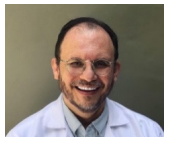
RIESGO CARDIOVASCULAR ABC DEL MANEJO HIPERTENSIÓN Y NEFROPROTECCIÓN

Miguel Urina-Triana PhD

PRESENTACION DR.MIGUEL URINA TRIANA



- 1981**- Médico General** y 1985**- Especialista en Medicina Interna** de la Pontificia Universidad Javeriana, Bogotá, Colombia; 1988- **Especialista en Cardiología** del Instituto Nacional de Cardiología 'Ignacio Chávez" y Universidad Nacional Autónoma de México, México DF; 1990**-Especialista en Diseño y Evaluación de Proyectos** y en 2001 **Gerencia en Servicios de Salud** de la Universidad del Norte, Barranquilla, Colombia;**2010**- **Magister en ensayos clínicos** de la Universidad de Sevilla España; **2020**-**Doctorado (PhD) en Investigación y Docencia** de la Universidad Centro Panamericano de Estudios Superiores, México
- **Investigador Senior** de Min ciencias y líder de grupo categoría A1; 2003-Actual **Profesor de la Facultad de Ciencias de la Salud** y 2021-Actual **Director del Programa de Especialización en Cardiología de la Universidad Simón Bolívar**
- 2001-2003 **Presidente nacional de la Sociedad Colombiana de Cardiología y Cirugía Cardiovascular** y de la **Fundación Colombiana del Corazón**; 2004-**FACC** y 2017-2019 **Gobernador del Capítulo de Colombia del American College of Cardiology**;2017-2019 **Member of International Heart Failure Working Group at ACC**; 2010 **Miembro Emérito de la Sociedad Colombiana de Cardiología y Cirugía Cardiovascular (SCC)**; 2014 **Miembro Emérito de la Asociación Colombiana Medicina Interna (ACMI**); 2010- **Miembro honorario de la Federación Diabetológica Colombiana** (FDC); 2015- **Miembro de la Sociedad latinoamericana de Hipertensión Arterial(LASH)**; 2016- **Member European Society of Cardiology(ESC) Council on Hypertension**; 2009- **Regular Member Heart Failure Association of ESC**; 2020 **Miembro Asociado Sociedad Española de Cardiología (SEC)**; 1988- **Miembro correspondiente de la Sociedad Mexicana de Cardiología**; 1988**-Miembro de SIBIC internacional;**2019-2022 **Miembro del Comité de la Junta**, **Sociedad Internacional de Aterosclerosis (IAS), Comité de la Junta Ejecutiva de la Federación Regional para las Américas**
- 2003- **Arbitro Par de la Revista Colombiana de Cardiología**; 2006- **Miembro comité editorial de la Revista Argentina de insuficiencia cardiaca;**2017- **Miembro del Comité Consultivo Científico de la Revista Colombiana de Nefrología**; 2019- **Miembro del Consejo Editorial / Editor de Sección / Editor Asociado, Revista Colombiana de Cardiología**; 2019- **Editorial Board Member and Reviewer BMC Cardiovascular Disorders**; 2019- **Member Reviewer Journal of Medical Case Reports;** 2020- **Member Reviewer of Evidence-Based Complementary and Alternative Medicine**
- Director de la Fundación del Caribe para la Investigación Biomédica (FUNDACION BIOS)

Conflicto de intereses declarado

Recibido por sí mismo o través de la Fundación Bios, apoyo financiero para la realización de estudios clínicos y/o honorarios como investigador, conferencista o “advisory board” de Abbott, Astra-Zeneca ,Bayer SA,, Boeringher Ingelheim, Bristol-Myers Squibb, Frosst Laboratories, Johnson and Johnson, Menarini, Novartis, Novonordisk, Pfizer, Procaps, Sanofi-Aventis, Servier, Tecnofarma

Objetivos de esta charla

- 1. Definir riesgo y revisar aspectos epidemiológicos de la HTA. Concepto de nefro-protección
- 2. Revisar algunos aspectos fisiopatológicos de la HTA importantes
- 3. Revisar aspectos terapéuticos de la HTA. Medicación dual y nefro protección

¿Qué es riesgo?

Contingencia o proximidad de un daño. Es una medida de la magnitud de los daños frente a una situación peligrosa

Urina-Triana MA, Urina-Jassir D, Urina-Jassir MA, Urina-Triana ME. Capitulo II. Valoración y estratificación del riesgo cardiovascular para la toma de decisiones. En el libro de Rehabilitación Cardiaca. Ed. Universidad Simón Bolívar Marzo 2017 ISBN 978-958-8930-72-5 pag 49-98

¿Qué es un Factor de riesgo?

Desde el punto de vista epidemiológico un ***factor de riesgo*** es una condición o característica de un individuo o población que está presente en forma temprana en la vida y se asocia con un riesgo aumentado de desarrollar una enfermedad futura.

Puede ser un comportamiento o hábito (ej. fumar, sedentarismo), un rasgo hereditario (historia familiar), una variable paraclínica (nivel sérico elevado de colesterol).

Para ser considerado causal el marcador en cuestión **debe preceder el comienzo de la enfermedad y tener plausibilidad biológica.**

Urina-Triana MA, Urina-Jassir D, Urina-Jassir MA, Urina-Triana ME. Capitulo II. Valoración y estratificación del riesgo cardiovascular para la toma de decisiones. En el libro de Rehabilitación Cardiaca. Ed. Universidad Simón Bolívar Marzo 2017 ISBN 978-958-8930-72-5 pág. 49-98

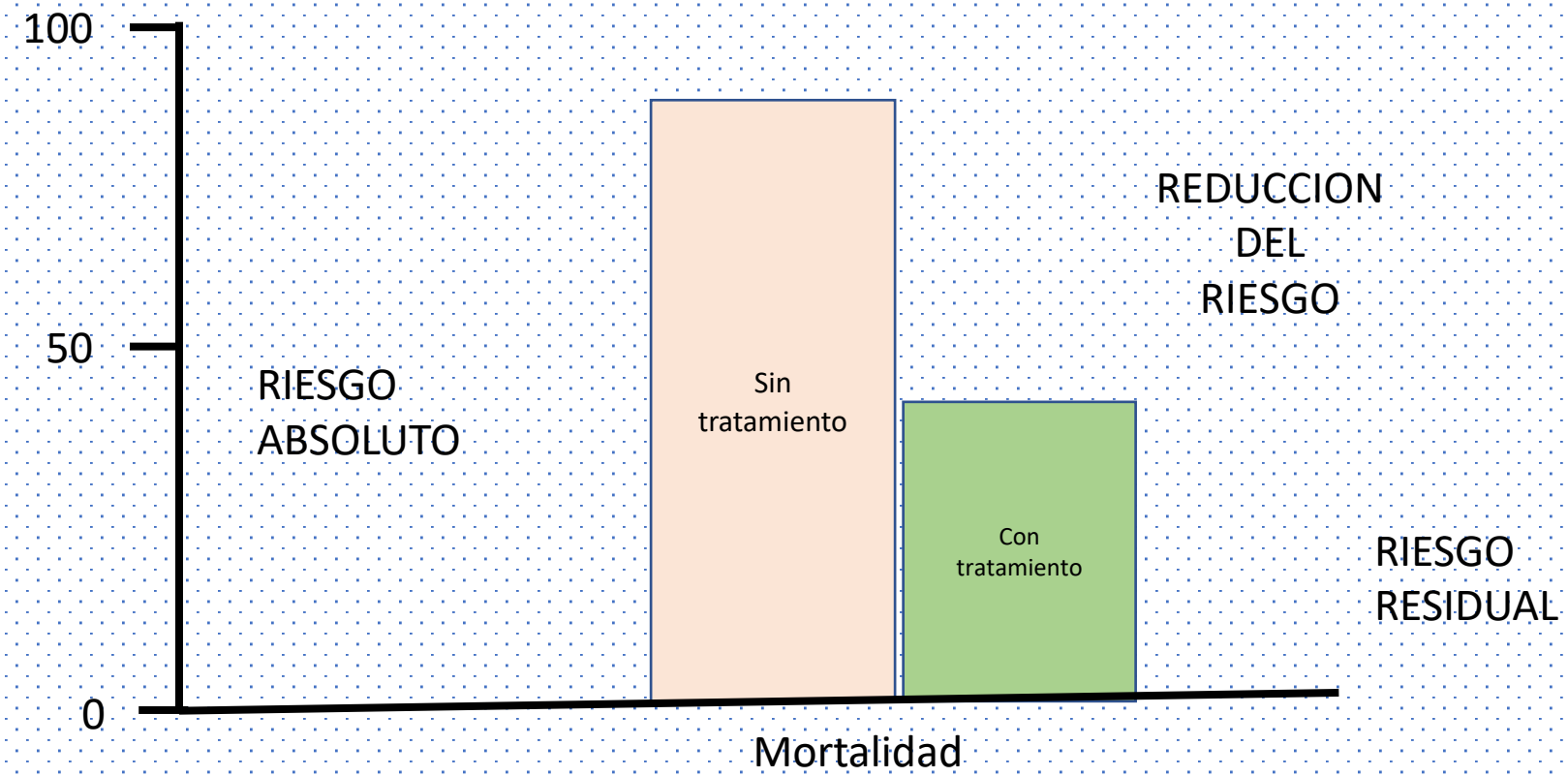
¿ PARA QUE SIRVE CALCULAR EL RIESGO?

1.- El objetivo fundamental es ayudarnos en la toma decisiones para la intervención con cambios en el estilo de vida, tratamiento con fármacos, o con otra medidas ej. Cirugía metabolica.

2.- Para “motivar” a los pacientes: podemos “mostrar” a los pacientes lo que pasaría en un futuro si modificara sus factores de riesgo.

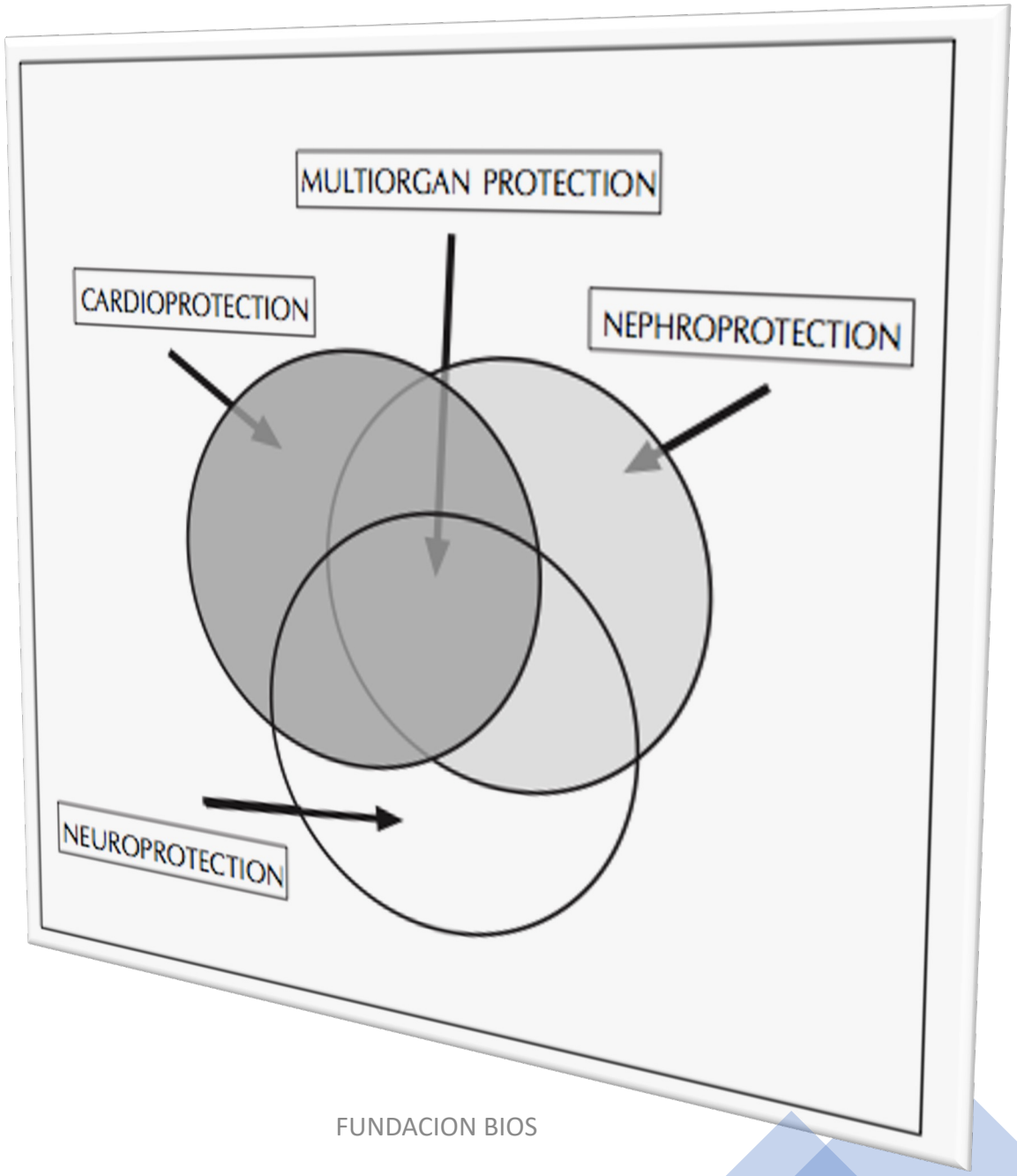
Urina-Triana MA, Urina-Jassir D, Urina-Jassir MA, Urina-Triana ME. Capitulo II. Valoración y estratificación del riesgo cardiovascular para la toma de decisiones. En el libro de Rehabilitación Cardiaca. Ed. Universidad Simón Bolívar Marzo 2017 ISBN 978-958-8930-72-5 pág. 49-98

FORMAS DE EXPRESAR EL RIESGO



Fuente: Urina-Triana M.

Concepto de nefroprotección


MULTIORGAN PROTECTION
CARDIOPROTECTION
NEPHROPROTECTION
NEUROPROTECTION

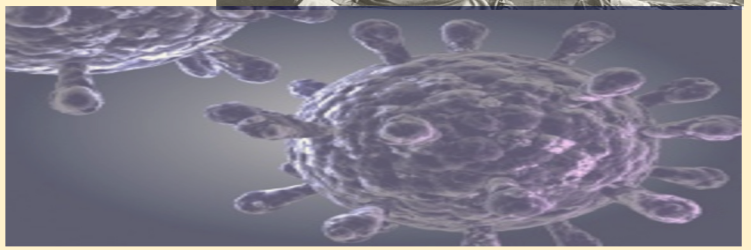
Aspectos epidemiológicos

LAS PEORES EPIDEMIAS

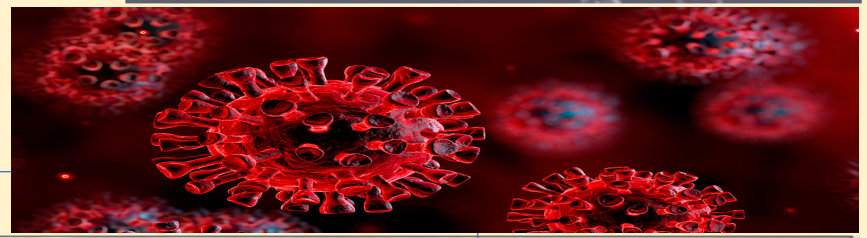
La Peste bubónica 1600

La Influenza 1919

EL SIDA 1990

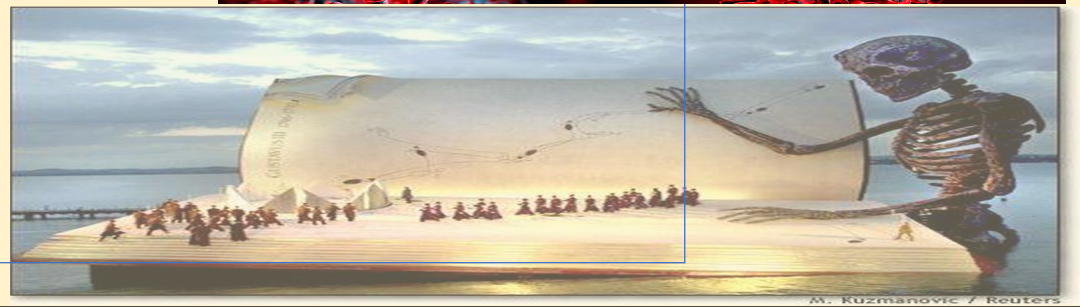


SARS-Cov-2 2020-actual

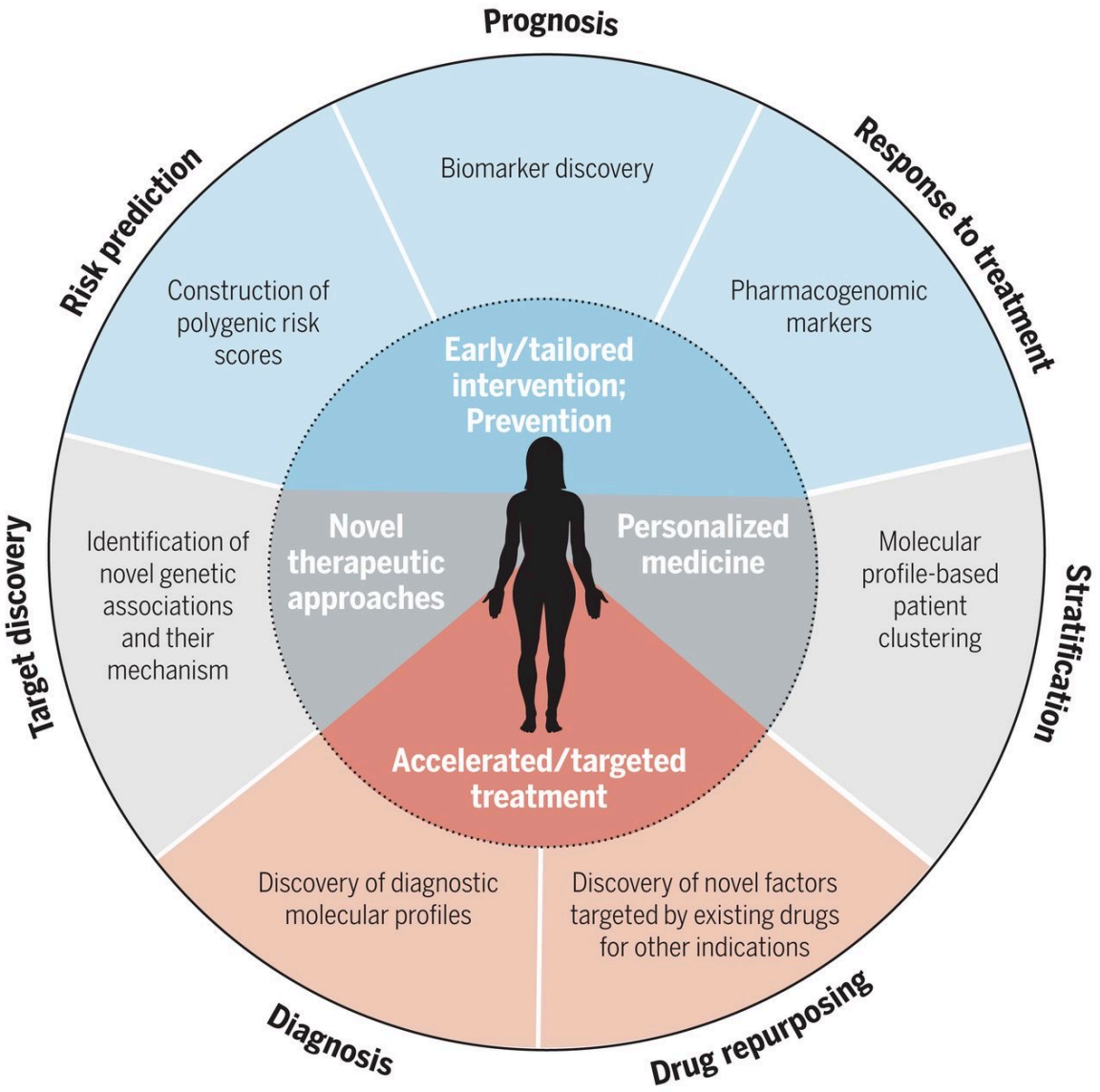


2023 las ECNT (**HTA**, DM, IRC): siglo XXI.

SERAN PRINCIPAL CAUSA DE MUERTE Y DISCAPACIDAD



¿DONDE ESTAMOS AHORA?



Aunque la medicina de precisión es muy prometedora, el verdadero potencial de este enfoque solo se realizará cuando los estudios de eficacia y efectividad demuestren que su aplicación reduce la incidencia de enfermedades cardíacas y accidentes cerebrovasculares, y mejora la salud cardiovascular de las poblaciones

https://doi.org/10.1126/science.aax4588

La magnitud del problema

¿La HTA una epidemia?

LA POBLACION CRECE

8.054.719.100 a las 12:00 pm 16 agosto 2023

LA POBLACION ENVEJECE

Naciones Unidas | Departamento de Asuntos Económicos y Sociales

Nota de Prensa

Sujeto a embargo hasta el 11 de julio de 2022, 12:01 am EDT

La población mundial llegará a 8.000 millones el 15 de noviembre de 2022

Mientras siguen cayendo las tasas de crecimiento poblacional, se proyecta que la población mundial alcanzará un máximo de 10.400 millones en la década de 2080

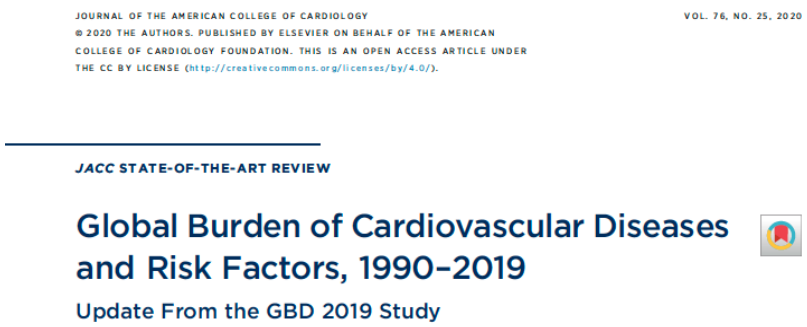
Nueva York, 11 de julio – Se prevé que la población mundial alcanzará los 8.000 millones el 15 de noviembre de 2022, y que India superará a China como el país más poblado del mundo en 2023, según el *World Population Prospects 2022*, publicado hoy en el Día Mundial de la Población.

+ Hipertensión arterial sistémica

https://www.worldometers.info/es/poblacion-mundial/#:~:text=Población%20Mundial%3A%207.9%20Billones%20de%20Personas%20(2023)%20%2D%20Worldometer

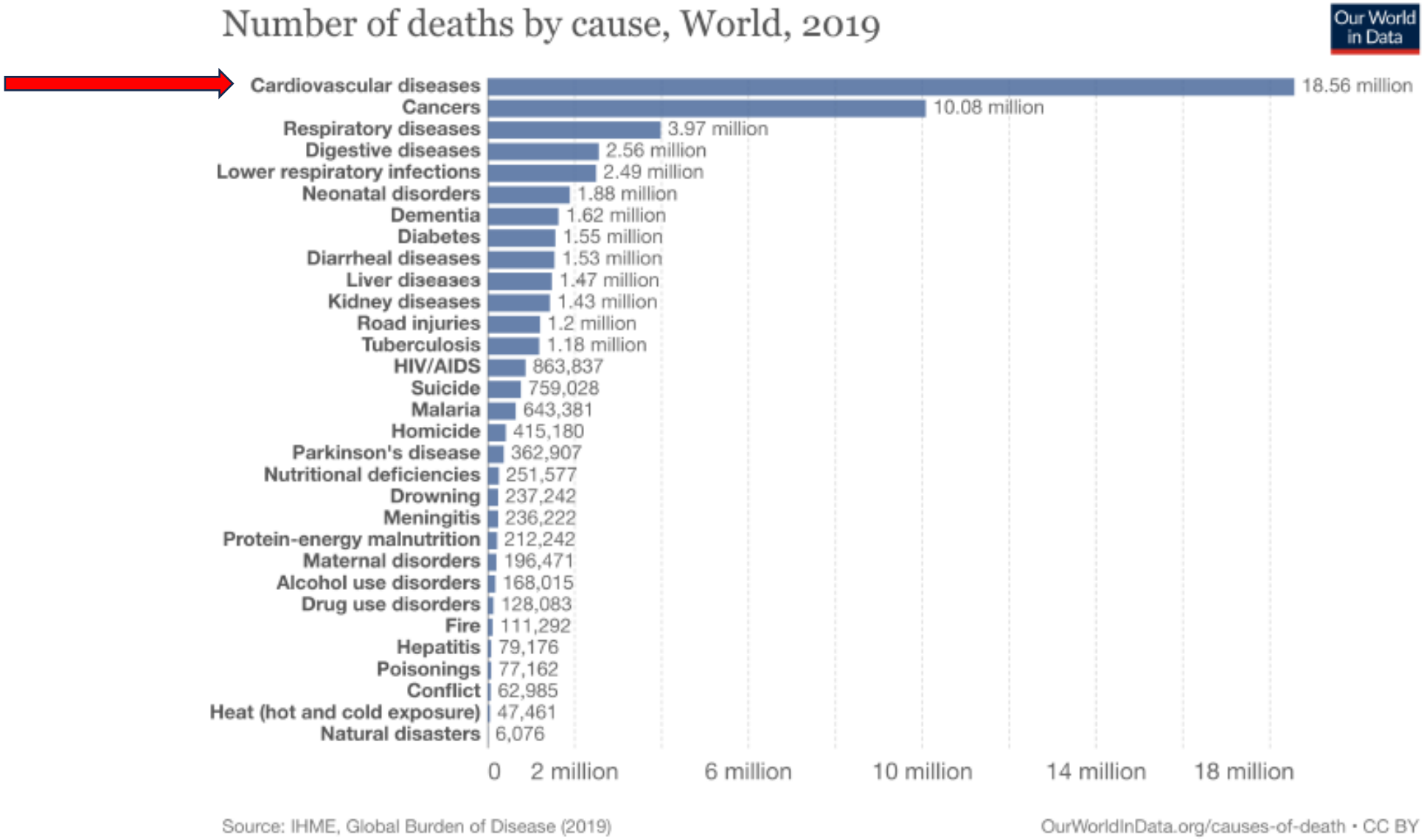
CARGA GLOBAL DE ENFERMEDADES CV Y FR

Roth, G.A. et al. J Am Coll Cardiol. 2020;76(25):2982-3021.

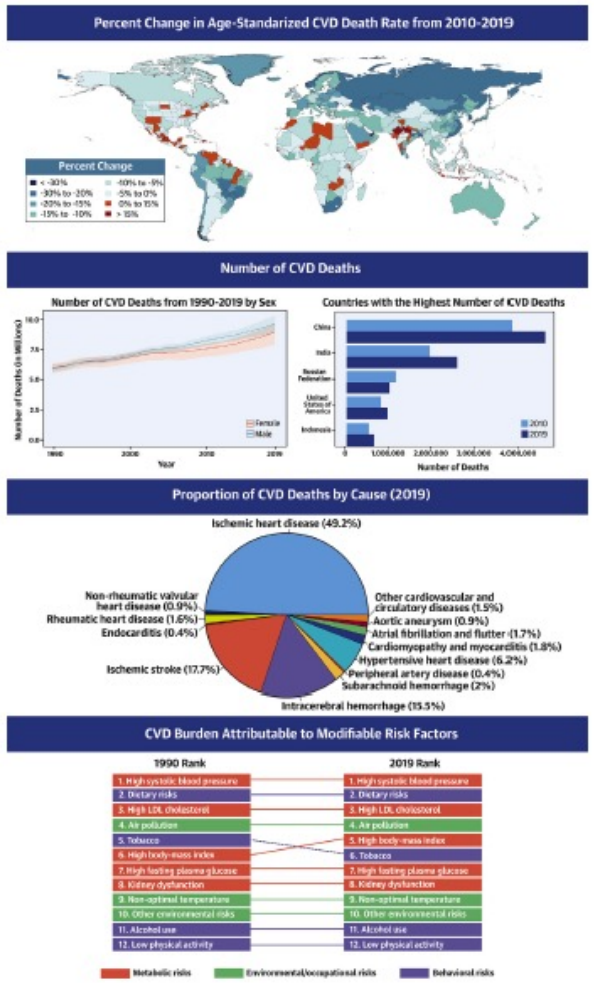


Este documento revisa la magnitud de la carga total de Enfermedad Cardiovascular (ECV), incluyendo 13 causas subyacentes de muerte cardiovascular y 9 factores de riesgo relacionados, utilizando estimaciones de la Estudio de la Carga Global de Morbilidad (GBD) 2019. GBD, una colaboración multinacional continua para proporcionar estimaciones comparables y consistentes de la salud de la población a lo largo del tiempo, utilizando todos los datos disponibles a nivel de población Fuentes sobre incidencia, prevalencia, letalidad, mortalidad y riesgos para la salud para producir estimaciones para 204 países y territorios de 1990 a 2019.

El estudio produce medidas epidemiológicas estándar, tales como: incidencia, prevalencia y tasas de mortalidad, así como medidas resumidas de salud, como los *DALYs*.


Number of deaths by cause, World, 2019
Our World in Data
Cardiovascular diseases 18.56 million
Cancers 10.08 million
Respiratory diseases 3.97 million
Digestive diseases 2.56 million
Lower respiratory infections 2.49 million
Neonatal disorders 1.88 million
Dementia 1.62 million
Diabetes 1.55 million
Diarrheal diseases 1.53 million
Liver diseases 1.47 million
Kidney diseases 1.43 million
Road injuries 1.2 million
Tuberculosis 1.18 million
HIV/AIDS 863,837
Suicide 759,028
Malaria 643,381
Homicide 415,180
Parkinson's disease 362,907
Nutritional deficiencies 251,577
Drowning 237,242
Meningitis 236,222
Protein-energy malnutrition 212,242
Maternal disorders 196,471
Alcohol use disorders 168,015
Drug use disorders 128,083
Fire 111,292
Hepatitis 79,176
Poisonings 77,162
Conflict 62,985
Heat (hot and cold exposure) 47,461
Natural disasters 6,076
0
2 million
6 million
10 million
14 million
18 million
Source: IHME, Global Burden of Disease (2019)
OurWorldInData.org/causes-of-death • CC BY

CENTRAL ILLUSTRATION Cardiovascular Disease Burden Across Time, Location, Cause, and Risk Factor

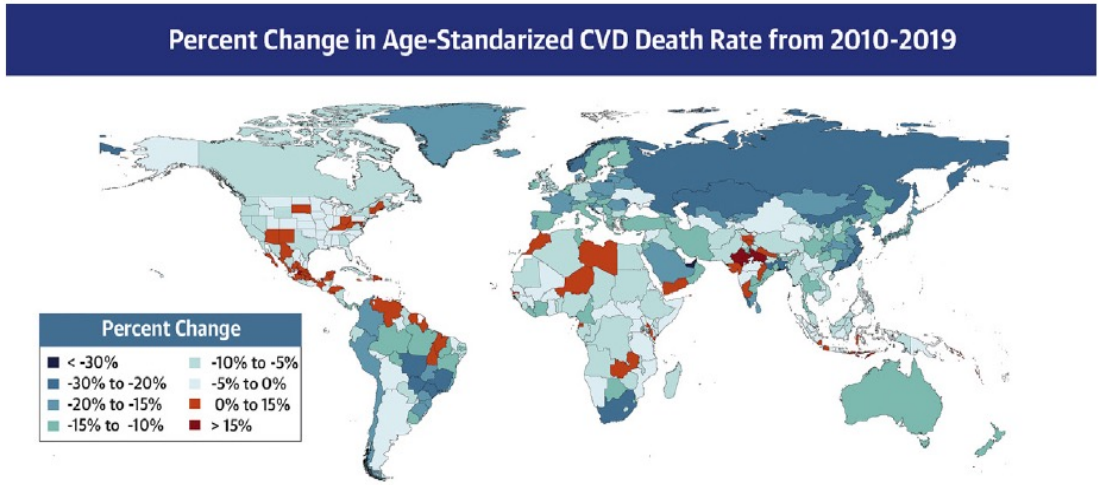


Roth, G.A. et al. J Am Coll Cardiol. 2020;76(25):2982-3021.

Percent Change in Age-Standardized CVD Death Rate from 2010-2019. Map of the percent change in age-standardized CVD mortality rate from 2010 to 2019. **Number of CVD Deaths.** Total number of deaths due to CVD by sex, 1990 to 2019; total number of deaths due to CVD in 2010 and 2019 among the countries with the highest number of CVD deaths in 2019. **Proportion of CVD Deaths by Cause (2019).** Proportion of total CVD deaths in 2019 by underlying causes. **CVD Burden Attributable to Modifiable Risk Factors.** Comparison of the rankings of CVD DALYs attributable to modifiable risk factors in 1990 and 2019. CVD = cardiovascular disease; DALYs = disability-adjusted life years; LDL = low-density lipoprotein.

Usemos una lupa

Roth, G.A. et al. J Am Coll Cardiol. 2020;76(25):2982-3021.



Prevalencia de ECV
Variación 1990 a 2019

1990
271 millones

2019
523 millones

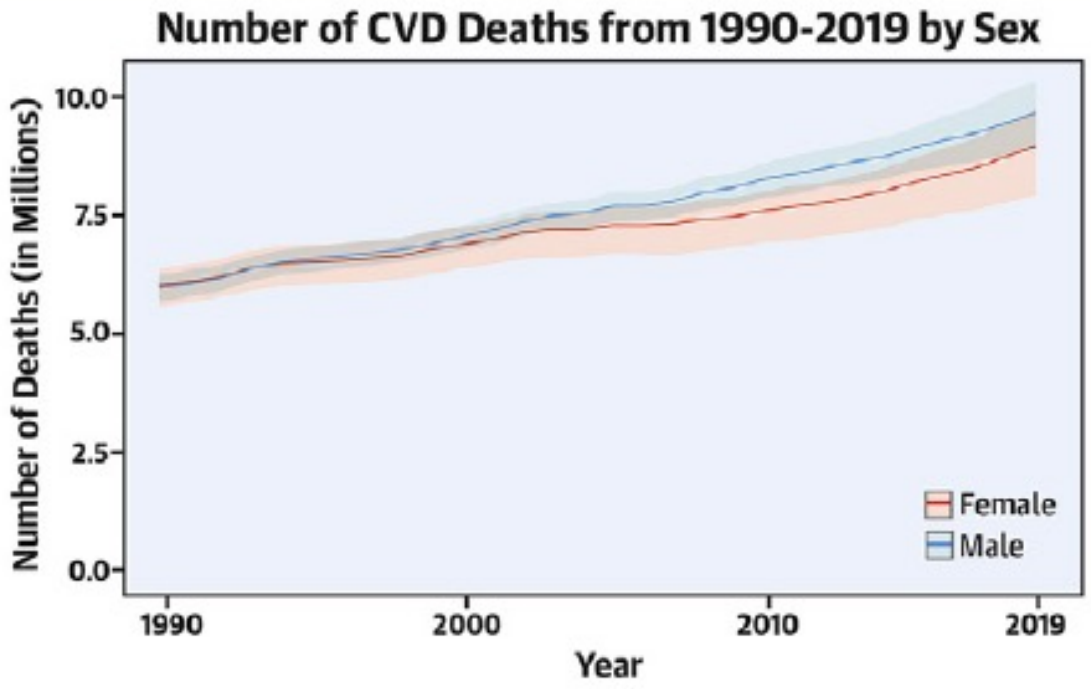
Mortalidad por ECV
Variación 1990-2019

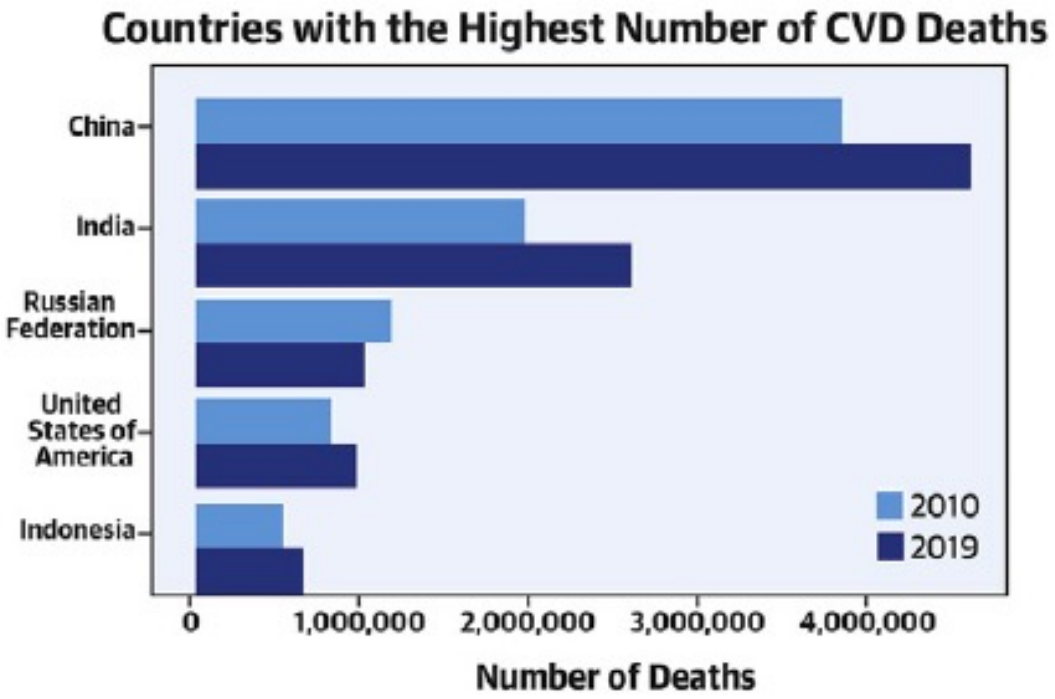
1990
12.1 millones

2019
18.6 millones

Roth, G.A. et al. J Am Coll Cardiol. 2020;76(25):2982-3021.

Number of CVD Deaths





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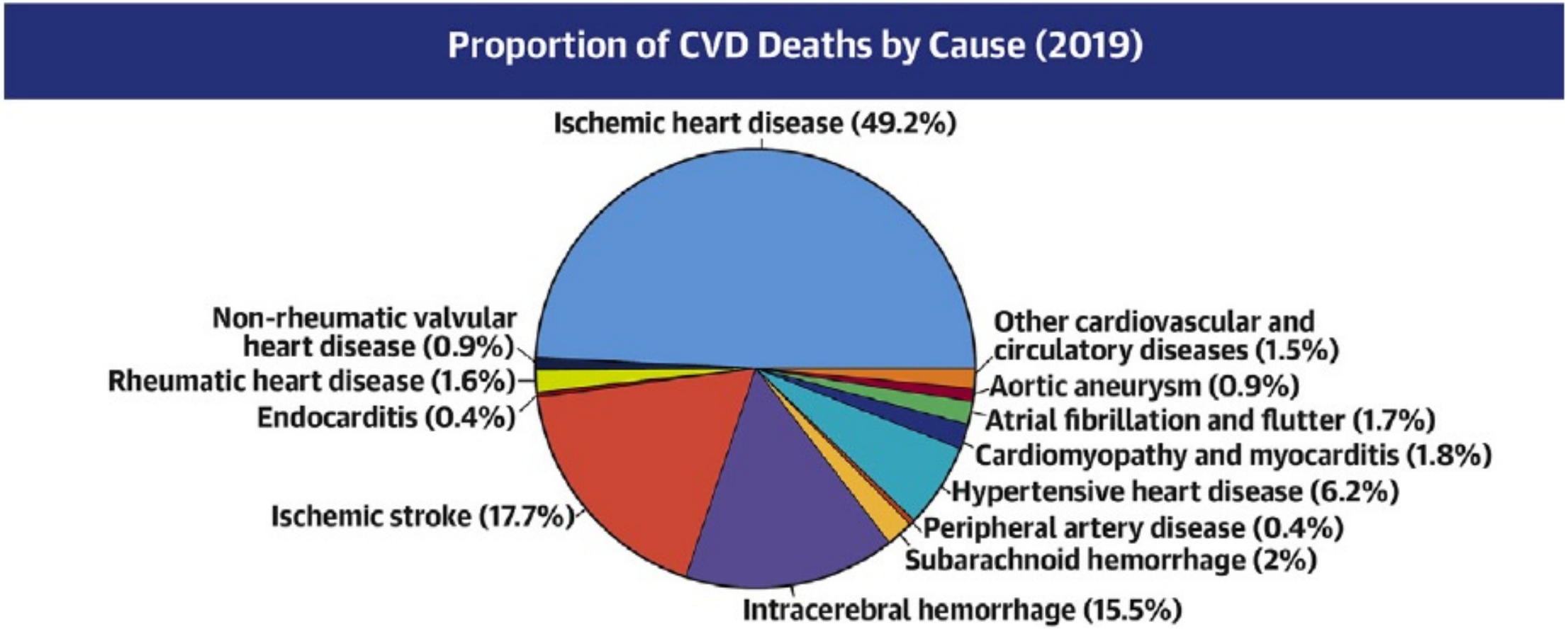

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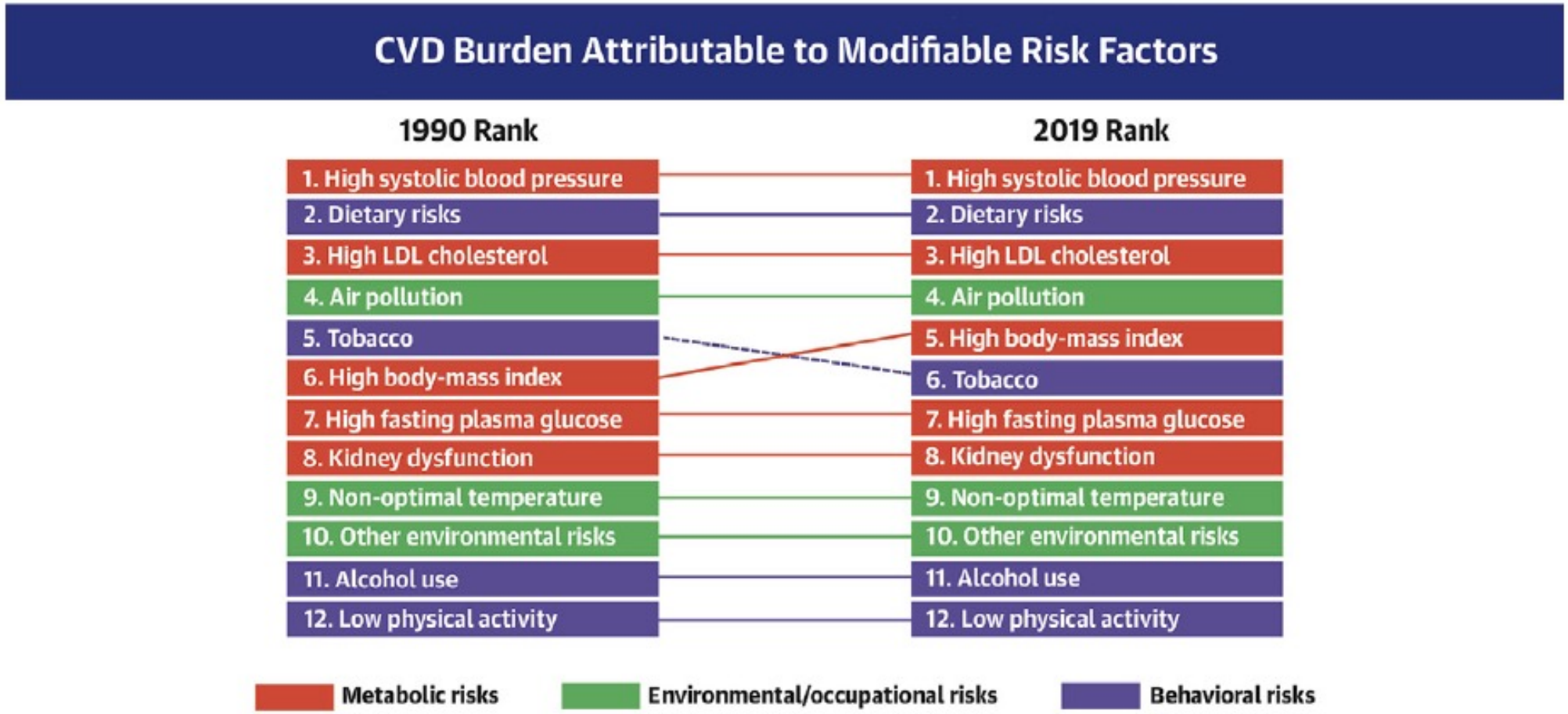


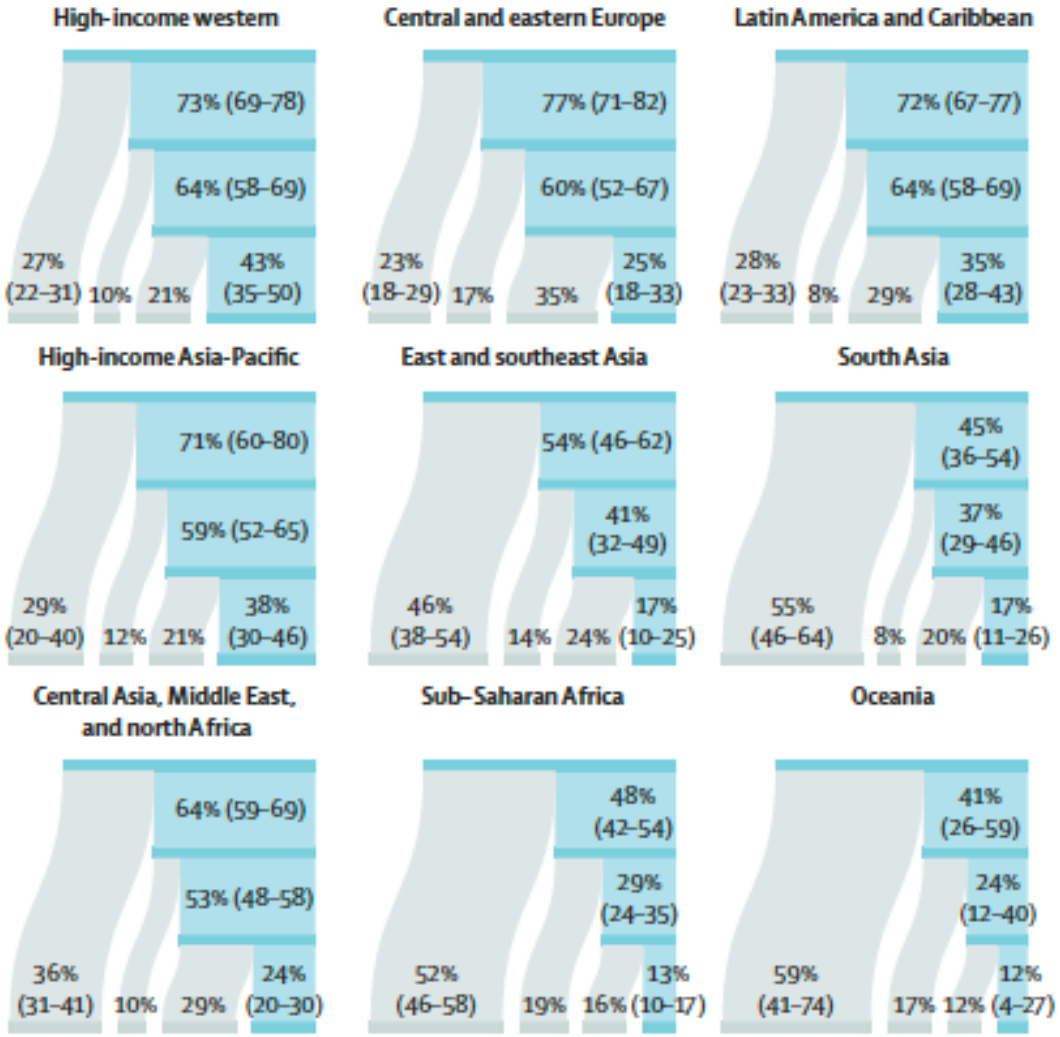
Roth, G.A. et al. J Am Coll Cardiol. 2020;76(25):2982-3021.

Hipertensión en las mujeres

Articles

Worldwide trends in hypertension prevalence and progress in treatment and control from 1990 to 2019: a pooled analysis of 1201 population-representative studies with 104 million participants

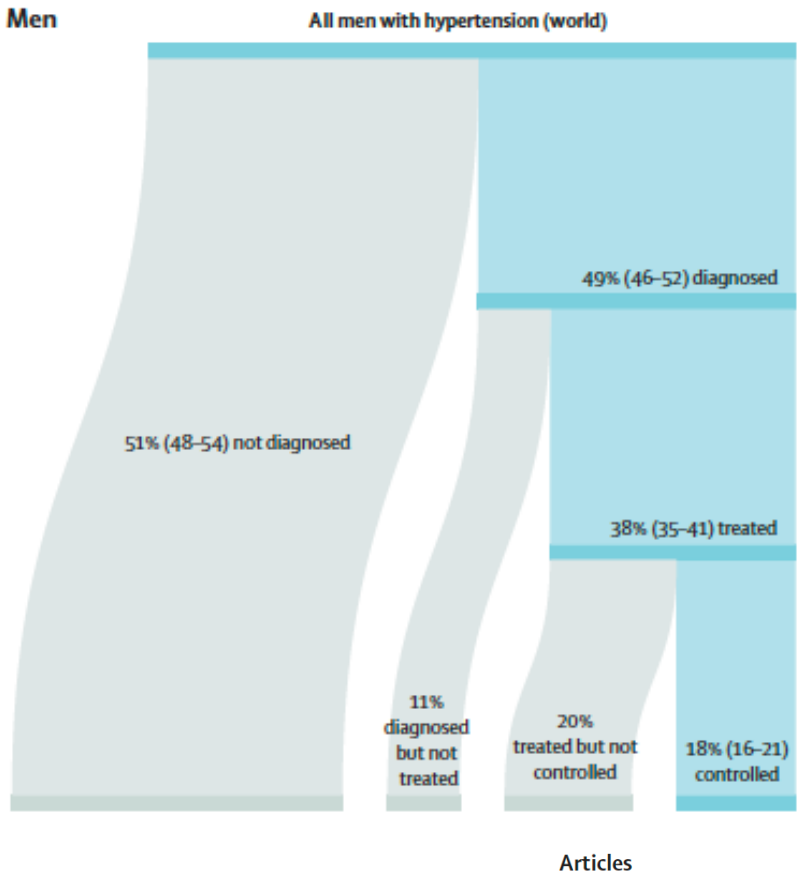
*NCD Risk Factor Collaboration (NCD-RisC)**



Latinoamérica Notable mejoría en el diagnóstico. Pero un rezago del 8 % con respecto. Al nivel de control comparado
Con los países desarrollados de occidente

Lancet 2021; 398: 957–80

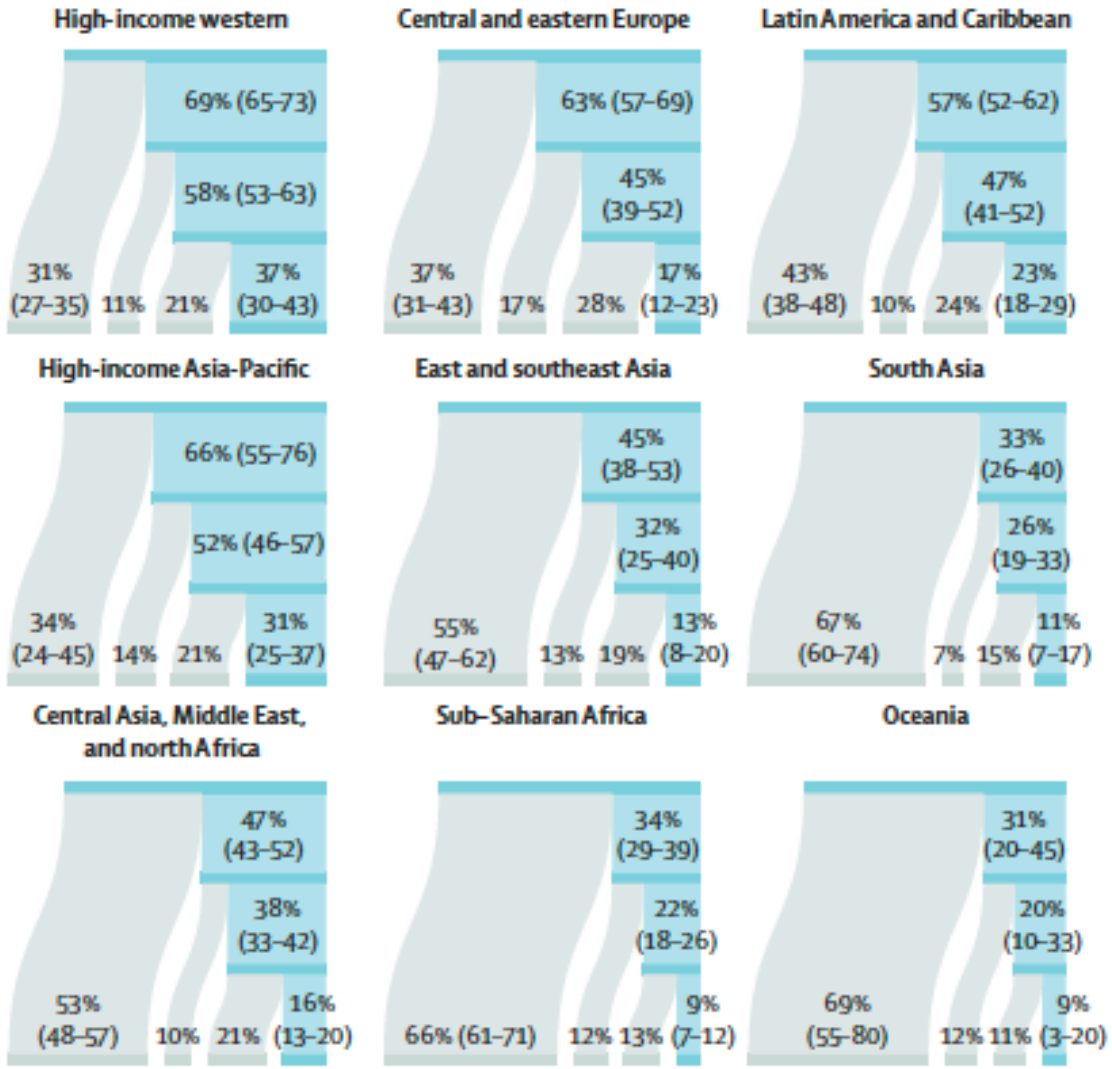
Hipertensión en hombres



Worldwide trends in hypertension prevalence and progress in treatment and control from 1990 to 2019: a pooled analysis of 1201 population-representative studies with 104 million participants

NCD Risk Factor Collaboration (NCD-RisC)*

Niveles de Diagnostico, tratamiento y Control menores que en las mujeres



Rezago marcado en todos los aspectos En hombres en Latinoamérica, comparados con las mujeres

Lancet 2021; 398: 957–80

Otros hallazgos

En 2019, la prevalencia global estandarizada por edad de La hipertensión en adultos de 30 a 79 años fue del 32% (95% CrI 30–34) en mujeres y 34% (32–37) en hombres, similar a los niveles de 1990 del 32% (30-35) en mujeres y 32% (30–35) en hombres

La estabilidad global de la prevalencia fue un efecto neto de una disminución de países de ingresos, y para las mujeres también en Europa del Este, y un aumento de algunos Países de ingresos medianos

Lancet 2021; 398: 957–80

Tendencias en el número de pacientes con HTA

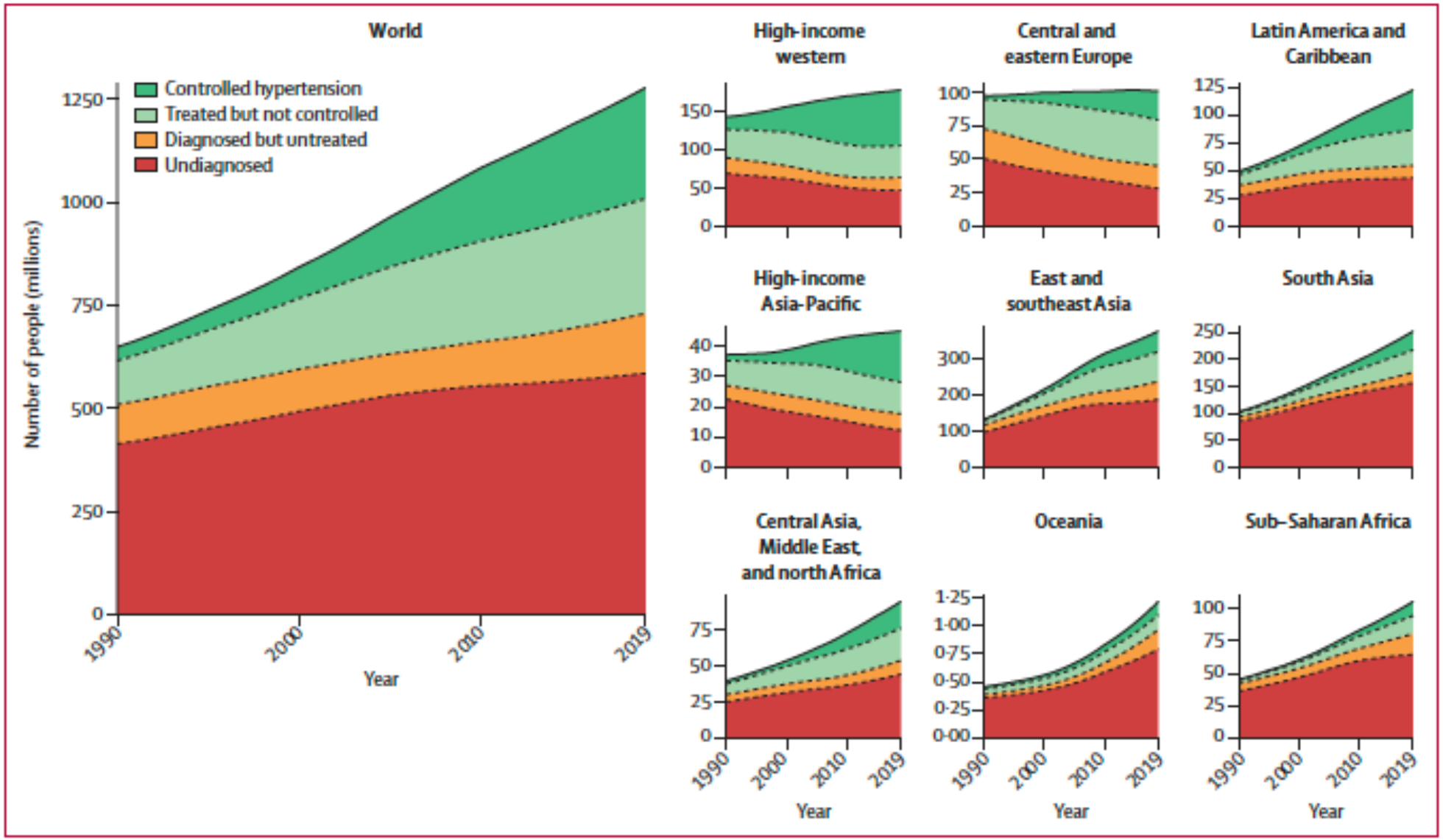


Figure 6: Trends in the number of people with hypertension who reported a diagnosis, who used treatment, and whose blood pressure was effectively controlled, globally and by region, 1990–2019

Lancet 2021; 398: 957–80

World Health Organization

- Se estima que 1280 millones de adultos de 30 a 79 años en todo el mundo tienen hipertensión, la mayoría (dos tercios) vive en países de ingresos bajos y medianos
- Se estima que el 46% de los adultos con hipertensión no saben que tienen la afección.
- Menos de la mitad de los adultos (42%) con hipertensión son diagnosticados y tratados.
- Aproximadamente 1 de cada 5 adultos (21%) con hipertensión la tiene bajo control.
- La hipertensión es una de las principales causas de muerte prematura en todo el mundo.
- Una de las metas mundiales para las enfermedades no transmisibles es reducir la prevalencia de la hipertensión en un 33 % entre 2010 y 2030.

https://www.who.int/news-room/fact-sheets/detail/hypertension#:~:text=An%20estimated%2046%25%20of%20adults,cause%20of%20premature%20death%20worldwide

.

Guidelines on the management of arterial hypertension and related comorbidities in Latin America

Milano, Milan, Italy.

Guidelines Task Force

Argentina
Guillermo Burlando (Departamento de Medicina, Hospital Tornú, Buenos Aires); Claudio Gonzalez [Department of Pharmacology (2nd Chair), School of Medicine, University of Buenos Aires, Buenos Aires]; Daniel Piskorz (Sanatorio Británico, Rosario); Agustín J. Ramirez (Arterial Hypertension and Metabolic Unit, University Hospital, Fundación Favaloro, Buenos Aires); Ramiro A. Sanchez (Arterial Hypertension and Metabolic Unit, University Hospital, Fundación Favaloro, Buenos Aires); Rosa Simsolo (Children's Hospital 'Ricardo Gutierrez', Buenos Aires); Liliana Voto (Obstetrics Unit, Hospital Fernández, Buenos Aires); Gabriel Darío Waissman (Hospital Italiano de Buenos Aires).

Brazil
Alexandre Alessi (Federal University of Curitiba); Celso Amodeo (Hypertension and Nephrology Section, Dante

Chile
Leonardo Cobos (Hospital El Pino, Santiago); Fernando Lanas (Universidad de La Frontera, Temuco); Raul Villar Moya (Integramedica, La Serena).

Colombia
José Luis Accini Mendoza (Unidad de Cuidados intensivos, Hospital Universidad del Norte, Barranquilla); Luis Hernando Garcia-Ortiz (Universidad Tecnológica de Pereira, Facultad de Ciencias de la Salud, Pereira); Patricio Lopez-Jaramillo, Clínica de Síndrome Metabólico, Prediabetes y Diabetes, FOSCAL, Bucaramanga, and Facultad de Ciencias de la Salud Eugenio Espejo, UTE, Quito, Ecuador); Dora Ines Molina (Universidad De Caldas, Clínica IPS Médicos Internistas De Caldas, Manizales); Gregorio Sanchez (Universidad del Quindío, Armenia); Miguel Urina-Triana (Universidad Simón Bolívar/Fundación del Caribe para la Investigación Biomédica, Barranquilla).

Cuba
Alberto Morales-Salinas (Cardiocentro 'Ernesto Che Guevara', Santa Clara).

African ancestors with increased salt sensitivity. In Mexican patients, a high prevalence of LV hypertrophy has been related to a high prevalence of obesity and metabolic syndrome [49,50].

Kidney diseases
The kidney has a key role in BP regulation and the pathogenesis of hypertension through control of sodium excretion, the RAAS and body fluid volume regulation [51]. Primary kidney disease is the most frequent cause of secondary hypertension, with renovascular disease representing 0.5–4% of the secondary causes of hypertension [52,53]. Renal artery stenosis can be seen frequently in older patients, as an atherosclerotic lesion, and can also be observed in young individuals, predominantly women, as fibro-muscular dysplasia. Renal artery stenosis does not invariably induce hypertension and in many cases is simply a casual finding in patients with essential hypertension. In other cases, it may induce severe hypertension with heart failure, recurrent pulmonary edema, renal dysfunction and chronic failure. Renovascular hypertension should be suspected in the presence of treatment resistant hypertension, malignant or accelerated hypertension, or an abdominal systo/diastolic murmur [52,53]. Although the study of renal arteries with color Doppler echography is examiner dependent, it can be used as a screening method. However, magnetic resonance angiography or computed tomography angiography must be used as confirmatory study [54,55]. Selective renal artery angiography is the gold standard method and applicable when revascularization is planned [56].

Kidney damage is also a consequence of high BP and hypertensive nephrosclerosis is the second cause of admission to chronic dialysis, after diabetes mellitus. The progression of renal disease appears to be related with the degree of BP control. In Latin America, renal replacement therapy for end-stage renal disease was followed-up in a registry including 20 countries, representing 99% of the region population. The prevalence of renal replacement therapy has increased in Latin America [57] from 119 patients per million in 1991 to 660 patients per million in 2010. The higher rates were observed in Puerto Rico (1366 per million) and in Argentina, Mexico, Uruguay and Chile (between 777 and 1136 patients per million).

Peripheral artery disease
The presence of PAD suggests the presence of an advanced arterial wall damage that may also involve coronary or cerebral arteries, even without clinical signs [58]. As a manifestation of atherosclerotic disease, PAD risk factors are the same as those identified for other vascular areas, with smoking and diabetes mellitus having a stronger association with PAD than with coronary or cerebral-vascular artery disease [59]. Measurement of the ankle-brachial index (ABI) performed with automated devices or with a continuous wave Doppler unit and a sphygmomanometer is strongly recommended [60]. An ABI less than 0.9 is a strong evidence of PAD and advanced atherosclerosis.

No information about prevalence of PAD in Latin America has been reported.

TABLE 1. Blood pressure classification

Classification	SBP (mmHg)	DBP (mmHg)
Normotension		
Optimal BP	<120	<80
Normal BP	120–129	80–84
High-normal BP	130–139	85–89
Hypertension		
Grade 1	140–159	90–99
Grade 2	160–179	100–109
Grade 3	≥180	≥110
Isolated systolic hypertension	≥140	<90

When SBP and DBP values are in different BP categories, the individual should be classified in the higher BP category. BP, blood pressure.

CLASSIFICATION, DIAGNOSIS, AND RISK STRATIFICATION IN ARTERIAL HYPERTENSION

Blood pressure classification
Hypertension is diagnosed when BP values are at least 140/90 mmHg. Above this value, hypertension can be subdivided in grade 1, 2 or 3 [61] as shown in Table 1.

When SBP and DBP values correspond to different grades, the higher grade should be used to classify the patient's hypertension. When SBP is at least 140 mmHg, and DBP less than 90 mmHg, isolated systolic hypertension is diagnosed and hypertension grade is classified according to the SBP values.

Hypertension can be subdivided in:

(1) *Primary, essential or idiopathic hypertension*: when BP is consistently higher than normal with no known underlying cause (around 90–95% of all cases).
(2) *Secondary hypertension*: when BP is increased as the result of an underlying, identifiable, often correctable cause (around 5–10% of the total hypertensive patients).

When office BP and ambulatory or home BP values are considered, four groups can be identified [62–64]:

(1) Patients with normal BP values with both methods (normotensives or sustained normotensives),
(2) Patients with increased BP values with both methods (hypertensive patients or sustained hypertensive patients),
(3) Those with normal BP values in the office and hypertensive values with the ABPM or at home (masked hypertensive patients), and
(4) Those with hypertensive values in the office and normal values with the ABPM or at home (white coat hypertensive patients).

Diagnosis
Office BP measurements should be performed using an auscultatory or oscillometric semiautomatic sphygmomanometer, validated and calibrated periodically. Today, the mercury sphygmomanometer is used less frequently and in

Journal of Hypertension. 35(8):1529–1545, AUG 2017

EL COSTO DE LA ATENCION HTA:
ESTA RELACIONADO CON SUS COMPLICACIONES

- Urina M. CAPITULO: COMPLICACIONES DE LA HTA. LIBRO SCC 2003

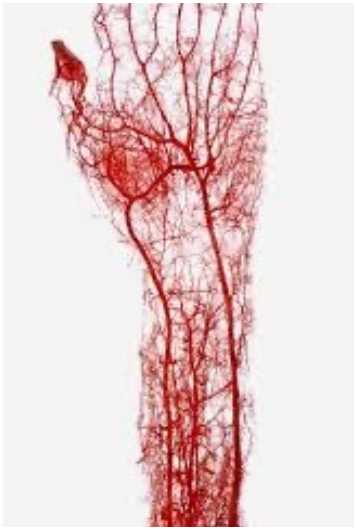
TABLA 32.1 Efectos sobre órganos producidos por la hipertensión arterial sistémica

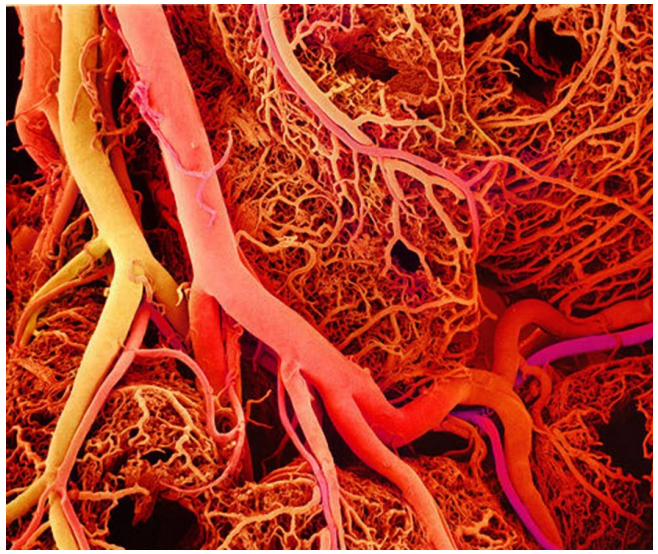
Efectos	Clase de efectos
Efectos sobre el corazón	Hipertrofia ventricular izquierda, angina de pecho o infarto miocárdico, por enfermedad coronaria, insuficiencia cardíaca.
Efectos neurológicos	Retinopatía hipertensiva, sistema nervioso central (infarto cerebral, hemorragia cerebral, disfunción del SNC), encefalopatía hipertensiva.
Efectos renales	Arteriosclerosis arterial aferente y eferente, lesión glomerular.
Efectos vasculares periféricos	Enfermedad vascular de miembros inferiores.
Efectos sobre el endotelio	Enfermedad hipertensiva por disfunción endotelial.
Efectos sobre la función sexual	Disfunción eréctil.

Tabla modificada del libro Cardiología 1999, pág. 379.

ASPECTOS FISIOPATOLOGICOS

Dr. Thomas Sydenham 1624-1689





EL HOMBRE TIENE LA EDAD QUE TIENEN SUS ARTERIAS

Sin embargo, los cambios en la función y estructura de las arterias con el envejecimiento, como rigidez y engrosamiento arterial, no ocurren a la misma medida en todas las personas.

A medida que envejecemos, el vaso sanguíneo se vuelve más susceptible a patologías que incluyen hipertensión y aterosclerosis

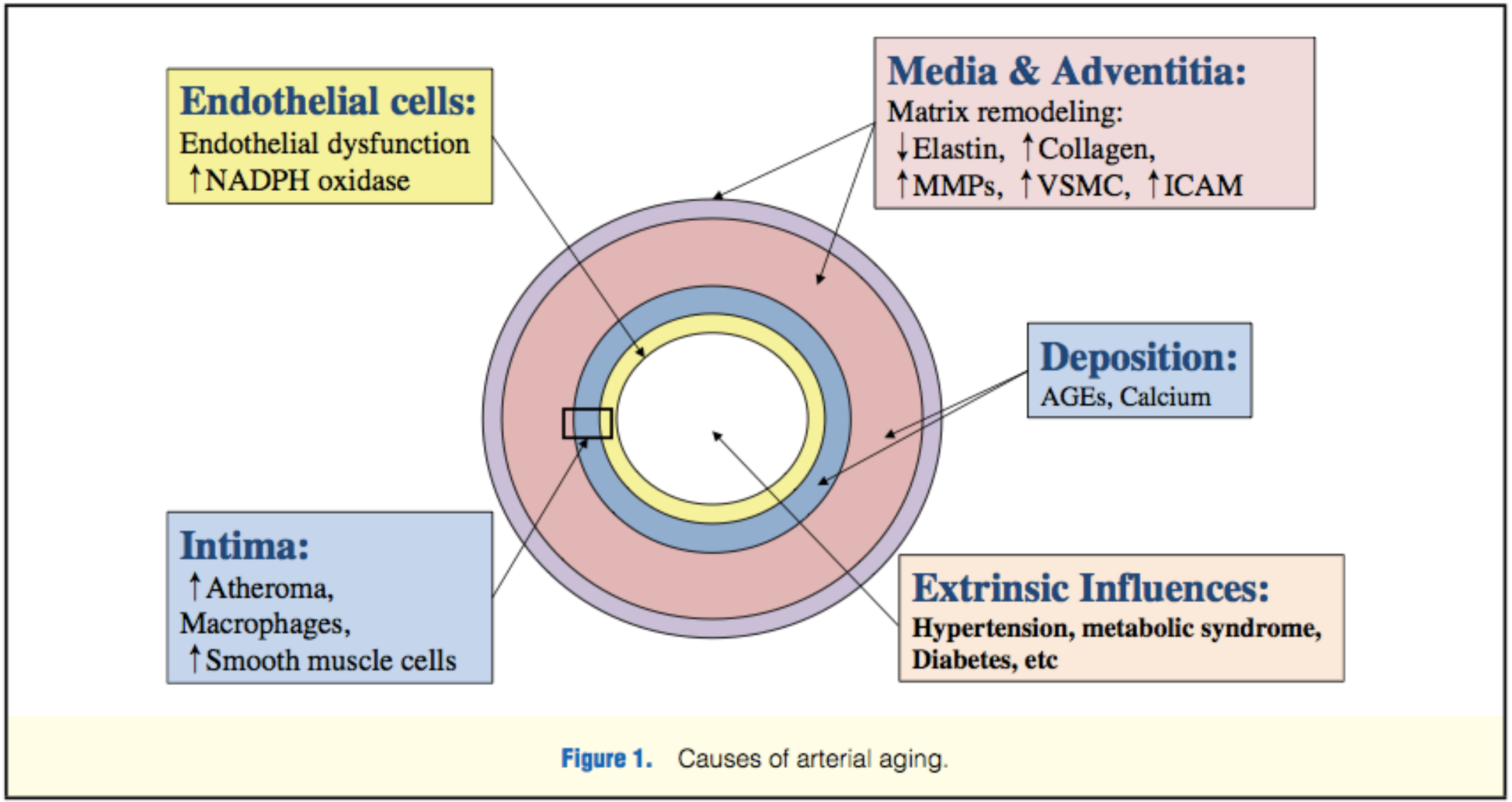
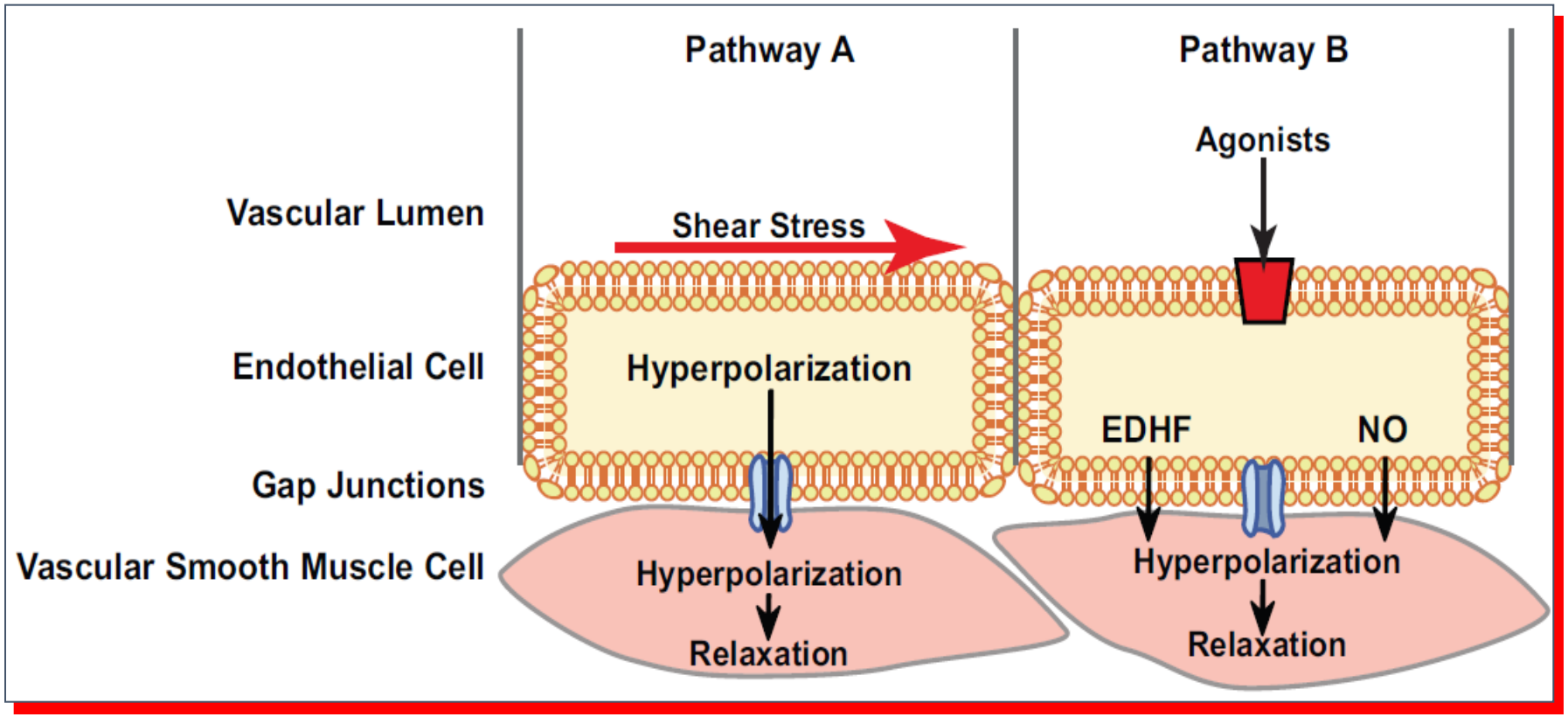


Figure 1. Causes of arterial aging.

RIGIDEZ ARTERIAL PREDICTOR DE ENFERMEDAD CARDIOVASCULAR

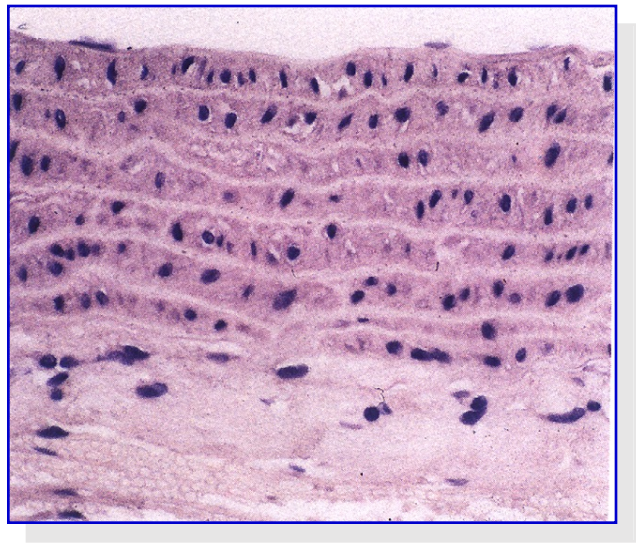


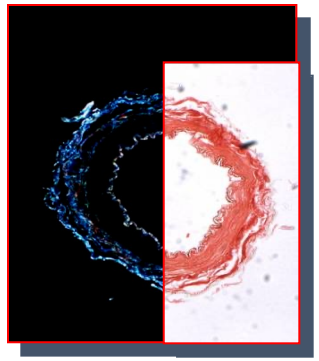
Duprez D. Hypertension 2010;55:612-13

HIPERTROFIA VASCULAR.

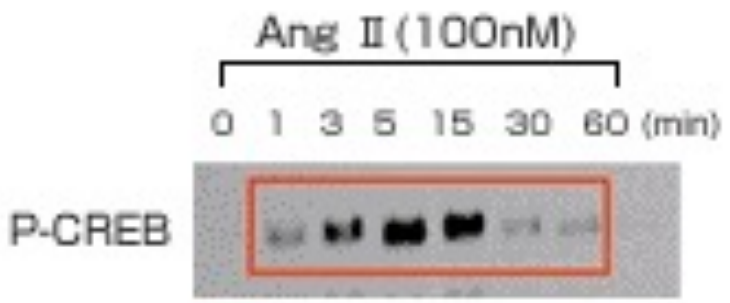
Resistance 0.54 units

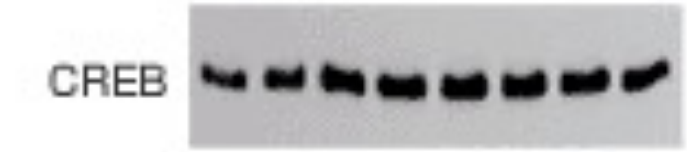
Resistance 218 units



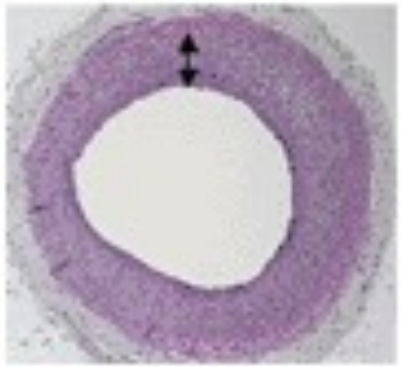


▼Research on the expression, signaling pathway and transcription factors of angiotensin II receptor

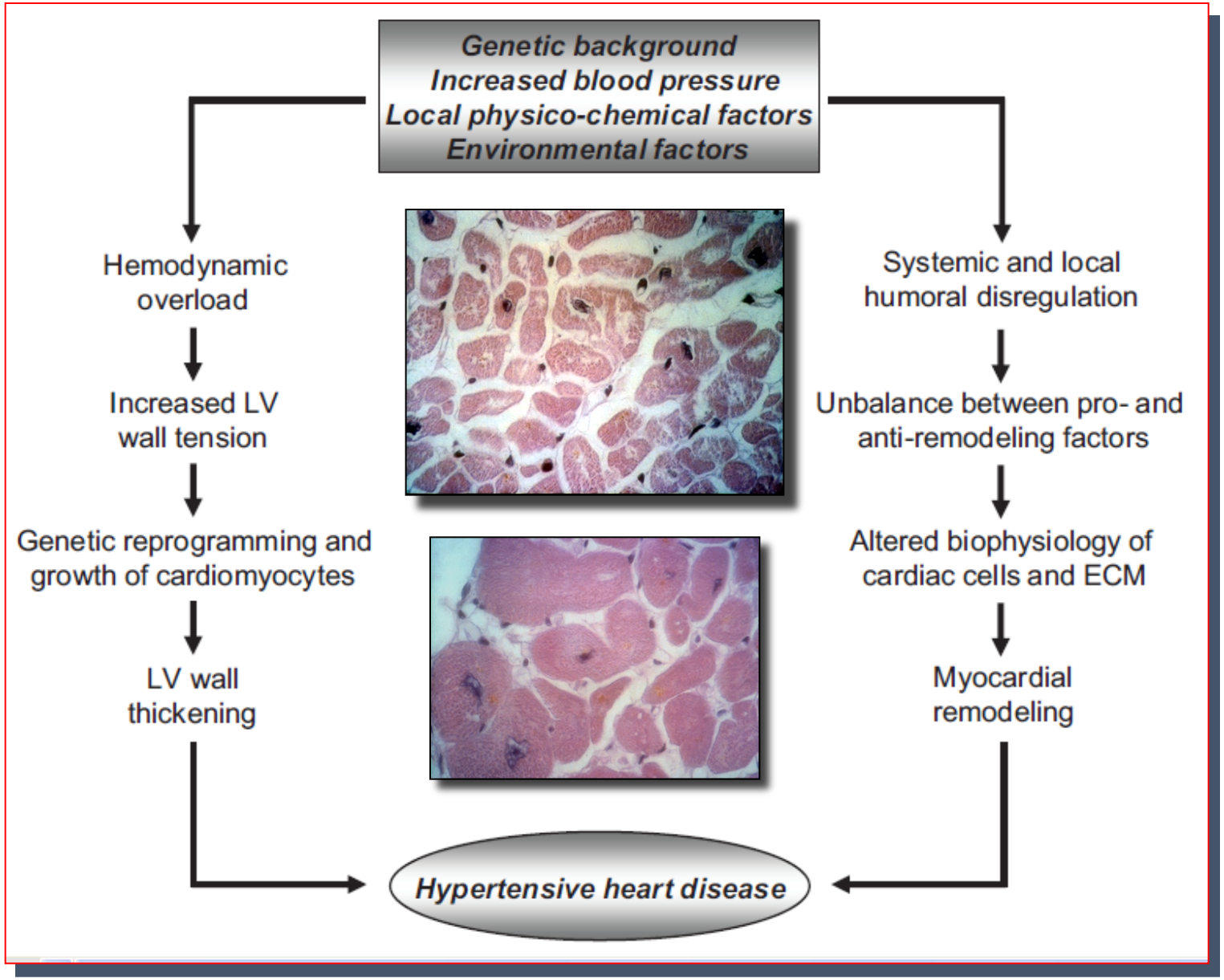




The transcription factor CREB is activated in the presence of angiotensin II



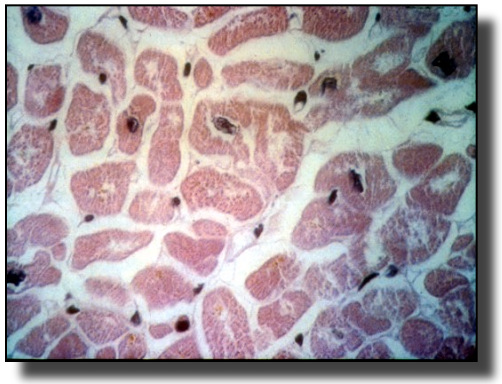
Transfer of the CREB mutant gene results in reduction of vascular lesion


Genetic background
Increased blood pressure
Local physico-chemical factors
Environmental factors
Hemodynamic overload
Increased LV wall tension
Genetic reprogramming and growth of cardiomyocytes
LV wall thickening
Systemic and local humoral disregulation
Unbalance between pro- and anti-remodeling factors
Altered biophysiology of cardiac cells and ECM
Myocardial remodeling
Hypertensive heart disease

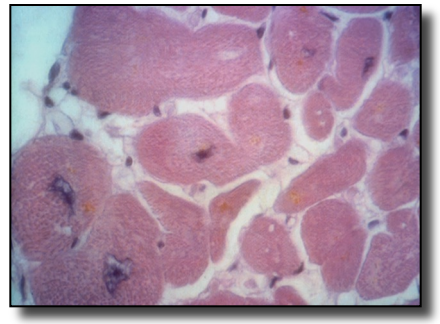
A Translational Approach to Hypertensive Heart Disease

Javier Díez, Edward D. Frohlich

Hipertrofia del cardiomiocito



genes de expresión



SINTESIS PROTEICA

Diez J. Hypertension 2010;55:1-8

A Translational Approach to Hypertensive Heart Disease

Javier Díez, Edward D. Frohlich

Table 1. Molecules Involved in Myocardial Remodeling in HHD

Proremodeling molecules

Vaso… …phrine,

Ho… …steron

Gr… …wth fa…

Cyto…

Othe… …endoge…

prolifĕrator-activated receptor-γ ligands)

Antiremodeling molecule…

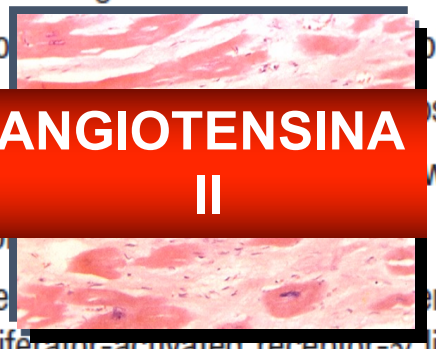
Vasoactive substance… …1-7])

Hormones (glucocort…

Growth factors (insuli…

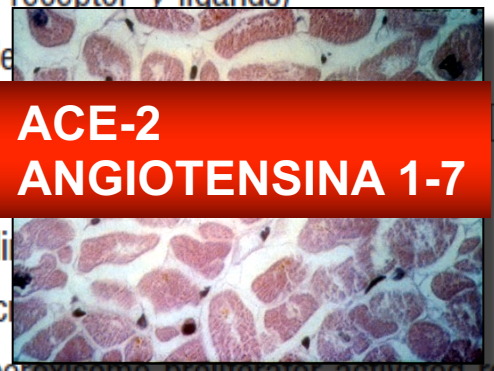
Cytokines (tumor nec…

Other (endogenous peroxisome proliferator-activated receptor−α ligands)



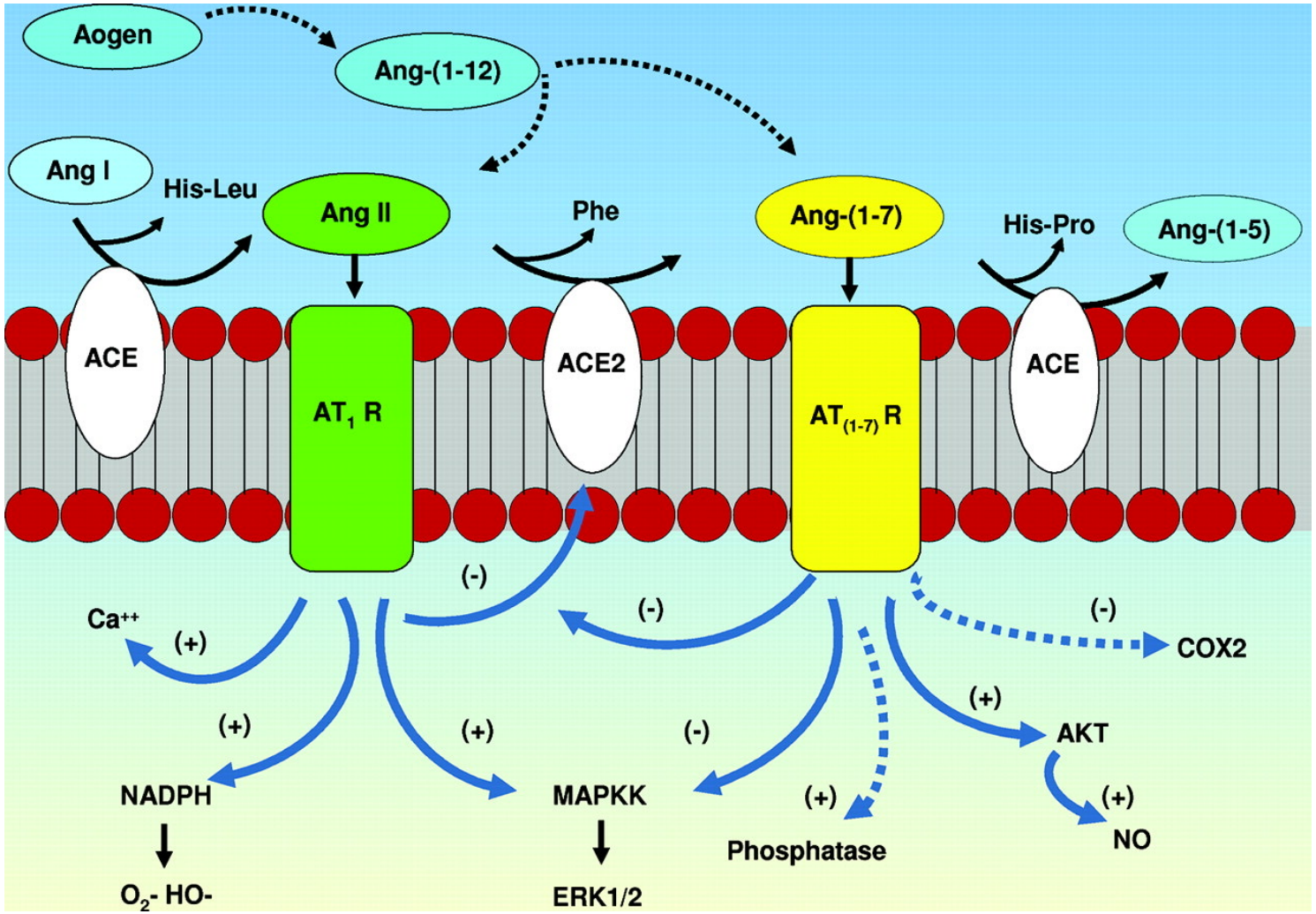
ANGIOTENSINA II

CARDIOTROFINA 1



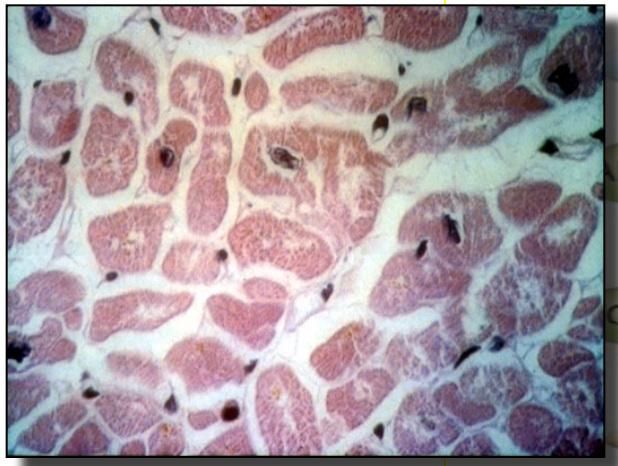
ACE-2
ANGIOTENSINA 1-7

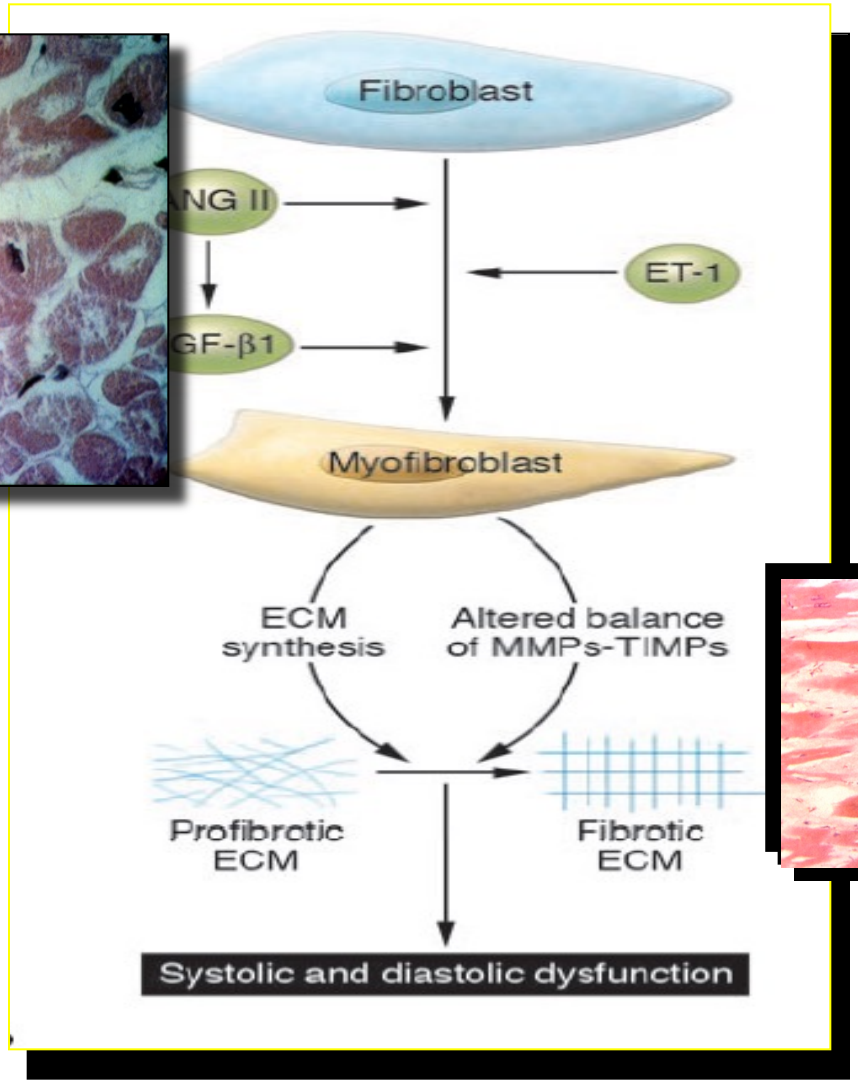
Figure. Cascade of the processing of angiotensin peptides and their interaction with AT1 and AT(1-7) receptor systems.

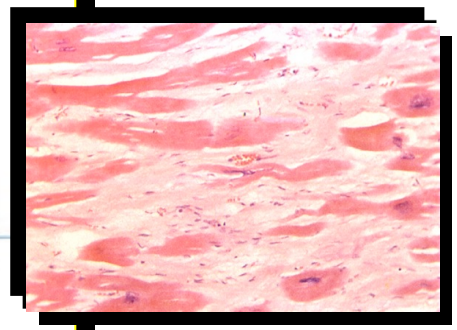


Chappell M C Hypertension 2007;50:596-599

ANGIOTENSIN II AND MYOCARDIAL FIBROSIS







Berk Bradford. J.Clin.Invest.2007;117:568-575

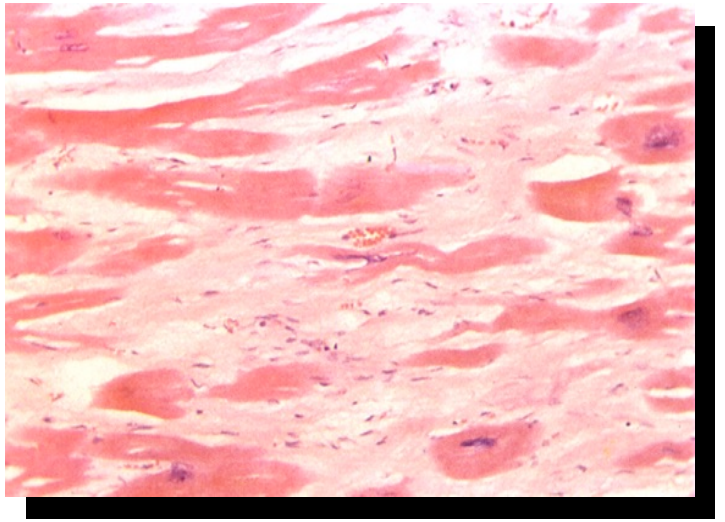
Cardiac Fibrosis

ANGIOTENSIN II

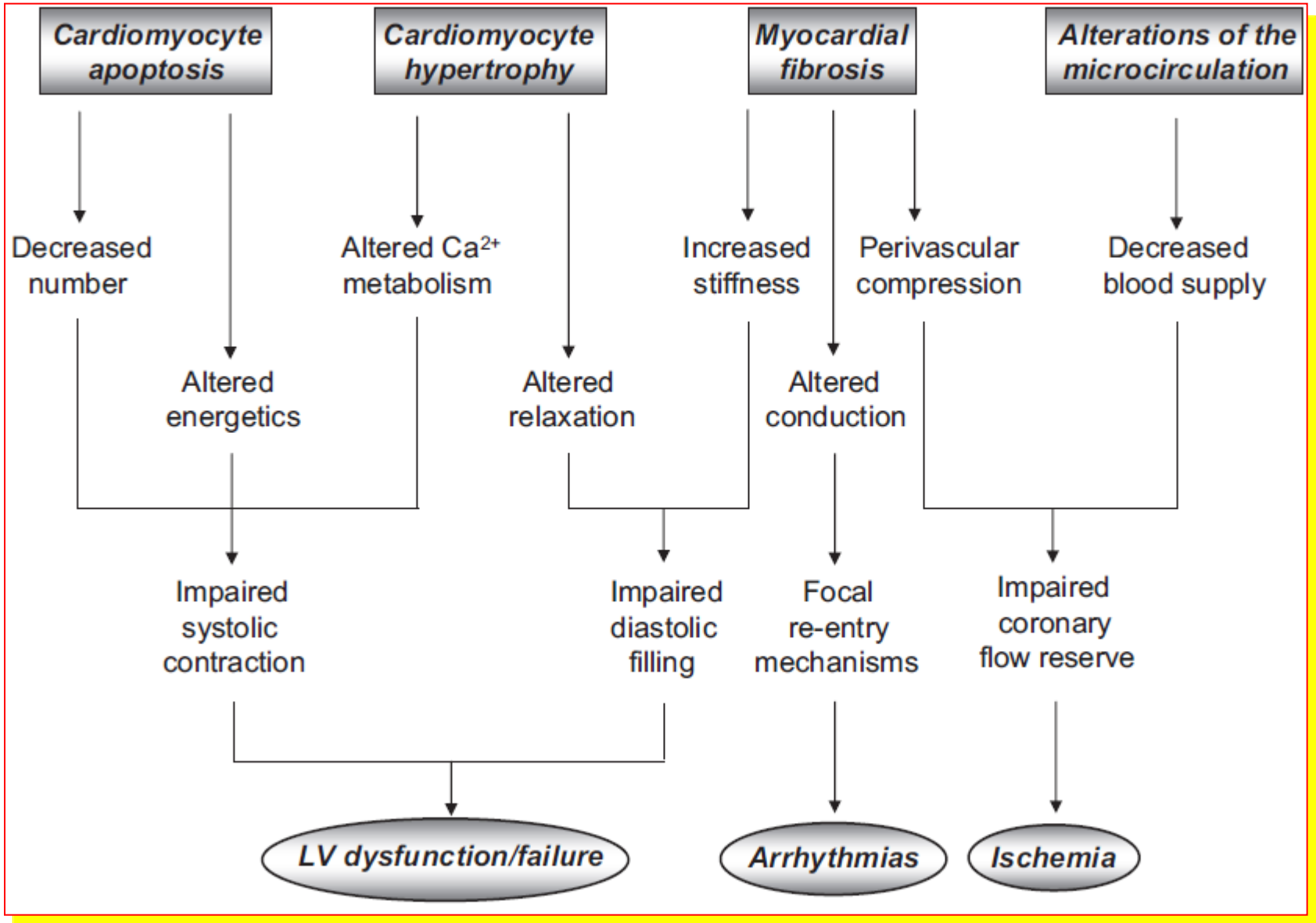
FIBROBLAST ACTIVATION

COLLAGEN DEPOSITION

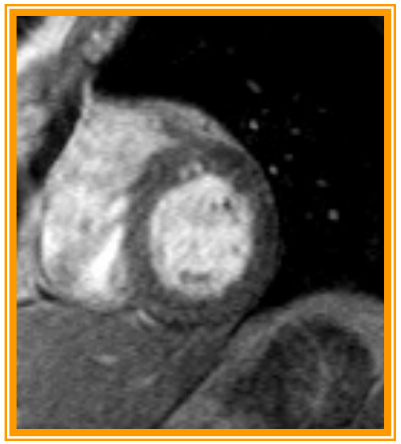
Aldosterone



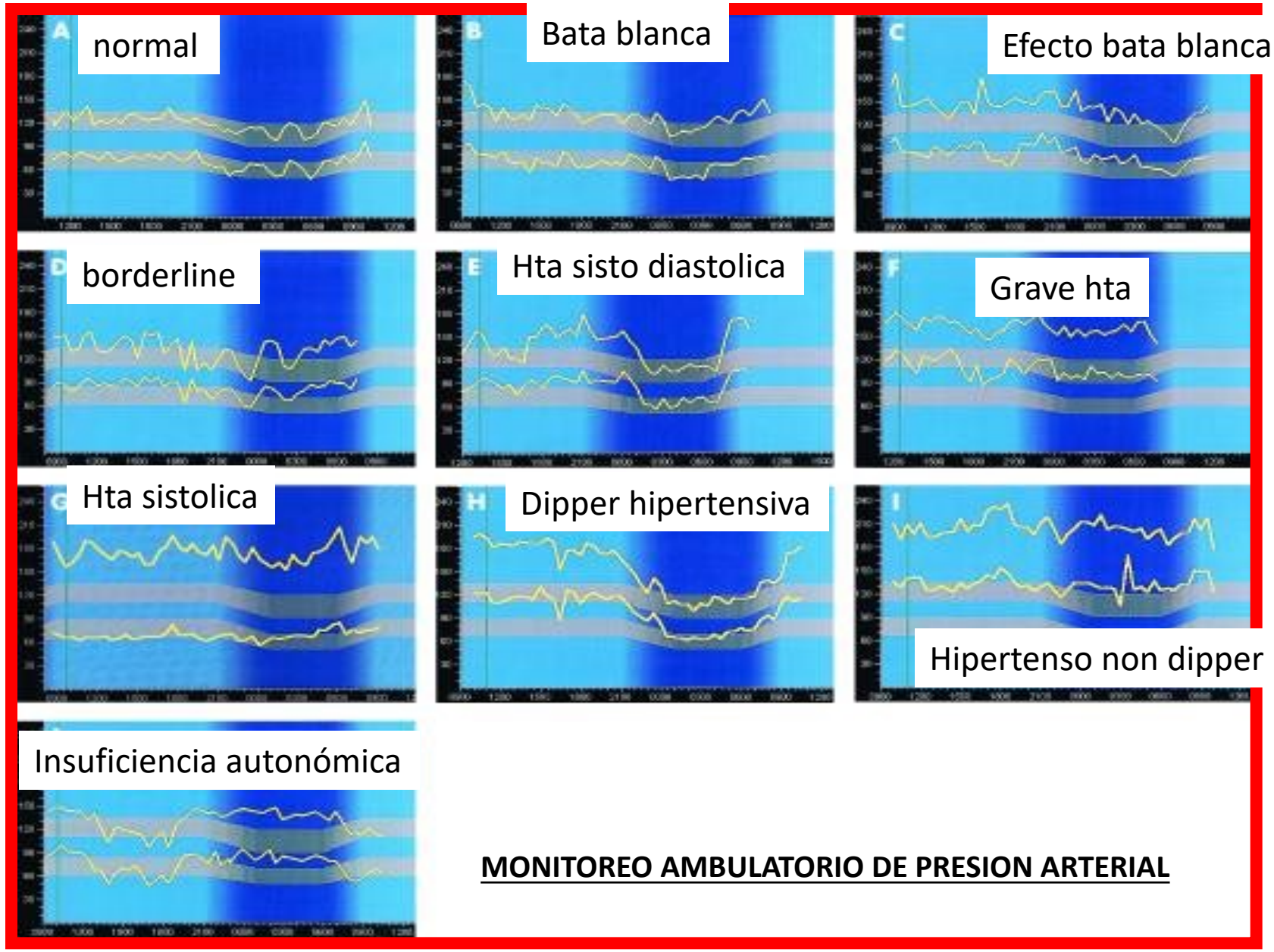
Endotelin - 1


Cardiomyocyte apoptosis
Cardiomyocyte hypertrophy
Myocardial fibrosis
Alterations of the microcirculation
Decreased number
Altered energetics
Altered Ca^{2+} metabolism
Altered relaxation
Increased stiffness
Altered conduction
Perivascular compression
Decreased blood supply
Impaired systolic contraction
Impaired diastolic filling
Focal re-entry mechanisms
Impaired coronary flow reserve
LV dysfunction/failure
Arrhythmias
Ischemia

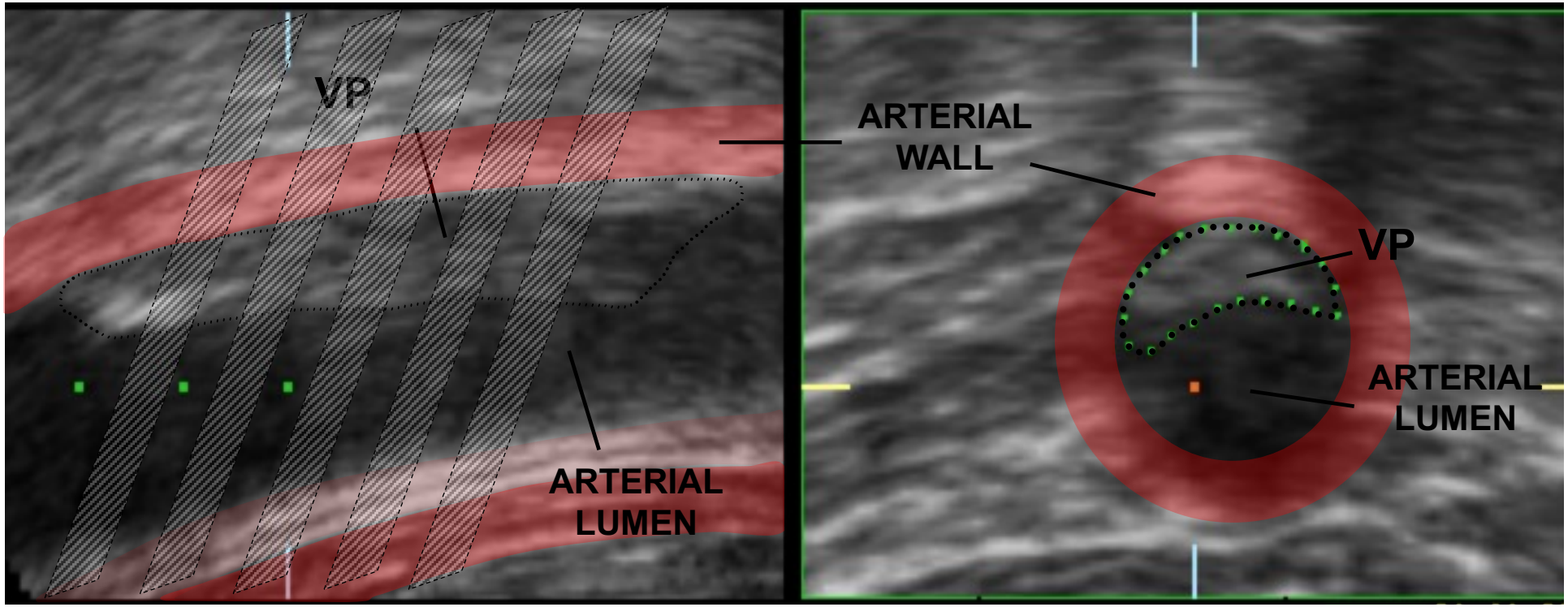
ESTRATEGIAS TEMPRANAS DE DETECCION DE HVI



- **MAPA 24 HRS (índice ambulatorio de rigidez arterial)**
- **Estudio genético del polimorfismo de la ECA y del receptor AT-1**
- **Medición de los niveles de Cardiotrofin 1 (CT-1)**
- **Echo 3-d o RMI**

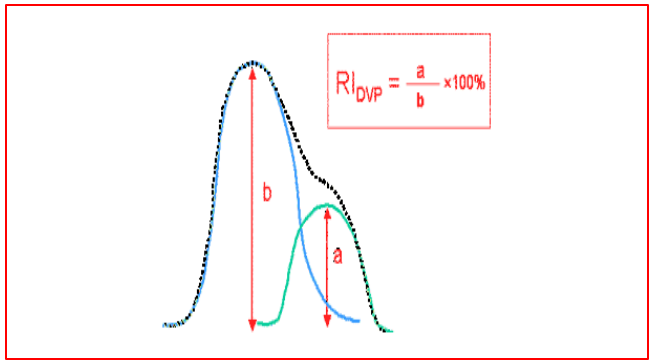


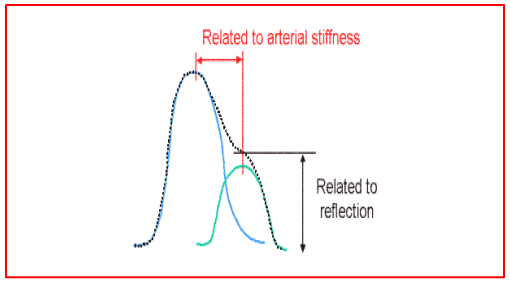
MONITOREO AMBULATORIO DE PRESION ARTERIAL


VP
ARTERIAL
LUMEN
ARTERIAL
WALL
VP
ARTERIAL
LUMEN

La rigidez arterial predice morbimortalidad cardiovascular y se puede evaluar con:

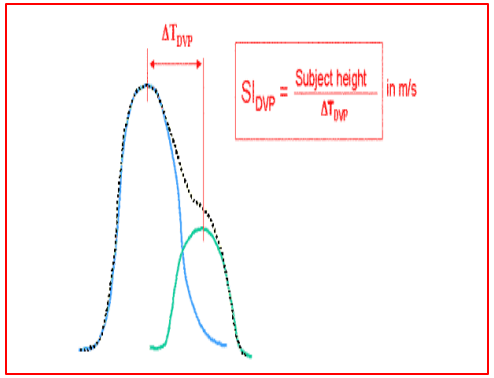
1. Velocidad de onda de pulso (VOP)
2. índice de rigidez arterial ambulatorio (AASI)
3. índice de aumento (IA)





El AASI se calcula como el resultado obtenido de restar a uno la pendiente de regresión de la TAD sobre la TAS, utilizándose el conjunto de datos de una completa monitorización de 24h.
El sym-AASI se calcula usando la fórmula 1-(1-AASI)/r, siendo r el coeficiente de correlación entre el conjunto de registros de TAS y TAD.
Representa la bisectriz entre las líneas de regresión de la TAD sobre la TAS y de su inversa, es decir, la TAS sobre la TAD19

.



La velocidad de onda de pulso y el envejecimiento arterial en sujetos con y sin diabetes mellitus tipo 2

Pulse wave velocity and arterial aging in subjects with and without type 2 diabetes mellitus

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[4]Residente de Tercer Año Medicina Interna Mount Sinai Miami Medical Center. E-mail: danielaurina@fundacionbios.org

[5]Fisioterapeuta. E-mail: lgaleano@unisimonbolivar.edu.co

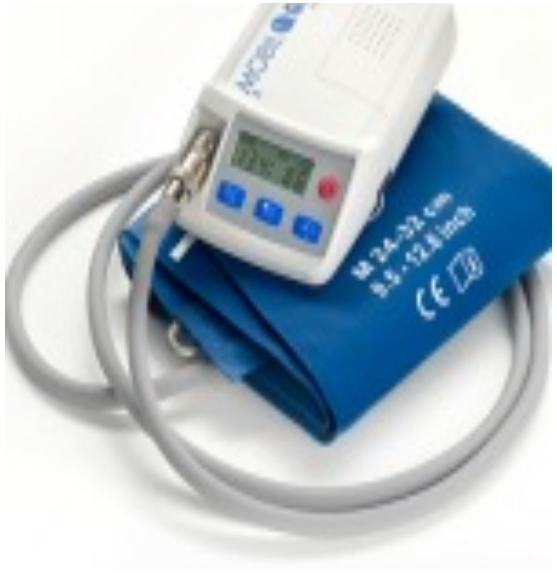
[6]Magister en Epidemiología Clínica. E-mail: manuelurina@fundacionbios.org

[7]Médico especialista en Cardiología adalbertoquintero@fundacionbios.org

Tabla 2. La VOP de acuerdo con el tiempo de evolución de la Diabetes Mellitus comparada con el sujeto sin Diabetes Mellitus*

	n			VOP (m/seg)	Desviación estándar
Sin Diabetes Mellitus	27			8,359	2,355
	n	n	Tiempo de evolución		
Con Diabetes Mellitus	12	8	De 5-9 años	9,700	2,171
		4	De 10-20 años	10,775	2,317
Promedio				8,882	2,408

*Correlación de Pearson: VOP= 1/tiempo de evolución= 0,350, Sig (unilateral) p=0,015



https://www.redalyc.org/journal/1702/170263336013/movil/

La correlación entre fragilidad y rigidez arterial con la capacidad funcional en adultos mayores con enfermedad crónica cardiovascular

Mantilla-Morrón M[1], Urina-Triana M[1], Quintero-Cruz V[1], Robles-Adriano A[2], Hernández-Sarabia C[2], Buelvas-Herazo J[3]

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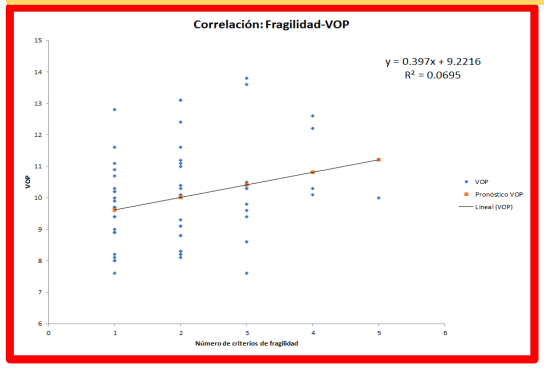
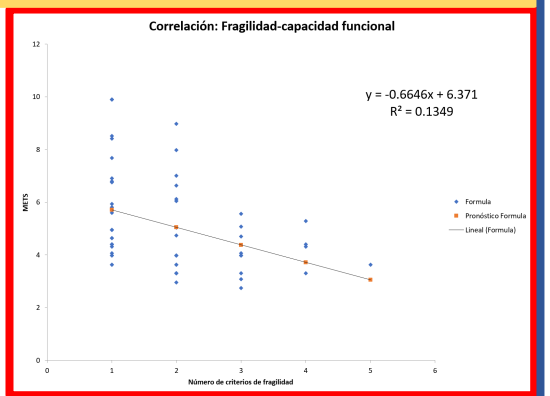
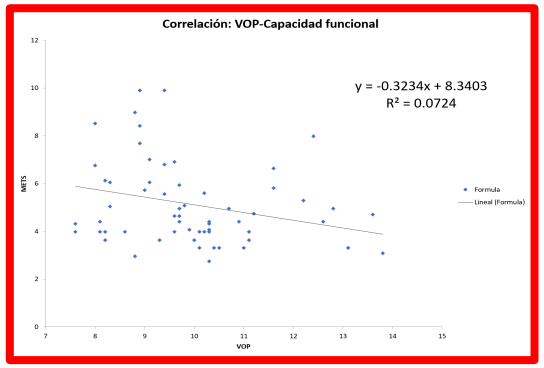
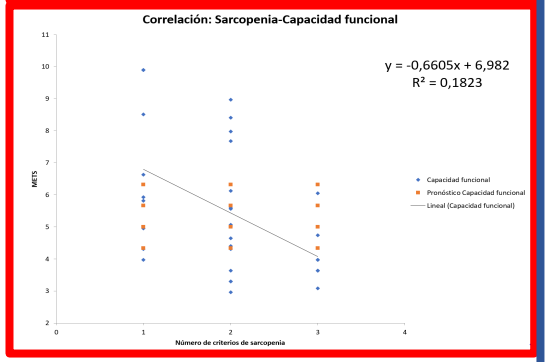
3 Cardiodiagnóstico SA









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NIT 830.009.650-1

LA

ASOCIACIÓN COLOMBIANA DE GERONTOLOGÍA Y GERIATRÍA

CERTIFICA QUE

LOS AUTORES

MIRARY MANTILLA MORRÓN
MIGUEL URINA TRIANA
JANNES BUELVAS HERAZO
MARIA VICTORIA QUINTERO
AMARILIS ROBLES-ADRIANO}
HERNÁNDEZ-SARABIA CRISTAL

Participaron como ponentes de Tema Libre en el XIV CONGRESO COLOMBIANO DE GERONTOLOGÍA Y GERIATRÍA / IV CURSO RESIDENTES DE GERIATRÍA / V CURSO TERAPÉUTICA EN GERIATRÍA / VI CONGRESO DE INTERDISCIPLINARIEDAD EN EL MANEJO DEL ANCIANO, celebrado en la ciudad de Cali del 5 al 7 de septiembre de 2019, en las instalaciones del Hotel Intercontinental.

Su participación tuvo lugar en la Sesión de Temas Libres poster del día jueves 5 de septiembre, con el tema: "**LA CORRELACIÓN ENTRE FRAGILIDAD Y RIGIDEZ ARTERIAL CON LA CAPACIDAD FUNCIONAL EN ADULTOS MAYORES CON ENFERMEDAD CRÓNICA CARDIOVASCULAR**"

La presente se expide, a los 7 días del mes de septiembre de 2019.

Cordialmente,

JAVIER CABRERA GUERRA
Presidente de la ACGG

Cra. 7C BIS No. 139-17 – Teléfono: 2320978 Bogotá, D.C. - Colombia
www.acgg.org.co e-mail: administrativo@acgg.org.co

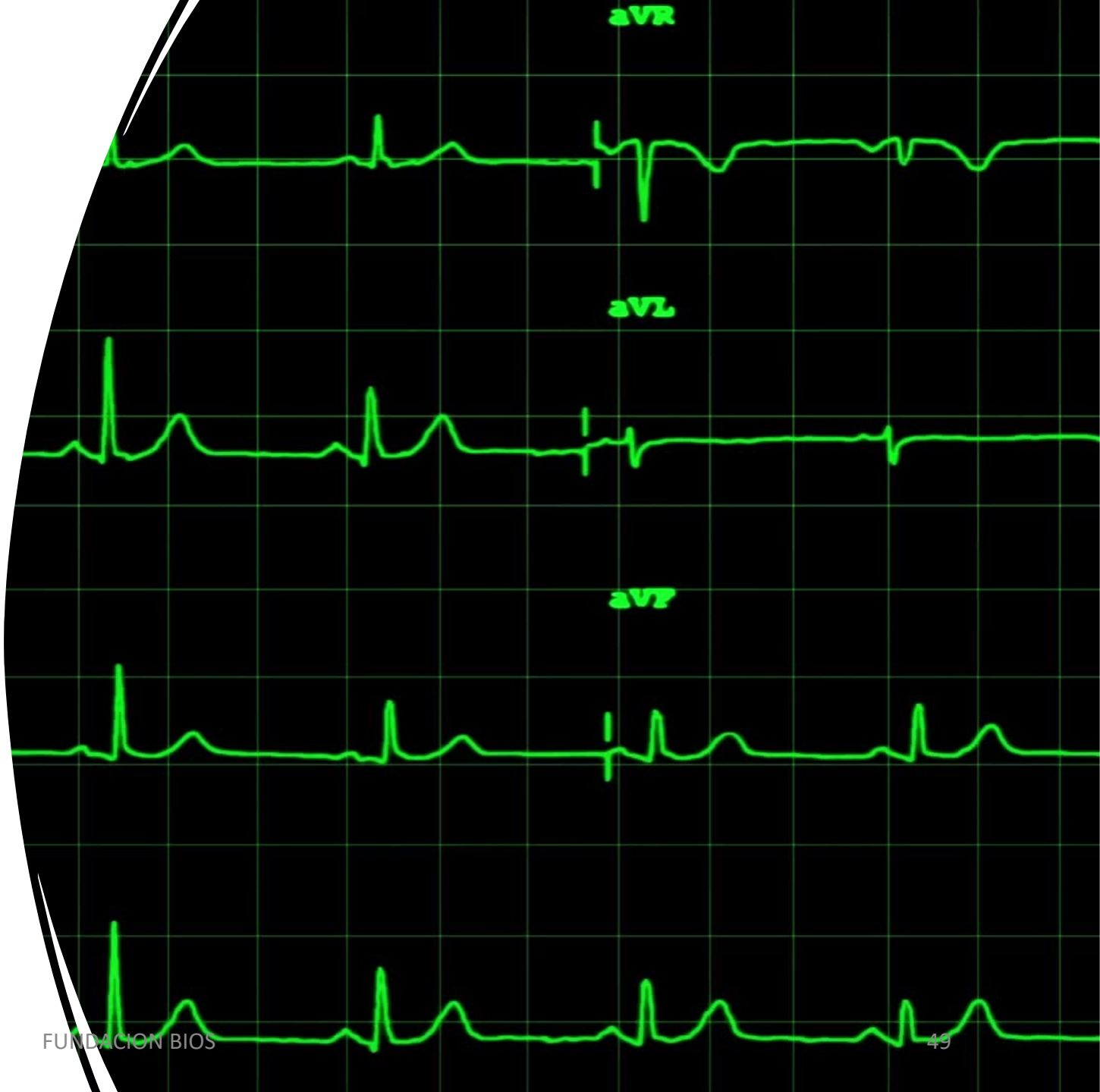
En este estudio (n=63) se demostró una **correlación inversa entre fragilidad, sarcopenia y velocidad de onda de pulso con capacidad funcional,** es decir, **a mayor fragilidad, sarcopenia y rigidez arterial, menor capacidad funcional** demostraron los pacientes.

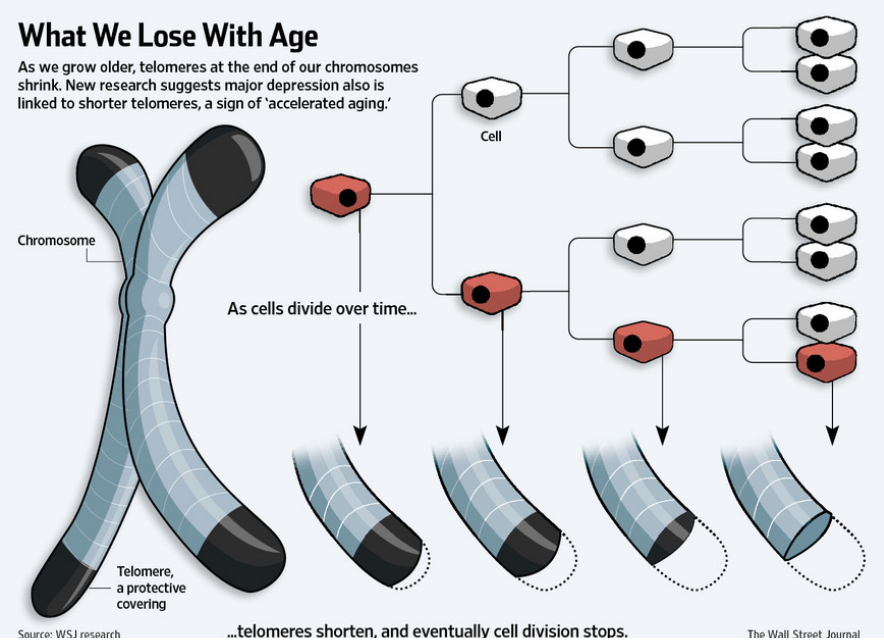
Efectos de un programa de ejercicio físico sobre la longitud telomérica, rigidez arterial, sarcopenia en sujetos con enfermedad cardiovascular

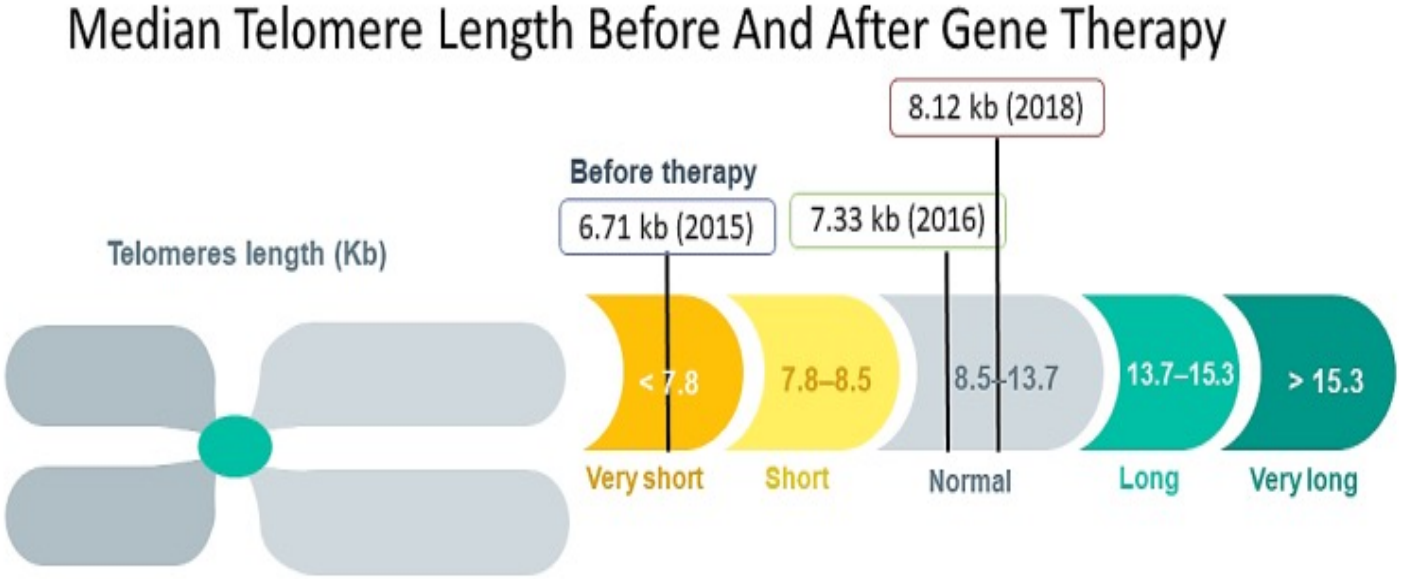
Investigador Principal:

Dr. Miguel Urina-Triana PhD, FACC

Centro de investigaciones Cardiodiagnóstico S.A./Fundación del Caribe para la investigación biomédica/ Grupo de Investigación en Genética/CICEFYNA



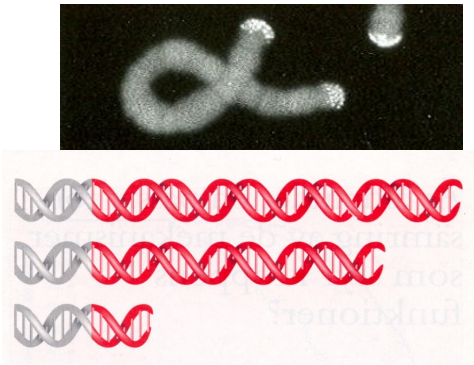




The infographic above depicts the increase in Liz Parrish's telomere length after taking the telomerase and myostatin gene therapy. Over the last three years since taking the therapy, Liz's median telomere has improved by over 20%, going from 6.71 kb in 2015, to 7.33 kb in 2016, to over 8.12 kb in 2018. According to Spectracell, an average 30-year-old has a telomere length of around 8.12 kb. Based on this result we think that Liz's biological age is closer to a 30-year-old or younger.

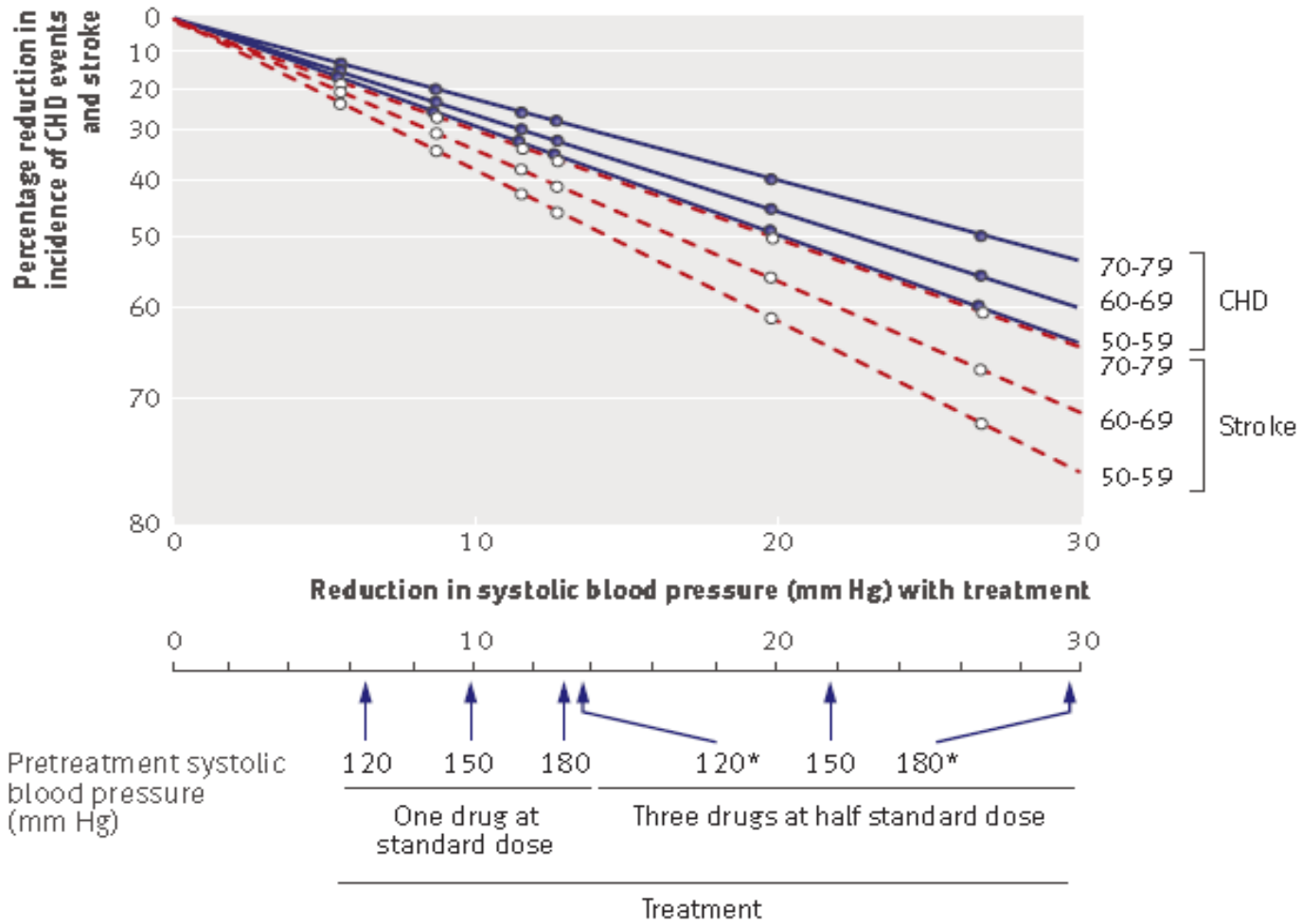
All telomere length measurements were conducted by Spectracell. Spectracell measures patient's average telomere length in peripheral whole blood cells. This average is then compared to telomere lengths from a population sample in the same age range as the patient to determine the patient's percentile score.

BioViva™



Telomeres at the end of the DNA helix

ASPECTOS TERAPEUTICOS papel del control del SRAA


Percentage reduction in incidence of CHD events and stroke
0
10
20
30
40
50
60
70
80
70-79
60-69
50-59
CHD
70-79
60-69
50-59
Stroke
0
10
20
30
Reduction in systolic blood pressure (mm Hg) with treatment
0
10
20
30
Pretreatment systolic blood pressure (mm Hg)
120
150
180
120*
150
180*
One drug at standard dose
Three drugs at half standard dose
Treatment

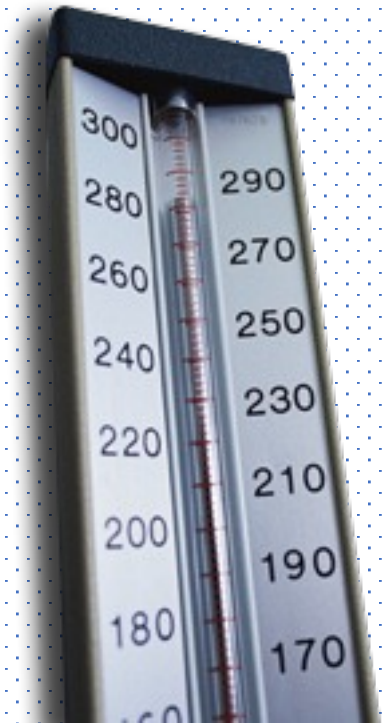
Los interrogantes pendientes

- Qué pasa con la falta de control ?
- Cuál es su impacto económico ?
- Se justifica la terapia combinada ?
- Cuál seria una combinación racional ?
- Qué es lo bueno de las nuevas combinaciones ?
- Son eficaces las combinaciones ?
- Cuales son los beneficios y ventajas de la terapia combinada que nos presentan hoy ?

Inhibición del SRAA más allá de la hipertensión

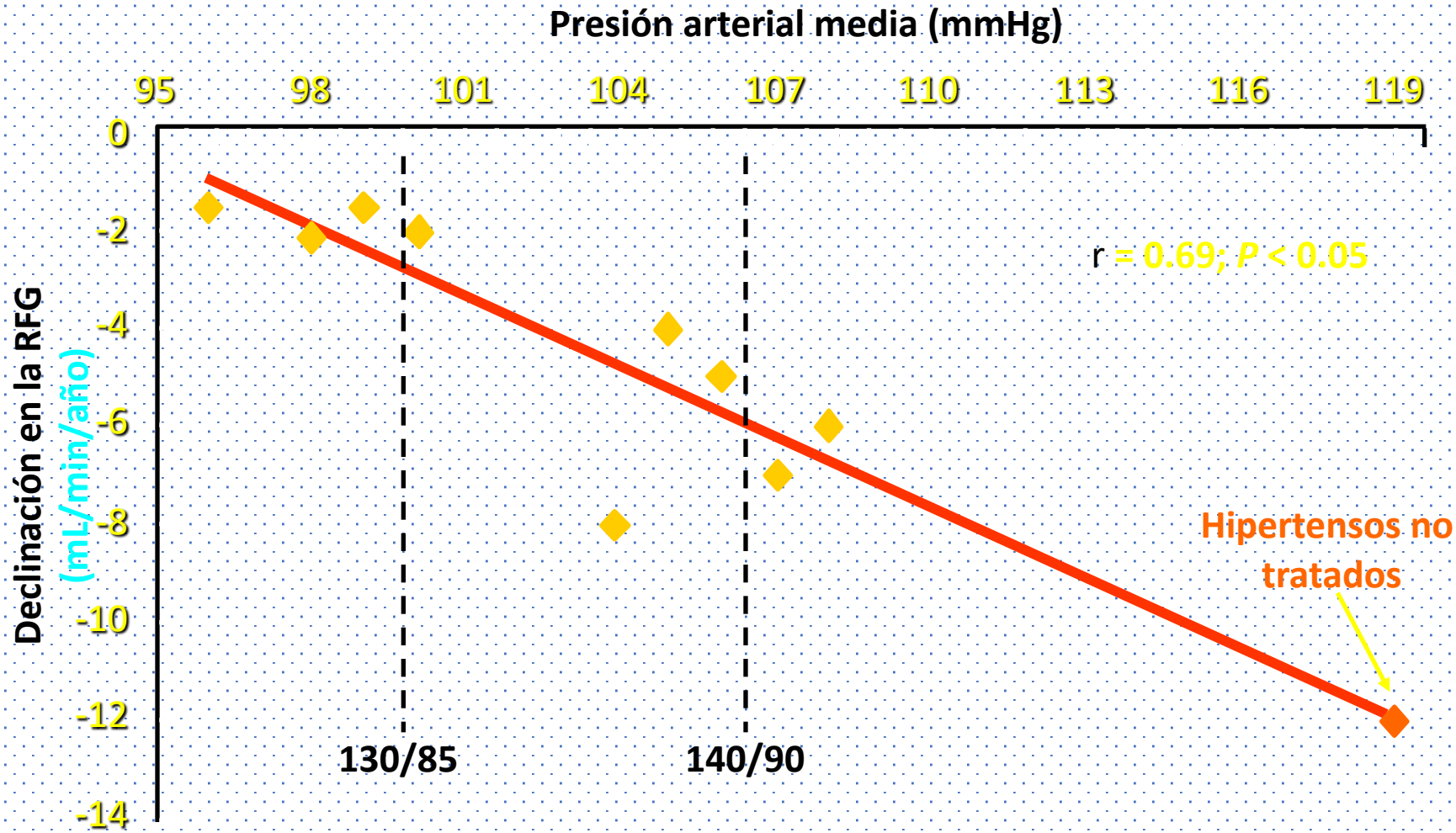
Los beneficios de la inhibición del SRAA incluyen:

- Tratamiento de la HTA
- Prevención de los eventos CV
- Tratamiento de la nefropatía diabética
- Prevención de la aparición de diabetes



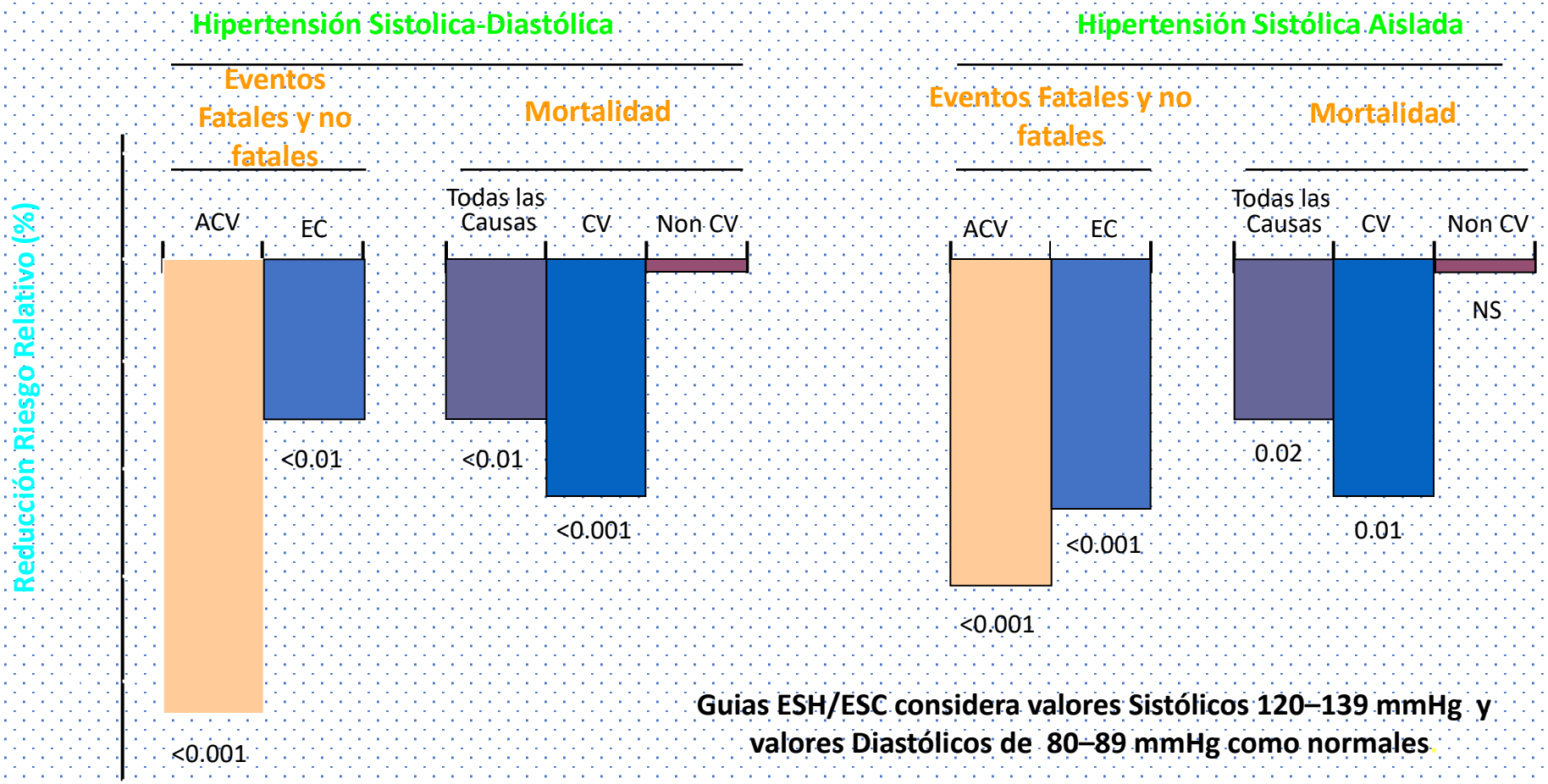
Fuente: Urina-Triana M

La reducción de la PA retarda la rata de declinación en el FG en diabéticos y no diabéticos



Bakris et al. Am J Kidney Dis 2000;36:646–661

La Morbilidad y Mortalidad Cardiovascular se reducen con un control efectivo de la Presión Arterial.

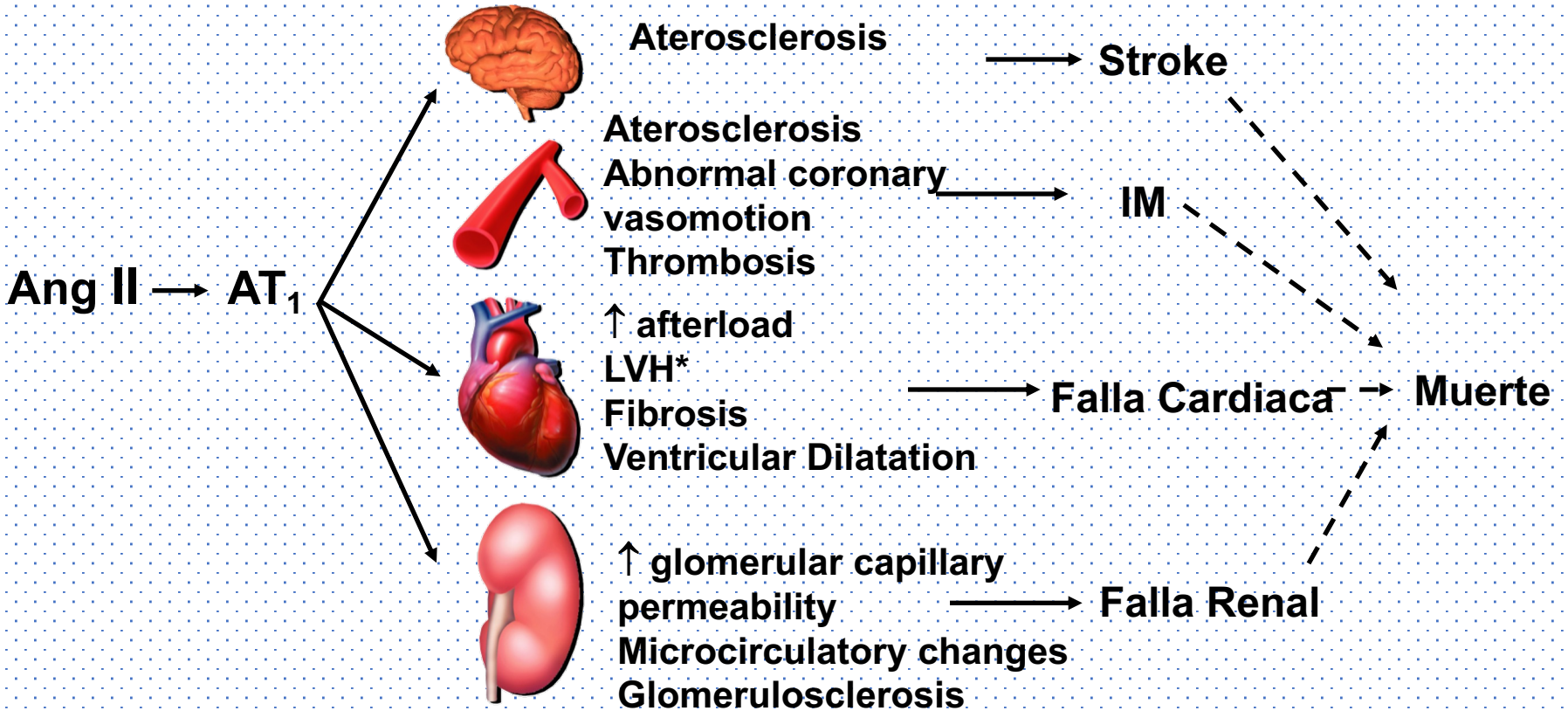


Guias ESH/ESC considera valores Sistólicos 120–139 mmHg y valores Diastólicos de 80–89 mmHg como normales.

CHD:coronary heart disease; CV:cardiovascular

1. Cifkova R et al. J Hypertens 2003;21:1011–1053

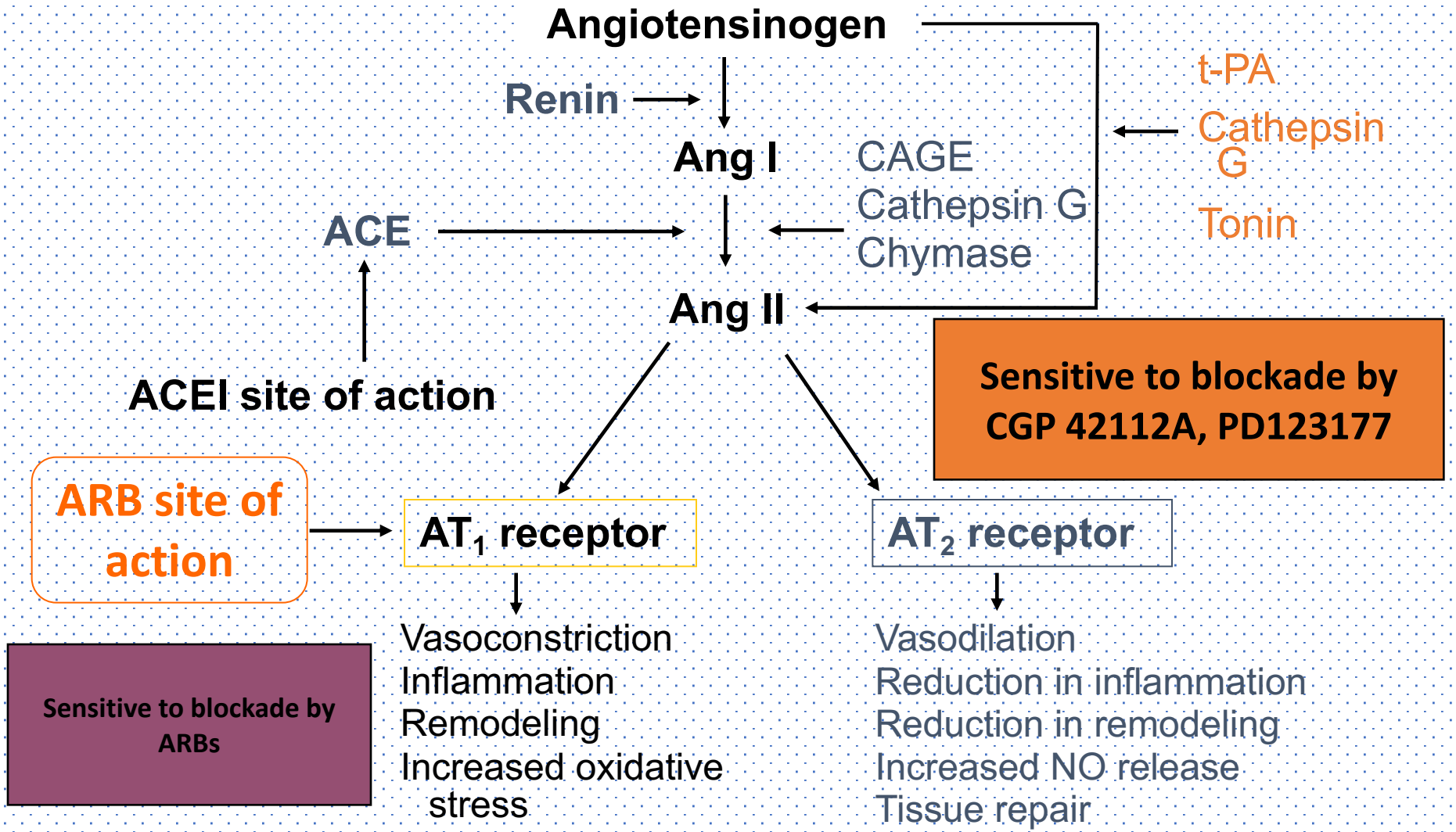
Angiotensina II juega un papel importante en el daño de organos



*LVH = left-ventricular hypertrophy.

Willenheimer R, et al. *Eur Heart J.* 1999;20:997-1008; Scheidegger KJ, Wood JM. *J Vasc Res.* 1997;34:436-446; Daugherty A et al. *J Clin Invest.* 2000;105:1605-1612; Hornig B et al. *Circulation.* 2001;103:799-805; Nickenig G, Harrison DG. *Circulation.* 2002;105:393-396; Sironi L et al. *Hypertension.* 2001;37:961-966; Oubina MP et al. *J Hypertens.* 2002;20:183-186; Jamali AH et al. *Arch Intern Med.* 2001;161:667-672; Dzau VJ. *Arch Intern Med.* 1993;153:937-942; Remuzzi G, Bertani T. *N Engl J Med.* 1998;339:1448-1456; Ruggenenti P et al. *Lancet.* 2001;357:1601-1608; Viberti G et al. *Circulation.* 2002;106:672-678.

Multiple Pathways of Angiotensin II Production



Dzau VJ. *J Hypertens.* 1989;7:933–936; de Gasparo M, Bottari S, Levens NR. In: *Hypertension. Pathophysiology, Diagnosis, and Management.* 2nd ed. New York, NY: Raven Press; 1995:1695-1720; Dzau VJ. *Hypertension.* 2001;37:1047-1052; Nickenig G, Harrison DG. *Circulation.* 2002;105:393-396.

Angiotensin II Contributes to the Development of Heart Failure

Incrementando la postcarga

- Incrementa la vasoconstriction
- Incrementa la norepinephrine
- Incrementa el PAI-1

Produce HVI

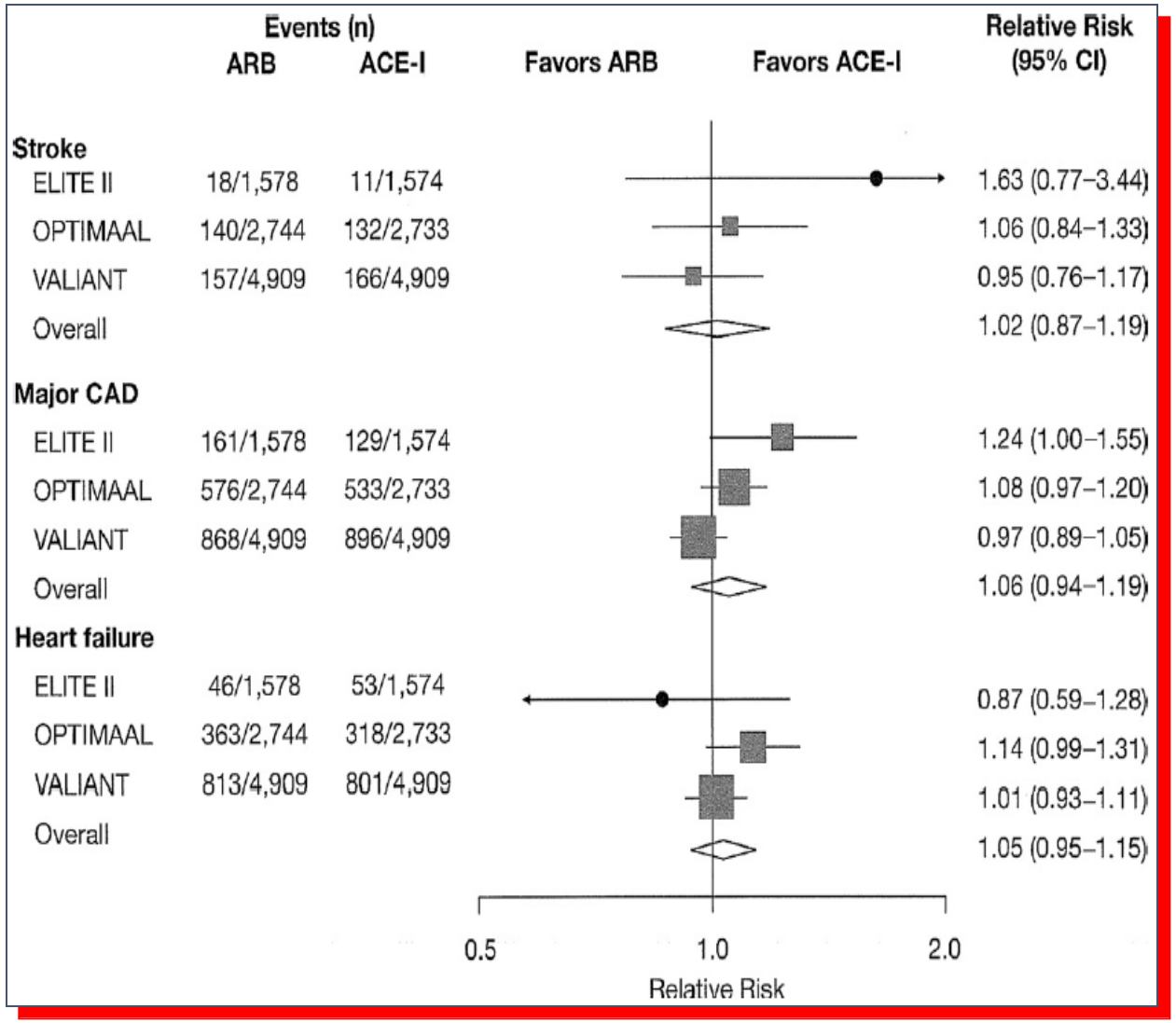
Produce remodelacion cardiaca

1. Jamali AH et al. *Arch Intern Med*. 2001;161:667-672.
2. Dzau VJ. *Arch Intern Med*.1993;153:937-942.

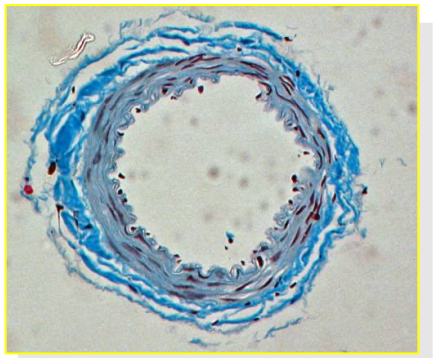
Are There Effects of Renin–Angiotensin System Antagonists Beyond Blood Pressure Control?

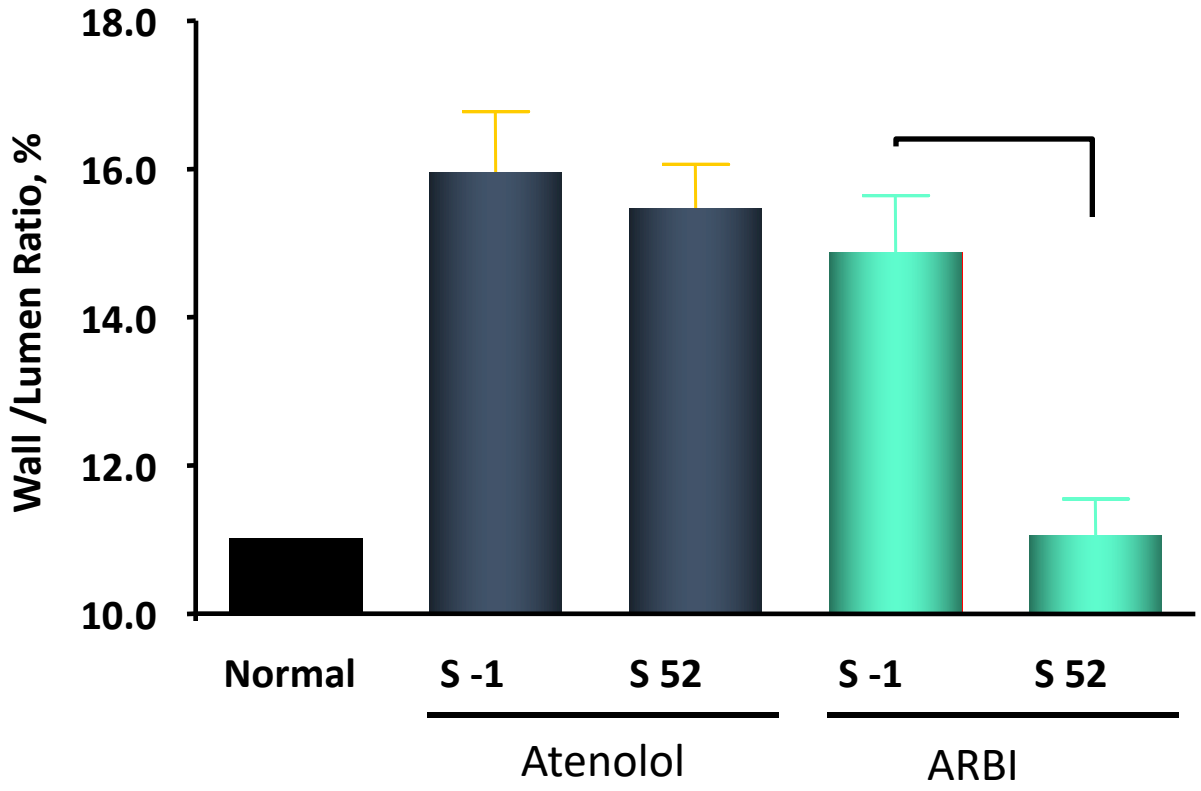
Jeffrey L. Probstfield, MD

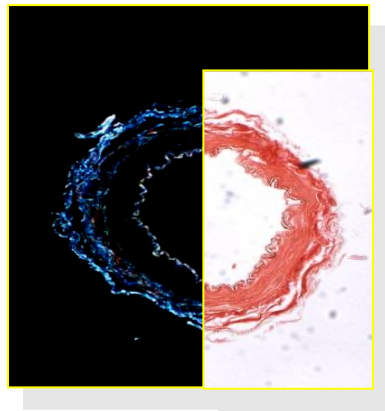
Michael A. Weber, MD



VIOS TRIAL



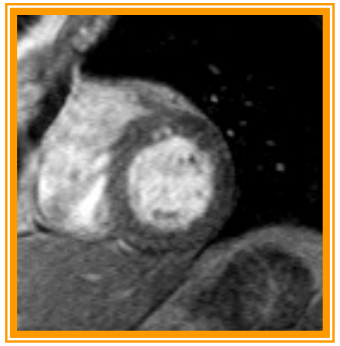


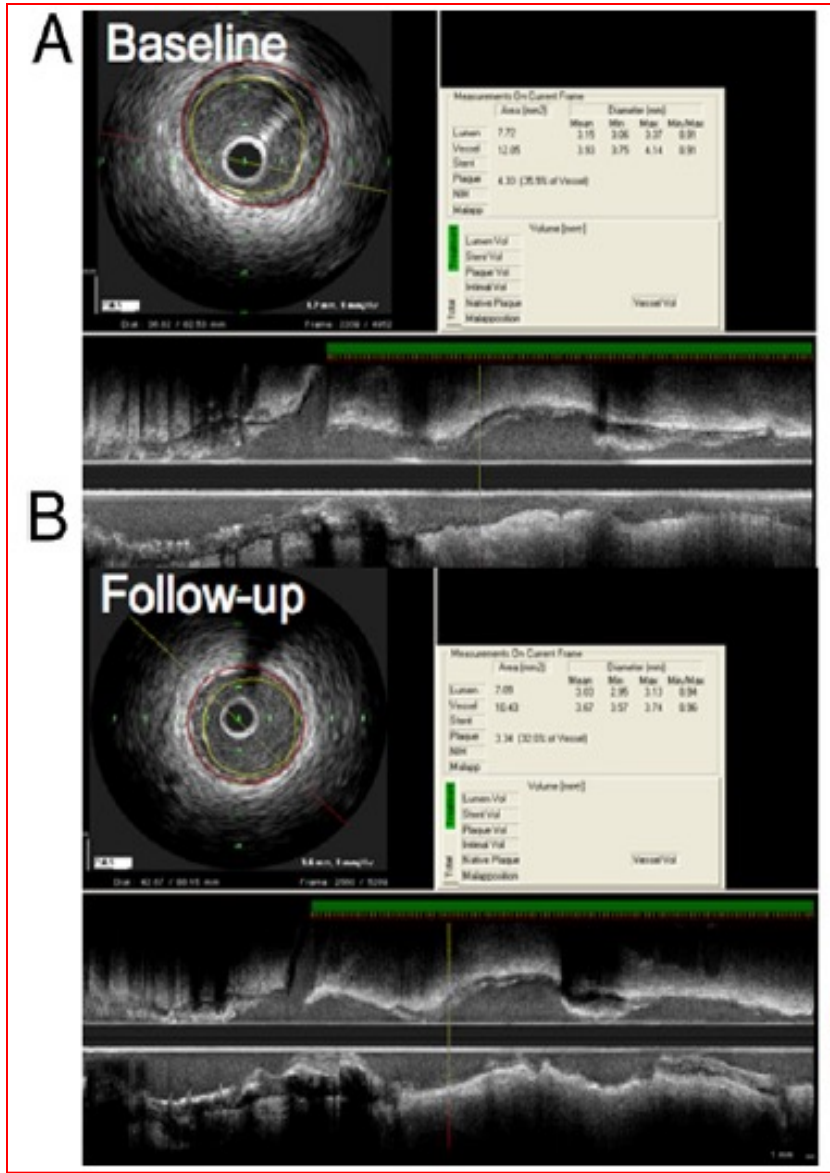


Smith R. J American Society Hyperten. 2008; 2(3):165-172

OLIVUS TRIAL

March 9, 2010, i*ournal of the American College of Cardiology*,
by **Dr Atsushi Hirohata**





Baseline

Measured lengths = 52mm

Lumen Volume = 320.7 mm3
Plaque Volume = 207.4 mm3
Vessel Volume = 528.1mm3
Percent Plaque Volume = 39.3%

14-months Follow-up

Measured lengths = 52mm

Lumen Volume = 309.1 mm3
Plaque Volume = 221.2 mm3
Vessel Volume = 530.3mm3
Percent Plaque Volume = 41.7%

Percent Change in Plaque Volume = 6.6%
Change in Percent Plaque Volume = 6.2%

OLIVUS TRIAL

Table 6 Changes in IVUS Parameters From Baseline to Follow-Up

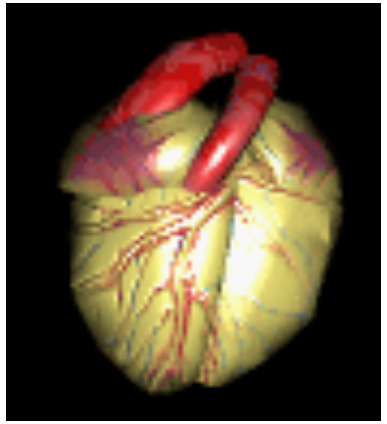
	Control (n = 121)	ARB (n = 126)	p Value
Nominal change			
Atheroma volume (mm^3)	7.1 (1.8–12.4)*	−2.6 (−7.9–2.8)	0.011
Lumen volume (mm^3)	0.3 (−8.7–9.3)	0.4 (−7.6–8.3)	0.989
Vessel volume (mm^3)	7.8 (2.5–10.5)	−2.1 (−8.5–2.5)	0.178
PAV (%)	1.1 (0.1–2.1)†	−0.1 (−0.9–0.8)	0.085
Change in total atheroma volume and PAV			
Total atheroma volume (%)	5.4 (2.4–8.5)	0.6 (−1.9–3.1)	0.016
PAV (%)	3.1 (0.7–5.6)	−0.7 (−3.4–2.0)	0.038

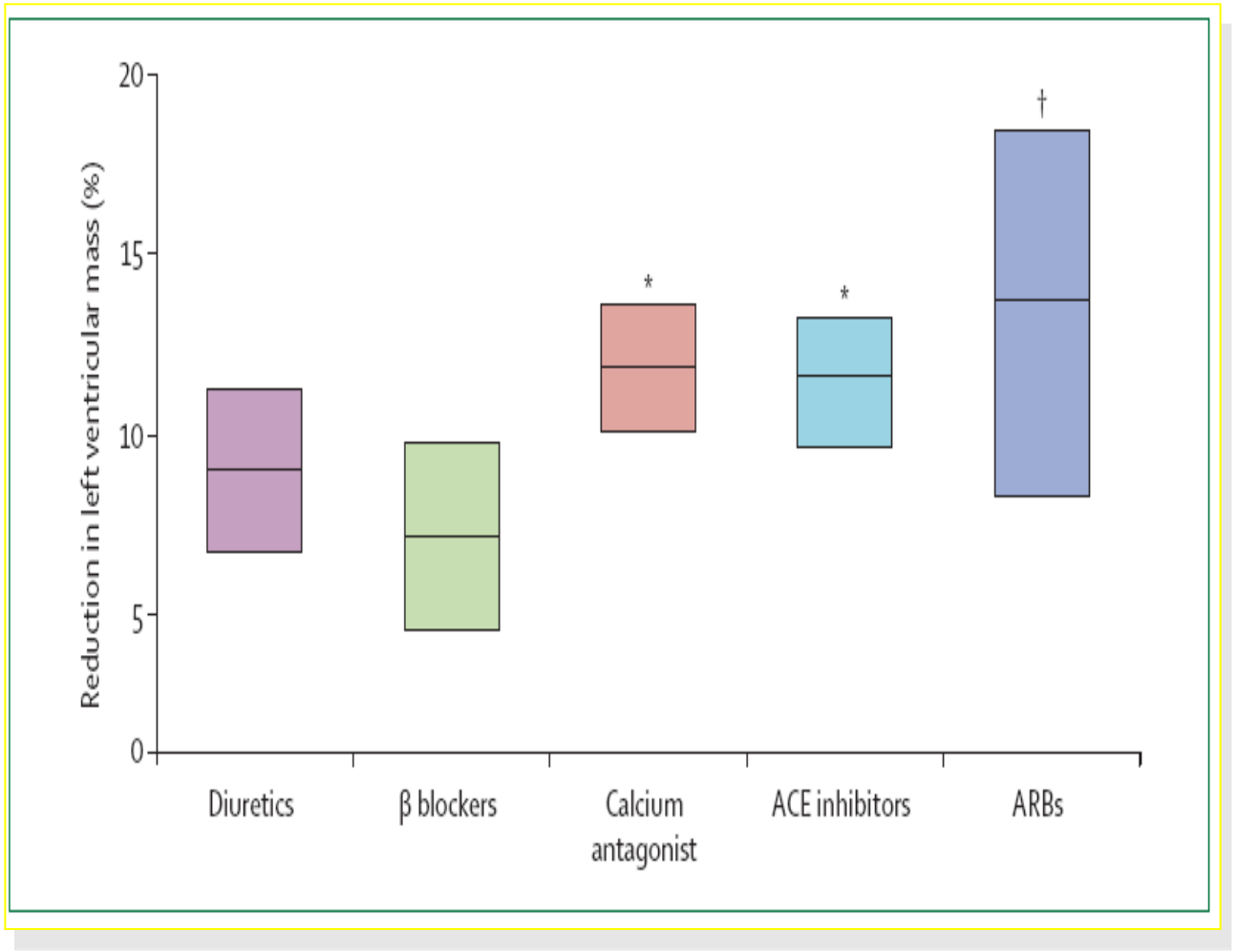
MEDICINA SUSTENTABLE EN LA EVIDENCIA

Table 3. Experimental and Clinical Evidence-Based Effects of Antihypertensive Agents in HHD

Pharmacological Class	Decrease of Blood Pressure	Regression of LVH	Repair of Remodeling
Diuretics	Yes	Mild effect	Proven for torsemide
β-Blockers	Yes	Mild-moderate effect	Apparently not
α-Blockers	Yes	Mild effect	Untested
Calcium antagonists	Yes	Marked effect	Apparently not
ACE inhibitors	Yes	Marked effect	Yes
Angiotensin receptor blockers	Yes	Marked effect	Yes
Aldosterone antagonists	Yes	Mild-moderate effect	Apparently yes
Direct renin inhibitors	Yes	Marked effect	Untested

CARDIORREPARACION





Schmieder R. Lancet 2007;369:1208-19

Table 3 Pharmacokinetic properties of ARBs [49,50,79]

	t_{max}(h)	Bioavailability (%)	$T_{1/2}$ (h)	Vd (L)	Interaction with food	Hepatic elimination (%)
Candesartan	3.0-5.0	42	9-13	0.13 (L/kg)	No	67
Eprosartan	2.0-6.0	13	5-7	308	No	90
Irbesartan	1.0-2.0	60-80	12-20	53-93	No	80
Losartan	1.0 (3.0-4.0)[1]	33	2 (4-6)[1]	34 (12)[1]	No	60
Olmesartan	1.4-2.8	26[2]	11.8-14.7	15-20	No	51-66[3]
Telmisartan	1	43	24	500	No	> 98[4]
Valsartan	2	23	7	17	No	83
Azilsartan	1.5-3.0	60	11	16	No	55

Abbreviations: ARB = angiotensin II receptor blocker; $t_{1/2}$ = terminal elimination half-life; t_{max} = time to maximum plasma concentration; Vd = volume of distribution

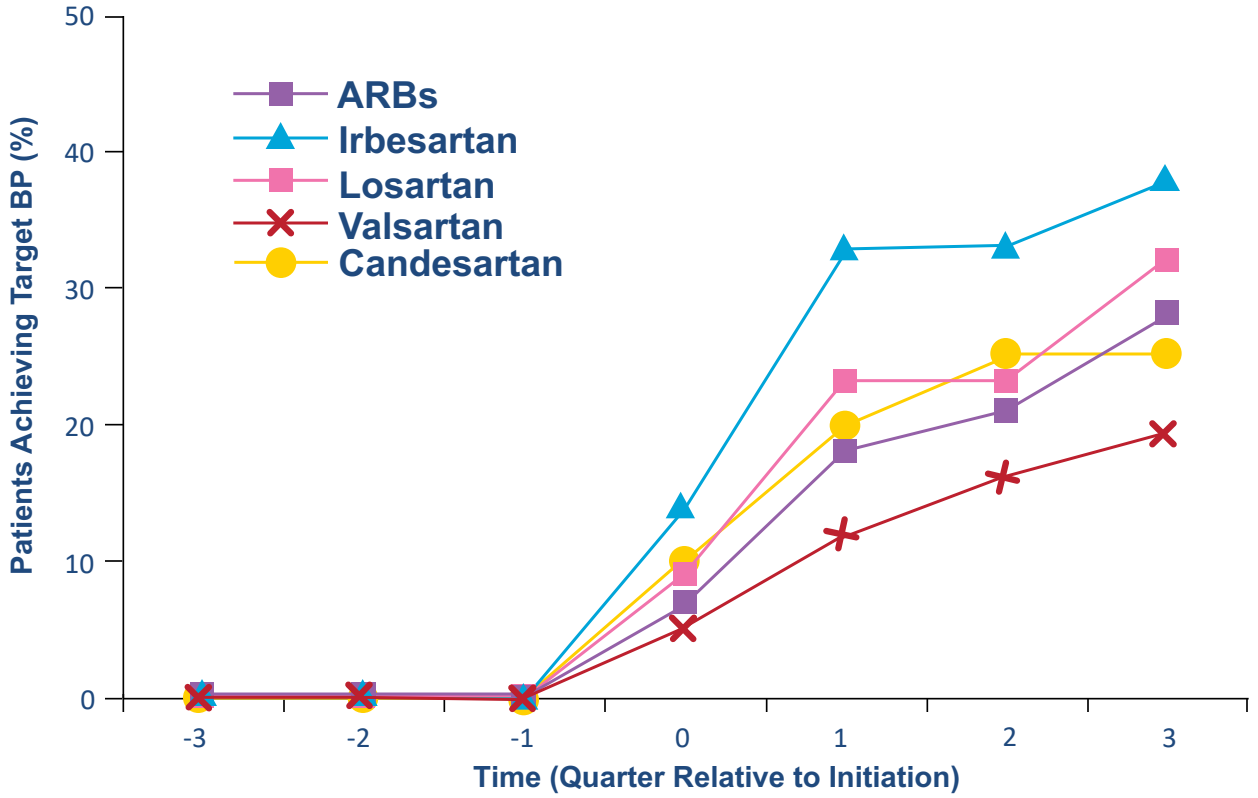
[1] Values in parentheses are for the active metabolite of losartan

[2] For olmesartan medoxomil

[3] Based on urinary recovery rate for intravenous olmesartan

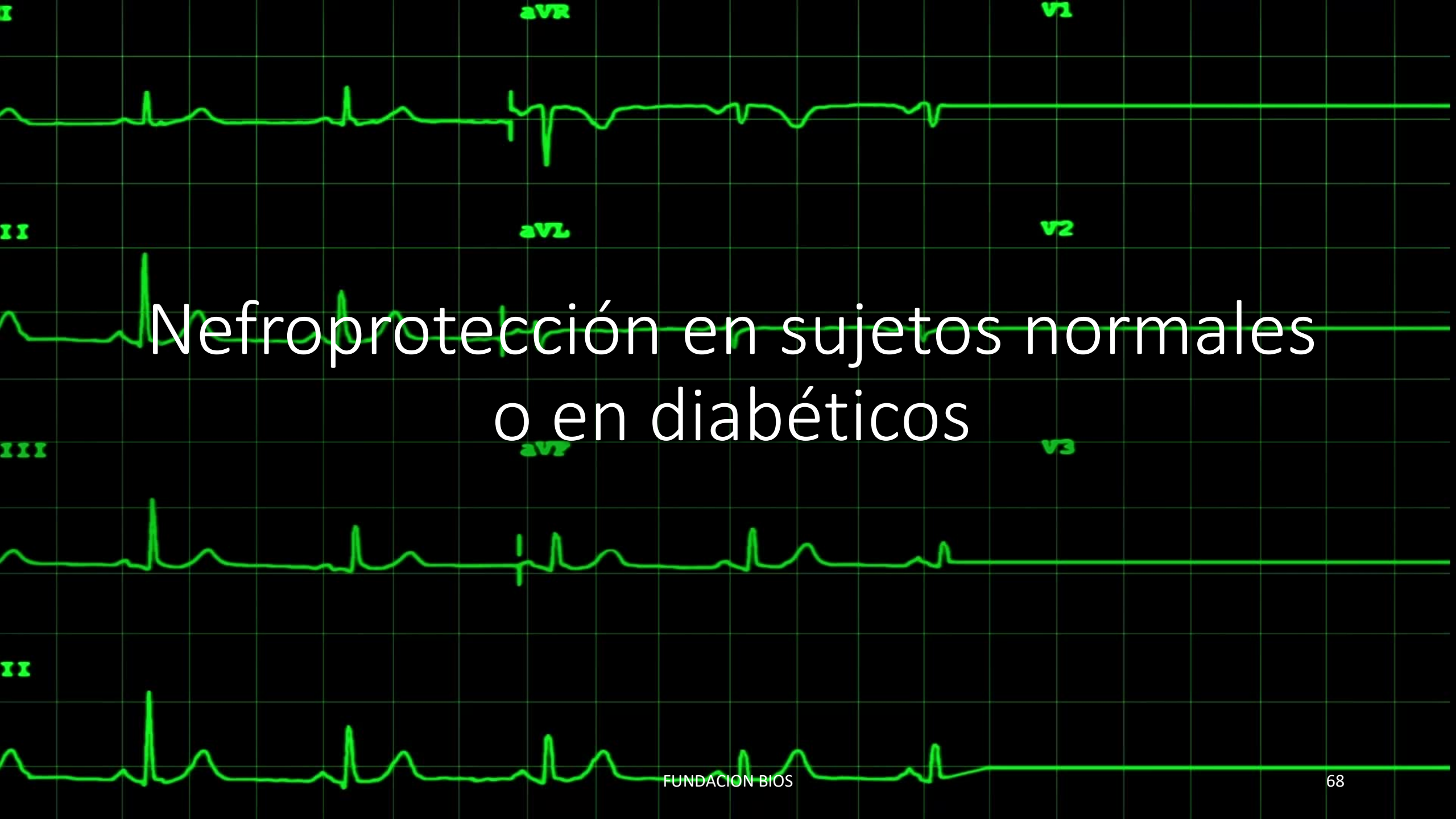
[4] Faecal recovery for telmisartan

Results: BP control with monotherapy (intraclass analysis)



R Petrella. European Cardiology, 2010; 6(3):33-8

P = 0.01 for irbesartan vs losartan
P = 0.001 for irbesartan vs valsartan
P = 0.001 for irbesartan vs candesartan

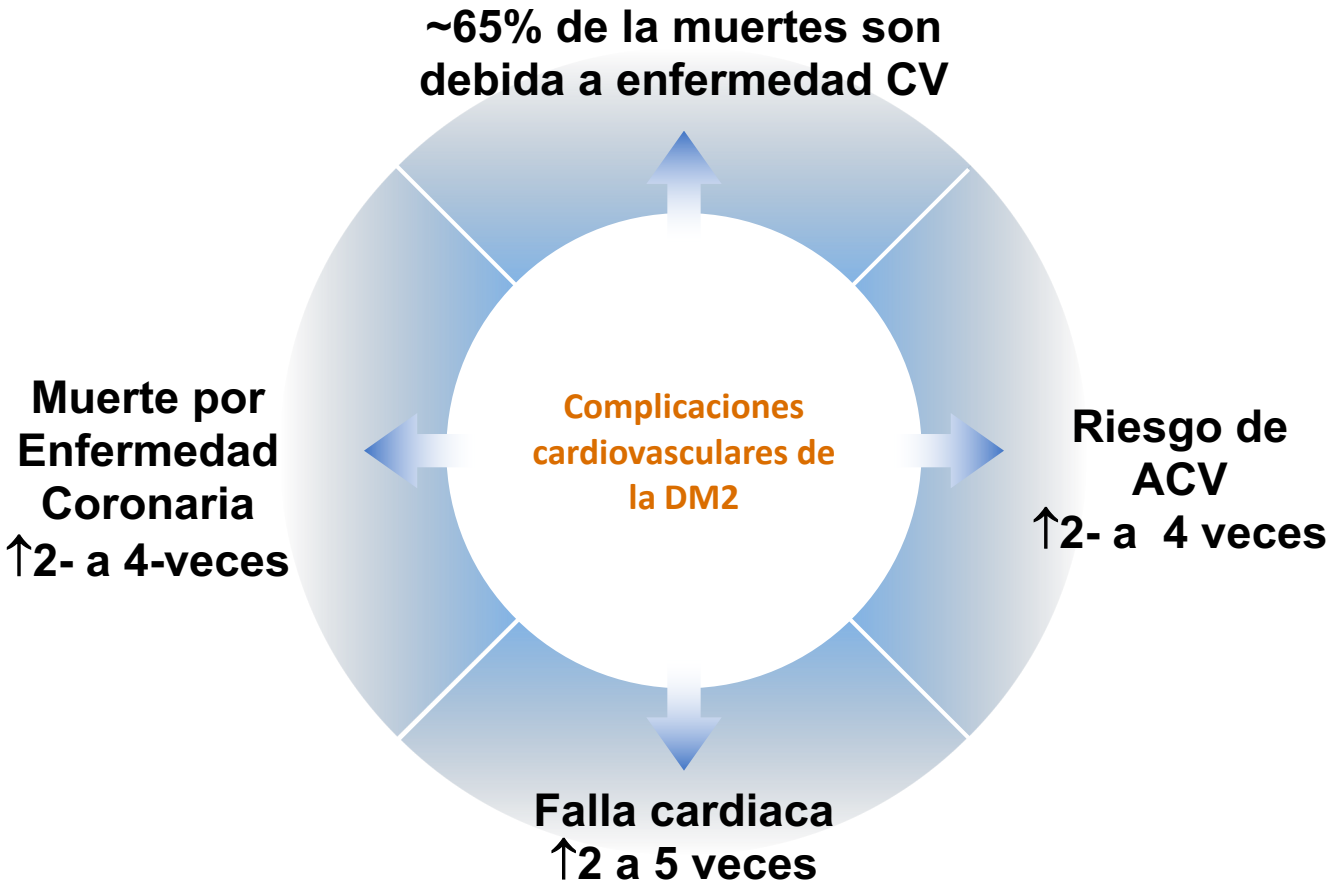


Nefroprotección en sujetos normales o en diabéticos

La famosa Diabetologa Finlandesa (Profesora Medicina Univ Helsinski , Hannele Yki-Järvinen, sostiene que:

"La Diabetes es una enfermedad cardiovascular la cual usted diagnostica al medir la glucosa en sangre"

Enfermedad Cardiovascular y Diabetes Mellitus



DM2 = diabetes mellitus tipo 2

Bell DSH. *Diabetes Care*. 2003;26:2433-41.
Centers for Disease Control (CDC). www.cdc.gov

N=969,531

Después de un seguimiento de 6 años, las personas que vivían con DM e HTA o ERC tenían un mejor control glucémico (niveles más bajos de HbA1c) que aquellas con obesidad. En ambos sujetos DM con HTA o ERC, los niveles de HbA1c fueron más bajos en aquellos que tenían la condición. Estos hallazgos fueron contrarios a los sujetos obesos/no obesos, donde encontramos niveles más altos de HbA1c cuando la obesidad estaba presente. Se observó una mayor brecha en los niveles de HbA1c entre la presencia o no de comorbilidades en las personas que viven con DM e HTA/no HTA que en los otros grupos. Al estimar la variación longitudinal de la HbA1c al cambiar el punto de corte de IMC ≥ 30 kg/m2 a 25 kg/m2, la tendencia fue opuesta a la observada en obesos, con un mejor control glucémico en aquellos con un IMC ≥ 25 kg/m2

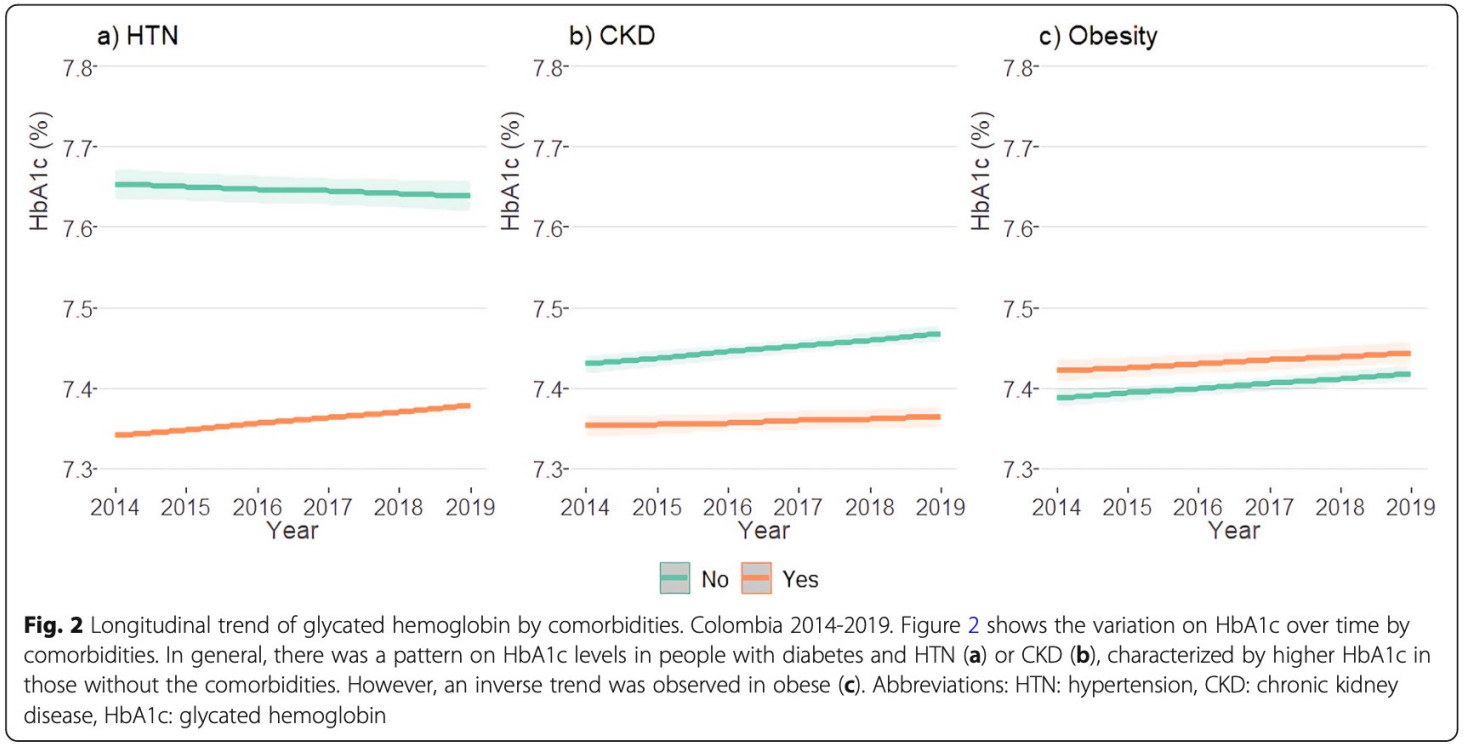


Fig. 2 Longitudinal trend of glycated hemoglobin by comorbidities. Colombia 2014-2019. Figure 2 shows the variation on HbA1c over time by comorbidities. In general, there was a pattern on HbA1c levels in people with diabetes and HTN (**a**) or CKD (**b**), characterized by higher HbA1c in those without the comorbidities. However, an inverse trend was observed in obese (**c**). Abbreviations: HTN: hypertension, CKD: chronic kidney disease, HbA1c: glycated hemoglobin

Urina-Jassir M, Herrera-Parra LJ, Hernández Vargas JA, Valbuena-García AM, Acuña-Merchán L, Urina-Triana M. The effect of comorbidities on glycemic control among Colombian adults with diabetes mellitus: a longitudinal approach with real-world data. BMC Endocr Disord. 2021 Jun 26;21(1):128. doi: 10.1186/s12902-021-00791-w. PMID: 34174843; PMCID: PMC8235812.

Clasificación de los estadios de enfermedad renal crónica*

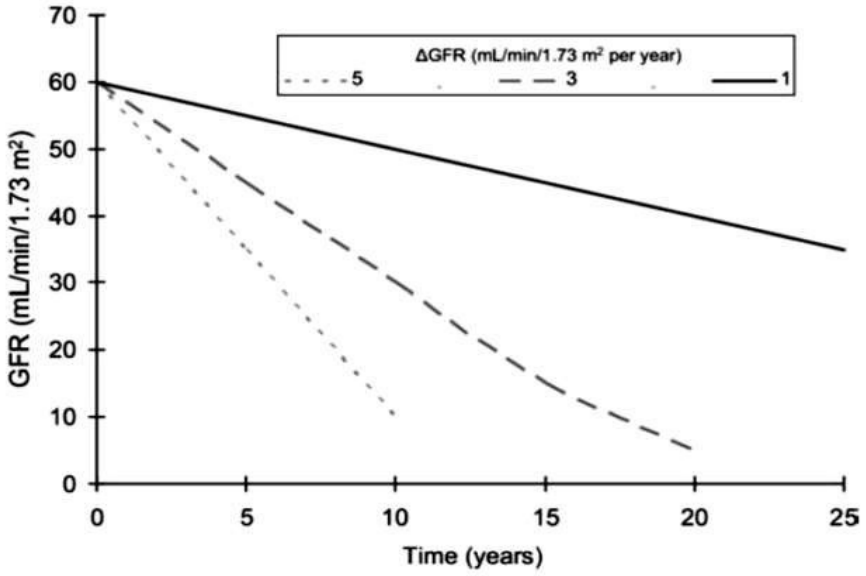
La ERC se clasifica por el filtrado glomerular (FG) de acuerdo a la siguiente tabla:

Etapa	Grado de Enfermedad Renal	FG (ml / min / 1.73m2)
5	Insuficiencia renal que requiere TSR	**< 15**
4	Insuficiencia renal severa	**15-29**
3	Insuficiencia renal moderada	**30-59**
2	Insuficiencia renal leve con proteinuria, hematuria o anormalidad estructural	**60-89**
1	Daño renal con proteinuria, hematuria o anormalidad estructural y FG normal	**≥ 90**

K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification and stratification. Am J Kidney Dis 2002; 39:S1-S266

Decline of GFR in patients with diabetic nephropathy

Rate of annual kidney function decrease has an important role on time to reach end-stage renal disease



Turgut F. et al.: Am. J. Kidney Dis., 2010; 55: 928-940

Outcomes in Framinghan Participants with Diabetes. The importance of Blood Pressure Cardiovascular

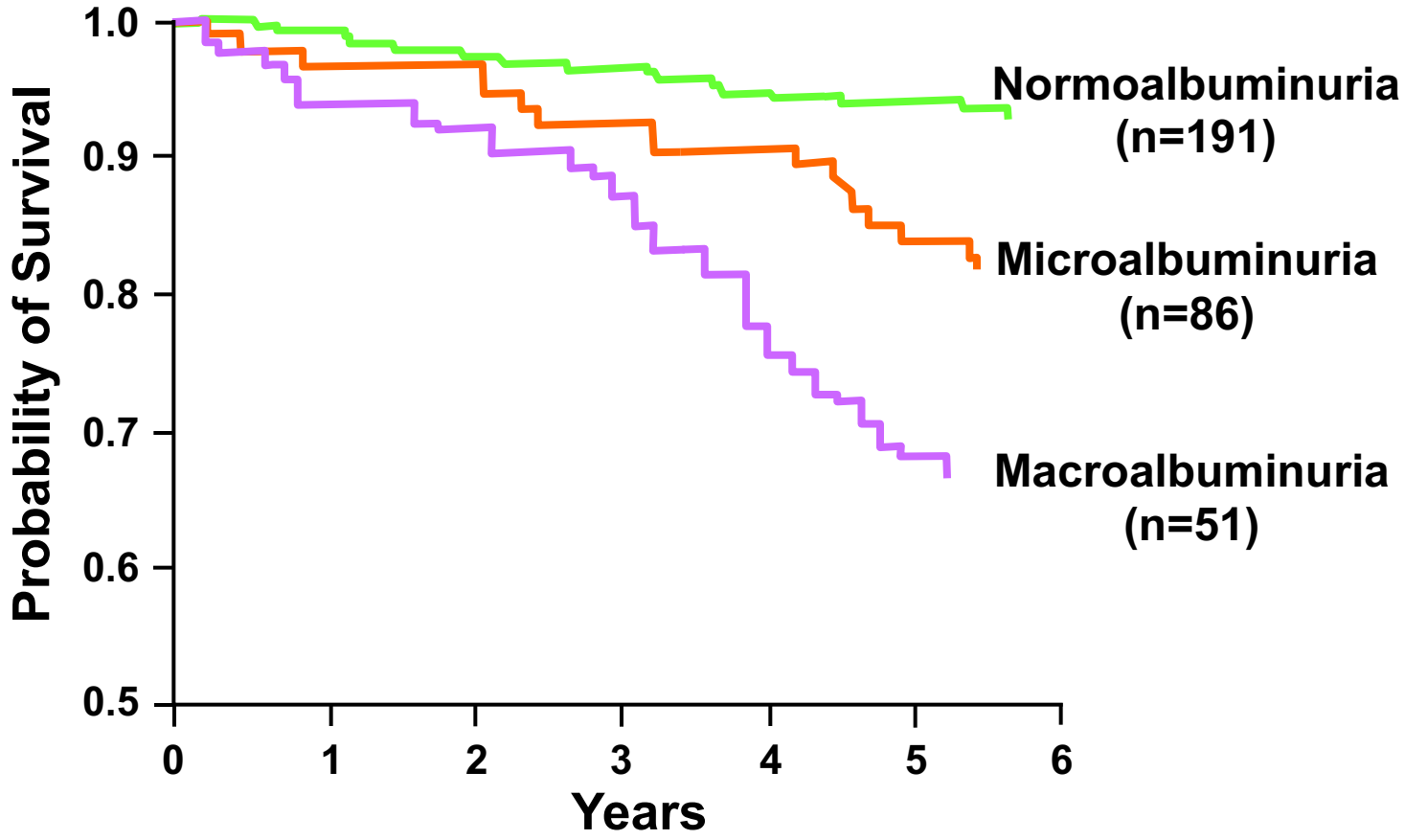
De 1145 sujetos diagnosticados de novo con DM y que no tenía historia previa de eventos cardiovasculares el 58% tuvo hipertensión arterial al momento de hacer el diagnóstico de DM.

En el seguimiento de cohortes de 1968-96 y de 1971 a 2001

Hypertension 2011; 57:891-897

Proteinuria Is an Independent Risk Factor for All-Cause Mortality in Type 2 Diabetes

P<0.01 normoalbuminuria vs microalbuminuria and macroalbuminuria.
P<0.05 microalbuminuria vs macroalbuminuria.



Gall MA et al. *Diabetes.* 1995;44:1303-1309.

Categories of Albuminuria

Albuminuria categories in CKD

Category	AER (mg/24 h)	ACR (approximate equivalent) (mg/mmol)	ACR (approximate equivalent) (mg/g)	Terms
A1	<30	<3	<30	Normal to mildly increased
A2	30–300	3–30	30–300	Moderately increased*
A3	>300	>30	>300	Severely increased**

Abbreviations: ACR, albumin-to-creatinine ratio; AER, albumin excretion rate; CKD, chronic kidney disease.

*Relative to young adult level.

**Including nephrotic syndrome (albumin excretion usually >2200 mg/24 h (ACR >2220 mg/g; >220 mg/mmol)).

Levin A., Stevens P.E., Kidney Int., 2014, 85, 49-61

Prognosis of CKD by GFR and albuminuria categories: KDIGO 2012

GFR categories (ml/min per 1.73 m²) Description and range				Persistent albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30–300 mg/g 3–30 mg/mmol	>300 mg/g >30 mg/mmol
	G1	Normal or high	⩾90			
	G2	Mildly decreased	60–89			
	G3a	Mildly to moderately decreased	45–59			
	G3b	Moderately to severely decreased	30–44			
	G4	Severely decreased	15–29			
	G5	Kidney failure	<15			

Green: low risk (if no other markers of kidney disease, no CKD); yellow: moderately increased risk; orange: high risk; red, very high risk.

Levin A., Stevens P.E., Kidney Int., 2014, 85, 49-61

Referral decison making by GFR and albuminuria)

GFR categories (ml/min per 1.73 m²) Description and range				Persistent albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30–300 mg/g 3–30 mg/mmol	>300 mg/g >30 mg/mmol
	G1	Normal or high	⩾90		Monitor	Refer*
	G2	Mildly decreased	60–89		Monitor	Refer*
	G3a	Mildly to moderately decreased	45–59	Monitor	Monitor	Refer
	G3b	Moderately to severely decreased	30–44	Monitor	Monitor	Refer
	G4	Severely decreased	15–29	Refer*	Refer*	Refer
	G5	Kidney failure	<15	Refer	Refer	Refer

Referral decision making by GFR and albuminuria. *Referring clinicians may wish to discuss with their nephrology service depending on local arrangements regarding monitoring or referring.

Levin A., Stevens P.E., Kidney Int., 2014, 85, 49-61

Guide to frequency of monitoring (number of times per year) by GFR and albuminuria category

GFR categories (ml/min per 1.73 m²) Description and range				Persistent albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30–300 mg/g 3–30 mg/mmol	>300 mg/g >30 mg/mmol
	G1	Normal or high	⩾90	1 if CKD	1	2
	G2	Mildly decreased	60–89	1 if CKD	1	2
	G3a	Mildly to moderately decreased	45–59	1	2	3
	G3b	Moderately to severely decreased	30–44	2	3	3
	G4	Severely decreased	15–29	3	3	4+
	G5	Kidney failure	<15	4+	4+	4+

GFR and albuminuria grid to reflect the risk of progression by intensity of coloring (green, yellow, orange, red, deep red). The numbers in the boxes are a guide to the frequency of monitoring (number of times per year).

Levin A., Stevens P.E., Kidney Int., 2014, 85, 49-61

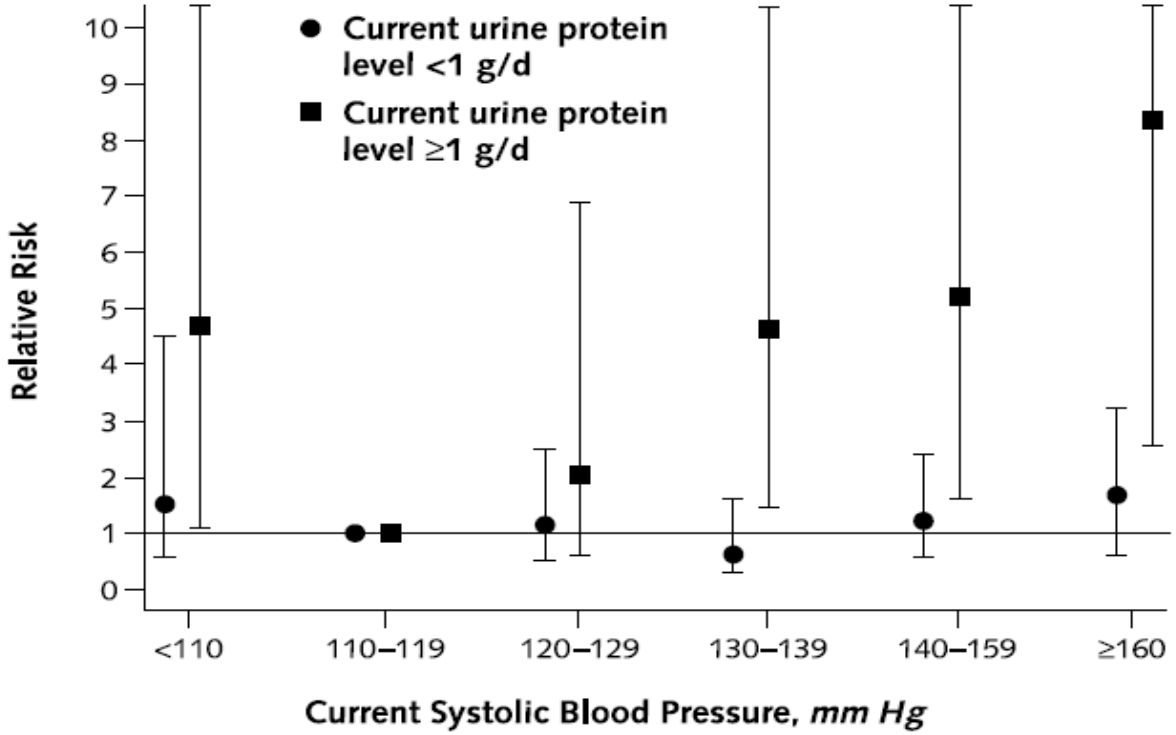
Nephroprotection: Where are we in 2023?

With confirmed beneficial effects:

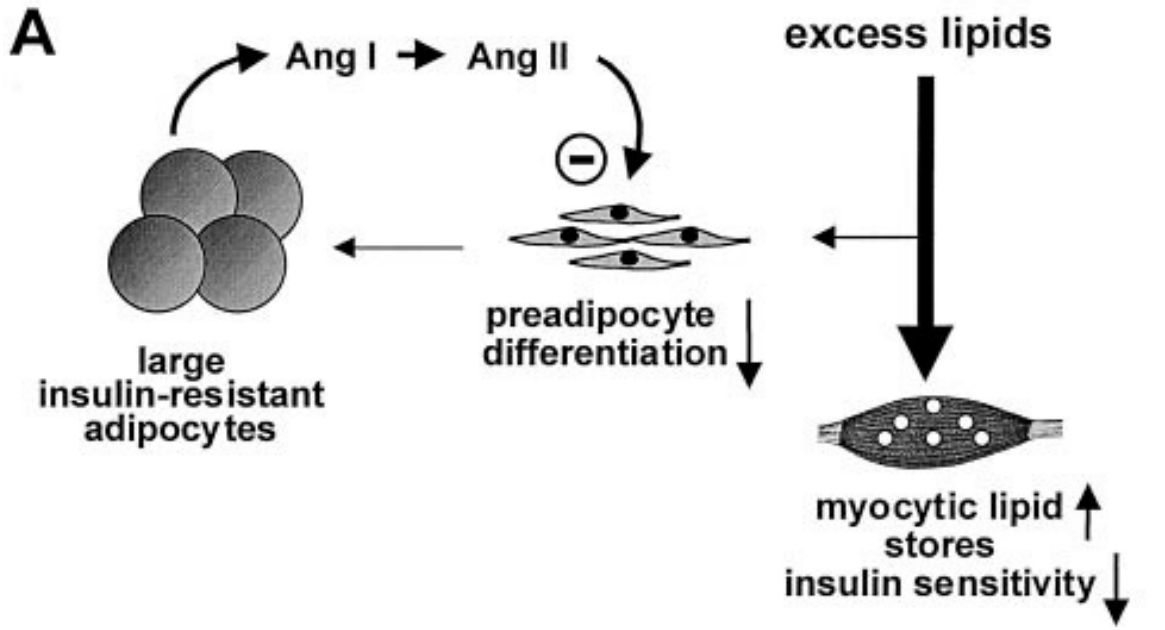
- Reduction of blood pressure
- Blockade of the RAAS (beyond BP reduction)
- Reduction of BMI
- Reduction of protein intake
- Correction of phosphatemia
- Correction of metabolic acidosis
- Correction of glucose (HBA1C) in DM patients
- Supplementation of Vitamin D
- Renal denervation

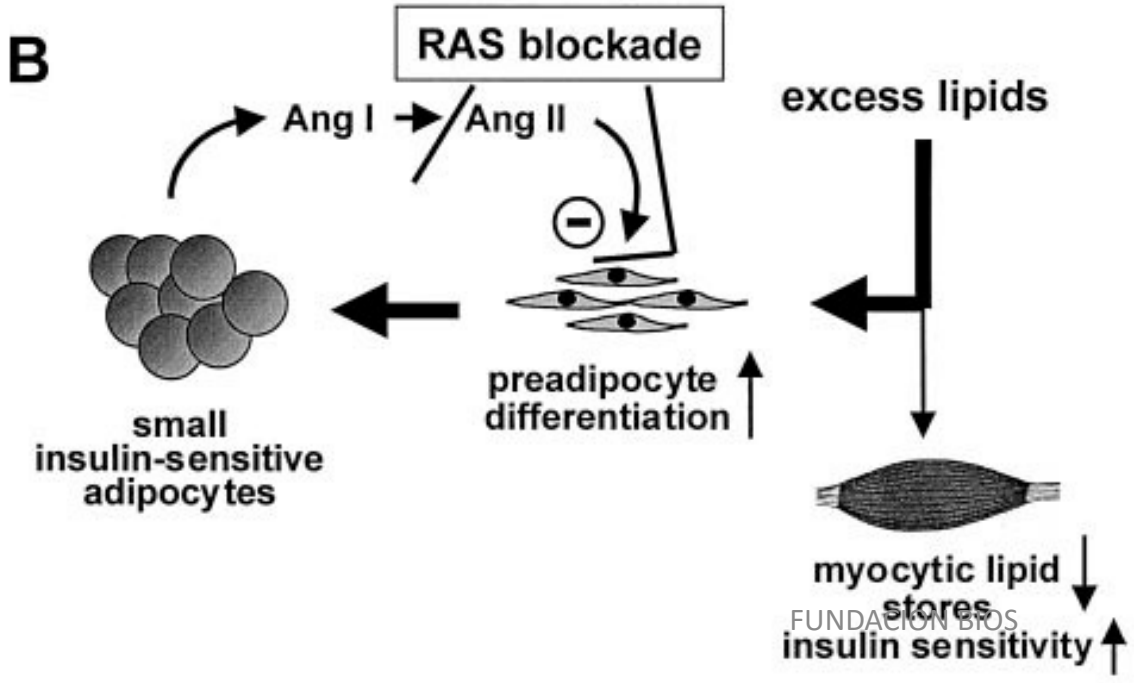
Relative risk for kidney disease progression based on current level of systolic blood pressure and current urine protein excretion

Jafar T.H. Ann Intern Med 2003, 193, 244-252



Progression of Chronic Kidney Disease: The Role of Blood Pressure Control, Proteinuria, and Angiotensin-Converting Enzyme Inhibition A Patient-Level Meta-Analysis





Angiotensin blockade prevents type 2 diabetes by formation of fat cells.

In Sharma, AM. et al. Hypertension 2002

Table 1 Potential antidiabetic mechanisms of interrupting the renin–angiotensin system

Both ACE inhibitors and ARBs may interfere with adverse effects of angiotensin II on:

- Insulin signaling
- Tissue blood flow
- Oxidative stress
- Sympathetic activity
- Adipogenesis

ACE, angiotensin-converting enzyme; ARBs, angiotensin receptor blockers.

Table 2 Antidiabetic mechanisms of ACE inhibitors and particular ARBs that may go beyond their effects on the renin–angiotensin system

ACE inhibitors may enhance glucose metabolism by:

- Activating bradykinin/nitric oxide pathways

Particular ARBs (e.g. telmisartan) may improve glucose and lipid metabolism by:

- Activating PPARγ

ACE, angiotensin-converting enzyme; ARBs, angiotensin receptor blockers; PPARγ, peroxisome proliferator activated receptor gamma.

Table 4 Results of clinical trials indicating the renoprotective nature of ARBs

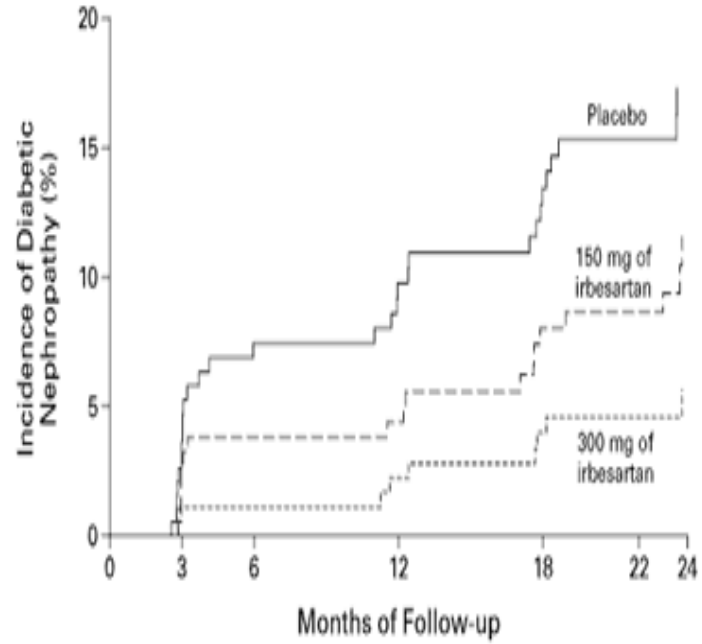
Study	Patients	n	Treatment	Duration	Principle findings
AMADEO® [122]	Hypertension and diabetic nephropathy	860	Telmisartan or losartan	52 weeks	Telmisartan was superior to losartan in reducing proteinuria
CALM [25]	Type 2 diabetes with hypertension and microalbuminuria	199	Candesartan, lisinopril or both	24 weeks	Candesartan was as effective as lisinopril in reducing UACR. Combined treatment was associated with a greater reduction in UACR than monotherapeutic treatment (statistically significant versus candesartan monotherapy)
DETAIL® [99]	Hypertension, Type 2 diabetes and early nephropathy	250	Telmisartan or enalapril	5 years	Telmisartan was not inferior to enalapril in providing long-term renoprotection
IDNT [119]	Hypertension and diabetic nephropathy	1715	Irbesartan, amlodipine or placebo	Mean 2.6 years	Irbesartan was superior to amlodipine and placebo in preventing the primary composite end point of: a doubling of the base-line serum creatinine concentration, the development of ESRD, or death from any cause. This was independent of BP
IRMA 2 [118]	Hypertension, type 2 diabetes and microalbuminuria	590	Irbesartan or Placebo	2 years	Irbesartan was superior to placebo in preventing diabetic nephropathy
MARVAL [121]	Diabetic nephropathy with and without hypertension	332	Valsartan or amlodipine	24 weeks	Valsartan was superior to amlodipine in reducing microalbuminuria
RENAAL [120]	Diabetic nephropathy	1513	Losartan or placebo	Mean 3.4 years	Losartan was superior to placebo in preventing increases in UACR and progression to ESRD. There was no difference in mortality
ROADMAP [123]	Type 2 diabetes with normoalbuminuria	4449	Olmesartan or placebo	Median 3.2 years	Olmesartan delayed the time to onset of microalbuminuria (statistical significance lost on adjustment for blood pressure difference)
VIVALDI® [124]	Hypertension and diabetic nephropathy	885	Telmisartan or valsartan*	52 weeks	Telmisartan and valsartan provided similar renoprotection

Abbreviations: AMADEO® = A trial to compare telMisartan 40 mg titrated to 80 mg versus losArtan 50 mg titrated to 100 mg in hypertensive type 2 DiabEtic patients with Overt nephropathy; ARB = angiotensin II receptor blocker; BP = blood pressure; CALM = CAndesartan and Lisinopril Microalbuminuria; DETAIL® = Diabetics Exposed to Telmisartan And enalaprIL study; ESRD, end-stage renal disease; IDNT = Irbesartan type II Diabetic Nephropathy Trial; IRMA2 = IRbesartan in patients with type 2 diabetes and MicroAlbuminuria; MARVAL = MicroAlbuminuria Reduction with VALsartan trial; RENAAL = Reduction of Endpoints in NIDDM with the Angiotensin II Antagonist Losartan; ROADMAP = Randomized Olmesartan And Diabetes Microalbuminuria Prevention; UACR = urine albumin:creatinine ratio; VIVALDI® = A trial to inVestigate the efficacy of telmIsartan versus VALsartan in hypertensive type 2 Diabetic patients with overt nephropathy.

*Additional hypertensive treatment was allowed in VIVALDI®.

The Effect of Irbesartan on the Development of Diabetic Nephropathy in Patients with Type 2 Diabetes

Volume 345:870-878 September 20, 2001



No. at Risk

Placebo	201	201	164	154	139	129	36
150 mg of irbesartan	195	195	167	161	148	142	45
300 mg of irbesartan	194	194	180	172	159	150	49

The Irbesartan in Diabetic Nephropathy Trial

IDNT overview

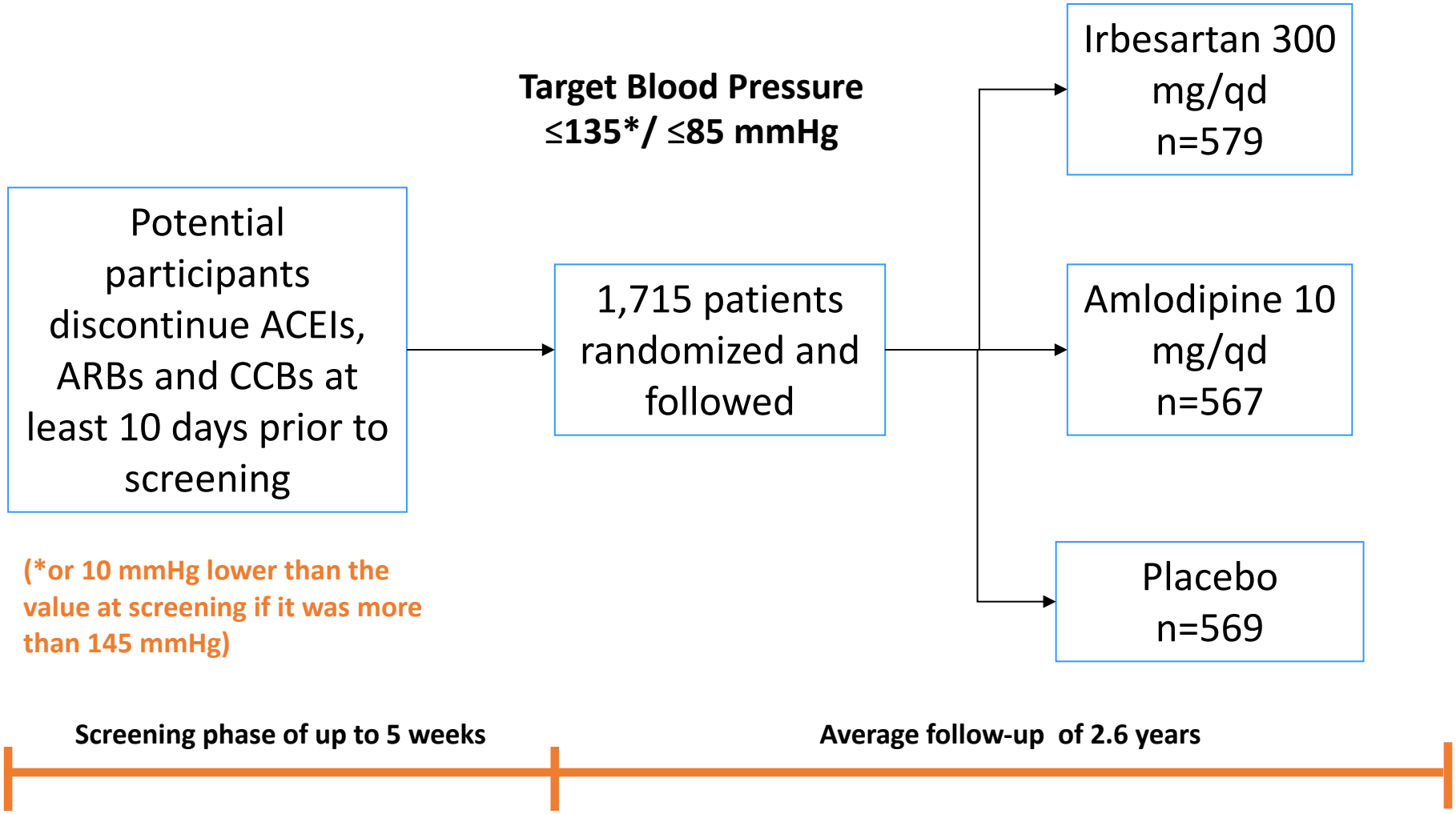
- Randomized, double-blind trial to determine if irbesartan, an angiotensin II receptor blocker, and amlodipine, a calcium channel blocker, slow the progression of nephropathy in type 2 diabetics

Population

- 1,715 patients (30 to 70 years old)
  - Diagnosed type 2 diabetes
  - Hypertension (systolic BP>135, diastolic BP >85 mmHg or treatment w/ antihypertensive agents)
  - Nephropathy (urinary protein excretion of at least 900 mg/24hrs and serum creatinine between 1.0–3.0 mg/dL in women, and 1.2–3.0 mg/dL in men)

Lewis EJ, et al. N Engl J Med. 2001;345(12):851-860.

IDNT Study Design



Lewis EJ, et al. N Engl J Med. 2001;345(12):851-860.

IDNT Endpoints

Primary Endpoint

- Composite of a doubling of serum creatinine, end stage renal disease (as indicated by starting dialysis, serum creatinine $\geq$ 6 mg/dl, or transplantation), or death

Secondary Cardiovascular Endpoint

- Composite of death from cardiovascular causes, nonfatal myocardial infarction, heart failure resulting in hospitalization, a permanent neurologic deficit caused by a cerebrovascular event, or lower limb amputation above the ankle

Lewis EJ, et al. N Engl J Med. 2001;345(12):851-860.

IDNT Baseline Characteristics*

	Irbesartan Group n=579	Amlodipine Group n=567	Placebo Group n=569
Mean age (yrs)	59.3	59.1	58.3
Male (%)	65	63	71
Mean Systolic BP (mmHg) Mean Diastolic BP (mmHg)	160 87	159 87	158 87
Mean BMI (kg/m^2)	31.0	30.9	30.5
Median urinary albumin excretion (g/24hr)	1.9	1.9	1.9
Mean serum creatinine (mg/dl)	1.67	1.65	1.69
Mean glycosylated hemoglobin (%)	8.1	8.2	8.2

***The differences between the treatment groups were not statistically significant, except for the smaller number of females in the placebo group (P=0.02)**

Lewis EJ, et al. N Engl J Med. 2001;345(12):851-860.

IDNT Irbesartan vs Amlodipine
Primary and Secondary Endpoints

	Irbesartan Group n=579		Amlodipine Group n=567		P value	Unadjusted relative risk (95% CI)
	n	%	n	%		
Primary composite endpoint*	189	32.6	233	41.1	0.006	0.77 (0.63-0.97)
• Doubling of serum creatinine	98	16.9	144	25.4	<0.001	0.63 (0.48-0.81)
• End stage renal disease	82	14.2	104	18.3	0.07	0.77 (0.57-1.03)
• Death	87	15.0	83	14.6	0.80	1.04 (0.77-1.40)
Secondary composite endpoint‡	138	23.8	128	22.6	0.79	1.03 (0.81-1.31)

*Composite of a doubling of serum creatinine, end stage renal disease, or death

‡Composite of death from cardiovascular causes, nonfatal myocardial infarction, heart failure resulting in hospitalization, a permanent neurologic deficit caused by a cerebrovascular event, or lower limb amputation above the ankle

Lewis EJ, et al. N Engl J Med. 2001;345(12):851-860.

IDNT Irbesartan vs Placebo
Primary and Secondary Endpoints

	Irbesartan Group n=579		Placebo Group n=569		P value	Unadjusted relative risk (95% CI)
	n	%	n	%		
Primary composite endpoint*	189	32.6	222	39.0	0.020	0.80 (0.66-0.97)
• Doubling of serum creatinine	98	16.9	135	23.7	0.003	0.67 (0.52-0.87)
• End stage renal disease	82	14.2	101	17.8	0.070	0.77 (0.57-1.03)
• Death	87	15.0	93	16.3	0.570	0.92 (0.69-1.23)
Secondary composite endpoint‡	138	23.8	144	25.3	0.400	0.91 (0.72-1.14)

*Composite of a doubling of serum creatinine, end stage renal disease, or death

‡Composite of death from cardiovascular causes, nonfatal myocardial infarction, heart failure resulting in hospitalization, a permanent neurologic deficit caused by a cerebrovascular event, or lower limb amputation above the ankle

Lewis EJ, et al. N Engl J Med. 2001;345(12):851-860.

IDNT Summary of Important Findings

Lewis EJ, et al. N Engl J Med. 2001;345(12):851-860.

In hypertensive, type 2 diabetics with nephropathy:

Irbesartan reduced the incidence of the primary composite endpoint of a doubling of serum creatinine, end stage renal disease, or death by 23% vs amlodipine (P=0.006) and 20% vs placebo (P=0.02)

Proteinuria was reduced 33% in the irbesartan group compared to 10% with placebo

These benefits were above and beyond those attributable to blood pressure reduction alone

Intensive Versus Conventional Therapy to Slow the Progression of Idiopathic Glomerular Diseases

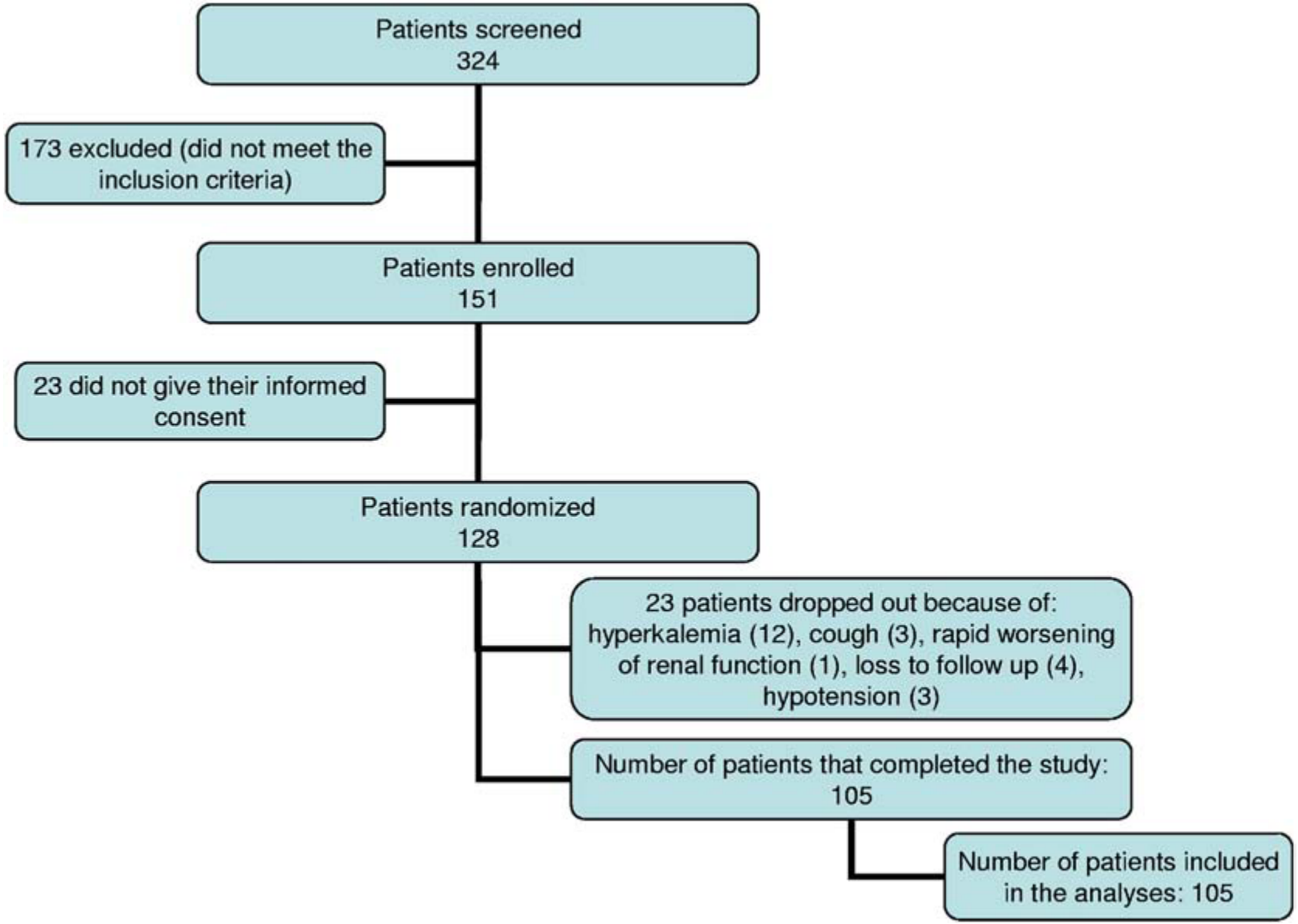
Stefano Bianchi, MD, Roberto Bigazzi, MD, and Vito M. Campese, MD

Am J Kidney Dis 55:671-681, abril 2010

Unità Operativa Nefrologia Spedali Riuniti di Livorno, Livorno, Italy; and Division of Nephrology, Keck, School of Medicine, USC, Los Angeles, CA.

	Intensivo	Convencional
Ramipril	10	10
Irbesartán	300	
Atorvastatina	20	10
Espironolactona	25	
PA	Menor posible	130/80
LDL	≤ 100	130

- *Am J Kidney Dis 55:671-681, abril 2010*

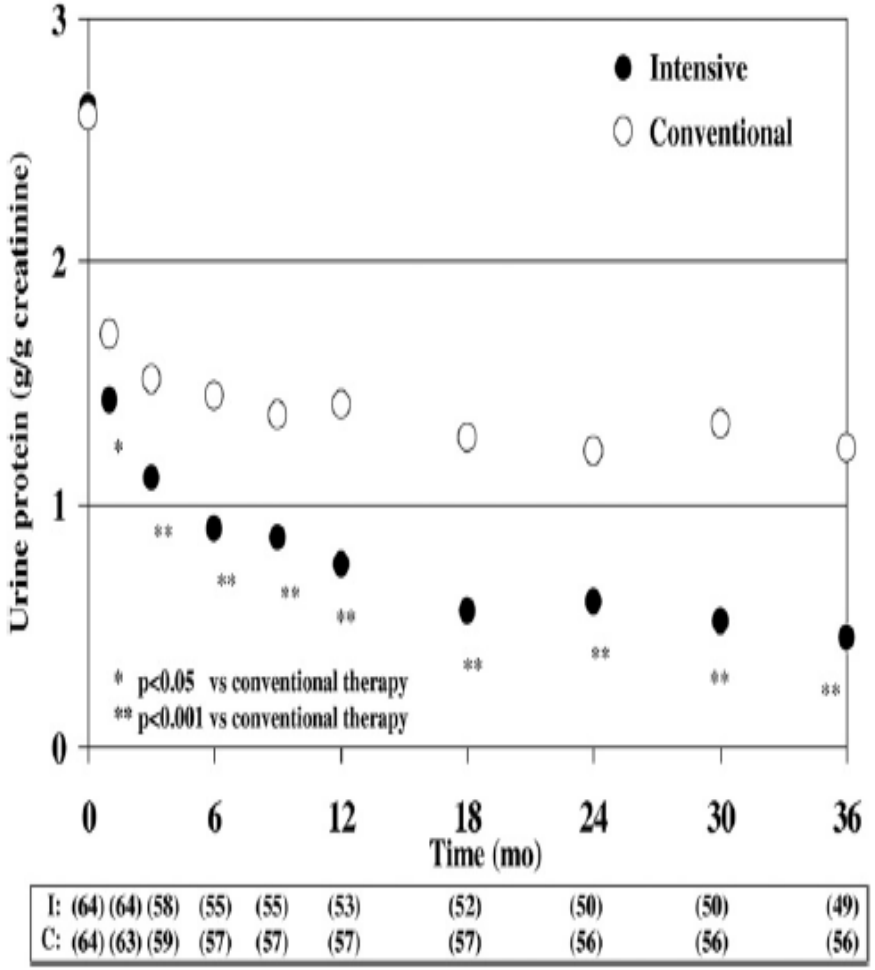


Am J Kidney Dis 55:671-681, abril 2010

Table 1. Baseline Clinical Characteristics

	All Patients	Conventional Therapy	Intensive Therapy
No. of patients (M/F)	128 (82/46)	64 (39/25)	64 (43/21)
Age (y)	53.2 ± 0.9	53.1 ± 1.1	53.1 ± 1.1
Body mass index (kg/m^2)	25.1 ± 0.3	25.3 ± 0.3	24.9 ± 0.3
Smokers (no/yes)	91/37	44/20	47/17
Kidney disease			
Chronic kidney disease	80	41	39
Focal segmental glomerulosclerosis	20	12	8
Immunoglobulin A	28	11	17
Systolic blood pressure (mm Hg)	156.1 ± 1.3	155.7 ± 1.6	156.6 ± 1.9
Diastolic blood pressure (mm Hg)	93.7 ± 0.7	93.3 ± 1.0	94.1 ± 1.0
Low-density lipoprotein cholesterol (mg/dL)	160.9 ± 4.7	161.6 ± 1.7	160.4 ± 2.2
High-density lipoprotein cholesterol (mg/dL)	45.5 ± 0.7	45.5 ± 1	45.6 ± 1.1
Mean blood pressure (mm Hg)	114.5 ± 0.8	114.0 ± 1.1	114.9 ± 1.1
Serum triglycerides (mg/dL)	169.6 ± 4.7	174.9 ± 6.4	164.3 ± 6.8
Serum sodium (mEq/L)	139.7 ± 0.2	139.4 ± 0.3	139.9 ± 0.3

Am J Kidney Dis 55:671-681, abril 2010



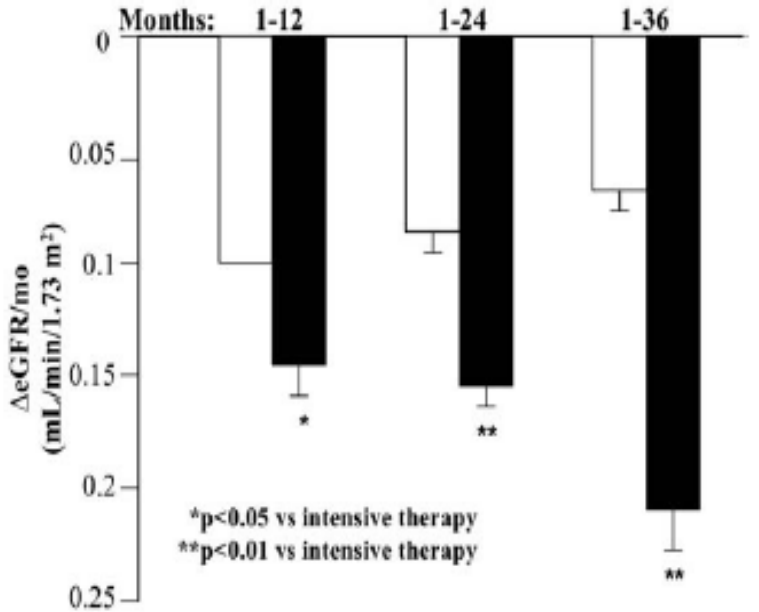


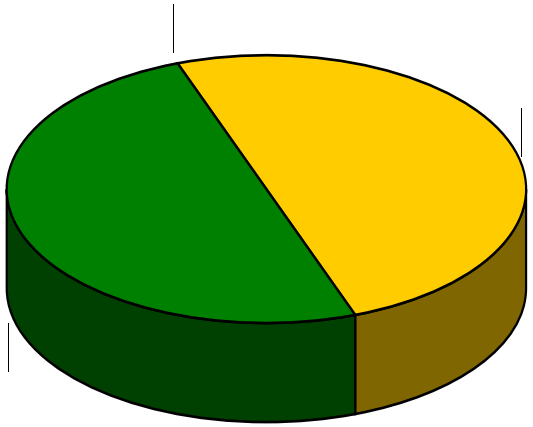
Figure 4. Rate of estimated glomerular filtration rate decrease (ΔeGFR; mL/min/1.73 m^2/mo) in patients treated with conventional (closed bars) or intensive therapy (open bars) during the first, second, or third year of treatment. Only the 49 patients on intensive therapy and the 56 on conventional therapy who completed the study are included in this analysis.

Am J Kidney Dis 55:671-681, abril 2010

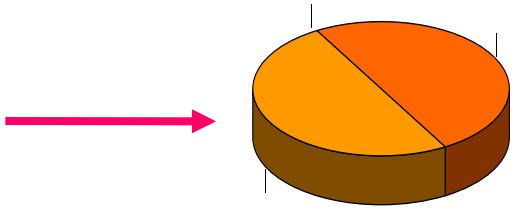
Terapia combinada o dual

EL MUNDO REAL

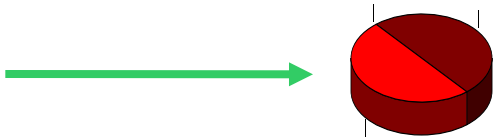
“ La Regla de las 3 Mitades ”



La mitad han
sido diagnosticados



La mitad de los
diagnosticados
está en tratamiento

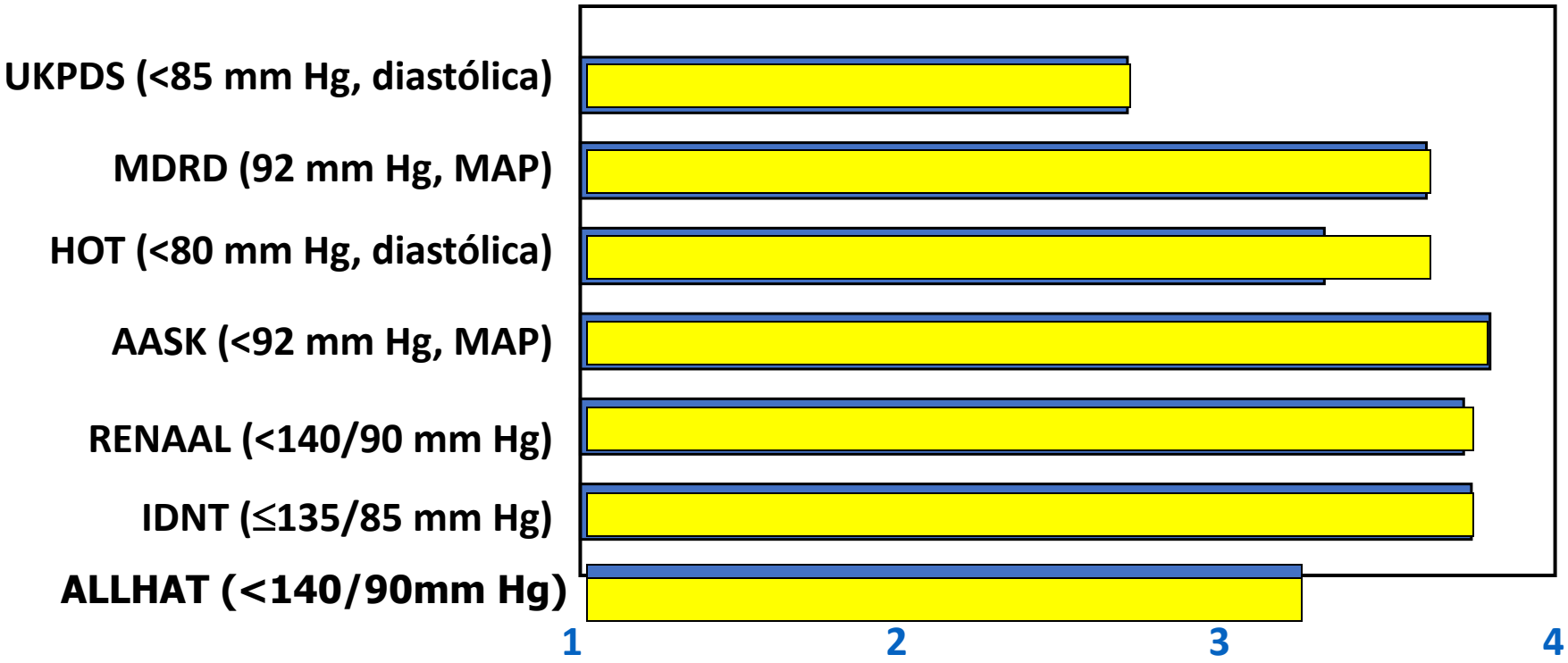


Sólo la mitad de los
tratados está en
control adecuado

Control : Sólo 12.5 %

WHO-ISH, 1999.

Número de agentes requeridos para alcanzar las metas de PA



NÚMERO DE MEDICAMENTOS

Bakris et al. *Am J Kidney Dis*. 2000;36:646-661; Brenner et al. *N Engl J Med*. 2001;345:861-869; Lewis et al. *N Engl J Med*. 2001;345:851-860.

Estudios clínicos utilizando Terapia combinada con irbesartan

Clinical Therapeutics/Volume 34, Number 8, 2012

I-COMBINE Study

Assessment of Efficacy and Safety Profile of Irbesartan/Amlodipine Fixed-Dose Combination Therapy Compared With Amlodipine Monotherapy in Hypertensive Patients Uncontrolled With Amlodipine 5 mg Monotherapy: A Multicenter, Phase III, Prospective, Randomized, Open-Label With Blinded–End Point Evaluation Study

Guillaume Bobrie, MD, for the I-COMBINE Study Investigators*

Department of Hypertension, Hôpital Européen Georges Pompidou, Paris, France

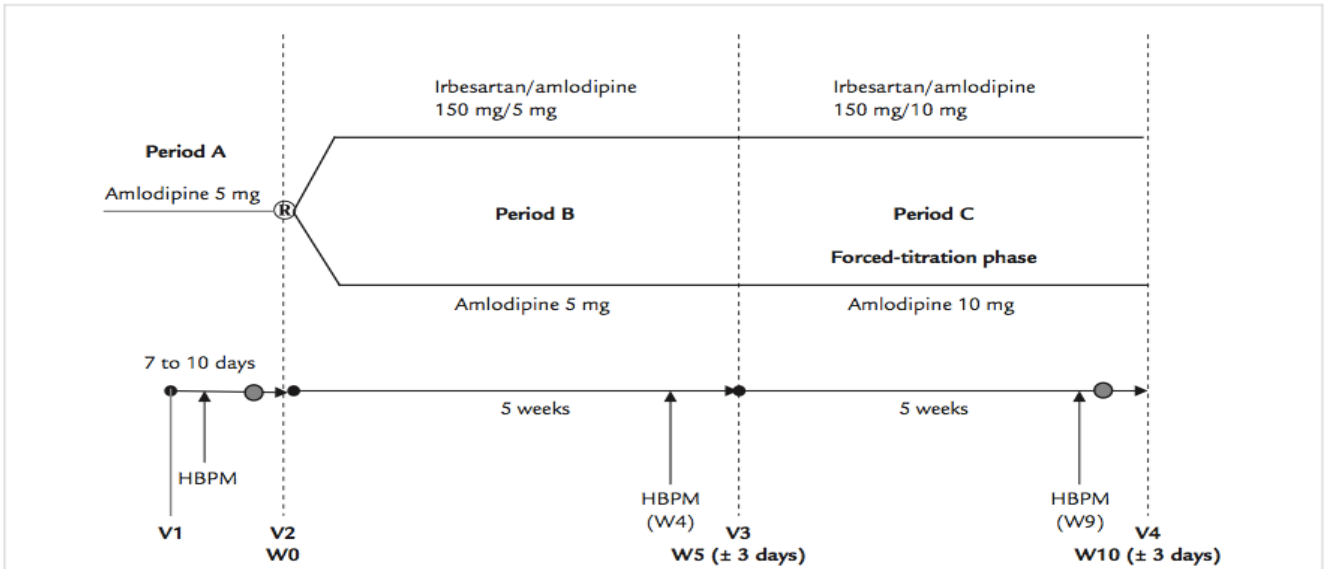
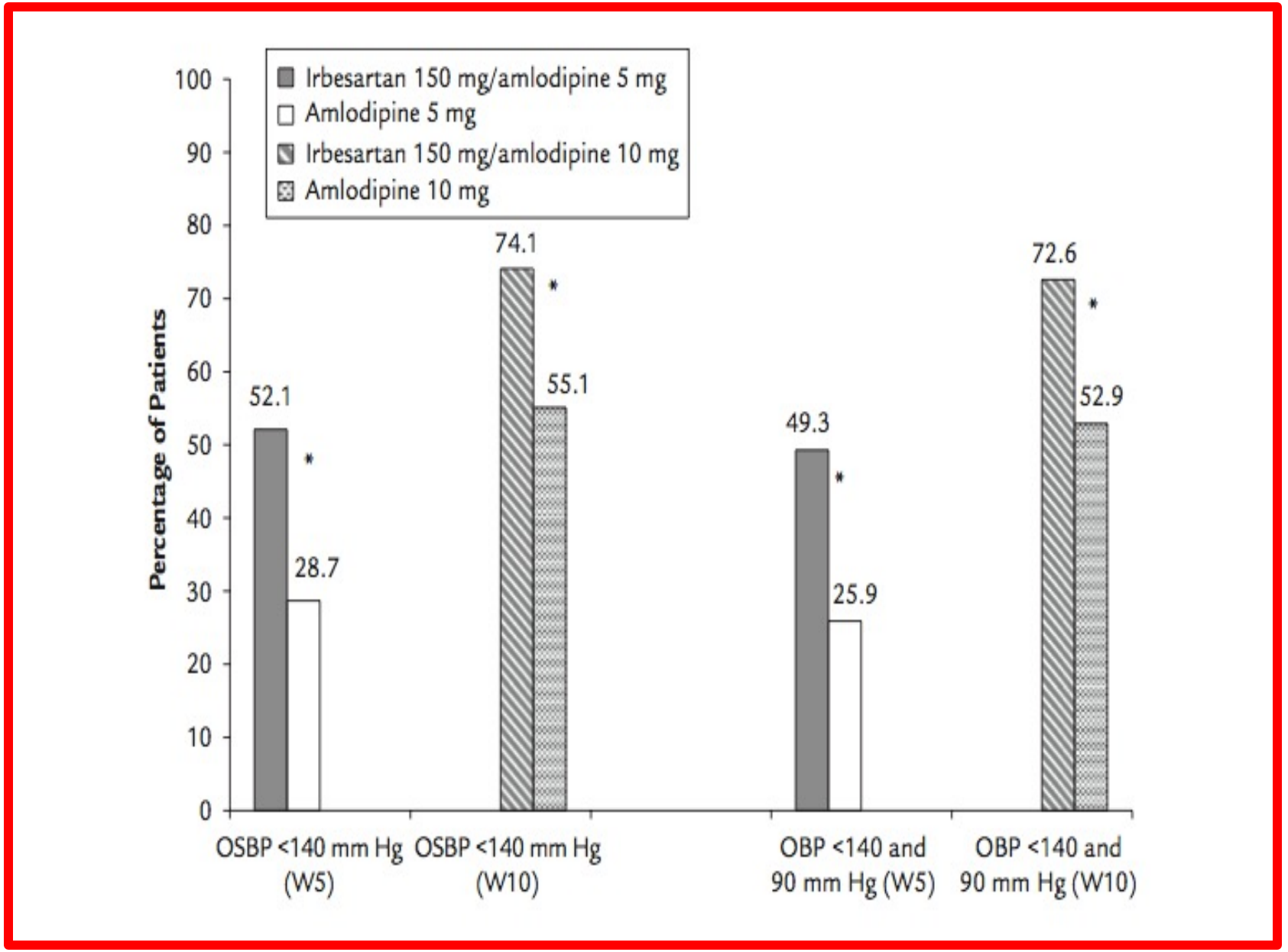


Figure 1. Study design. HBPM = home blood pressure measurement. V = visit; W = week.


Irbesartan 150 mg/amlodipine 5 mg
Amlodipine 5 mg
Irbesartan 150 mg/amlodipine 10 mg
Amlodipine 10 mg
Percentage of Patients
100
90
80
70
60
50
40
30
20
10
0
52.1
28.7
74.1
55.1
49.3
25.9
72.6
52.9
*
OSBP <140 mm Hg (W5)
OSBP <140 mm Hg (W10)
OBP <140 and 90 mm Hg (W5)
OBP <140 and 90 mm Hg (W10)

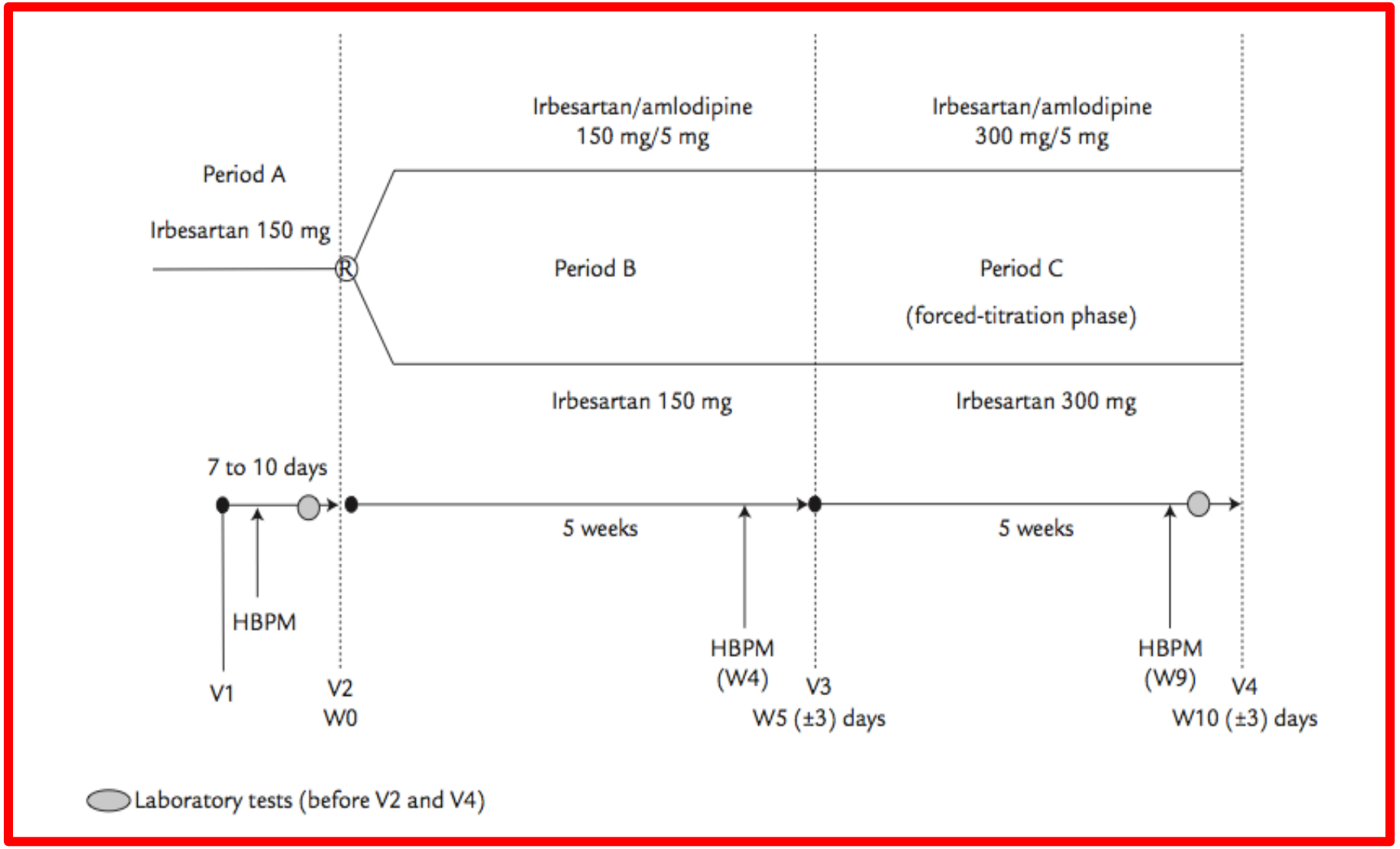
Clinical Therapeutics/Volume 34, Number 8, 2012

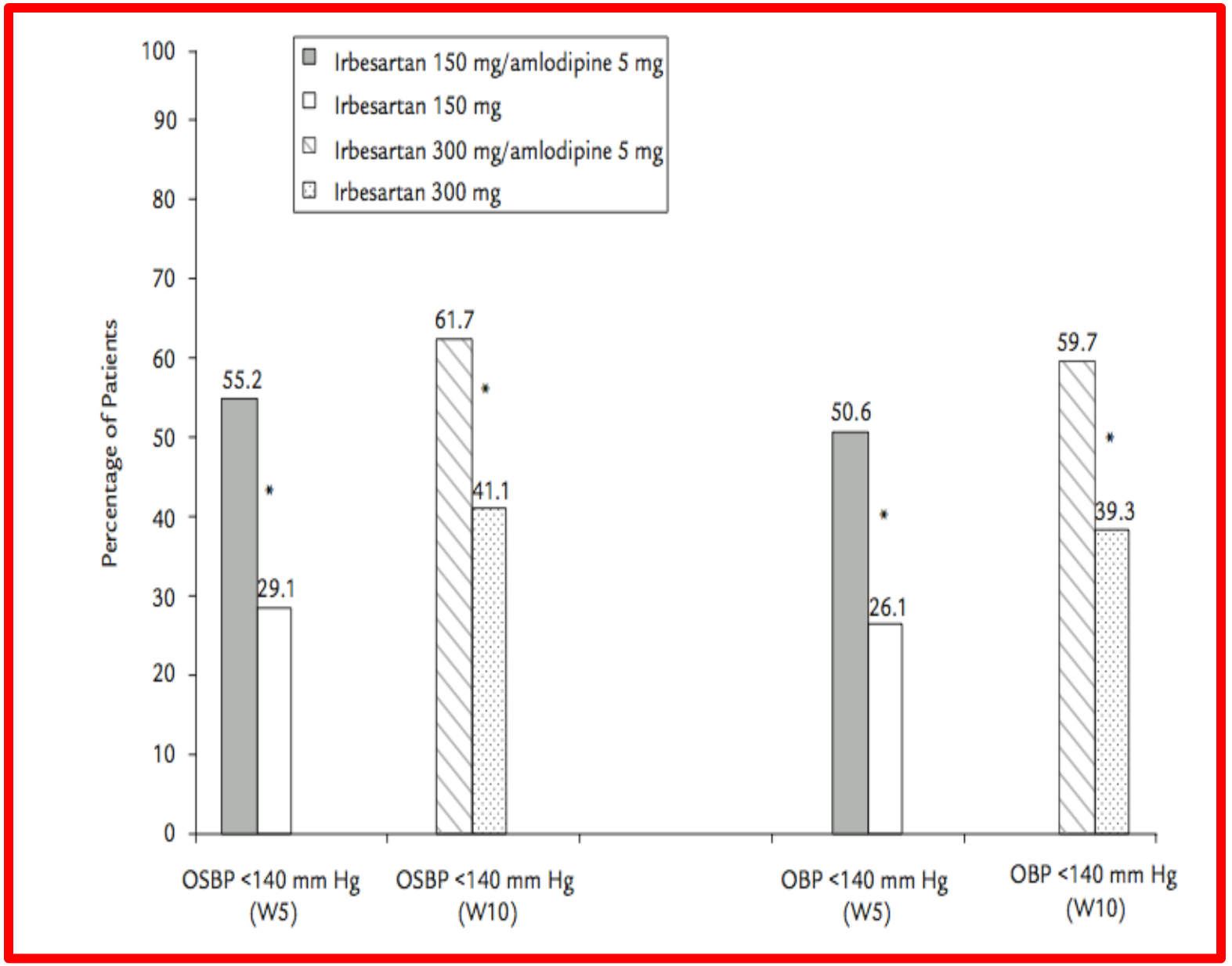
I-ADD Study: Assessment of Efficacy and Safety Profile of Irbesartan/Amlodipine Fixed-Dose Combination Therapy Compared With Irbesartan Monotherapy in Hypertensive Patients Uncontrolled With Irbesartan 150 mg Monotherapy: A Multicenter, Phase III, Prospective, Randomized, Open-Label With Blinded–End Point Evaluation Study

Guillaume Bobrie, MD, for the I-ADD Study Investigators*

Department of Hypertension Hôpital Européen Georges Pompidou, Paris, France

ardo Maffini, Universidade de Caxias do Sul; Mota Gomes Marco Antonio, Clinicor; Tarcísio Freire Antonio, Santa Casa de Belo Horizonte; Colombia: Elias Maria, Cardiolab; Manzur Fernando, Centro de Diagnostico Cardiologico; Miguel Urina, Fundacion BIOS; Guatemala: Alonzo Omar, Hospital General San Juan de


Irbesartan/amlodipine
150 mg/5 mg
Irbesartan/amlodipine
300 mg/5 mg
Period A
Irbesartan 150 mg
R
Period B
Period C
(forced-titration phase)
Irbesartan 150 mg
Irbesartan 300 mg
7 to 10 days
5 weeks
5 weeks
HBPM
HBPM
(W4)
HBPM
(W9)
V1
V2
W0
V3
W5 (±3) days
V4
W10 (±3) days
Laboratory tests (before V2 and V4)


Irbesartan 150 mg/amlodipine 5 mg
Irbesartan 150 mg
Irbesartan 300 mg/amlodipine 5 mg
Irbesartan 300 mg
Percentage of Patients
100
90
80
70
60
50
40
30
20
10
0
55.2
29.1
61.7
41.1
50.6
26.1
59.7
39.3
*
OSBP <140 mm Hg (W5)
OSBP <140 mm Hg (W10)
OBP <140 mm Hg (W5)
OBP <140 mm Hg (W10)

2023 ESH Guidelines for the management of arterial hypertension The Task Force for the Management of arterial hypertension of the European Society of Hypertension Endorsed by the European Renal Association (ERA) and the International Society of Hypertension (ISH).

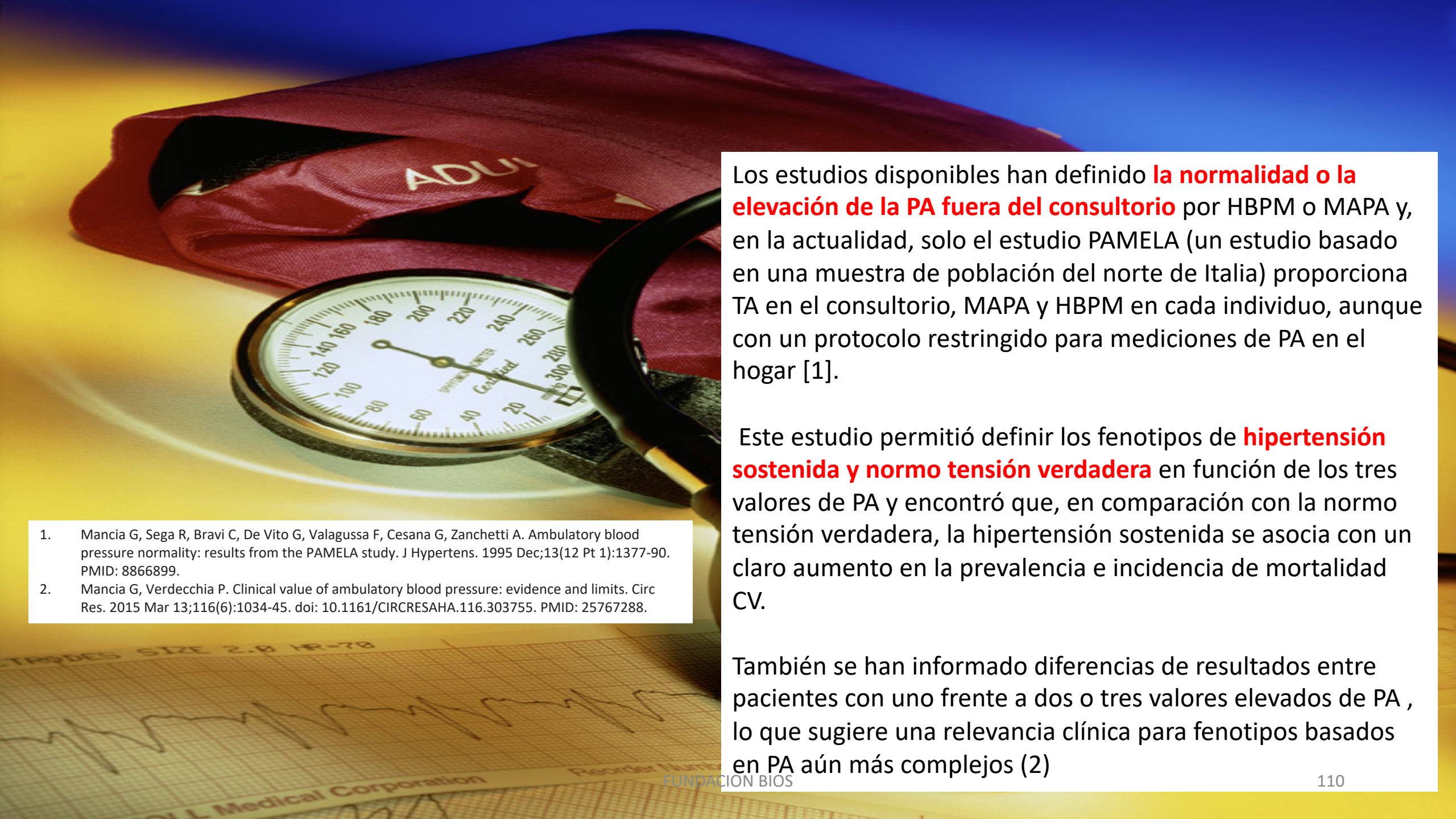
Journal of Hypertension ():10.1097/HJH.0000000000003480, June 21, 2023. | DOI: 10.1097/HJH.0000000000003480

El uso de mediciones de PA fuera del consultorio por HBPM y/o MAPA permite identificar fenotipos de PA que eran desconocidos cuando las mediciones de PA se limitaban a la PA en consultorio.

- Un fenotipo se denomina **hipertensión sostenida** y consiste en una elevación de la PA en el consultorio y fuera del consultorio.

- Otra es la **normo tensión verdadera**, que se caracteriza por la normalidad de la PA en el consultorio y fuera del consultorio.

- Estos fenotipos pueden referirse no solo a los individuos no tratados sino también a los tratados, donde indican un control prolongado de la PA en el consultorio y fuera del consultorio o ningún control de todos estos valores de PA.



Los estudios disponibles han definido **la normalidad o la elevación de la PA fuera del consultorio** por HBPM o MAPA y, en la actualidad, solo el estudio PAMELA (un estudio basado en una muestra de población del norte de Italia) proporciona TA en el consultorio, MAPA y HBPM en cada individuo, aunque con un protocolo restringido para mediciones de PA en el hogar [1].

Este estudio permitió definir los fenotipos de **hipertensión sostenida y normo tensión verdadera** en función de los tres valores de PA y encontró que, en comparación con la normo tensión verdadera, la hipertensión sostenida se asocia con un claro aumento en la prevalencia e incidencia de mortalidad CV.

También se han informado diferencias de resultados entre pacientes con uno frente a dos o tres valores elevados de PA , lo que sugiere una relevancia clínica para fenotipos basados en PA aún más complejos (2)

1. Mancia G, Sega R, Bravi C, De Vito G, Valagussa F, Cesana G, Zanchetti A. Ambulatory blood pressure normality: results from the PAMELA study. J Hypertens. 1995 Dec;13(12 Pt 1):1377-90. PMID: 8866899.
2. Mancia G, Verdecchia P. Clinical value of ambulatory blood pressure: evidence and limits. Circ Res. 2015 Mar 13;116(6):1034-45. doi: 10.1161/CIRCRESAHA.116.303755. PMID: 25767288.

2023 ESH Guidelines for the management of arterial hypertension The Task Force for the management of arterial hypertension of the European Society of Hypertension Endorsed by the European Renal Association (ERA) and the International Society of Hypertension (ISH)

doi: 10.1097/HJH.0000000000003480

Class of Recommendation		Level of Evidence		
	Definition		Definition	Interpretation
I	Evidence or general agreement that a treatment/test/procedure is beneficial, useful or effective AND that potential benefits clearly outweigh potential risk	A	- RCT or meta-analysis of RCTs with CVD outcomes - Single trial enough if sufficient power and without important limitations[a]	Strong evidence. Evidence of high certainty. Unlikely that future studies will change the effect estimate substantially
II	Conflicting evidence oropinion about the benefit, usefulness and effectiveness of a treatment/ test/procedure OR uncertainty about benefit-risk balance	B	- RCT with surrogate measures (BP, HMOD) - Observational studies with CVD outcomes and no major limitations[a] - Meta-analyses including the above study types	Moderate evidence. Evidence with some Future studies may modify, at least the magnitude of, the effect estimate
III	Evidence or general agreement that a treatment/test/procedure is not beneficial, useful or effective OR that potential risks outweigh the potential benefit	C	- Observational studies of surrogate measures - Any study type may be downgraded to level C due to limitations[a] - Expert opinion (EO)	Weak evidence. Evidence of low certainty. Future studies may change the effect estimate substantially

Class of recommendation (CoR) and level of evidence (LoE). BP, blood pressure, CVD, cardiovascular disease, HMOD, hypertension mediated organ damage, RCT, randomized controlled trial. aLimitations affecting level of evidence include (but may not be limited to) high risk of bias, inability to account for important confounding factors in observational studies, questionable external validity and uncertain effect estimates (confidence intervals including negligible effect).

RECOMENDACIONES DE LA GUIA

Lifestyle interventions

Recommendations and statements	CoR	LoE
In adults with elevated BP who are overweight or obese, weight reduction is recommended to reduce BP and improve CV outcomes.	I	A
Preferred dietary products include vegetables, fruits, beans, nuts, seeds, vegetable oils, and fish and poultry among meat products. Fatty meats, full-fat dairy, sugar, sweetened beverages, and sweets should be limited. Overall, a healthy dietary patterns including more plant-based and less animal-based food is recommended.	I	B
In adults with hypertension consuming a high sodium diet (most Europeans), salt substitutes replacing part of the NaCl with KCl is recommended to reduce BP and the risk for CVD.	I	A
Dietary salt (NaCl) restriction is recommended for adults with elevated BP to reduce BP. Salt (NaCl) restriction to < 5 g (~2g sodium) per day is recommended.	I	B
Increased potassium consumption, preferably via dietary modification, is recommended for adults with elevated BP, except for patients with advanced CKD.	I	B
Daily physical activity and structured exercise is recommended for adults with elevated BP to reduce BP and improve cardiovascular risk profile. It is recommended to strive for at least 150-300 minutes of aerobic exercise a week of moderate intensity, or 75-150 minutes a week of aerobic exercise of vigorous intensity or an equivalent combination. Sedentary time should also be reduced and supplemented with dynamic resistance exercise (2-3 times per week).	I	B

White-coat hypertension (WCH)

Recommendations and statements	CoR	LoE
Out-of-office BP measurement by ABPM and/or HBPM should be done when WCH is suspected, particularly in people with grade 1 hypertension.	I	B
In patients with WCH, assessment of CV risk factors and HMOD is recommended.	I	B
Out-of-office BP measurements should be done by ABPM and/or HBPM and repeated during follow up to timely identify sustained hypertension or new HMOD.	I	B
In patients with WCH, lifestyle interventions to reduce CV risk and close follow are recommended.	I	B
Whether BP lowering drug treatment should be used is still unresolved, but it can be considered in patients with HMOD and high CV risk.	IIb	C

Masked hypertension (MH)

Recommendations and statements	CoR	LoE
Out-of-office BP measurement by ABPM and/or HBPM should be done in people with high normal blood pressure to identify MH.	I	B
In patients with MH, lifestyle interventions and close follow up are recommended to reduce CV risk and to timely identify sustained hypertension and new HMOD.	I	C
Whether BP lowering drug treatment should be used in MH is still unresolved, but it can be considered in patients with HMOD and high CV risk.	IIb	C

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White-coat uncontrolled hypertension (WUCH) and masked uncontrolled hypertension (MUCH)

Recommendations and statements	CoR	LoE
The recommendations for WCH and MH apply to WUCH and MUCH, respectively, except that WUCH and MUCH refer to treated people.	I	C
Considering the limitations of available evidence on WUCH and MUCH, uptitration of drug treatment can be done in both conditions to ideally control both BP phenotypes if well tolerated.	II	C

Isolated systolic hypertension in the young (ISHY)

Recommendations and statements	CoR	LoE
Due to the frequent presence of a pronounced white-coat effect, out-of-office BP measurement is recommended.	I	C
Central BP measurement can be considered to identify ISHY individuals at low CV risk to detect spurious hypertension, if available.	II	C
Close follow-up and lifestyle interventions are recommended.	I	C
In individuals with high out-of-office BP or high central BP, particularly with other CV risk factors or HMOD, BP lowering drug treatment can be considered.	II	C

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Isolated diastolic hypertension (IDH)

Recommendations and statements	CoR	LoE
Periodic BP evaluation and lifestyle interventions are recommended for all patients with IDH.	I	C
Despite the absence of dedicated RCTs in IDH, it is recommended that the BP lowering drug treatment should follow the general treatment strategy.	II	C

Night-time hypertension and BP phenotypes

Recommendations and statements	CoR	LoE
It is recommended to assess night-time BP using ABPM because it is more predictive for outcomes than daytime BP, and because nocturnal hypertension, non-dipping and reverse dipping are associated with increased CV risk	I	B
For the identification of night-time BP phenotypes, repeating ABPM is necessary, because of poor reproducibility.	I	B
Elevated night-time BP may be reduced by antihypertensive treatment.	II	C
In the general hypertensive population morning dosing or bedtime dosing results in similar outcome.	I	B

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Mancia(Chairperson), Giuseppe; Kreutz(Co-Chair), Reinhold; Brunström, Mattias; Burnier, Michel;

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Baroreflex failure and autonomic failure

Recommendations	Class	Level
The diagnosis of (afferent) baroreflex failure should be considered in patients with highly volatile hypertension that is exacerbated by psychological and physiological stress, particularly in those with predisposing conditions (e.g., following neck dissection or radiation therapy).	I	C
The diagnosis of (afferent) baroreflex failure should be confirmed by baroreflex testing preferably in specialized centers	I	C
Long-acting sympatholytic drugs can be prescribed to attenuate hypertensive episodes in patients with (afferent) baroreflex failure.	II	C
In patients with (efferent) autonomic failure, the underlying causes should be sought for to identify potentially treatable conditions and to gauge prognosis.	I	C
For patients with (efferent) autonomic failure and symptomatic orthostatic hypotension, non-pharmacological treatments such as increased sodium ingestion, sufficient water ingestion, and venous compression garments should be instituted first. Medications worsening orthostatic hypotension (e.g., diuretics, alpha-1 blockers, vasodilators) should be discontinued whenever possible.	I	C
Anti-hypotensive medications (e.g., alpha-adrenoreceptor agonists) may be considered for patients with (efferent) autonomic failure who remain symptomatic on non-pharmacological treatments, however, the treatment can worsen hypertension in the supine position.	II	C
In patients with (efferent) autonomic failure and hypertension in the supine position, sleeping with the head of the bed tilted up can improve BP. Pharmacological therapy of supine hypertension can be considered in selected patient after individual risk-benefit consideration weighing potential benefits on cardiovascular risk against risk of fall and overall prognosis of the underlying disease.	II	C

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PA EN CONSULTORIO RESUMEN DE LA GUIA

En pacientes de 18 a 79 años, el umbral de consultorio recomendado para inicio de tratamiento farmacológico es 140 mmHg para PAS y/o 90 mmHg para PAD.	I	A
En pacientes ≥80 años, el umbral de PAS recomendado en el consultorio para inicio de tratamiento farmacológico es de 160 mmHg.	I	B

Los umbrales de PAS y PAD de la oficina para el inicio del tratamiento farmacológico en pacientes frágiles debe ser individualizado.	I	C
En pacientes adultos con antecedentes de CVD, predominantemente CAD, medicamentos el tratamiento debe iniciarse en el rango de PA normal-alta (PAS ≥130 o PAD ≥80 mmHg).	I	A

OBJETIVO

Pacientes de 18 a 64 años		
El objetivo es reducir la PA en el consultorio a <130/80 mmHg	I	A
Pacientes de 65 a 79 años		
El objetivo principal del tratamiento es reducir la PA a <140/80 mmHg	I	A
Sin embargo, se puede considerar bajar la PA por debajo de 130/80 mmHg si el tratamiento es bien tolerado.	I	B
Pacientes de 65 a 79 años con ISH		
El objetivo principal del tratamiento es reducir la PAS en el rango de 140 a 150 mmHg.	I	A
Sin embargo, se puede considerar una reducción de la PAS en el consultorio en el rango de 130 a 139 mmHg si se tolera bien, aunque con precaución si la PAD ya está por debajo de 70 mmHg.	Yo	B
Pacientes ≥80 años		
La PA en el consultorio debe reducirse a una PAS en el rango de 140 a 150 mmHg ya una PAD <80 mmHg.	I	A

RECOMENDACIONES DE INTERVENCION DE LA GUIA

Recomendaciones y declaraciones	CDR	Noroeste
La reducción de la PA debe priorizarse sobre medicamentos antihipertensivos porque el be medida en la reducción de la PA.		
Cinco clases principales de fármacos, inclui los diuréticos tiazídicos/similares a las tiazid de presión arterial y CV en los ECA. Estos f recomiendan como base de las estrategias		
Se recomienda iniciar el tratamiento con una c de los pacientes hipertensos. Las combinacion RAS (ya sea un inhibidor de la ECA o un ARB) la tiazida. Se pueden usar otras combinaciones		
Se puede considerar el inicio con monoterapia hipertensión grado 1 y de bajo riesgo elevada (PAS inferior a 150 mmHg y riesgo CV muy alto, fragilidad y/o ed		
Si la PA no se controla con la combinación inicial de dos fármacos mediante el uso de la dosis máxima recomendada y tolerada de los respectivos componentes, el tratamiento debe aumentarse a una combinación de tres fármacos, generalmente un bloqueador del SRA + BCC + tiazida/diurético similar a la tiazida.	I	A
Si la PA no se controla con una combinación de tres fármacos usando la dosis máxima recomendada y tolerada de los respectivos componentes, se recomienda extender el tratamiento de acuerdo con las recomendaciones para la hipertensión resistente.	I	A
Se debe preferir el uso de combinaciones de un solo comprimido (SPC) en cualquier paso del tratamiento, es decir, durante el inicio de la terapia con una combinación de dos fármacos y en cualquier otro paso del tratamiento.	I	B
Los BB deben usarse al inicio de la terapia o en cualquier paso del tratamiento como GDMT, ejemplos: Insuficiencia cardiaca con fracción de eyección reducida HFrEF Terapia antiisquémica en síndromes coronarios crónicos Control de la frecuencia cardíaca en la fibrilación auricular	I	A
Los BB pueden considerarse en presencia de varias otras condiciones en las que su uso puede ser favorable como se resume en la Tabla xx.	I	C

RECOMENDACIONES DE INTERVENCION DE LA GUIA

Recomendaciones y declaraciones	CoR LoE	
La decisión de iniciar un tratamiento para reducir el colesterol LDL, así como como objetivos del tratamiento, debe basarse en una estimación del CV total riesgo, dando prioridad a los pacientes de alto riesgo.	I	A
Se recomienda el tratamiento con estatinas en pacientes con hipertensión y elevado riesgo CV.	I	A
Se pueden considerar inhibidores de PCSK9 y siRNA dirigidos a PCSK9 en pacientes seleccionados de alto riesgo que no alcanzan el colesterol LDL objetivo niveles con la terapia de combinación de estatinas/ezetimiba.	Yo	A
Uso de una polipíldora que contiene dos fármacos antihipertensivos y una estatina para Se puede considerar la reducción del colesterol LDL en hipertensos pacientes para la prevención primaria.	Yo	A

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SPECIAL ARTICLE

Latin American Consensus on management of residual cardiometabolic risk. A consensus paper prepared by the Latin American Academy for the Study of Lipids and Cardiometabolic Risk (ALALIP) endorsed by the Inter-American Society of Cardiology (IASC), the International Atherosclerosis Society (IAS), and the Pan-American College of Endothelium (PACE)

Consenso latinoamericano para el manejo del riesgo residual cardiometabólico. Consenso realizado por la Academia Latinoamericana de Lipidología y Riesgo Cardiometabólico (ALALIP), con el aval de la Sociedad Interamericana de Cardiología (SIAC), la Sociedad Interamericana de Aterosclerosis (IAS) y el Colegio Panamericano de Endotelio (PACE)

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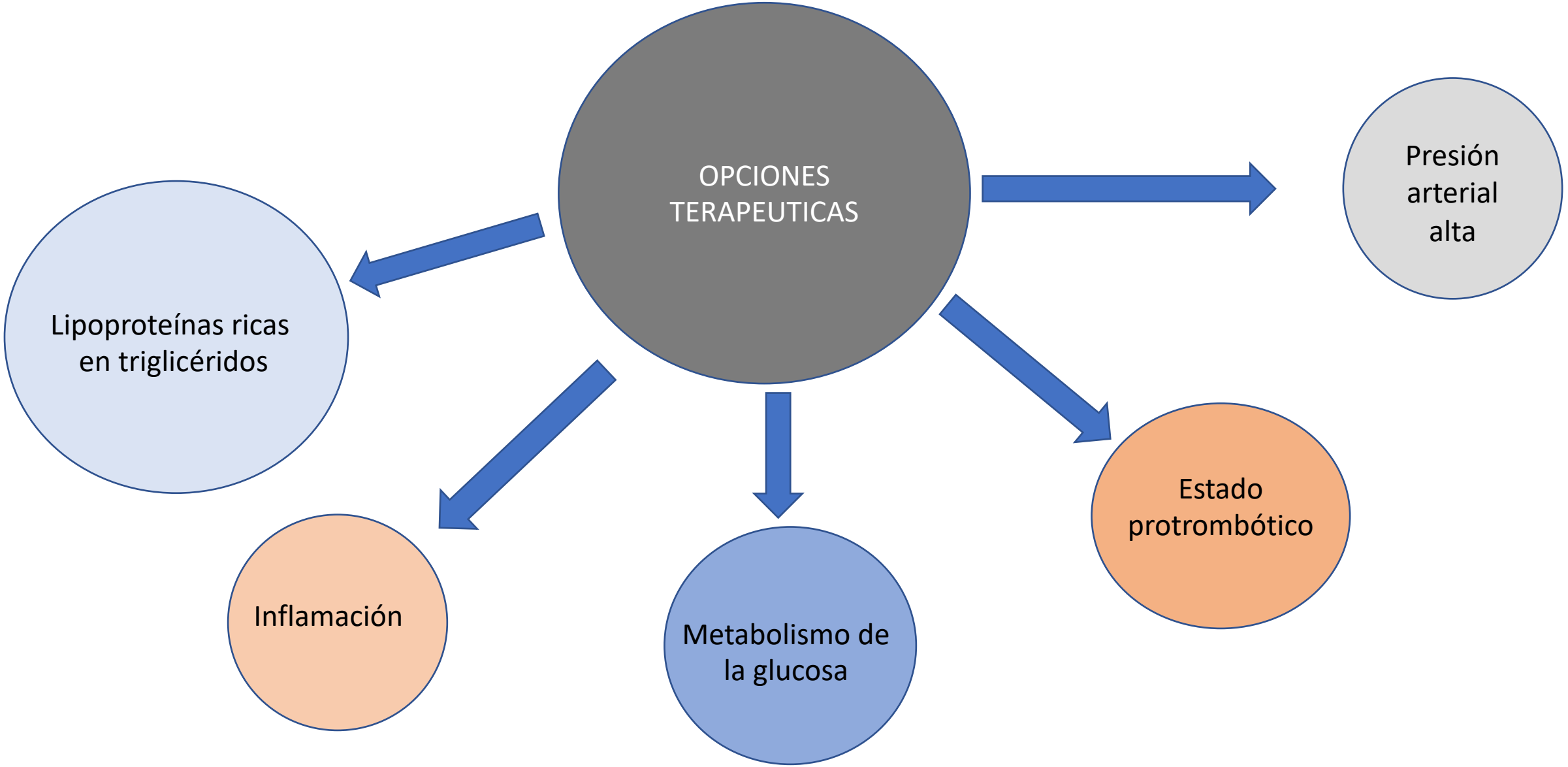
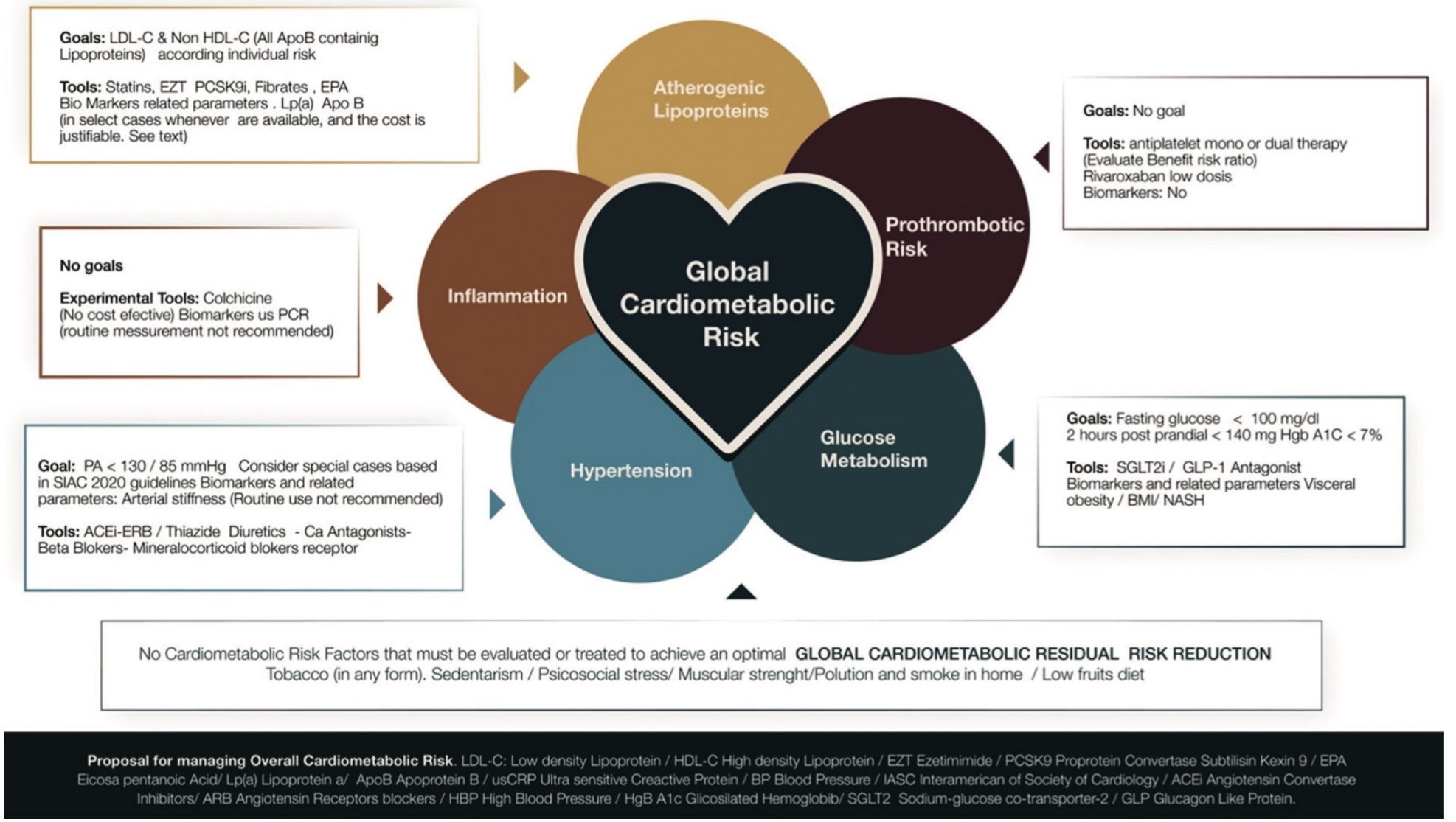


La reducción del RR debe considerar opciones terapéuticas adaptadas a las necesidades específicas del paciente, basadas en **5 objetivos de tratamiento.**

RIESGO CARDIOMETABOLICO RESIDUAL: ¿Cómo manejarlo?



Proposal for managing Overall Cardiometabolic Risk. LDL-C: Low density Lipoprotein / HDL-C High density Lipoprotein / EZT Ezetimimide / PCSK9 Proprotein Convertase Subtilisin Kexin 9 / EPA Eicosa pentanoic Acid/ Lp(a) Lipoprotein a/ ApoB Apoprotein B / usCRP Ultra sensitive Creactive Protein / BP Blood Pressure / IASC Interamerican of Society of Cardiology / ACEi Angiotensin Convertase Inhibitors/ ARB Angiotensin Receptors blockers / HBP High Blood Pressure / HgB A1c Glicosilated Hemoglobib/ SGLT2 Sodium-glucose co-transporter-2 / GLP Glucagon Like Protein.

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Gracias