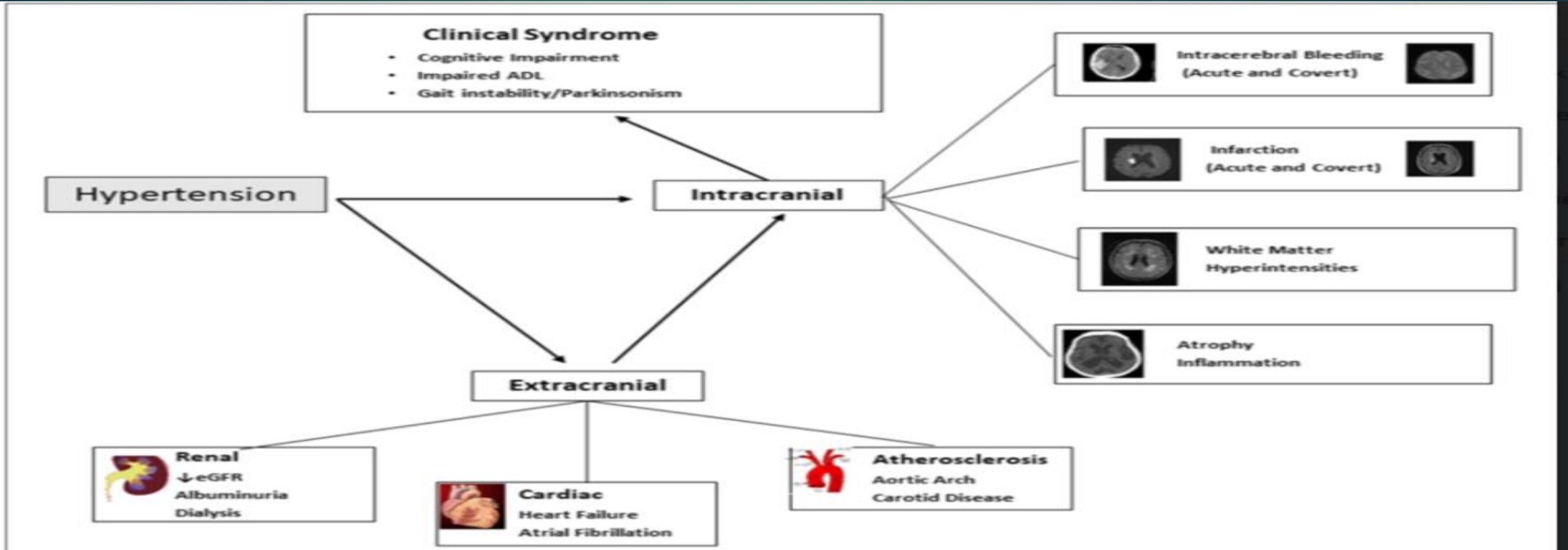


FIGURE 1 | Hypertension and cognitive impairment and dementia. Figure illustrates the intracranial and extracranial mechanisms through with hypertension results in cognitive impairment and dementia (and illustrates other non-cognitive clinical consequences that are part of the dementia syndrome). ADL, Activities of Daily Living; eGFR, Estimated Glomerular Filtration Rate.

doi: 10.3389/fneur.2022.821135

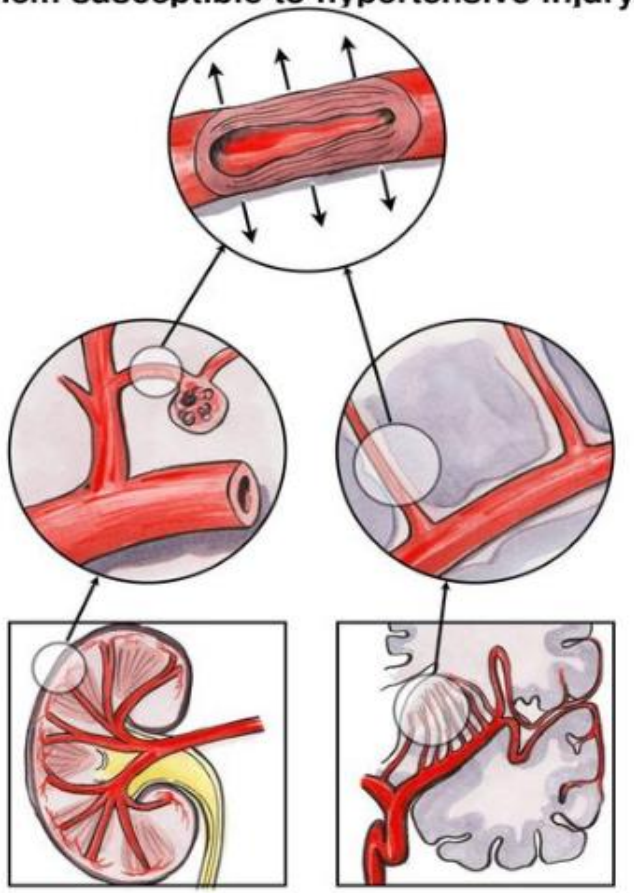
Relationship Between Aortic Stiffening and Microvascular Disease in Brain and Kidney

Cause and Logic of Therapy

Michael F. O'Rourke, Michel E. Safar

Abstract—A close relationship has been established between microvascular damage in brain and kidney and indices of age and hypertension (pulse pressure, aortic pulse wave velocity, and augmentation index). The mechanism of such association has not been established, nor has rationale for prevention and treatment of microvascular damage. A logical pathophysiological explanation can be offered on the basis of differential input impedance in the brain and kidney compared with other systemic vascular beds. Torrential flow and low resistance to flow in these organs exposes small arterial vessels to the high-pressure fluctuations that exist in the carotid, vertebral, and renal arteries. Such fluctuations, measurable as central pulse pressure, increase 3- to 4-fold with age. Exposure of small vessels to highly pulsatile pressure and flow explains microvascular damage and resulting renal insufficiency and intellectual deterioration, according to the mechanism established by Byrom >50 years ago. The logical approach to prevention and treatment requires reduction of central pulse pressure. Because the aorta and large arteries are not directly affected by drugs, this entails reduction of wave reflection by dilation of conduit arteries elsewhere in the body. This can be accomplished by regular exercise and by drugs such as nitrates, calcium channel blockers, angiotensin-converting enzyme inhibitors, and angiotensin receptor blockers. The explanation given here accounts for greater and earlier vascular damage in diabetes mellitus (relative microvascular fragility) and is similar to that given for vascular changes of pulmonary hypertension caused by ventricular septal defects and other congenital vascular shunts. (*Hypertension*. **2005;46:200-204.**)

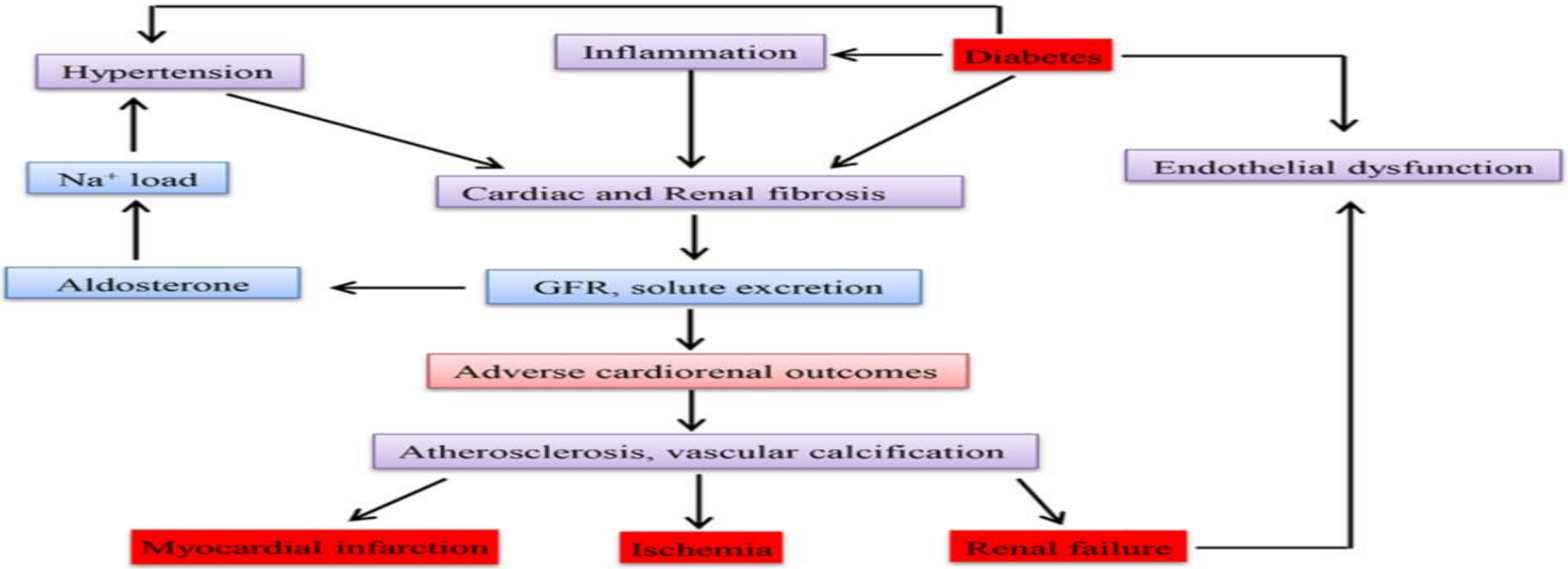
The strain vessel hypothesis: juxtamedullary afferent arterioles and cerebral perforating arteries are both exposed to high pressure and have to maintain large pressure gradients, rendering them susceptible to hypertensive injury.



Dearbhla Kelly, and Peter Malcolm Rothwell J Neurol Neurosurg Psychiatry doi:10.1136/jnnp-2019-320526

JNNP

ΣΑΚΧΑΡΩΔΗΣ ΔΙΑΒΗΤΗΣ ΚΑΙ ΚΑΡΔΙΑΓΓΕΙΑΚΟΣ ΚΙΝΔΥΝΟΣ



Schematic representation of clinical link between chronic kidney disease, diabetes mellitus, and cardiovascular disease.

doi: 10.3389/fimmu.2015.00340

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Intensive Blood Glucose Control and Vascular Outcomes in Patients with Type 2 Diabetes

The ADVANCE Collaborative Group*

ABSTRACT

BACKGROUND

In patients with type 2 diabetes, the effects of intensive glucose control on vascular outcomes remain uncertain.

METHODS

We randomly assigned 11,140 patients with type 2 diabetes to undergo either standard glucose control or intensive glucose control, defined as the use of gliclazide (modified release) plus other drugs as required to achieve a glycated hemoglobin value of 6.5% or less. Primary end points were composites of major macrovascular events (death from cardiovascular causes, nonfatal myocardial infarction, or nonfatal stroke) and major microvascular events (new or worsening nephropathy or retinopathy), assessed both jointly and separately.

This article (10.1056/NEJMoa0802987) was published at www.nejm.org on June 6, 2008.

N Engl J Med 2008;358:2560-72.

RESULTS

After a median of 5 years of follow-up, the mean glycated hemoglobin level was lower in the intensive-control group (6.5%) than in the standard-control group (7.3%). Intensive control reduced the incidence of combined major macrovascular and microvascular events (18.1%, vs. 20.0% with standard control; hazard ratio, 0.90; 95% confidence interval [CI], 0.82 to 0.98; P=0.01), as well as that of major microvascular events (9.4% vs. 10.9%; hazard ratio, 0.86; 95% CI, 0.77 to 0.97; P=0.01), primarily because of a reduction in the incidence of nephropathy (4.1% vs. 5.2%; hazard ratio, 0.79; 95% CI, 0.66 to 0.93; P=0.006), with no significant effect on retinopathy (P=0.50). There were no significant effects of the type of glucose control on major macrovascular events (hazard ratio with intensive control, 0.94; 95% CI, 0.84 to 1.06; P=0.32), death from cardiovascular causes (hazard ratio with intensive control, 0.88; 95% CI, 0.74 to 1.04; P=0.12), or death from any cause (hazard ratio with intensive control, 0.93; 95% CI, 0.83 to 1.06; P=0.28). Severe hypoglycemia, although uncommon, was more common in the intensive-control group (2.7%, vs. 1.5% in the standard-control group; hazard ratio, 1.86; 95% CI, 1.42 to 2.40; P<0.001).

CONCLUSIONS

A strategy of intensive glucose control, involving gliclazide (modified release) and other drugs as required, that lowered the glycated hemoglobin value to 6.5% yielded a 10% relative reduction in the combined outcome of major macrovascular and microvascular events, primarily as a consequence of a 21% relative reduction in nephropathy. (ClinicalTrials.gov number, NCT00145925.)

ΣΑΚΧΑΡΩΔΗΣ ΔΙΑΒΗΤΗΣ ΚΑΙ ΓΝΩΣΙΑΚΗ ΛΥΣΛΕΙΤΟΥΡΓΙΑ

ELSEVIER

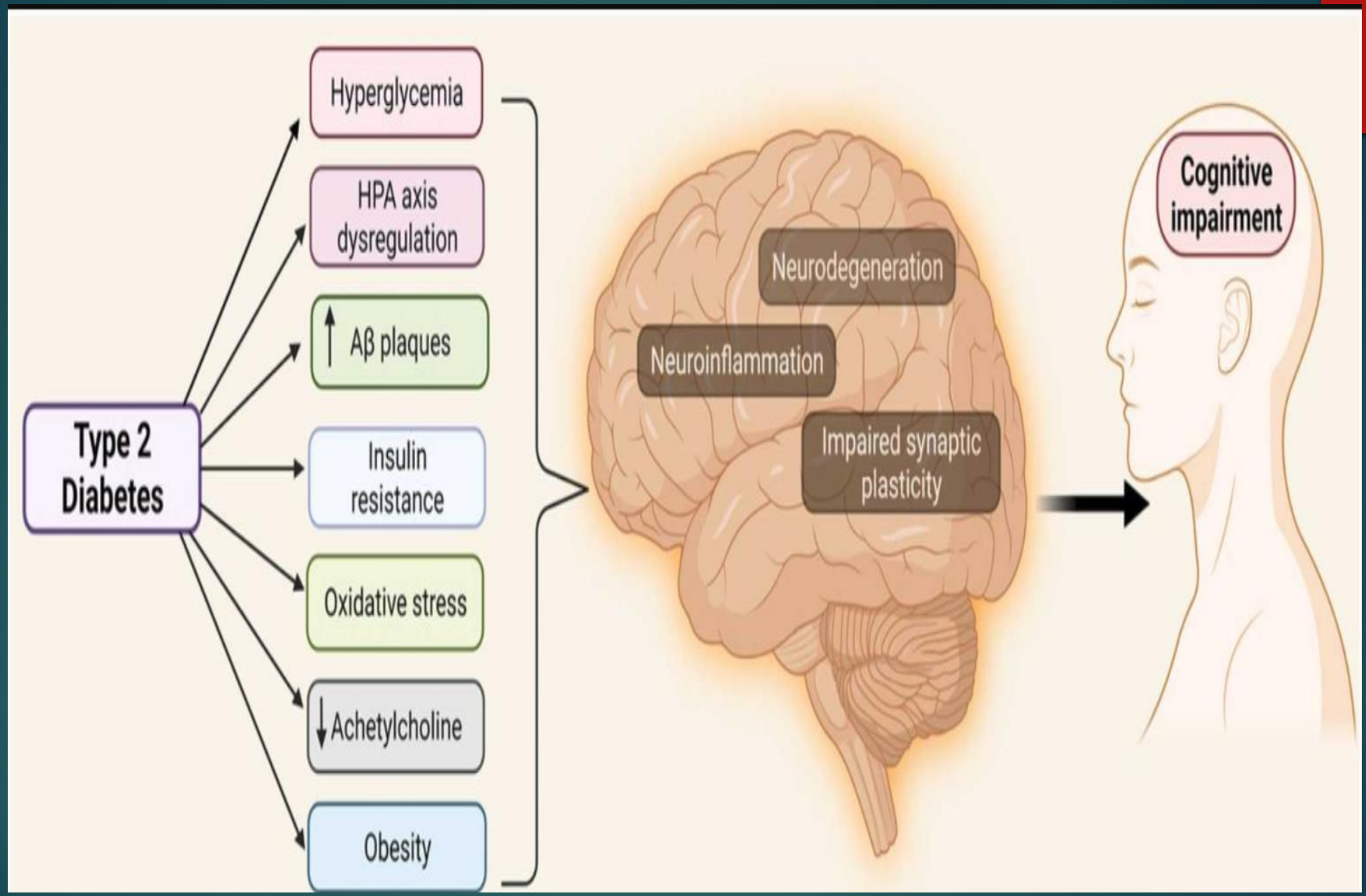
Pharmacological Research

Volume 182, August 2022, 106358

Review

Inside the diabetic brain: Insulin resistance and molecular mechanism associated with cognitive impairment and its possible therapeutic strategies

Bhaskar Jyoti Dutta, Shamsher Singh, Sanket Seksaria, Ghanshyam Das Gupta, Amrita Singh



https://doi.org/10.1016/j.phrs.2022.106358

ΔΥΣΛΙΠΙΔΑΙΜΙΑ

- Παθολογική κατάσταση των λιπιδίων στο αίμα.
- Αύξηση ολικής χοληστερόλης ,LDL, τριγλυκεριδίων όσο και έλάττωση HDL

ΤΥΠΟΙ ΔΥΣΛΙΠΙΔΑΙΜΙΑΣ

- Πρωτοπαθής : Γονιδιακή (κληρονομικής αιτιολογίας).
- Δευτεροπαθής : Παθολογικές καταστάσεις (ΣΔ,ΧΝΑ, παχυσαρκία), διατροφή.

Table 4 **Cardiovascular risk categories**

Very-high-risk	People with any of the following: Documented ASCVD, either clinical or unequivocal on imaging. Documented ASCVD includes previous ACS (MI or unstable angina), stable angina, coronary revascularization (PCI, CABG, and other arterial revascularization procedures), stroke and TIA, and peripheral arterial disease. Unequivocally documented ASCVD on imaging includes those findings that are known to be predictive of clinical events, such as significant plaque on coronary angiography or CT scan (multivessel coronary disease with two major epicardial arteries having >50% stenosis), or on carotid ultrasound. DM with target organ damage,[a] or at least three major risk factors, or early onset of T1DM of long duration (>20 years). Severe CKD (eGFR <30 mL/min/1.73 m^2). A calculated SCORE ≥10% for 10-year risk of fatal CVD. FH with ASCVD or with another major risk factor.
High-risk	People with: Markedly elevated single risk factors, in particular TC >8 mmol/L (>310 mg/dL), LDL-C >4.9 mmol/L (>190 mg/dL), or BP ≥180/110 mmHg. Patients with FH without other major risk factors. Patients with DM without target organ damage,[a] with DM duration ≥10 years or another additional risk factor. Moderate CKD (eGFR 30—59 mL/min/1.73 m^2). A calculated SCORE ≥5% and <10% for 10-year risk of fatal CVD.
Moderate-risk	Young patients (T1DM <35 years; T2DM <50 years) with DM duration <10 years, without other risk factors. Calculated SCORE ≥1 % and <5% for 10-year risk of fatal CVD.
Low-risk	Calculated SCORE <1% for 10-year risk of fatal CVD.

Recommendations for treatment goals for low-density lipoprotein cholesterol

Recommendations	Class[a]	Level[b]
In secondary prevention for patients at very-high risk,[c] an LDL-C reduction of ≥50% from baseline[d] and an LDL-C goal of <1.4 mmol/L (<55 mg/dL) are recommended.[33–35,119,120]	I	A
In primary prevention for individuals at very-high risk but without FH,[c] an LDL-C reduction of ≥50% from baseline[d] and an LDL-C goal of <1.4 mmol/L (<55 mg/dL) are recommended.[34–36]	I	C
In primary prevention for individuals with FH at very-high risk, an LDL-C reduction of ≥50% from baseline and an LDL-C goal of <1.4 mmol/L (<55 mg/dL) should be considered.	IIa	C
For patients with ASCVD who experience a second vascular event within 2 years (not necessarily of the same type as the first event) while taking maximally tolerated statin-based therapy, an LDL-C goal of <1.0 mmol/L (<40 mg/dL) may be considered.[119,120]	IIb	B
In patients at high risk,[c] an LDL-C reduction of ≥50% from baseline[d] and an LDL-C goal of <1.8 mmol/L (<70 mg/dL) are recommended.[34,35]	I	A
In individuals at moderate risk,[c] an LDL-C goal of <2.6 mmol/L (<100 mg/dL) should be considered.[34]	IIa	A
In individuals at low risk,[c] an LDL-C goal <3.0 mmol/L (<116 mg/dL) may be considered.[36]	IIb	A

doi:10.1093/eurheartj/ehz455

ΔΥΣΛΙΠΙΔΑΙΜΙΑ ΚΑΙ ΧΡΟΝΙΑ ΝΕΦΡΙΚΗ ΑΝΕΠΑΡΚΕΙΑ

- ΥΠΕΡΤΡΙΓΛΥΚΕΡΙΔΑΙΜΙΑ
- ΧΑΜΗΛΗ HDL
- ΦΥΣΙΟΛΟΓΙΚΗ LDL

https://doi.org/10.3390/ijms22158201

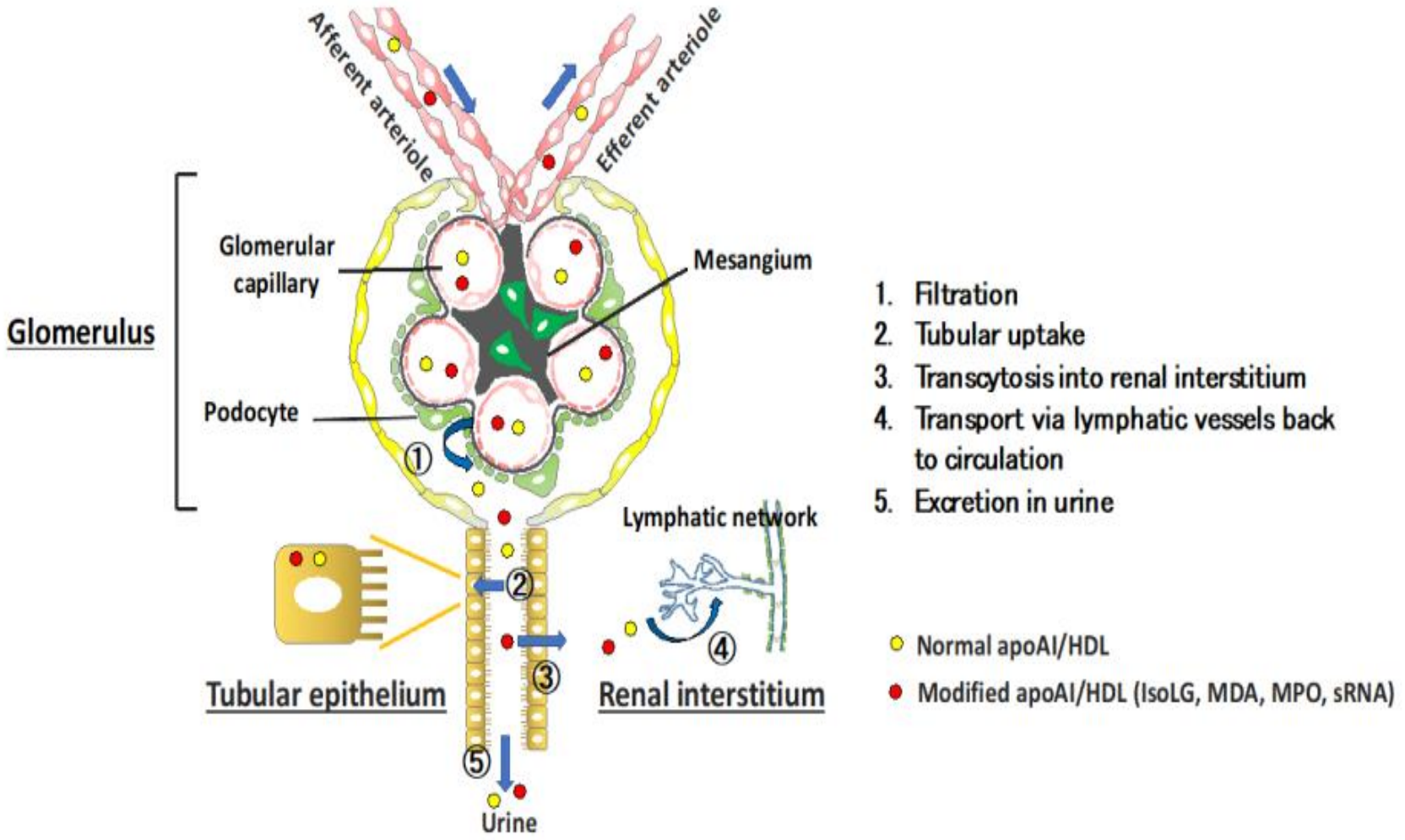


Figure 1. Kidney handling of normal and modified apoAI/HDL by the glomerulus, tubule epithelium, and renal interstitium involves (1) filtration (2) tubular uptake (3) transcytosis (4) transport by lymphatic vascular network in interstitium and (5) urinary excretion.

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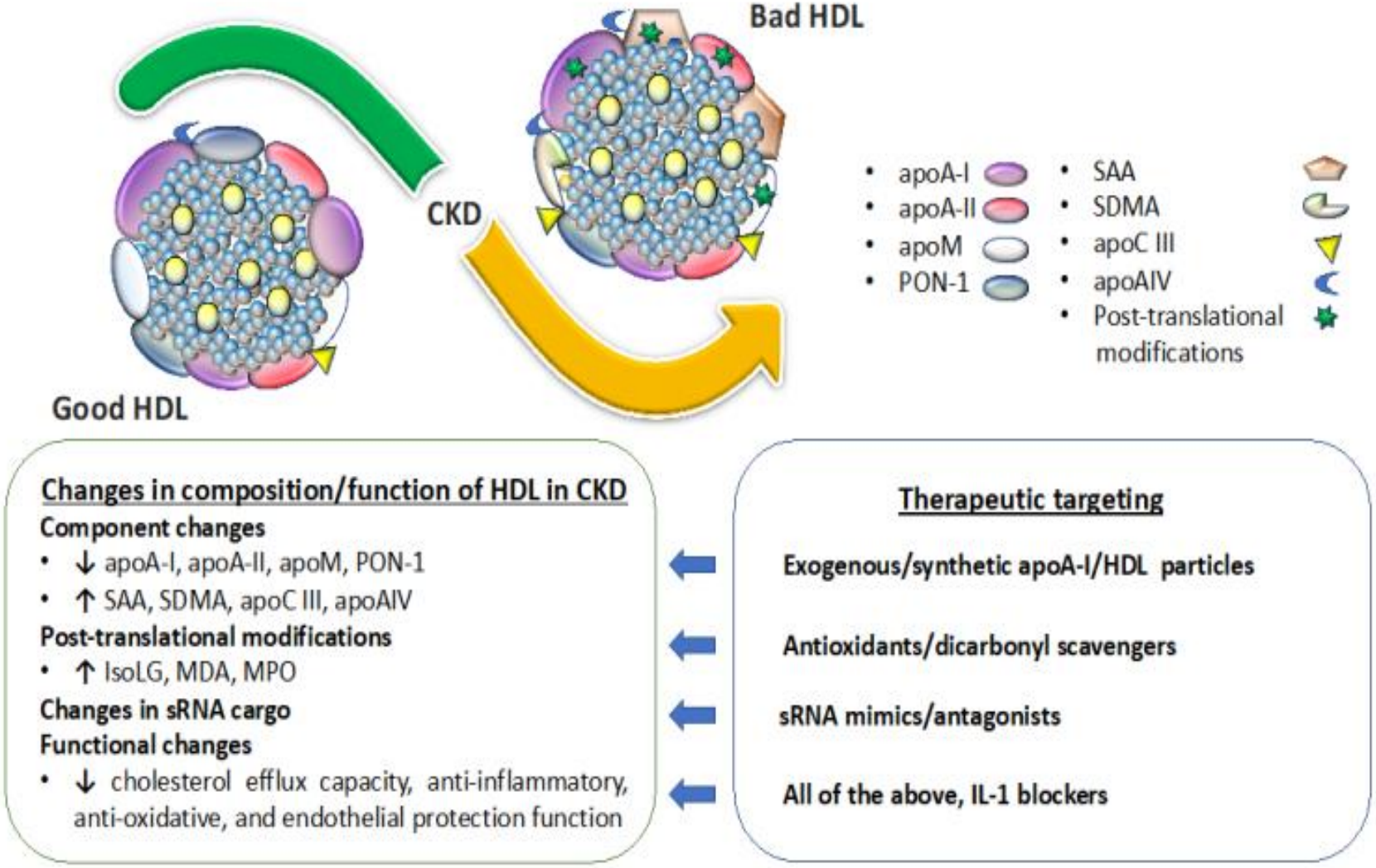
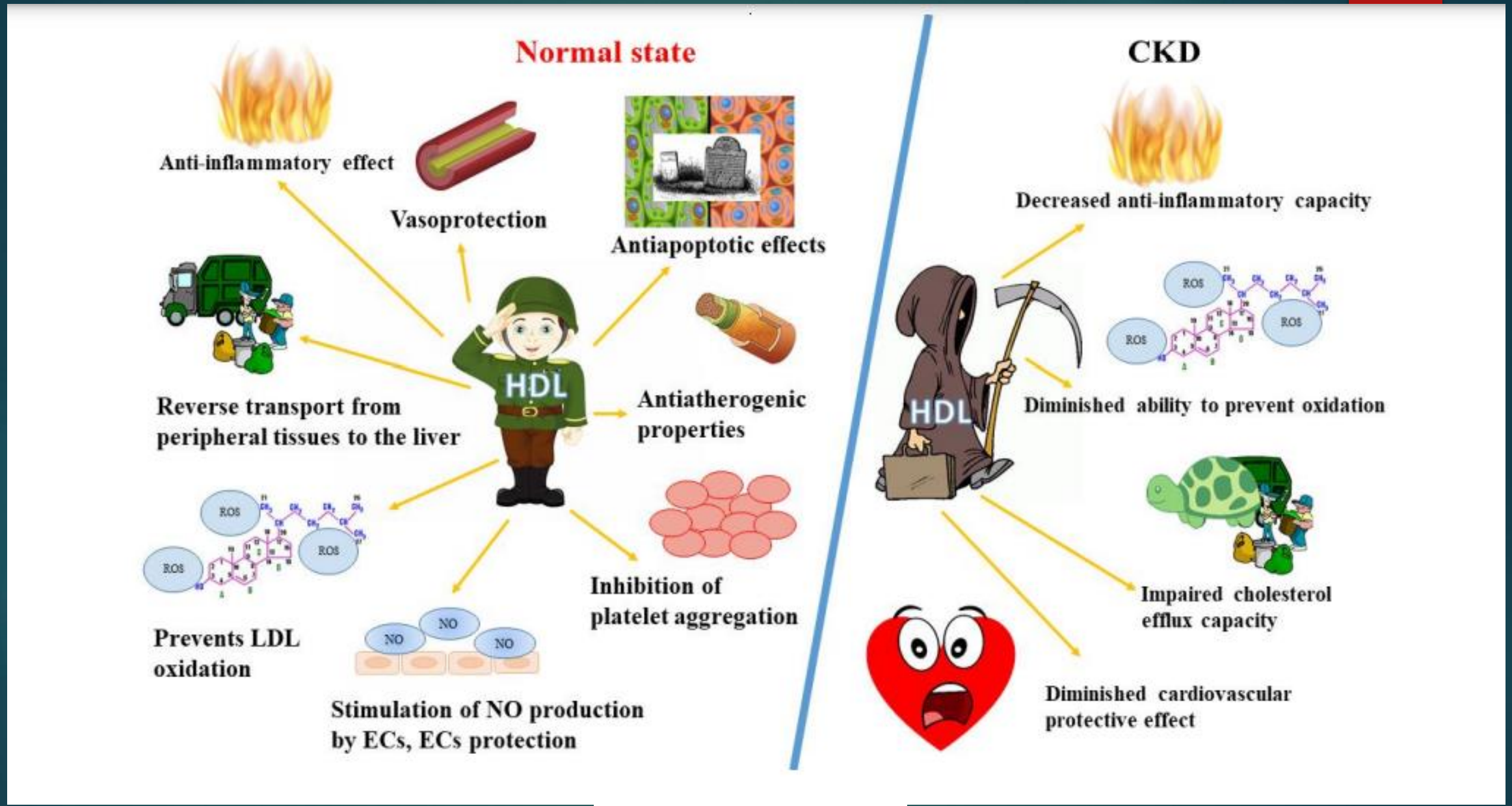


Figure 2. The effects of CKD on HDL structure and function, and the therapeutic implications.

https://doi.org/10.3390/ijms22158201



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Alzheimer's & Dementia
Diagnosis, Assessment & Disease Monitoring

RESEARCH ARTICLE

The relationship between midlife dyslipidemia and lifetime incidence of dementia: A systematic review and meta-analysis of cohort studies

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Abstract

Introduction: We conducted a systematic review and meta-analysis to review the relationship between midlife dyslipidemia and lifetime incident dementia.

Methods: The databases Medline, Embase, Scopus, Web of Science, and Cochrane were searched from inception to February 20, 2022. Longitudinal studies examining the relationship between midlife lipid levels on dementia, dementia subtypes, and/or cognitive impairment were pooled using inverse-variance weighted random-effects meta-analysis.

Results: Seventeen studies (1.2 million participants) were included. Midlife hypercholesterolemia was associated with increased incidence of mild cognitive impairment (effect size [ES] = 2.01; 95% confidence interval [CI] 1.19 to 2.84; $I^2 = 0.0\%$) and all-cause dementia (ES = 1.14; 95% CI: 1.07 to 1.21; $I^2 = 0.0\%$). Each 1 mmol/L increase in low-density lipoprotein was associated with an 8% increase (ES = 1.08, 95% CI: 1.03 to 1.14; $I^2 = 0.3\%$) in incidence of all-cause dementia.

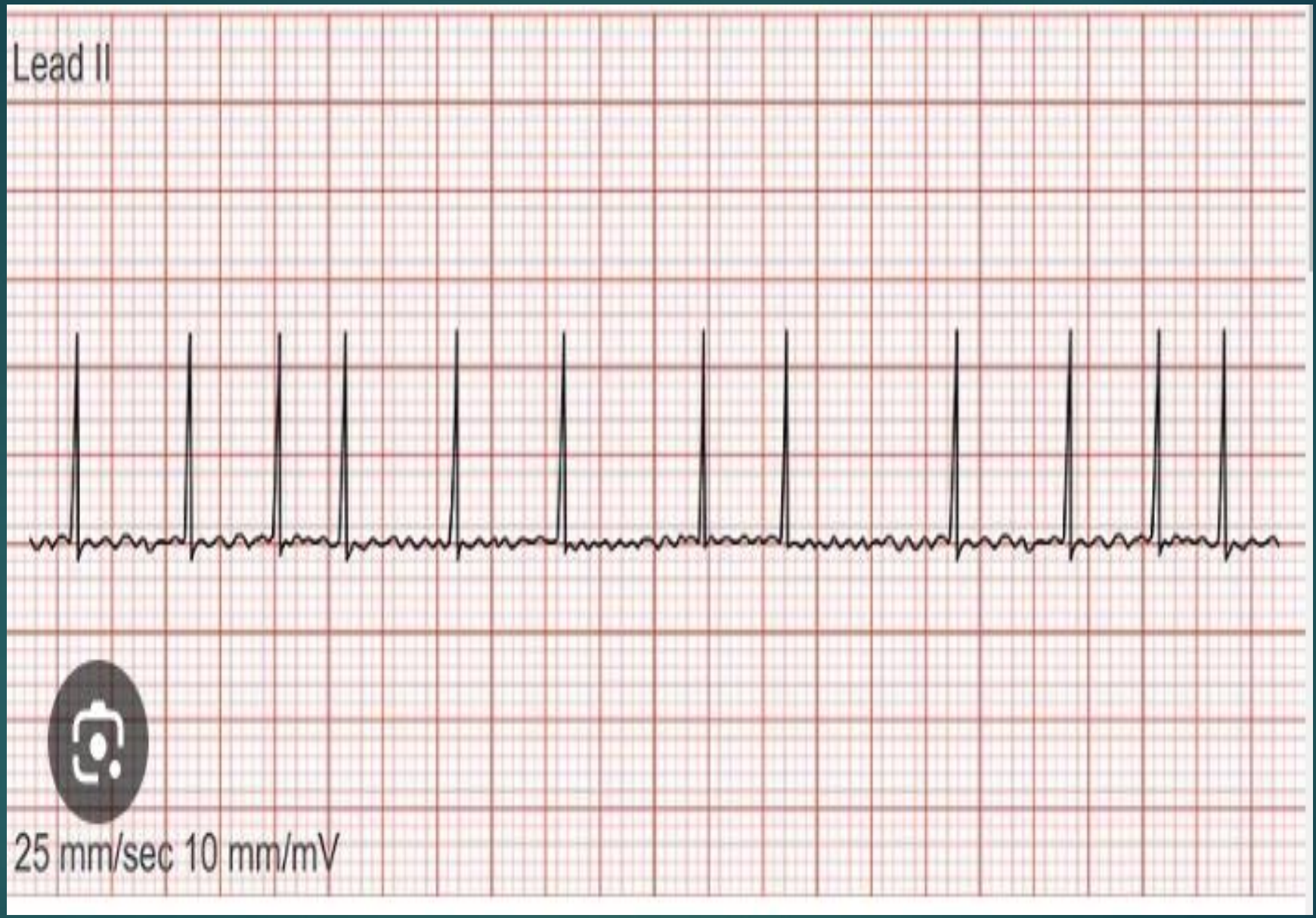
Discussion: Midlife dyslipidemia is associated with an increased risk of cognitive impairment in later life.

KEYWORDS

aging, cholesterol, cognitive, cognitive dysfunction, dementia, dyslipidemia, midlife

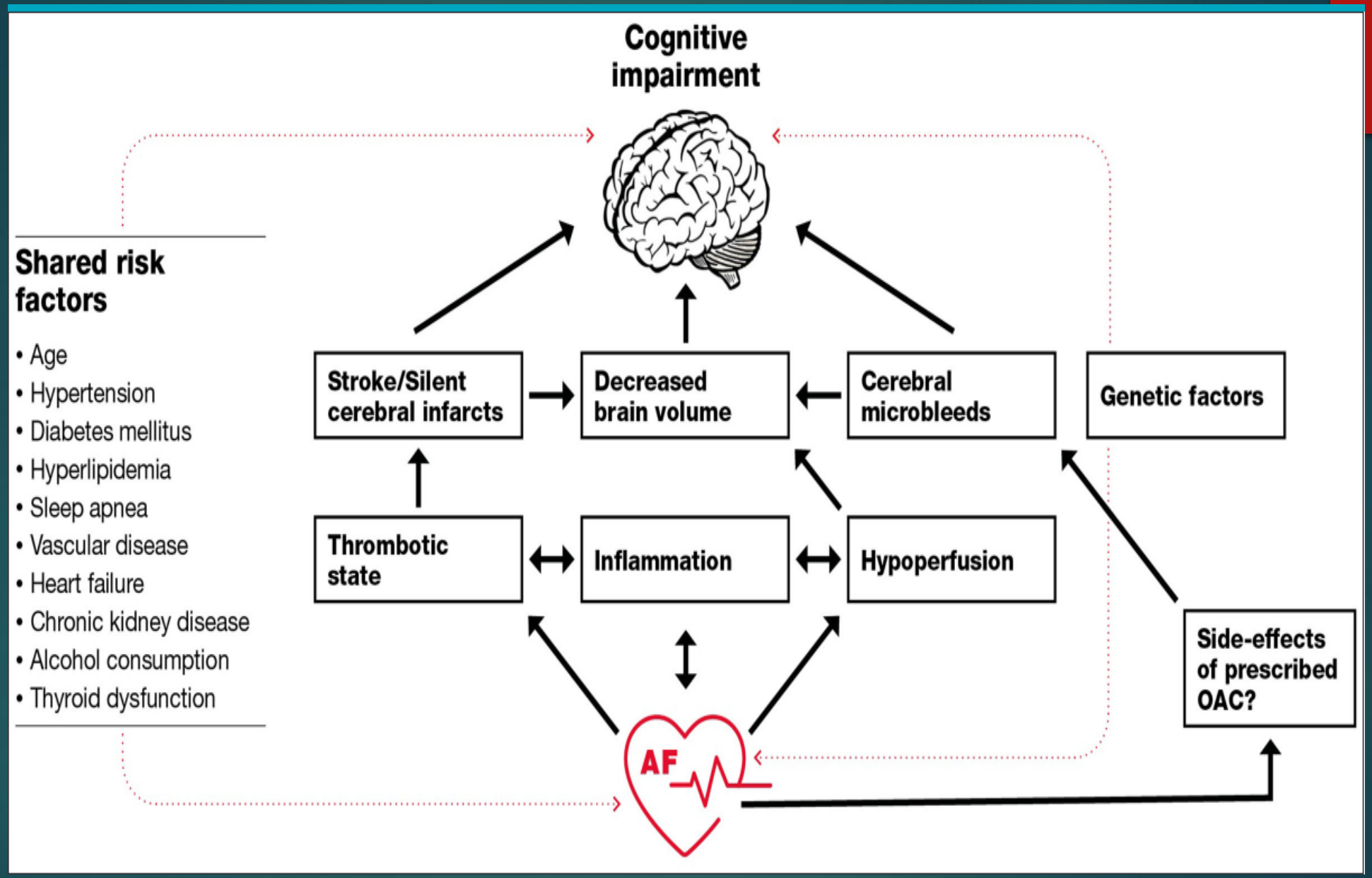
ΚΟΛΠΙΚΗ ΜΑΡΜΑΡΥΓΗ ΚΑΙ ΓΝΩΣΙΑΚΗ ΔΥΣΛΕΙΤΟΥΡΓΙΑ

- Η πιο συχνή καρδιακή αρρυθμία.
- Ακανόνιστος καρδιακός ρυθμός ο οποίος μπορέι να είναι πολύ γρήγορος.
- Επεισόδια απότομης έναρξης και ανάταξης.(παροξυσμική κολπική μαρμαρυγή) ή συνεχής (μόνιμη κολπική μαρμαρυγή).


Lead II
25 mm/sec 10 mm/mV

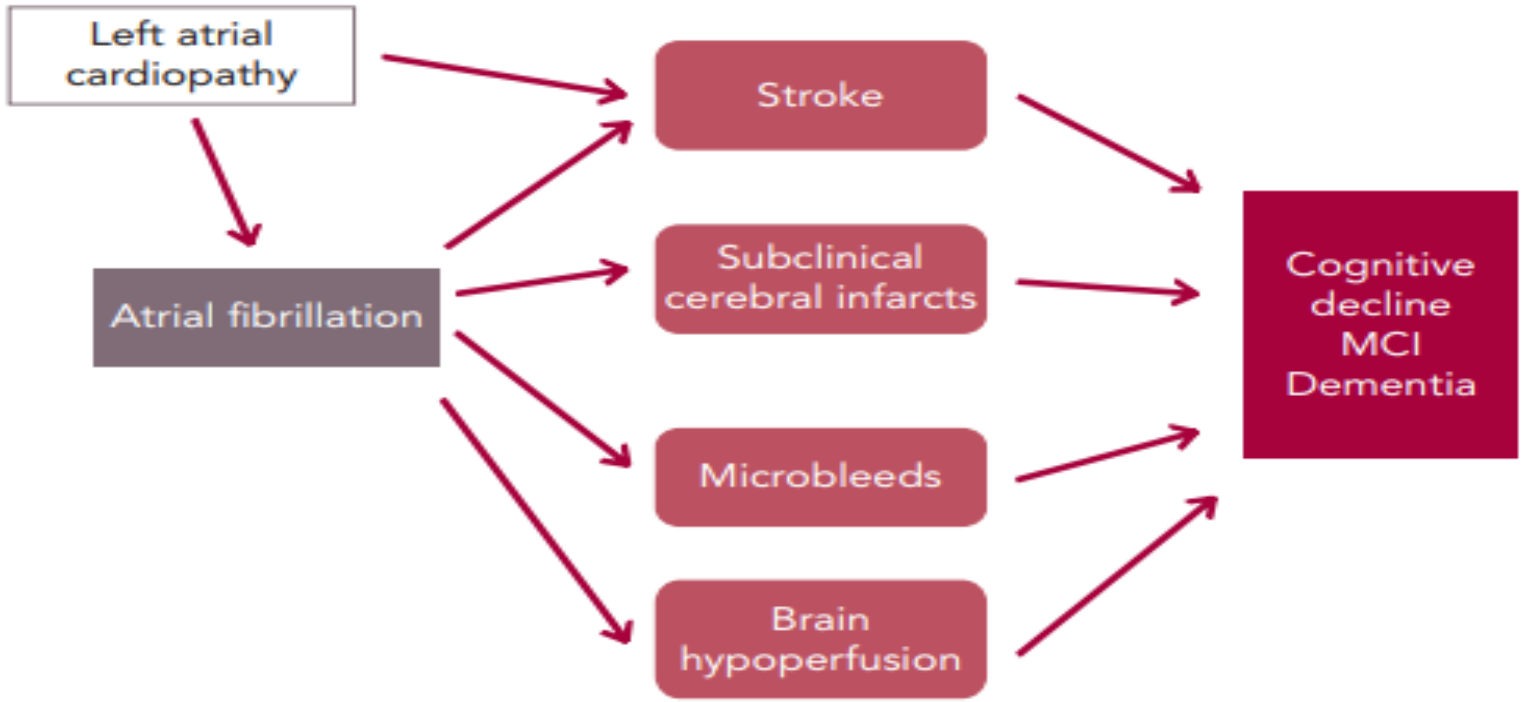
ΣΥΜΠΤΩΜΑΤΑ

- Αίσθημα παλμών
- Δύσπνοια
- Θωρακικό άλγος
- Απώλεια αισθήσεων.
- Ζάλη
- Κόπωση
- Θρομβοεμβολικά επεισόδια



https://doi.org/10.1161/CIRCULATIONAHA.121.055018

Figure 1: Potential Mechanisms Linking Atrial Fibrillation (AF) with Cognitive Decline and Dementia



AF increases the risk of stroke and subclinical cerebral infarcts and can lead to brain hypoperfusion. Oral anticoagulation in patients with AF can lead to new or worsening cerebral microbleeds. In turn, these conditions lead to cognitive decline and increased risk of mild cognitive impairment (MCI) and dementia. Independently, left atrial cardiopathy increases the risk of AF and is associated with stroke risk independent of AF.

https://doi.org/10.15420/ecr.2016:13:2

ESC European Society of Cardiology

Europace (2022) **24**, 1229–1239
https://doi.org/10.1093/europace/euac003

CLINICAL RESEARCH
Atrial fibrillation

Predictive role of atrial fibrillation in cognitive decline: a systematic review and meta-analysis of 2.8 million individuals

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Aims To systematic review and meta-analyse the association and mechanistic links between atrial fibrillation (AF) and cognitive impairment.

Methods and results PubMed, EMBASE, and Cochrane Library were searched up to 27 March 2021 and yielded 4534 citations. After exclusions, 61 were analysed; 15 and 6 studies reported on the association of AF and cognitive impairment in the general population and post-stroke cohorts, respectively. Thirty-six studies reported on the neuro-pathological changes in patients with AF; of those, 13 reported on silent cerebral infarction (SCI) and 11 reported on cerebral microbleeds (CMB). Atrial fibrillation was associated with 39% increased risk of cognitive impairment in the general population [$n = 15$: 2 822 974 patients; hazard ratio = 1.39; 95% confidence interval (CI) 1.25–1.53, $I^2 = 90.3\%$; follow-up 3.8–25 years]. In the post-stroke cohort, AF was associated with a 2.70-fold increased risk of cognitive impairment [adjusted odds ratio (OR) 2.70; 95% CI 1.66–3.74, $I^2 = 0.0\%$; follow-up 0.25–3.78 years]. Atrial fibrillation was associated with cerebral small vessel disease, such as white matter hyperintensities and CMB ($n = 8$: 3698 patients; OR = 1.38; 95% CI 1.11–1.73, $I^2 = 0.0\%$), SCI ($n = 13$: 6188 patients; OR = 2.11; 95% CI 1.58–2.64, $I^2 = 0\%$), and decreased cerebral perfusion and cerebral volume even in the absence of clinical stroke.

Conclusion Atrial fibrillation is associated with increased risk of cognitive impairment. The association with cerebral small vessel disease and cerebral atrophy secondary to cardioembolism and cerebral hypoperfusion may suggest a plausible link in the absence of clinical stroke. PROSPERO CRD42018109185.

Γνωσιακή δυσλειτουργία και καρδιακή ανεπάρκεια

Heart Failure

- Cerebral hypoperfusion and impaired autoregulation
- Systemic inflammation
- Proteotoxicity from protein misfolding*
- Thrombo-embolic disease and infarction

Brain Changes

Cognitive	Anatomical
Impairments in Memory, Executive function, Psychomotor speed etc.	Total and regional grey matter atrophy, Increased white matter lesions

Cognitive impairment in heart failure associated with poorer outcomes
Reduced self-care, Reduced functional independence, Increased rehospitalisation, Increased mortality

Figure 1. Summary of the reported pathophysiology, brain changes and impact of cognitive impairment in heart failure. *Proteotoxicity may be a shared disease pathology between specific cardiomyopathies and CI.

https://doi.org/10.3390/biology11020179

ΣΥΜΠΕΡΑΣΜΑΤΑ

- ΚΑΡΔΙΑ-ΕΓΚΕΦΑΛΟΣ- ΝΕΦΡΟΣ
- ΑΠΑΡΑΙΤΗΤΗ Η ΣΥΝΕΡΓΑΣΙΑ ΟΛΩΝ ΤΩΝ ΙΑΤΡΙΚΩΝ ΕΙΔΙΚΟΤΗΤΩΝ ΠΑΝΤΑ ΜΕ ΣΤΟΧΟ ΤΟ ΟΦΕΛΟΣ ΤΟΥ ΑΣΘΕΝΟΥΣ