Seminario Web en español



Dra. Adriana Cortés

¿Qué tienen en común la salud de tu corazón y la de tu cerebro?

Fecha: martes, 25 de febrero de 2025

Hora: 6:00 p.m. - 7:00 p.m. (hora este/ Miami)





¿Qué tienen en común la salud de tu corazón y la de tu cerebro?

Dra. **ADRIANA CORTÉS**

Whole to Heal
INTERNATIONAL

Dra.

ADRIANA CORTÉS



- ✓ Médica de la Pontificia Universidad Javeriana - 2002
- ✓ Maestría en Salud Pública Internacional de la Universidad de Sydney, Australia - 2005
- ✓ Traductora de www.nutritionstudies.org desde 2017
- ✓ Cofundadora de **Bien Plantado** - 2016
- ✓ Creadora del Sistema TEN - Transformando tu Entendimiento sobre Nutrición
- ✓ Directora y Fundadora de **Whole to Heal International**
- ✓ **Miembro activo de la Medical Advisory Board** del Centro de Estudios en Nutrición del Dr. T Colin Campbell
- ✓ Conferencista para el Certificado de Nutrición Basada en Plantas



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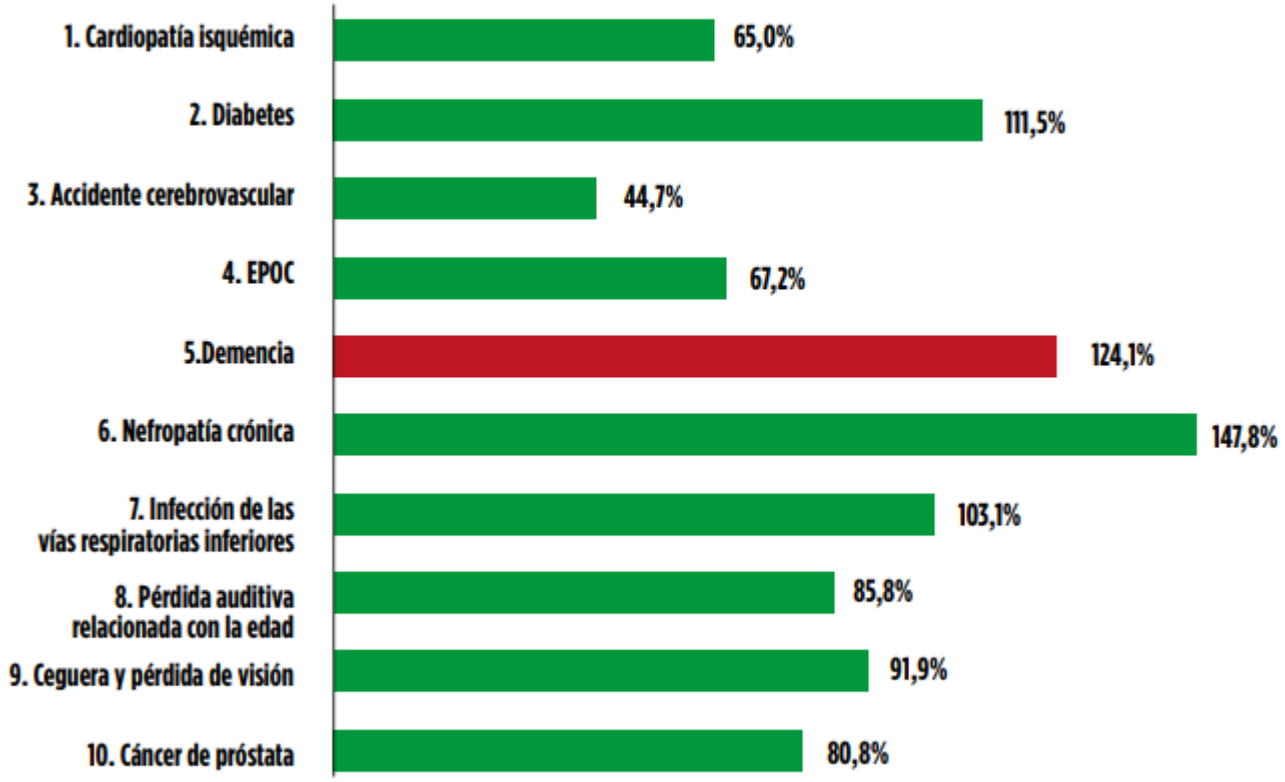
¿Qué aprenderemos hoy?

- El común denominador de tu salud cardiaca y la salud de tu cerebro
- Evidencia de reversión de enfermedad cardiovascular
- ¿Por qué se genera el deterioro cognitivo?
- Reversión de deterioro cognitivo

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Años de vida ajustados por discapacidad

Figura 6. Las diez afecciones responsables del mayor número de AVAD en las personas de 70 años o más en América Latina y el Caribe en el 2019, y aumento porcentual entre el 2000 y el 2019



Fuente: Instituto de Sanimetría y Evaluación Sanitaria. GBD Compare. Seattle: University of Washington; 2022. Disponible en: http://ihmeuw.org/5n3e.

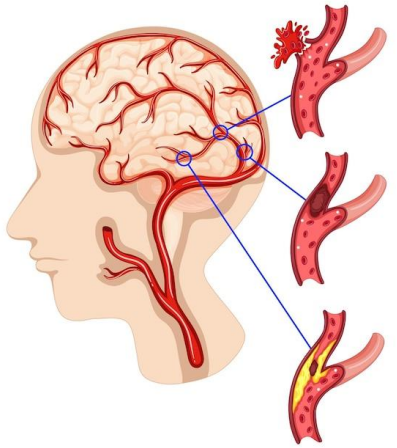
Organización Panamericana de la Salud. La demencia en América Latina y el Caribe: prevalencia, incidencia, repercusiones y tendencias a lo largo del tiempo. Washington, DC: OPS; 2023. Disponible en: https://doi.org/10.37774/9789275326657

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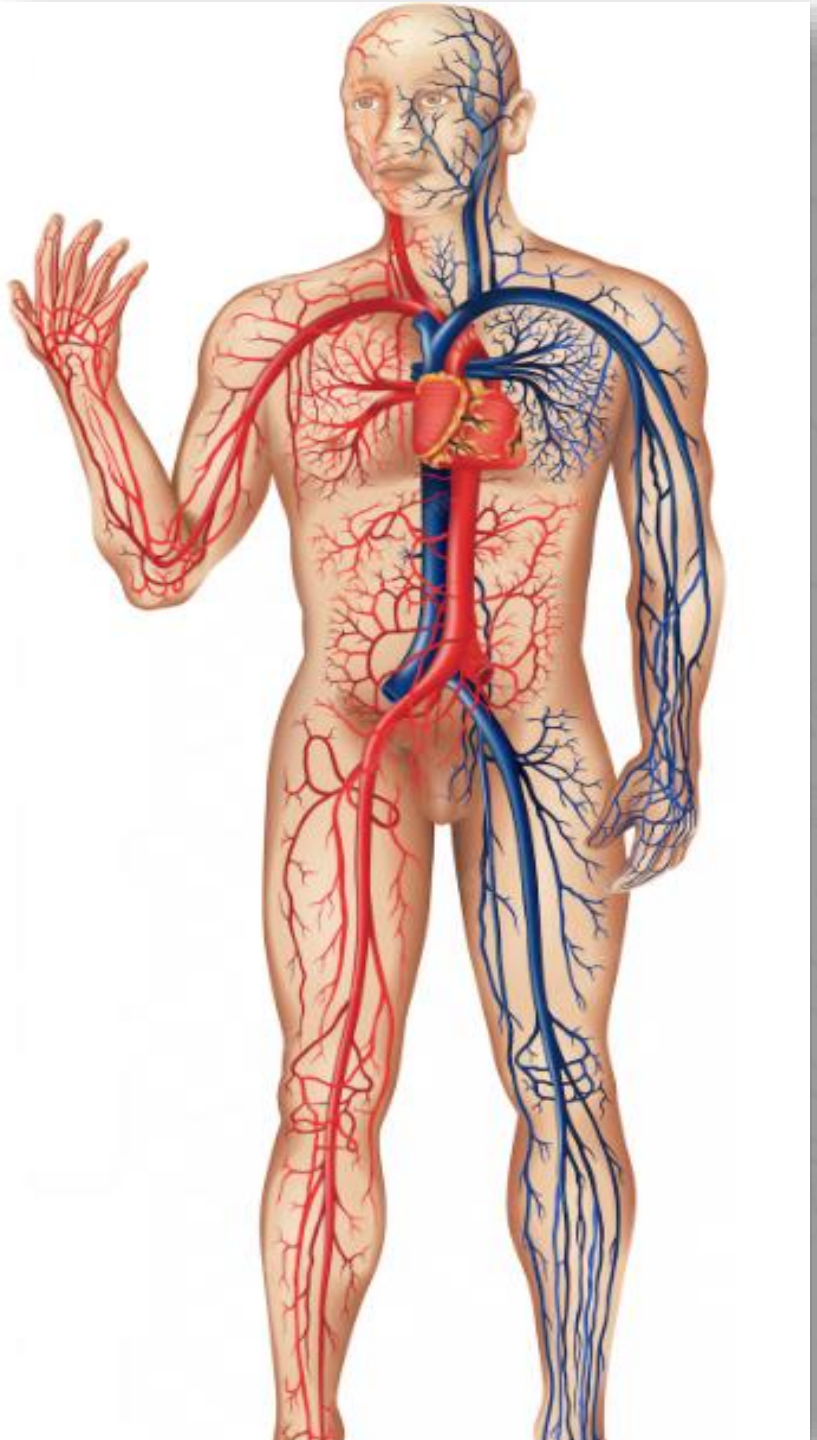
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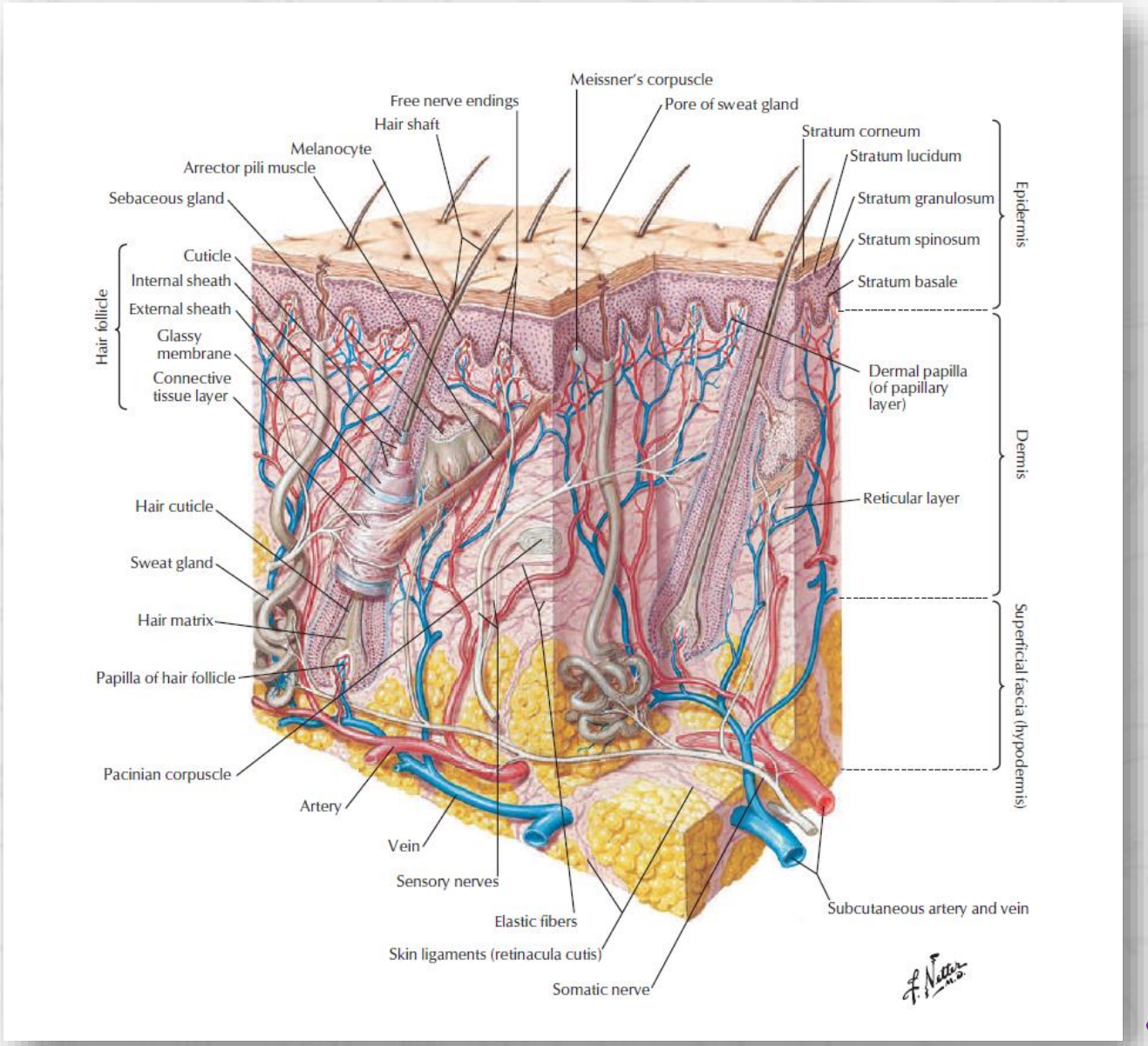
El común denominador

de tu salud cardíaca y cerebral





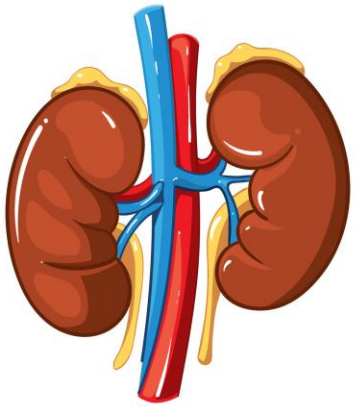




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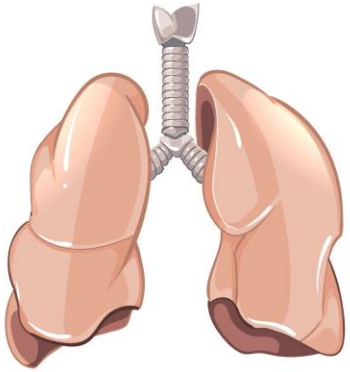
BRAIN



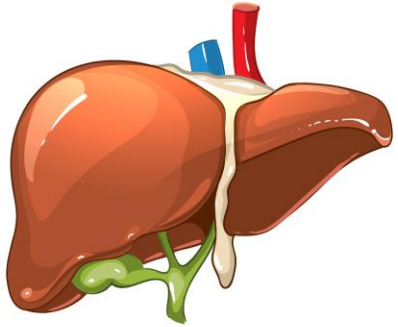
KIDNEY



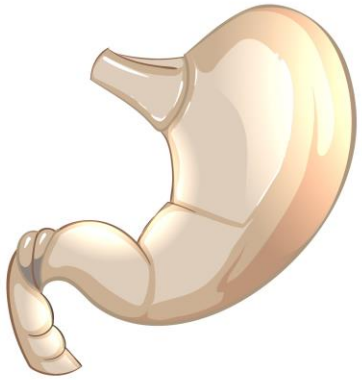
HEART



LUNGS



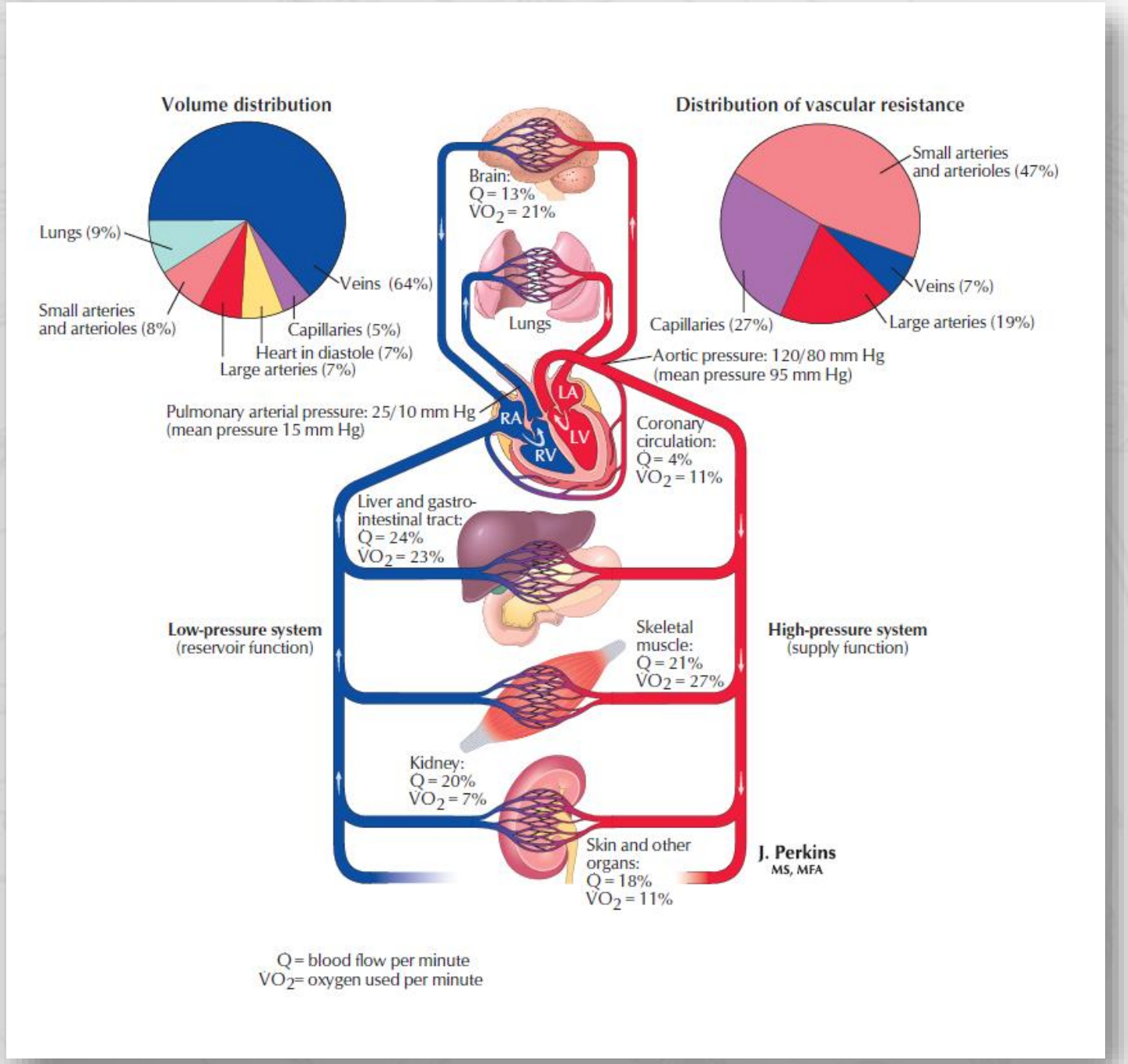
LIVER



STOMACH

Por TODO tu cuerpo

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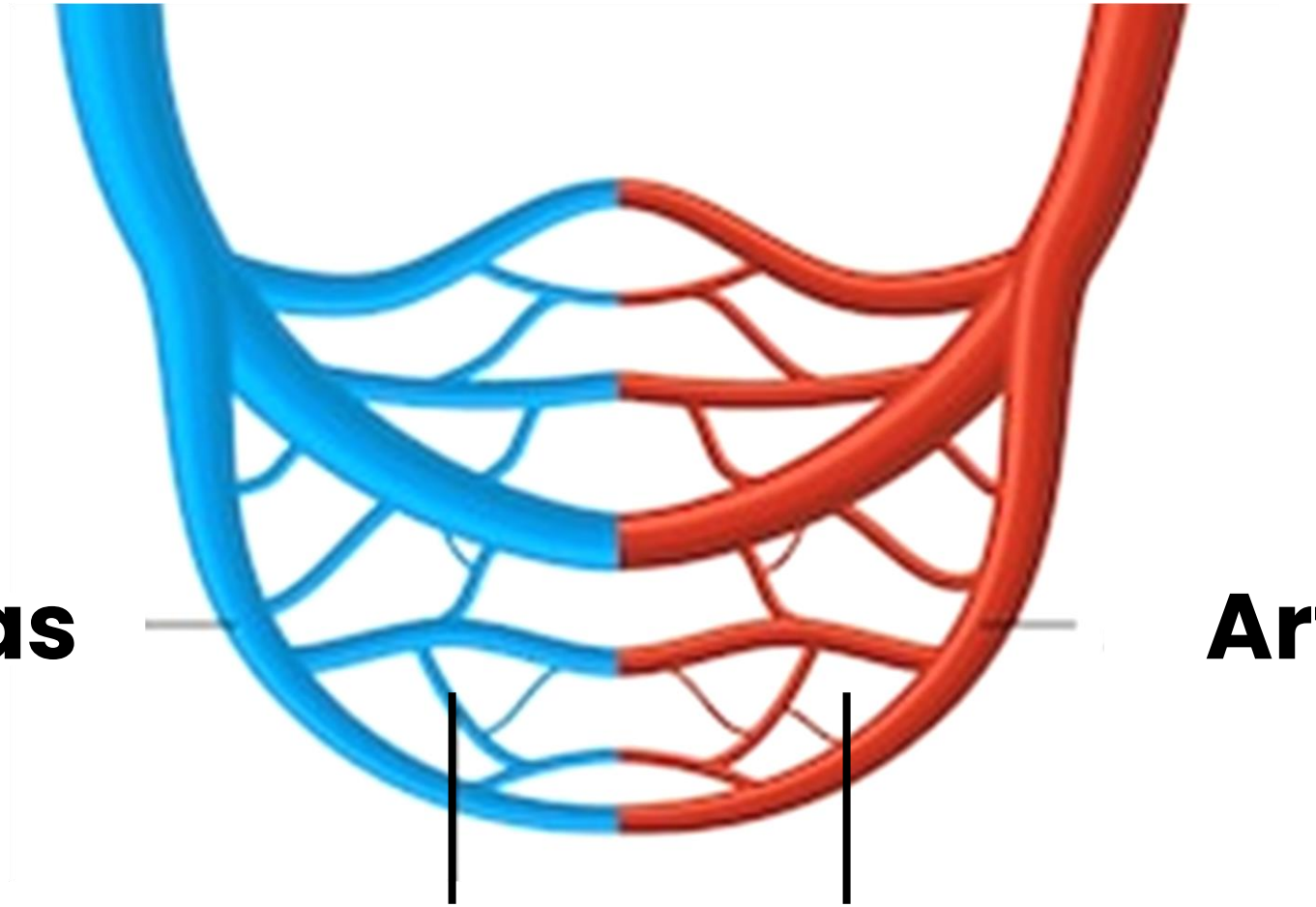


100,000 kilómetros de distancia



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Vénulas

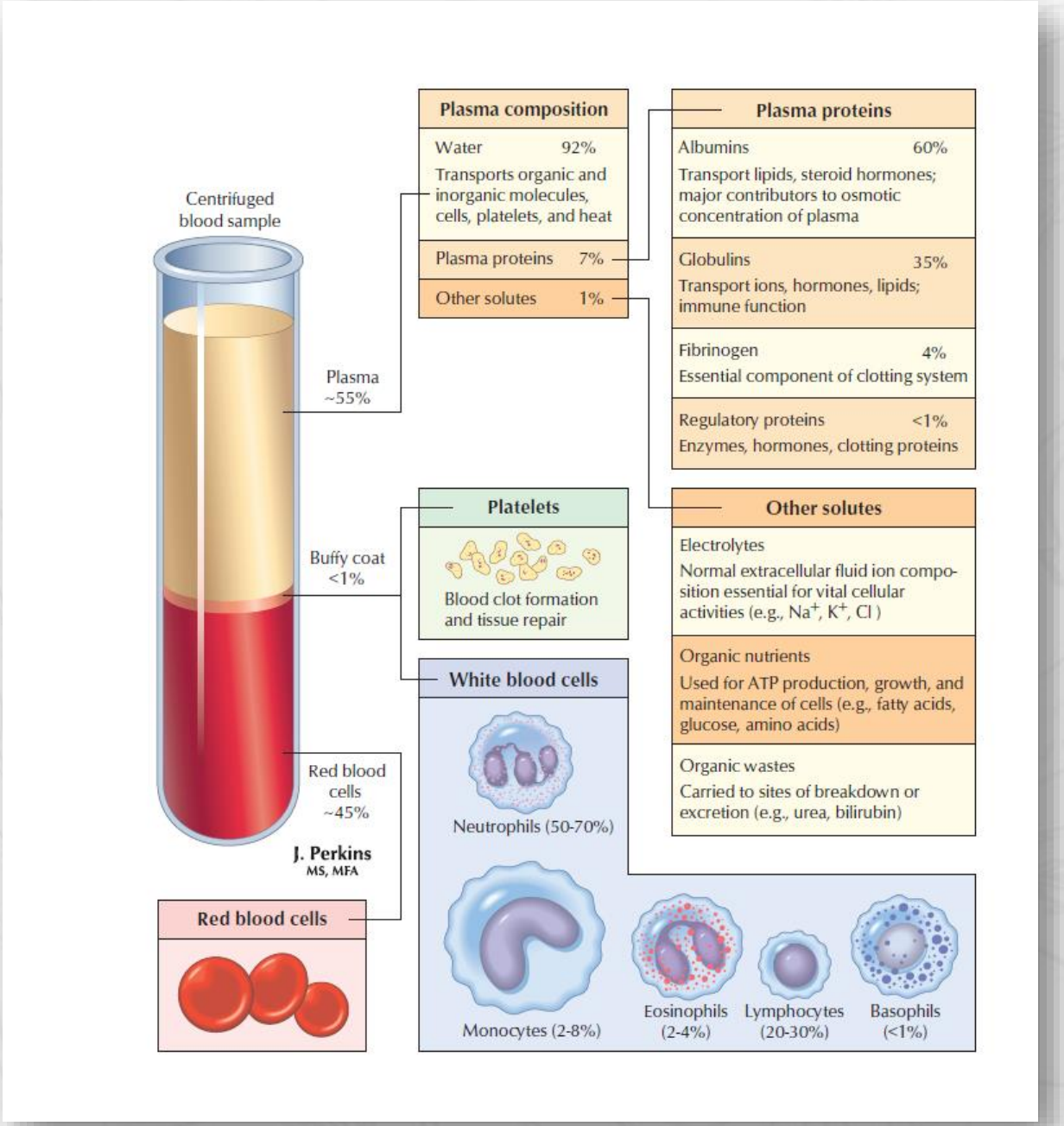


Arteriolas

CAPILARES

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Tu sangre ...
el río de la vida



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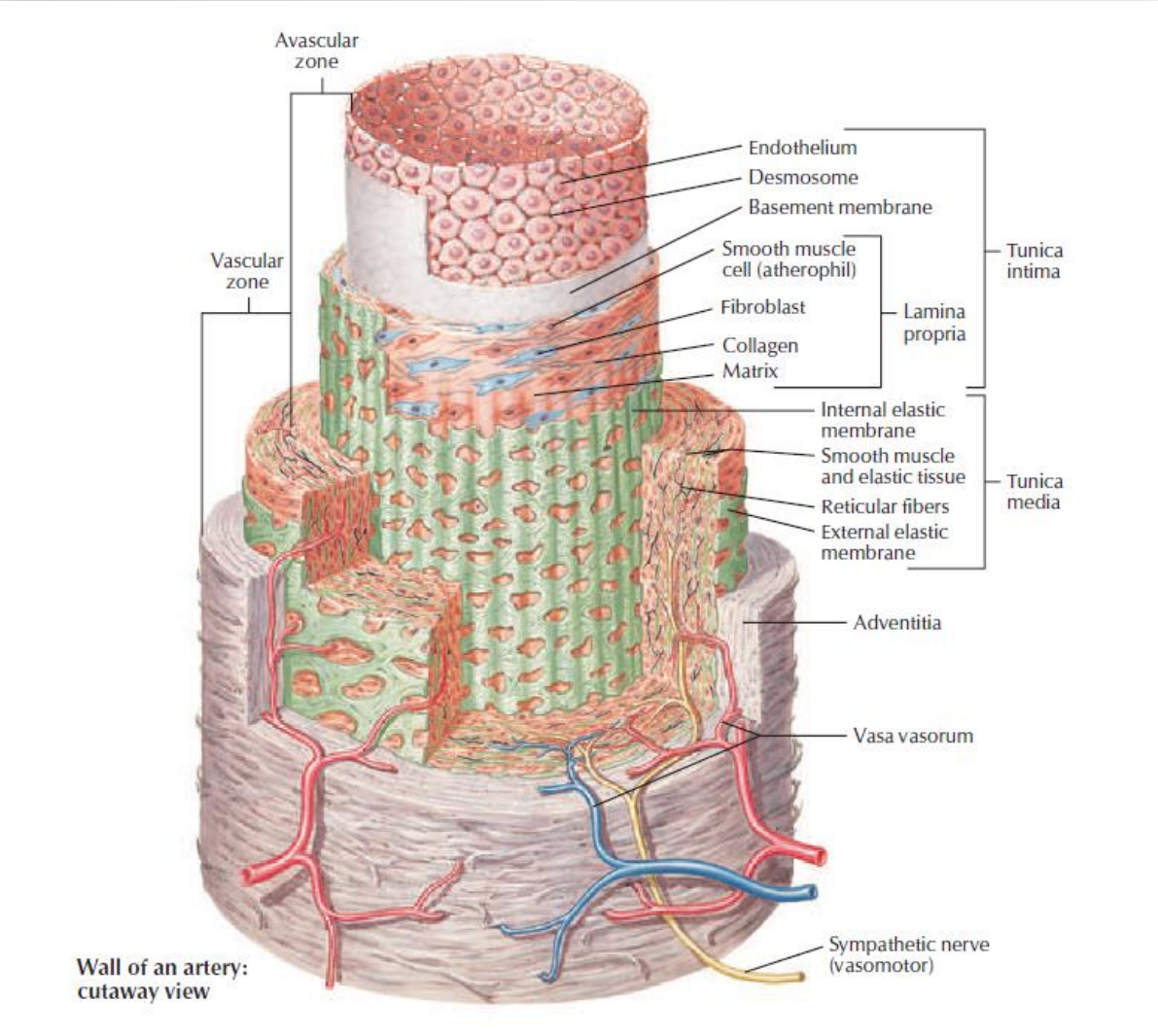
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Tus arterias por dentro

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Wall of an artery: cutaway view


Healthy artery

Aterosclerosis



Arteria saludable



Depósitos iniciales de grasa



Placa obstruyendo el flujo

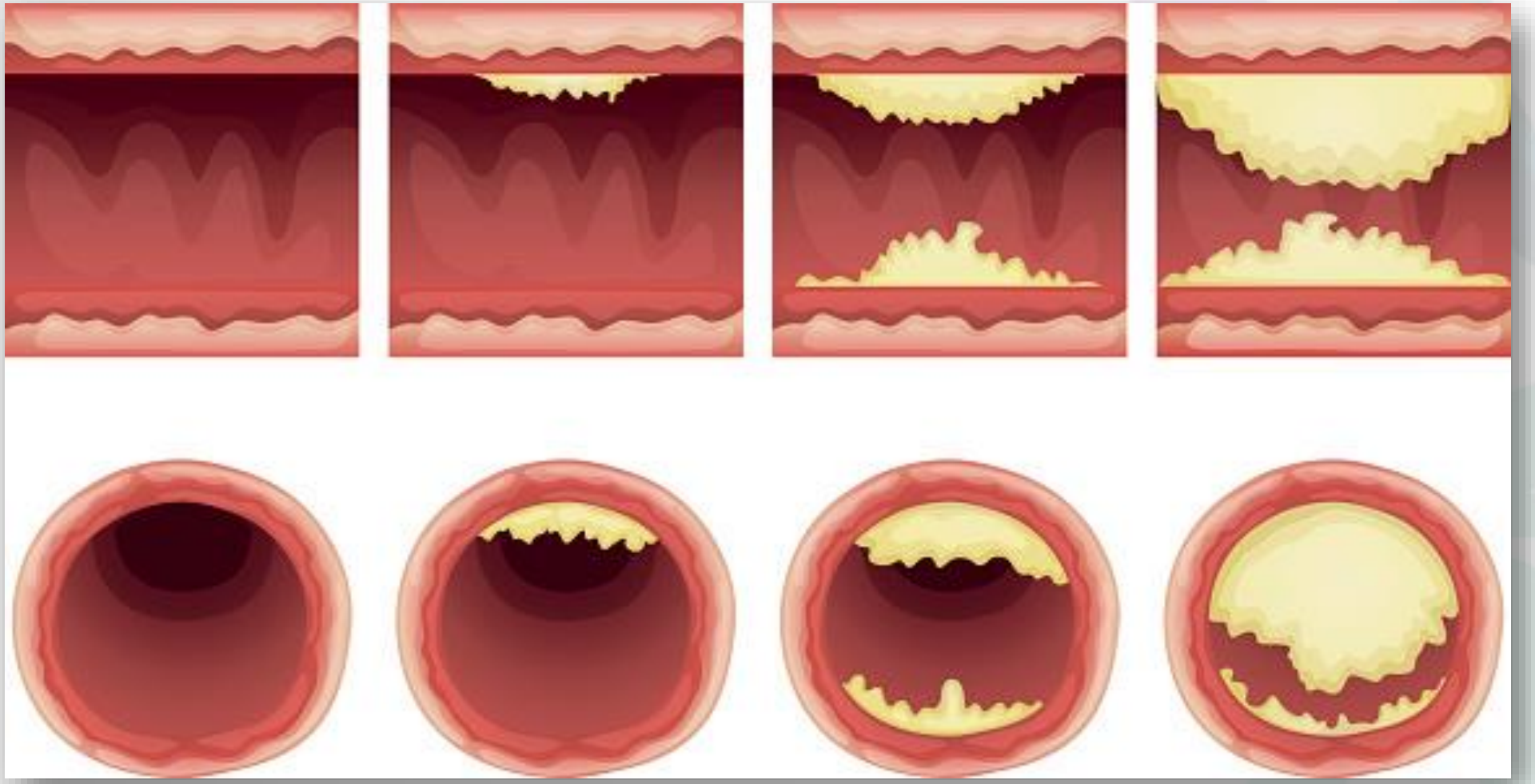


Obstrucción casi completa

Roher AE, Tyas SL, Maarouf CL, et al. Intracranial atherosclerosis as a contributing factor to Alzheimer's disease dementia. Alzheimers Dement. 2011;7(4):436-444.

Aterosclerosis

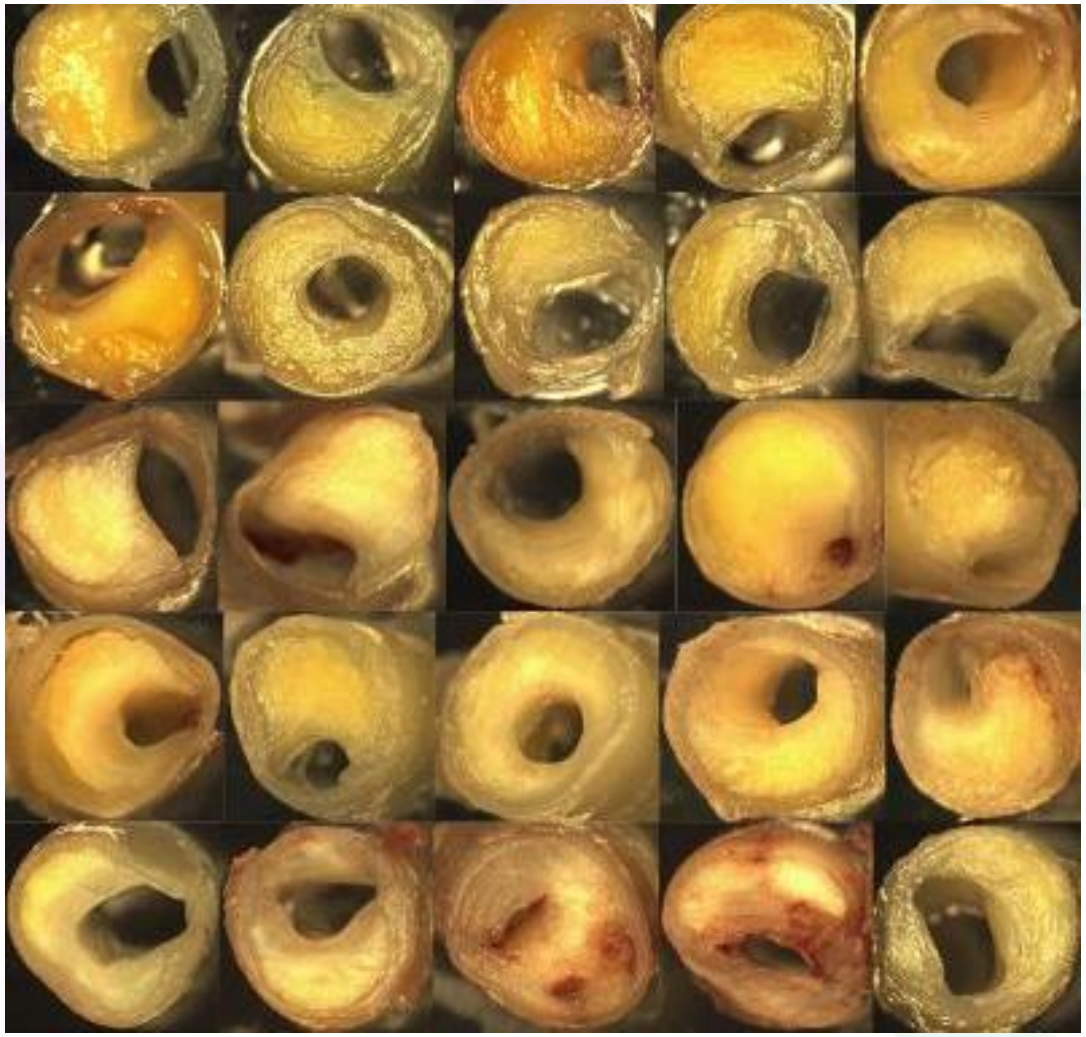
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Las arterias con aterosclerosis (de verdad)

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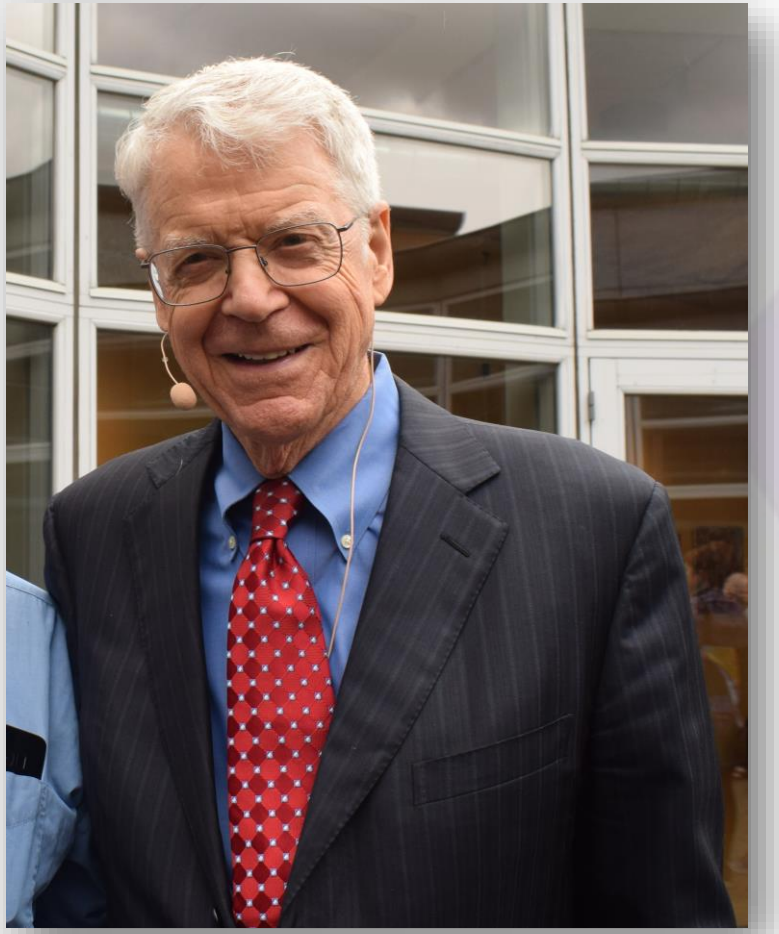


Evidencia científica de

reversión de aterosclerosis en el corazón

Reversión de enfermedad cardiovascular

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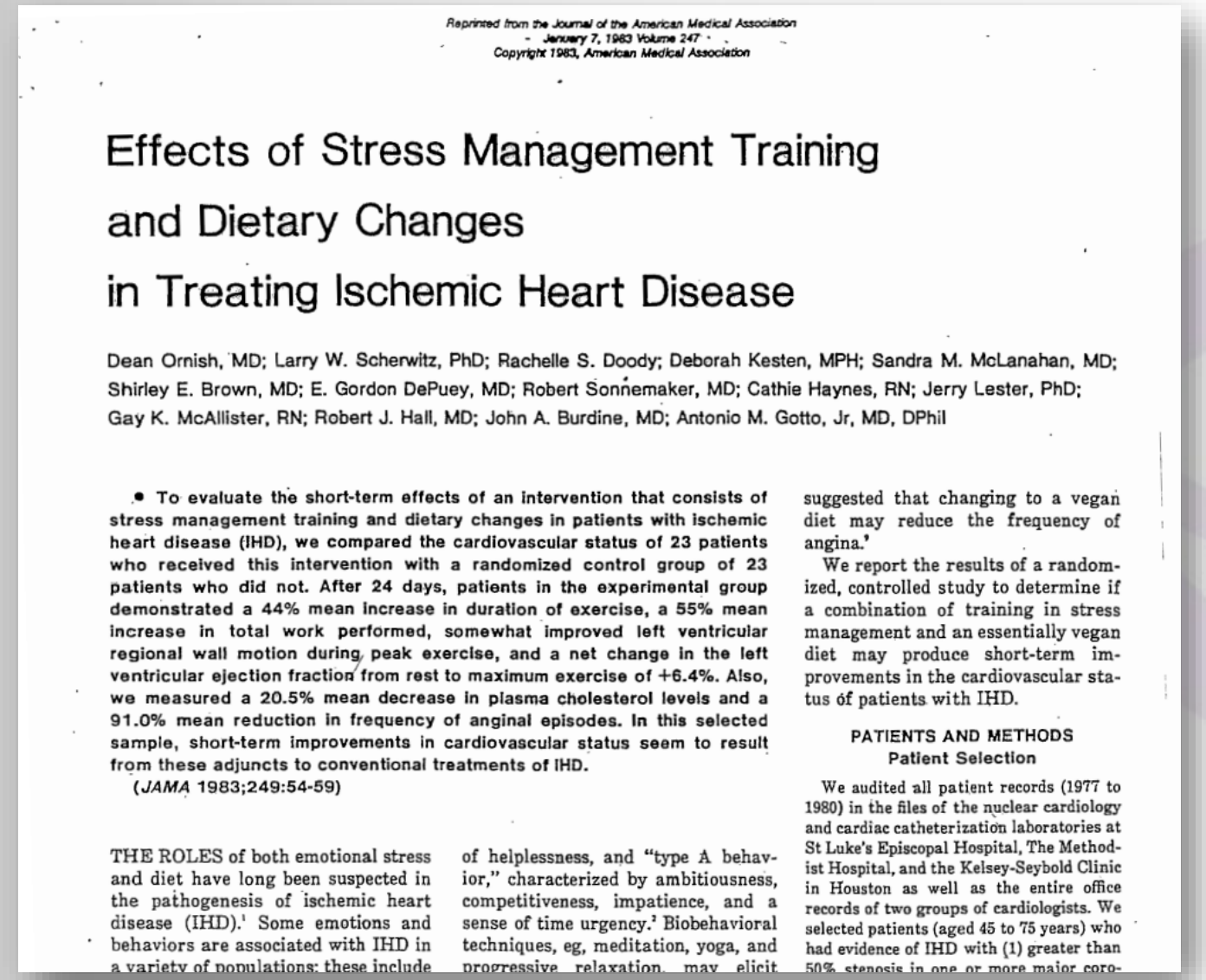
Dr. Caldwell Esselstyn



Dr. Dean Ornish

1983

Dr. Dean Ornish



Reprinted from the Journal of the American Medical Association
January 7, 1983 Volume 247
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Effects of Stress Management Training and Dietary Changes in Treating Ischemic Heart Disease

Dean Ornish, MD; Larry W. Scherwitz, PhD; Rachelle S. Doody; Deborah Kesten, MPH; Sandra M. McLanahan, MD; Shirley E. Brown, MD; E. Gordon DePuey, MD; Robert Sonnemaker, MD; Cathie Haynes, RN; Jerry Lester, PhD; Gay K. McAllister, RN; Robert J. Hall, MD; John A. Burdine, MD; Antonio M. Gotto, Jr, MD, DPhil

• To evaluate the short-term effects of an intervention that consists of stress management training and dietary changes in patients with ischemic heart disease (IHD), we compared the cardiovascular status of 23 patients who received this intervention with a randomized control group of 23 patients who did not. After 24 days, patients in the experimental group demonstrated a 44% mean increase in duration of exercise, a 55% mean increase in total work performed, somewhat improved left ventricular regional wall motion during peak exercise, and a net change in the left ventricular ejection fraction from rest to maximum exercise of +6.4%. Also, we measured a 20.5% mean decrease in plasma cholesterol levels and a 91.0% mean reduction in frequency of anginal episodes. In this selected sample, short-term improvements in cardiovascular status seem to result from these adjuncts to conventional treatments of IHD.
(*JAMA* 1983;249:54-59)

THE ROLES of both emotional stress and diet have long been suspected in the pathogenesis of ischemic heart disease (IHD).[1] Some emotions and behaviors are associated with IHD in a variety of populations; these include of helplessness, and "type A behavior," characterized by ambitiousness, competitiveness, impatience, and a sense of time urgency.[2] Biobehavioral techniques, eg, meditation, yoga, and progressive relaxation, may elicit suggested that changing to a vegan diet may reduce the frequency of angina.[3]

We report the results of a randomized, controlled study to determine if a combination of training in stress management and an essentially vegan diet may produce short-term improvements in the cardiovascular status of patients with IHD.

PATIENTS AND METHODS

Patient Selection

We audited all patient records (1977 to 1980) in the files of the nuclear cardiology and cardiac catheterization laboratories at St Luke's Episcopal Hospital, The Methodist Hospital, and the Kelsey-Seybold Clinic in Houston as well as the entire office records of two groups of cardiologists. We selected patients (aged 45 to 75 years) who had evidence of IHD with (1) greater than 50% stenosis in one or more major coro-

- 23 pacientes

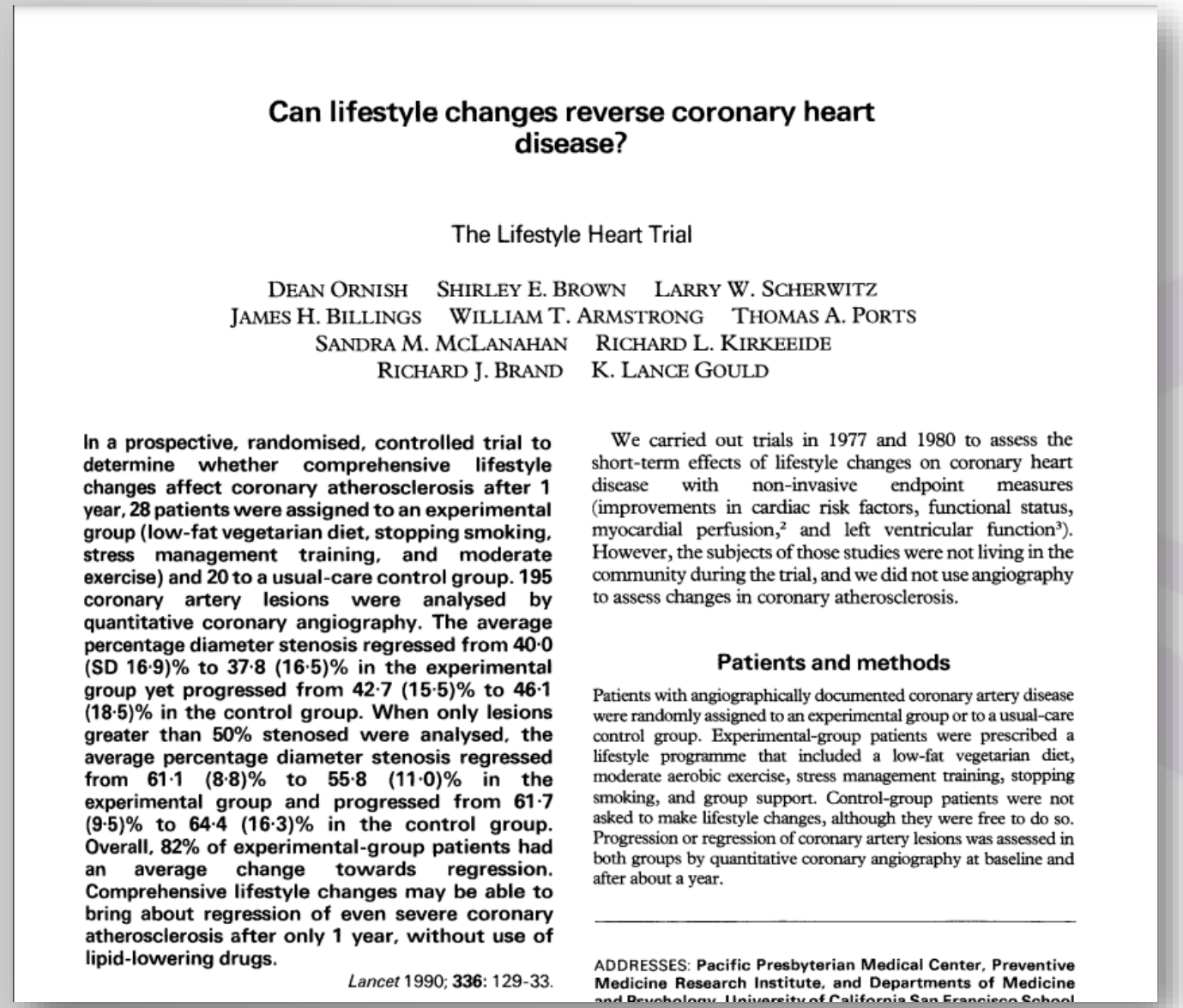
Grupo experimental:

- 44% duración de ejercicio
- Mejoría de función cardiaca
- 20% disminución de colesterol
- 91% reducción de frecuencia de angina

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1990

Dr. Dean Ornish



Can lifestyle changes reverse coronary heart disease?

The Lifestyle Heart Trial

DEAN ORNISH SHIRLEY E. BROWN LARRY W. SCHERWITZ
JAMES H. BILLINGS WILLIAM T. ARMSTRONG THOMAS A. PORTS
SANDRA M. MCLANAHAN RICHARD L. KIRKEEIDE
RICHARD J. BRAND K. LANCE GOULD

In a prospective, randomised, controlled trial to determine whether comprehensive lifestyle changes affect coronary atherosclerosis after 1 year, 28 patients were assigned to an experimental group (low-fat vegetarian diet, stopping smoking, stress management training, and moderate exercise) and 20 to a usual-care control group. 195 coronary artery lesions were analysed by quantitative coronary angiography. The average percentage diameter stenosis regressed from 40·0 (SD 16·9)% to 37·8 (16·5)% in the experimental group yet progressed from 42·7 (15·5)% to 46·1 (18·5)% in the control group. When only lesions greater than 50% stenosed were analysed, the average percentage diameter stenosis regressed from 61·1 (8·8)% to 55·8 (11·0)% in the experimental group and progressed from 61·7 (9·5)% to 64·4 (16·3)% in the control group. Overall, 82% of experimental-group patients had an average change towards regression. Comprehensive lifestyle changes may be able to bring about regression of even severe coronary atherosclerosis after only 1 year, without use of lipid-lowering drugs.

Lancet 1990; **336**: 129-33.

We carried out trials in 1977 and 1980 to assess the short-term effects of lifestyle changes on coronary heart disease with non-invasive endpoint measures (improvements in cardiac risk factors, functional status, myocardial perfusion,[2] and left ventricular function[3]). However, the subjects of those studies were not living in the community during the trial, and we did not use angiography to assess changes in coronary atherosclerosis.

Patients and methods

Patients with angiographically documented coronary artery disease were randomly assigned to an experimental group or to a usual-care control group. Experimental-group patients were prescribed a lifestyle programme that included a low-fat vegetarian diet, moderate aerobic exercise, stress management training, stopping smoking, and group support. Control-group patients were not asked to make lifestyle changes, although they were free to do so. Progression or regression of coronary artery lesions was assessed in both groups by quantitative coronary angiography at baseline and after about a year.

ADDRESSES: **Pacific Presbyterian Medical Center, Preventive Medicine Research Institute, and Departments of Medicine and Psychology, University of California San Francisco School**

- 48 pacientes
- Estenosis >50% revirtió de 61,1 a 55,8 en grupo de intervención
- Estenosis empeoró en grupo control de 61,7 a 64,4
- 82% tuvo reversión

Whole to Heal
INTERNATIONAL

1992

Dr. Dean Ornish

CORONARY ARTERY DISEASE

The American Journal of Cardiology

APRIL 1, 1992, VOL. 69, NO. 10

Improved Stenosis Geometry by Quantitative Coronary Arteriography After Vigorous Risk Factor Modification

K. Lance Gould, MD, Dean Ornish, MD, Richard Kirkeeide, PhD, Shirley Brown, MD, Yvonne Stuart, Martin Buchi, MD, James Billings, PhD, William Armstrong, MD, Thomas Ports, MD, and Larry Scherwitz, PhD

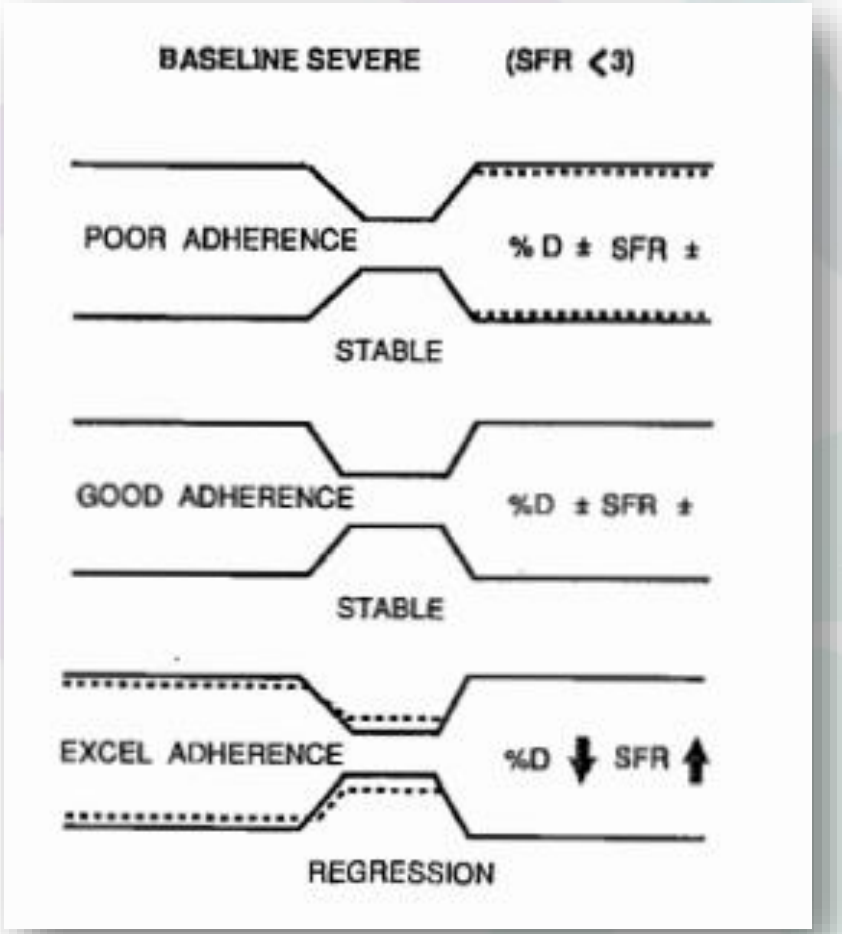
This study is a randomized, controlled, blinded, arteriographic trial to determine the effects of a low-cholesterol, low-fat, vegetarian diet, stress management and moderate aerobic exercise on geometric dimensions, shape and fluid dynamic characteristics of coronary artery stenoses in humans. Complex changes of different primary stenosis dimensions in opposite directions or to different degrees cause stenosis shape change with profound effects on fluid dynamic severity, not accounted for by simple percent narrowing. Accordingly, all stenosis dimensions were analyzed, including proximal, minimal, distal diameter, integrated length, exit angles and exit effects, determining stenosis shape and a single integrated measure of stenosis severity, stenosis flow reserve reflecting functional severity. In the control group, complex shape change and a stenosis—molding characteristic of statistically significant progressing severity occurred with worsening of stenosis flow reserve. In the treated group, complex shape change and stenosis molding characteristic of significant regressing severity was observed with improved stenosis flow reserve, thereby documenting the multidimensional characteristics of regressing coronary artery disease in humans.

(Am J Cardiol 1992;69:845–853)

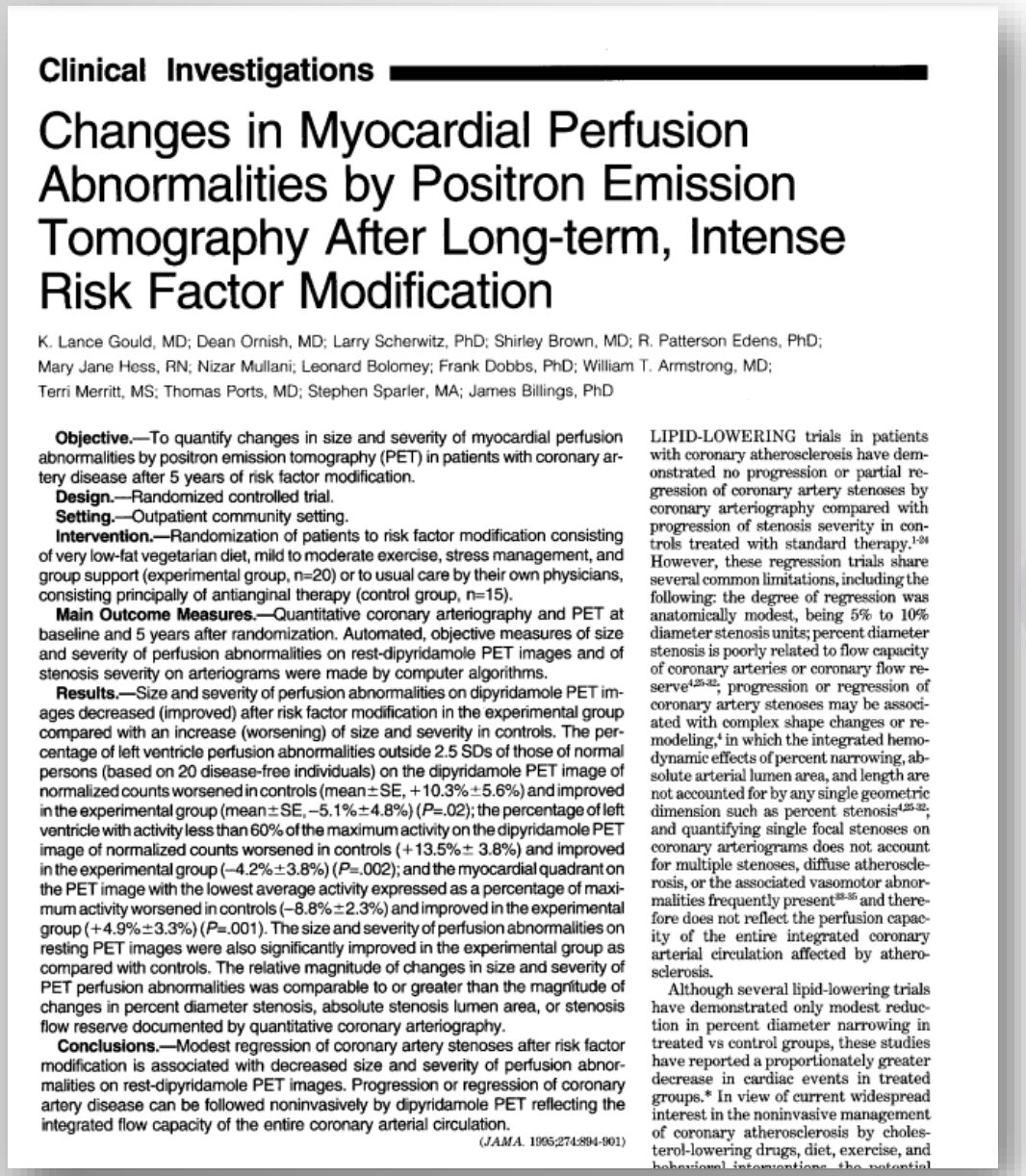
The Lifestyle Heart Trial[1] is a randomized, controlled, blinded, arteriographic trial to test the hypothesis that (1) vigorous modification of risk factors stops progression or causes regression of coronary artery stenosis, and (2) stenosis shape changes or undergoes remolding with progression or regression of coronary atherosclerosis in humans to the extent that no single dimension reflects the essential changes observed.

The initial report of this trial[1] describes the experimental design, risk factor intervention and changes in percent diameter stenosis because percent diameter stenosis is the most widely used measure of stenosis severity. However, percent stenosis is an incomplete measure of stenosis severity because length, absolute lumen area and shape effects are not accounted for and correlate poorly with the functional measure of coronary artery stenoses, coronary flow reserve.[2–10] The cumulative effects of multidimensional geometric changes on fluid dynamic characteristics of stenoses suggest that simple concepts of progression/regression need to be redefined in terms of shape changes or remolding of stenoses. Therefore, to definitively address whether progression or regression occurs in humans with risk factor modification, we performed multidimensional analysis of arteriographic stenoses to determine stenosis flow reserve as a single functional measure of severity incorporating all geometric dimensions of each stenosis.

Background: Seven previous coronary arteriographic trials of cholesterol lowering by diet and drugs have been reported.[11–17] Kuo et al[11] and the Leiden Interven-



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Clinical Investigations

Changes in Myocardial Perfusion Abnormalities by Positron Emission Tomography After Long-term, Intense Risk Factor Modification

K. Lance Gould, MD; Dean Ornish, MD; Larry Scherwitz, PhD; Shirley Brown, MD; R. Patterson Edens, PhD; Mary Jane Hess, RN; Nizar Mullani; Leonard Bolomey; Frank Dobbs, PhD; William T. Armstrong, MD; Terri Merritt, MS; Thomas Ports, MD; Stephen Sparler, MA; James Billings, PhD

Objective.—To quantify changes in size and severity of myocardial perfusion abnormalities by positron emission tomography (PET) in patients with coronary artery disease after 5 years of risk factor modification.

Design.—Randomized controlled trial.

Setting.—Outpatient community setting.

Intervention.—Randomization of patients to risk factor modification consisting of very low-fat vegetarian diet, mild to moderate exercise, stress management, and group support (experimental group, n=20) or to usual care by their own physicians, consisting principally of antianginal therapy (control group, n=15).

Main Outcome Measures.—Quantitative coronary arteriography and PET at baseline and 5 years after randomization. Automated, objective measures of size and severity of perfusion abnormalities on rest-dipyridamole PET images and of stenosis severity on arteriograms were made by computer algorithms.

Results.—Size and severity of perfusion abnormalities on dipyridamole PET images decreased (improved) after risk factor modification in the experimental group compared with an increase (worsening) of size and severity in controls. The percentage of left ventricle perfusion abnormalities outside 2.5 SDs of those of normal persons (based on 20 disease-free individuals) on the dipyridamole PET image of normalized counts worsened in controls (mean±SE, +10.3%±5.6%) and improved in the experimental group (mean±SE, −5.1%±4.8%) (P=.02); the percentage of left ventricle with activity less than 60% of the maximum activity on the dipyridamole PET image of normalized counts worsened in controls (+13.5%± 3.8%) and improved in the experimental group (−4.2%±3.8%) (P=.002); and the myocardial quadrant on the PET image with the lowest average activity expressed as a percentage of maximum activity worsened in controls (−8.8%±2.3%) and improved in the experimental group (+4.9%±3.3%) (P=.001). The size and severity of perfusion abnormalities on resting PET images were also significantly improved in the experimental group as compared with controls. The relative magnitude of changes in size and severity of PET perfusion abnormalities was comparable to or greater than the magnitude of changes in percent diameter stenosis, absolute stenosis lumen area, or stenosis flow reserve documented by quantitative coronary arteriography.

Conclusions.—Modest regression of coronary artery stenoses after risk factor modification is associated with decreased size and severity of perfusion abnormalities on rest-dipyridamole PET images. Progression or regression of coronary artery disease can be followed noninvasively by dipyridamole PET reflecting the integrated flow capacity of the entire coronary arterial circulation.

(*JAMA*. 1995;274:894-901)

LIPID-LOWERING trials in patients with coronary atherosclerosis have demonstrated no progression or partial regression of coronary artery stenoses by coronary arteriography compared with progression of stenosis severity in controls treated with standard therapy.[1-24] However, these regression trials share several common limitations, including the following: the degree of regression was anatomically modest, being 5% to 10% diameter stenosis units; percent diameter stenosis is poorly related to flow capacity of coronary arteries or coronary flow reserve[4,25-32]; progression or regression of coronary artery stenoses may be associated with complex shape changes or remodeling,[4] in which the integrated hemodynamic effects of percent narrowing, absolute arterial lumen area, and length are not accounted for by any single geometric dimension such as percent stenosis[4,25-32]; and quantifying single focal stenoses on coronary arteriograms does not account for multiple stenoses, diffuse atherosclerosis, or the associated vasomotor abnormalities frequently present[33-35] and therefore does not reflect the perfusion capacity of the entire integrated coronary arterial circulation affected by atherosclerosis.

Although several lipid-lowering trials have demonstrated only modest reduction in percent diameter narrowing in treated vs control groups, these studies have reported a proportionately greater decrease in cardiac events in treated groups.* In view of current widespread interest in the noninvasive management of coronary atherosclerosis by cholesterol-lowering drugs, diet, exercise, and behavioral interventions, the potential

1995

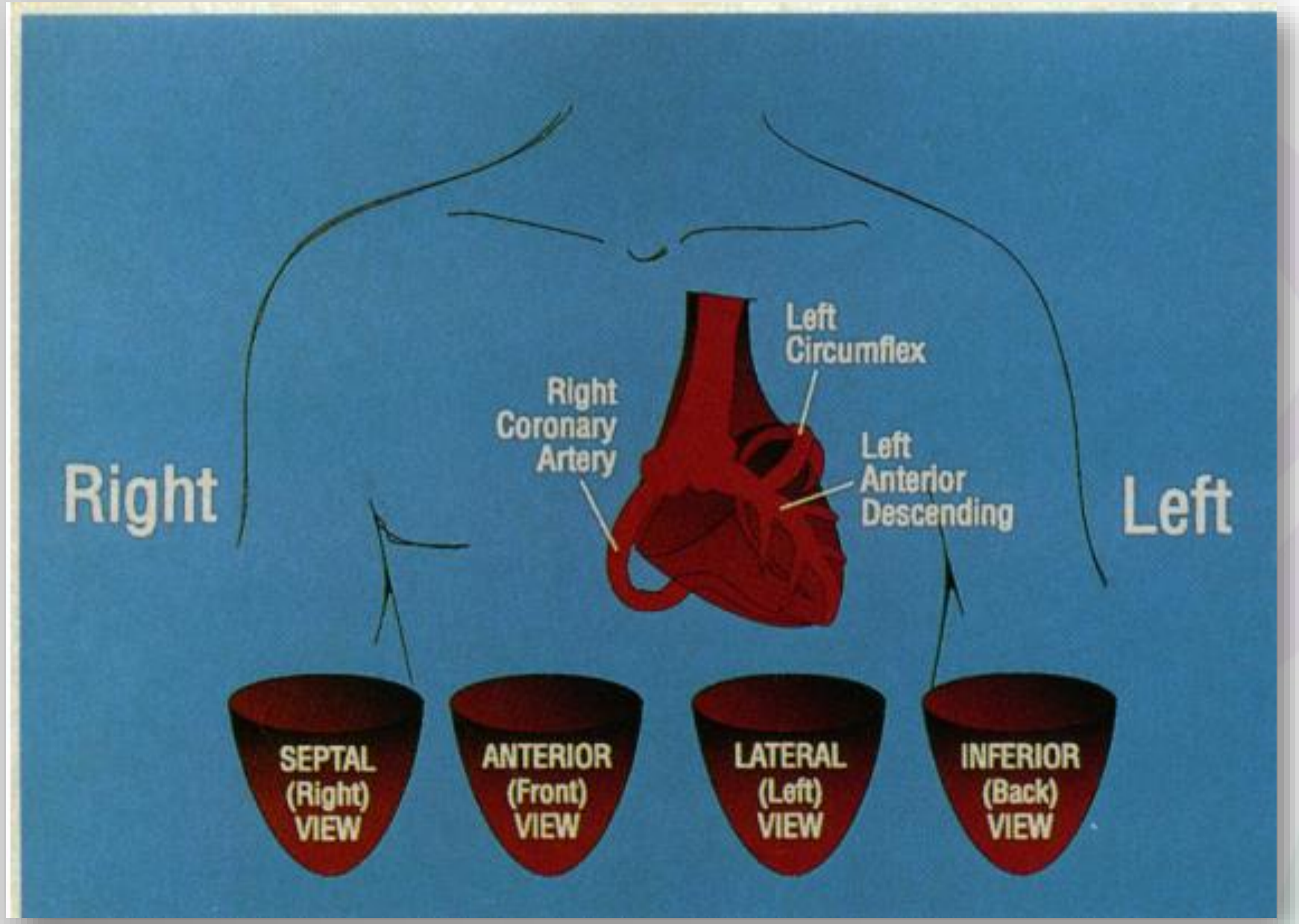
Dr. Dean Ornish

- Cambios en la perfusión miocárdica en PET scans después de 5 años
- Tamaño y severidad de anormalidades de perfusión mejoraron significativamente en grupo de tratamiento

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1995

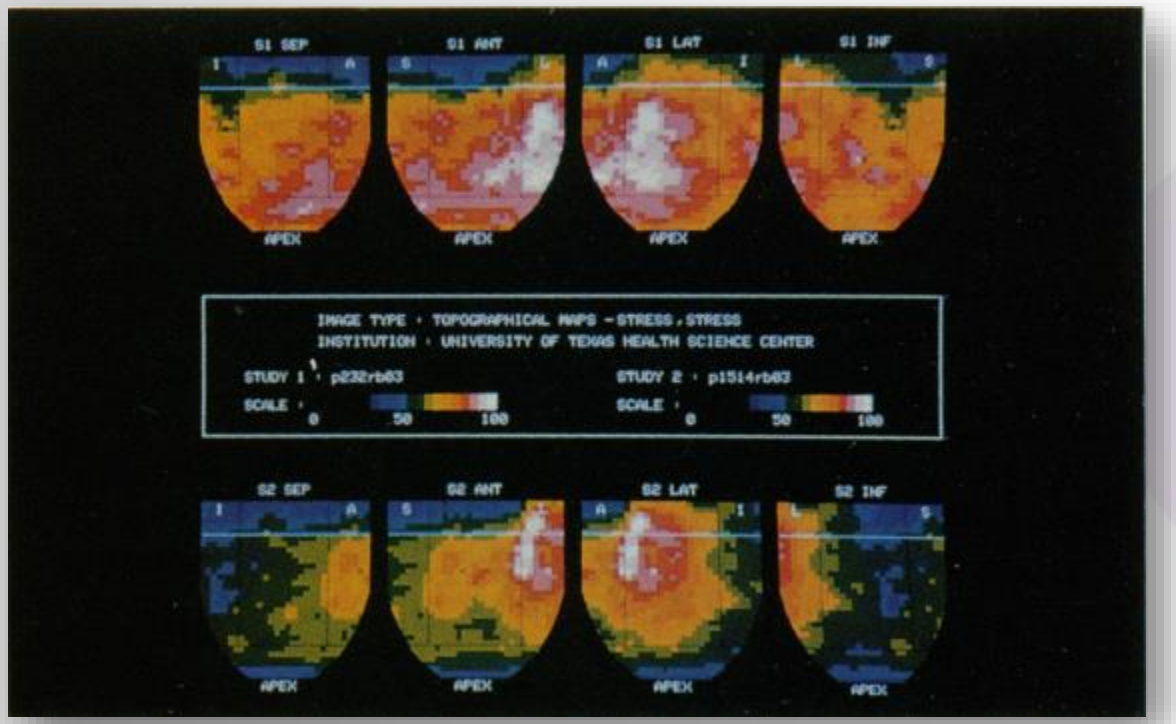
Dr. Dean Ornish



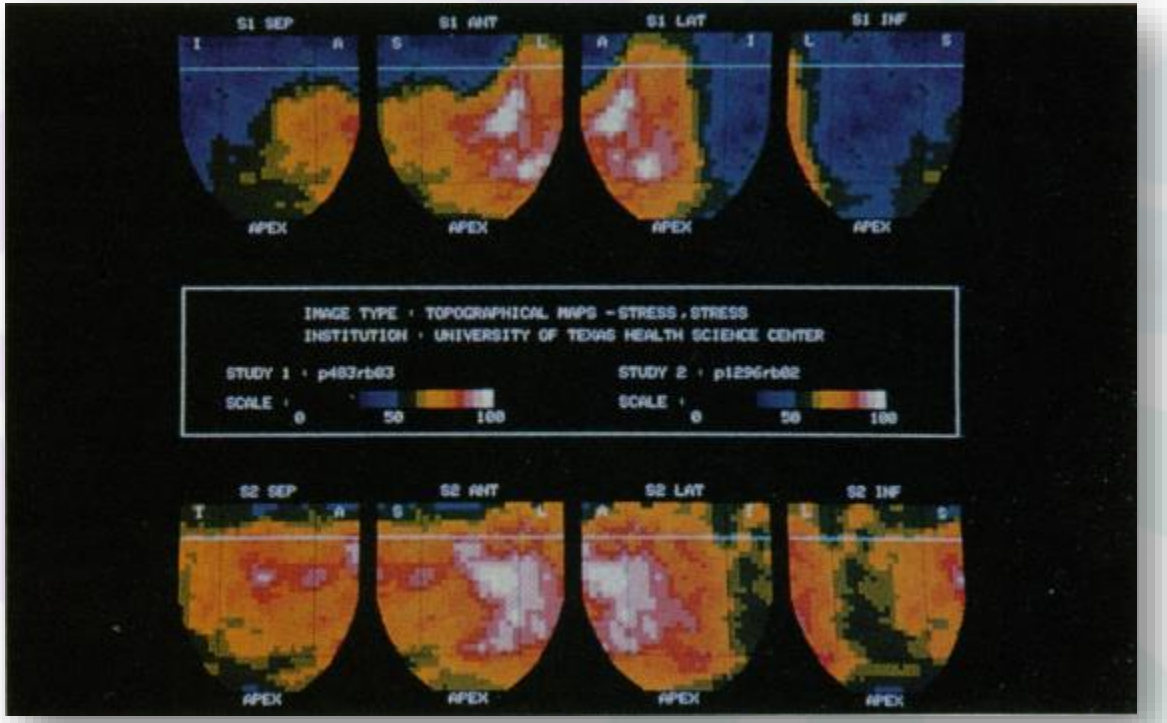
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1995

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Paciente control

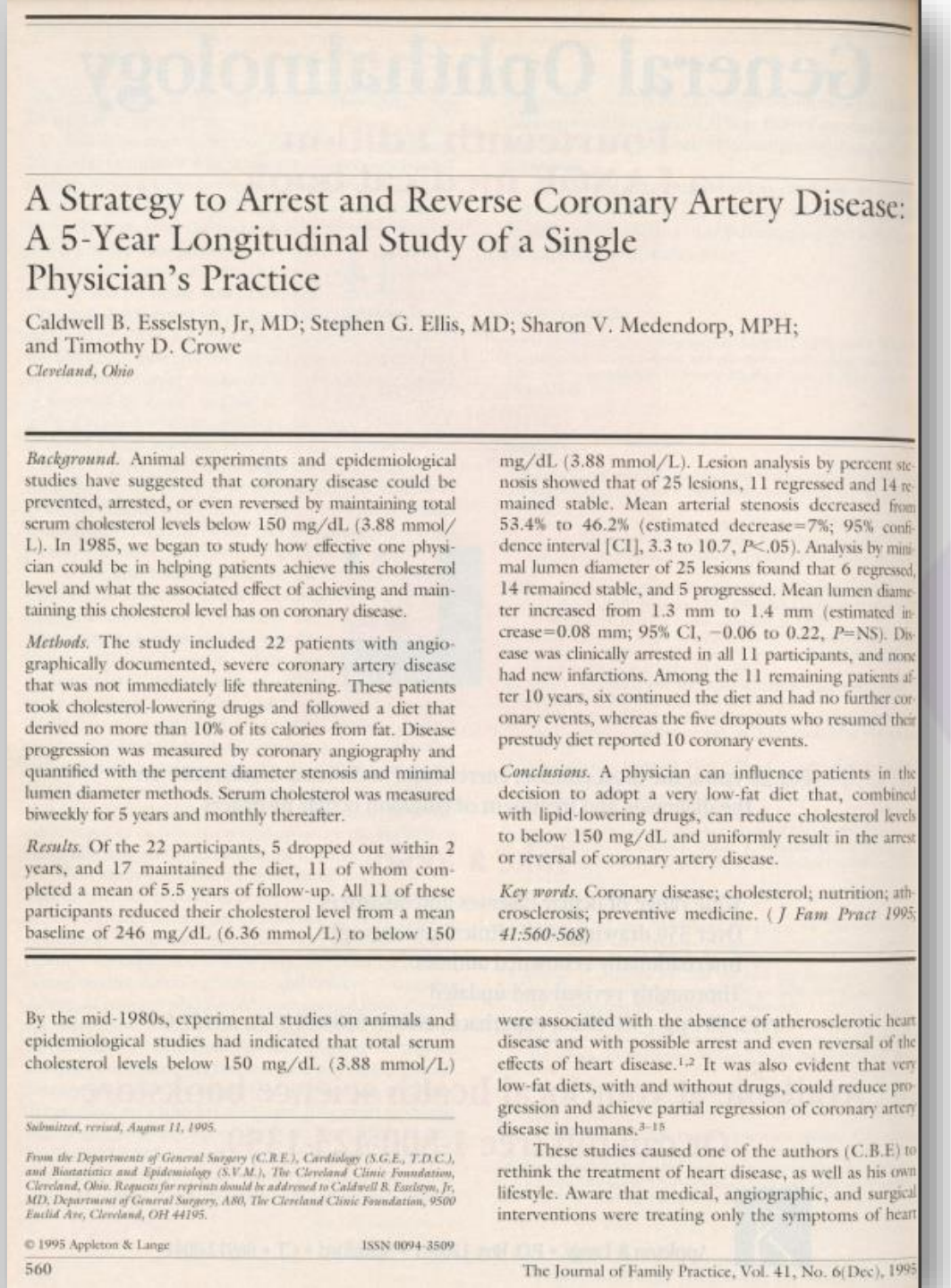


Paciente tratado

Whole to Heal
INTERNATIONAL

1995

Dr. Esselstyn



A Strategy to Arrest and Reverse Coronary Artery Disease: A 5-Year Longitudinal Study of a Single Physician's Practice

Caldwell B. Esselstyn, Jr, MD; Stephen G. Ellis, MD; Sharon V. Medendorp, MPH; and Timothy D. Crowe
Cleveland, Ohio

Background. Animal experiments and epidemiological studies have suggested that coronary disease could be prevented, arrested, or even reversed by maintaining total serum cholesterol levels below 150 mg/dL (3.88 mmol/L). In 1985, we began to study how effective one physician could be in helping patients achieve this cholesterol level and what the associated effect of achieving and maintaining this cholesterol level has on coronary disease.

Methods. The study included 22 patients with angiographically documented, severe coronary artery disease that was not immediately life threatening. These patients took cholesterol-lowering drugs and followed a diet that derived no more than 10% of its calories from fat. Disease progression was measured by coronary angiography and quantified with the percent diameter stenosis and minimal lumen diameter methods. Serum cholesterol was measured biweekly for 5 years and monthly thereafter.

Results. Of the 22 participants, 5 dropped out within 2 years, and 17 maintained the diet, 11 of whom completed a mean of 5.5 years of follow-up. All 11 of these participants reduced their cholesterol level from a mean baseline of 246 mg/dL (6.36 mmol/L) to below 150 mg/dL (3.88 mmol/L). Lesion analysis by percent stenosis showed that of 25 lesions, 11 regressed and 14 remained stable. Mean arterial stenosis decreased from 53.4% to 46.2% (estimated decrease=7%; 95% confidence interval [CI], 3.3 to 10.7, $P<.05$). Analysis by minimal lumen diameter of 25 lesions found that 6 regressed, 14 remained stable, and 5 progressed. Mean lumen diameter increased from 1.3 mm to 1.4 mm (estimated increase=0.08 mm; 95% CI, −0.06 to 0.22, P=NS). Disease was clinically arrested in all 11 participants, and none had new infarctions. Among the 11 remaining patients after 10 years, six continued the diet and had no further coronary events, whereas the five dropouts who resumed their prestudy diet reported 10 coronary events.

Conclusions. A physician can influence patients in the decision to adopt a very low-fat diet that, combined with lipid-lowering drugs, can reduce cholesterol levels to below 150 mg/dL and uniformly result in the arrest or reversal of coronary artery disease.

Key words. Coronary disease; cholesterol; nutrition; atherosclerosis; preventive medicine. (*J Fam Pract 1995; 41:560-568*)

By the mid-1980s, experimental studies on animals and epidemiological studies had indicated that total serum cholesterol levels below 150 mg/dL (3.88 mmol/L) were associated with the absence of atherosclerotic heart disease and with possible arrest and even reversal of the effects of heart disease.[1,2] It was also evident that very low-fat diets, with and without drugs, could reduce progression and achieve partial regression of coronary artery disease in humans.[3-15]

These studies caused one of the authors (C.B.E) to rethink the treatment of heart disease, as well as his own lifestyle. Aware that medical, angiographic, and surgical interventions were treating only the symptoms of heart

Submitted, revised, August 11, 1995.

From the Departments of General Surgery (C.B.E.), Cardiology (S.G.E., T.D.C.), and Biostatistics and Epidemiology (S.V.M.), The Cleveland Clinic Foundation, Cleveland, Ohio. Requests for reprints should be addressed to Caldwell B. Esselstyn, Jr, MD, Department of General Surgery, A80, The Cleveland Clinic Foundation, 9500 Euclid Ave, Cleveland, OH 44195.

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ISSN 0094-3509

- 22 pacientes con enfermedad coronaria severa
- Seguimiento de 5 años en 11 pacientes
- Colesterol bajo de 246 a 150 mg/dL
- 11 estenosis revirtieron, 14 quedaron estables
- Nunca volvieron a tener infartos

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Intensive Lifestyle Changes for Reversal of Coronary Heart Disease

Dean Ornish, MD; Larry W. Scherwitz, PhD; James H. Billings, PhD, MPH; K. Lance Gould, MD; Terri A. Merritt, MS; Stephen Sparler, MA; William T. Armstrong, MD; Thomas A. Ports, MD; Richard L. Kirkeeide, PhD; Charissa Hogeboom, PhD; Richard J. Brand, PhD

Context.—The Lifestyle Heart Trial demonstrated that intensive lifestyle changes may lead to regression of coronary atherosclerosis after 1 year.

Objectives.—To determine the feasibility of patients to sustain intensive lifestyle changes for a total of 5 years and the effects of these lifestyle changes (without lipid-lowering drugs) on coronary heart disease.

Design.—Randomized controlled trial conducted from 1986 to 1992 using a randomized invitational design.

Patients.—Forty-eight patients with moderate to severe coronary heart disease were randomized to an intensive lifestyle change group or to a usual-care control group, and 35 completed the 5-year follow-up quantitative coronary arteriography.

Setting.—Two tertiary care university medical centers.

Intervention.—Intensive lifestyle changes (10% fat whole foods vegetarian diet, aerobic exercise, stress management training, smoking cessation, group psychosocial support) for 5 years.

Main Outcome Measures.—Adherence to intensive lifestyle changes, changes in coronary artery percent diameter stenosis, and cardiac events.

Results.—Experimental group patients (20 [71%] of 28 patients completed 5-year follow-up) made and maintained comprehensive lifestyle changes for 5 years, whereas control group patients (15 [75%] of 20 patients completed 5-year follow-up) made more moderate changes. In the experimental group, the average percent diameter stenosis at baseline decreased 1.75 absolute percentage points after 1 year (a 4.5% relative improvement) and by 3.1 absolute percentage points after 5 years (a 7.9% relative improvement). In contrast, the average percent diameter stenosis in the control group increased by 2.3 percentage points after 1 year (a 5.4% relative worsening) and by 11.8 percentage points after 5 years (a 27.7% relative worsening) ($P = .001$ between groups. Twenty-five cardiac events occurred in 28 experimental group patients vs 45 events in 20 control group patients during the 5-year follow-up (risk ratio for any event for the control group, 2.47 [95% confidence interval, 1.48-4.20]).

Conclusions.—More regression of coronary atherosclerosis occurred after 5 years than after 1 year in the experimental group. In contrast, in the control group, coronary atherosclerosis continued to progress and more than twice as many cardiac events occurred.

JAMA. 1998;280:2001-2007

THE LIFESTYLE Heart Trial was the first randomized clinical trial to investigate whether ambulatory patients could be motivated to make and sustain comprehensive lifestyle changes and, if so, whether the progression of coronary atherosclerosis could be stopped or reversed without using lipid-lowering drugs as measured by computer-assisted quantitative coronary arteriography. This study derived from earlier studies that used noninvasive measures.[1,2]

After 1 year, we found that experimental group participants were able to make and maintain intensive lifestyle changes and had a 37.2% reduction in low-density lipoprotein (LDL) cholesterol levels and a 91% reduction in the frequency of anginal episodes.[3] Average percent diameter stenosis regressed from 40.0% at baseline to 37.8% 1 year later, a change that was correlated with the degree of lifestyle change. In contrast, patients in the usual-care control group made more moderate changes in lifestyle, reduced LDL cholesterol levels by 6%, and had a 165% increase in the frequency of reported anginal episodes. Average percent diameter stenosis progressed from 42.7% to 46.1%.

Given these encouraging findings, we extended the study for an additional 4 years to determine (1) the feasibility of patients sustaining intensive changes in diet and lifestyle for a much longer time, and (2) the effects of these changes on risk factors, coronary atherosclero-

1998

Dr. Dean Ornish

- 48 pacientes con enfermedad arterial coronaria moderada y severa
- 35 completo 5 años
- 20 en grupo de intervención

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1998

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- Lifestyle Heart Trial
- 25 eventos cardiovasculares en grupo de tratamiento (28) vs 45 en 20 controles

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Lifestyle Heart Trial

1998
Dr. Dean Ornish

Control

- Ninguno pudo revertir enfermedad cardiovascular
- 165% de aumento de frecuencia del dolor de pecho
- Colesterol malo peor que el grupo de tratamiento

Tratamiento

- 82% pudo revertir enfermedad cardiovascular
- 91% reducción de frecuencia del dolor de pecho
- Colesterol malo bajo de 152mg/dl a 95mg/dl

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1999

Dr. Esselstyn

Am J Cardiol. 1999 Aug 1;84(3):339-41, A8. doi: 10.1016/s0002-9149(99)00290-8.

Updating a 12-year experience with arrest and reversal therapy for coronary heart disease (an overdue requiem for palliative cardiology)

C B Esselstyn Jr [1]

Affiliations + expand

PMID: 10496449 DOI: 10.1016/s0002-9149(99)00290-8

Abstract

Coronary artery disease is essentially nonexistent in cultures whose nutrition assures cholesterol levels <150 mg/dl. Patients with advanced coronary artery disease may abolish disease progression through a plant-based diet and cholesterol-lowering medication to achieve and maintain a total cholesterol <150 mg/dl.

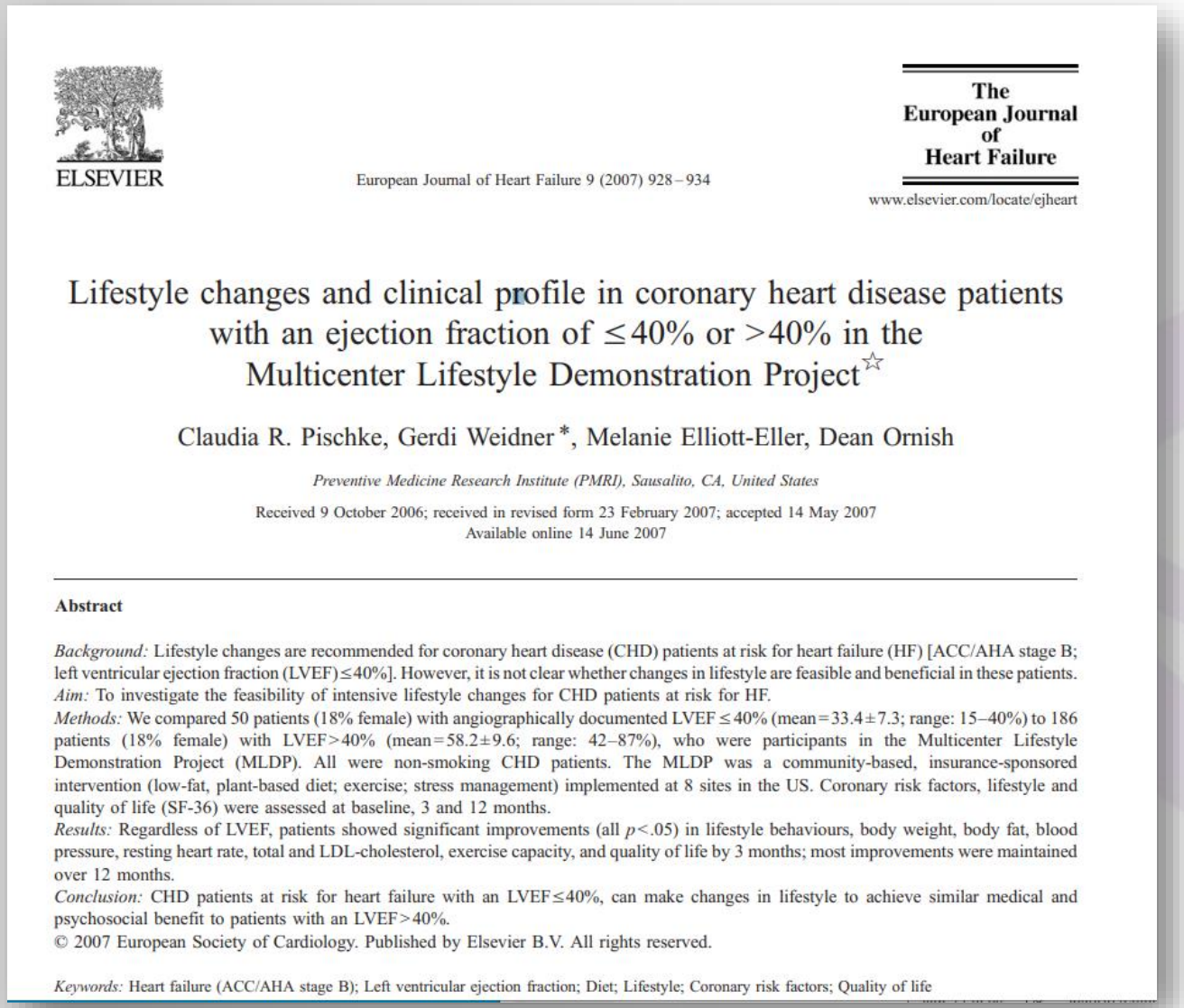
PubMed Disclaimer

- 18 pacientes se adhirieron después de 12 años
- Antes del estudio habían tenido 49 eventos cardiacos
- 17 de los 18 NO tuvieron ningún evento durante los 12 años

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2007

Dr. Dean Ornish



ELSEVIER

European Journal of Heart Failure 9 (2007) 928–934

The European Journal of Heart Failure

www.elsevier.com/locate/ejheart

Lifestyle changes and clinical profile in coronary heart disease patients with an ejection fraction of ≤40% or >40% in the Multicenter Lifestyle Demonstration Project☆

Claudia R. Pischke, Gerdi Weidner*, Melanie Elliott-Eller, Dean Ornish

Preventive Medicine Research Institute (PMRI), Sausalito, CA, United States

Received 9 October 2006; received in revised form 23 February 2007; accepted 14 May 2007
Available online 14 June 2007

Abstract

Background: Lifestyle changes are recommended for coronary heart disease (CHD) patients at risk for heart failure (HF) [ACC/AHA stage B; left ventricular ejection fraction (LVEF)≤40%]. However, it is not clear whether changes in lifestyle are feasible and beneficial in these patients.
Aim: To investigate the feasibility of intensive lifestyle changes for CHD patients at risk for HF.
Methods: We compared 50 patients (18% female) with angiographically documented LVEF ≤40% (mean=33.4±7.3; range: 15–40%) to 186 patients (18% female) with LVEF>40% (mean=58.2±9.6; range: 42–87%), who were participants in the Multicenter Lifestyle Demonstration Project (MLDP). All were non-smoking CHD patients. The MLDP was a community-based, insurance-sponsored intervention (low-fat, plant-based diet; exercise; stress management) implemented at 8 sites in the US. Coronary risk factors, lifestyle and quality of life (SF-36) were assessed at baseline, 3 and 12 months.
Results: Regardless of LVEF, patients showed significant improvements (all $p<.05$) in lifestyle behaviours, body weight, body fat, blood pressure, resting heart rate, total and LDL-cholesterol, exercise capacity, and quality of life by 3 months; most improvements were maintained over 12 months.
Conclusion: CHD patients at risk for heart failure with an LVEF≤40%, can make changes in lifestyle to achieve similar medical and psychosocial benefit to patients with an LVEF>40%.
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Keywords: Heart failure (ACC/AHA stage B); Left ventricular ejection fraction; Diet; Lifestyle; Coronary risk factors; Quality of life

- 50 pacientes con falla cardiaca crónica FE<40%
- Mejoría en peso, grasa corporal, tensión arterial, frecuencia cardiaca, perfil lipídico, capacidad de ejercicio y calidad de vida

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2008

Dr. Dean Ornish



Angina Pectoris and Atherosclerotic Risk Factors in the Multisite Cardiac Lifestyle Intervention Program

Joanne Frattaroli, PhD, Gerdi Weidner, PhD*, Terri A. Merritt-Worden, MS, Steven Frenda, BA, and Dean Ornish, MD

Cardiovascular symptom relief is a major indicator for revascularization procedures. To examine the effects of intensive lifestyle modification on symptom relief, we investigated changes in angina pectoris, coronary risk factors, quality of life, and lifestyle behaviors in patients with stable coronary artery disease enrolled in the multisite cardiac lifestyle intervention program, an ongoing health insurance–covered lifestyle intervention conducted at 22 sites in the united states. Patients with coronary artery disease (nonsmokers; 757 men, 395 women; mean age 61 years) were asked to make changes in diet (10% calories from fat, plant based), engage in moderate exercise (3 hours/week), and practice stress management (1 hour/day). At baseline, 108 patients (43% women) reported mild angina and 174 patients (37% women) reported limiting angina. By 12 weeks, 74% of these patients were angina free, and an additional 9% moved from limiting to mild angina. This improvement in angina was significant for patients with mild and limiting angina at baseline regardless of gender ($p < 0.01$). Significant improvements in cardiac risk factors, quality of life, and lifestyle behaviors were observed, and patients with angina who became angina free by 12 weeks showed the greatest improvements in exercise capacity, depression, and health-related quality of life ($p < 0.05$). In conclusion, the observed improvements in angina in patients making intensive lifestyle changes could drastically reduce their need for revascularization procedures. © 2008 Elsevier Inc. All rights reserved. (Am J Cardiol 2008;101:911–918)

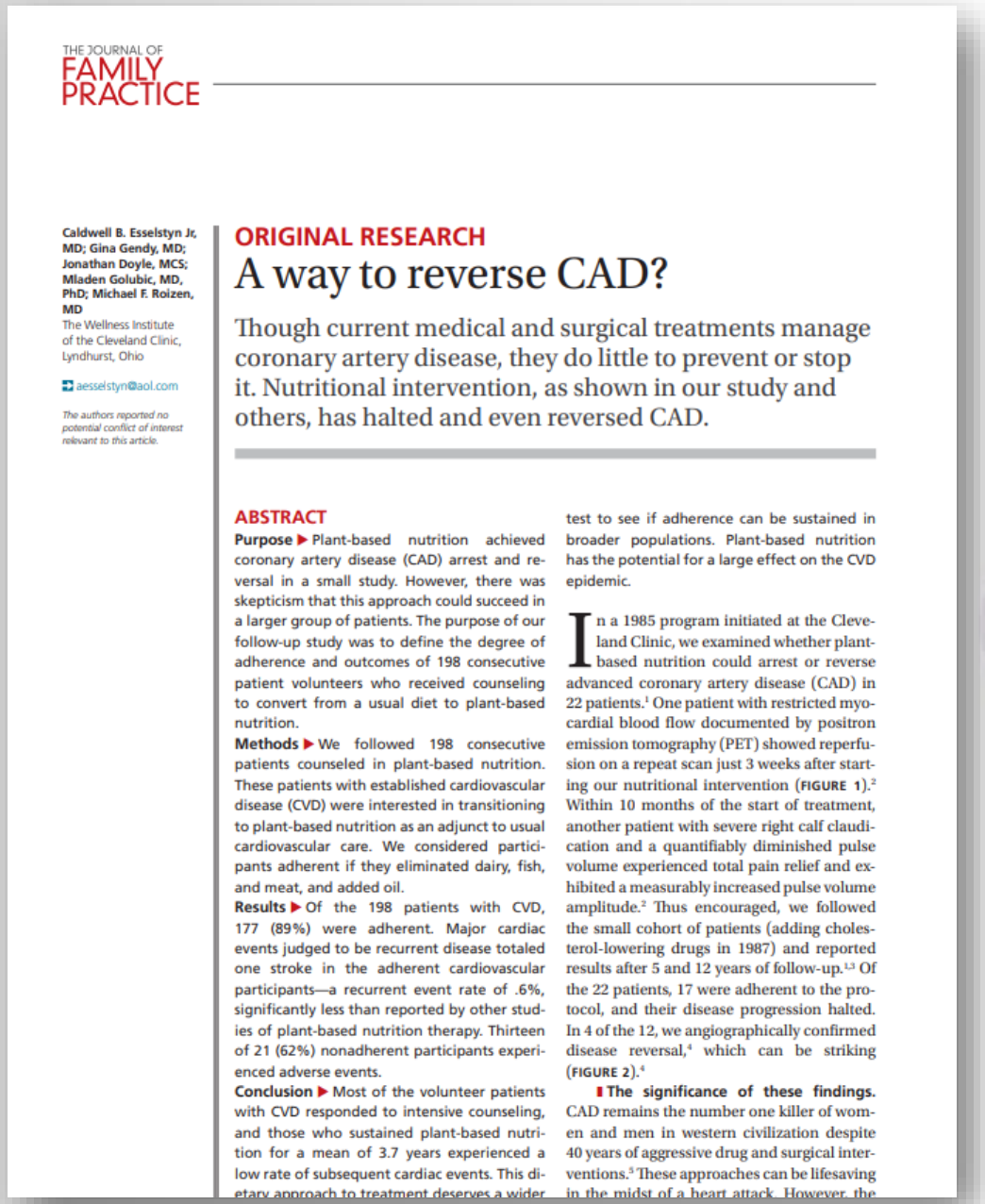
Although the prognostic significance of angina pectoris in women was questioned,[1] a recent large-scale population study found that women aged <75 years with stable angina pectoris had higher 4-year cardiac mortality rates than their male counterparts.[2] It is important to examine angina reduction in both men and women who are making comprehensive lifestyle changes. In addition, it is unknown whether patients with limiting/severe angina show benefits from making lifestyle changes similar to those with mild angina. We examined angina symptoms, coronary risk factors, quality of life, and lifestyle behaviors in the Multisite Cardiac Lifestyle Intervention Program during a 12-week period.

Methods

lifestyle change program for primary and secondary prevention of CAD administered by insurance companies. All patients were individually prescribed exercise levels (typically walking) according to their baseline treadmill test results. Target training heart rates were calculated using the Karvonen formula at intensities of 45% to 80% of maximal heart rate achieved on the exercise test. If ischemia occurred, the upper training heart rate was limited to 10 beats/min lower than the heart rate at which 1-mm ST-segment depression was documented on the electrocardiogram. Patients were also trained to identify exertion levels using the Borg rate of perceived exertion scale, with intensity goals of 11 to 14 (fairly light to somewhat hard). Patients were asked to exercise for a minimum of 3 hours/

- 22 sitios en USA
- 757 hombres y 395 mujeres
- 108 con angina leve
- 174 con angina limitante
- A las 12 semanas, 74% de los pacientes estaban libres de angina
- Aumento capacidad de ejercicio, mejoró depresión y calidad de vida

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THE JOURNAL OF FAMILY PRACTICE

Caldwell B. Esselstyn Jr, MD; Gina Gendy, MD; Jonathan Doyle, MCS; Mladen Golubic, MD, PhD; Michael F. Roizen, MD
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The authors reported no potential conflict of interest relevant to this article.

ORIGINAL RESEARCH

A way to reverse CAD?

Though current medical and surgical treatments manage coronary artery disease, they do little to prevent or stop it. Nutritional intervention, as shown in our study and others, has halted and even reversed CAD.

ABSTRACT

Purpose ▶ Plant-based nutrition achieved coronary artery disease (CAD) arrest and reversal in a small study. However, there was skepticism that this approach could succeed in a larger group of patients. The purpose of our follow-up study was to define the degree of adherence and outcomes of 198 consecutive patient volunteers who received counseling to convert from a usual diet to plant-based nutrition.

Methods ▶ We followed 198 consecutive patients counseled in plant-based nutrition. These patients with established cardiovascular disease (CVD) were interested in transitioning to plant-based nutrition as an adjunct to usual cardiovascular care. We considered participants adherent if they eliminated dairy, fish, and meat, and added oil.

Results ▶ Of the 198 patients with CVD, 177 (89%) were adherent. Major cardiac events judged to be recurrent disease totaled one stroke in the adherent cardiovascular participants—a recurrent event rate of .6%, significantly less than reported by other studies of plant-based nutrition therapy. Thirteen of 21 (62%) nonadherent participants experienced adverse events.

Conclusion ▶ Most of the volunteer patients with CVD responded to intensive counseling, and those who sustained plant-based nutrition for a mean of 3.7 years experienced a low rate of subsequent cardiac events. This dietary approach to treatment deserves a wider test to see if adherence can be sustained in broader populations. Plant-based nutrition has the potential for a large effect on the CVD epidemic.

In a 1985 program initiated at the Cleveland Clinic, we examined whether plant-based nutrition could arrest or reverse advanced coronary artery disease (CAD) in 22 patients.[1] One patient with restricted myocardial blood flow documented by positron emission tomography (PET) showed reperfusion on a repeat scan just 3 weeks after starting our nutritional intervention (FIGURE 1).[2] Within 10 months of the start of treatment, another patient with severe right calf claudication and a quantifiably diminished pulse volume experienced total pain relief and exhibited a measurably increased pulse volume amplitude.[2] Thus encouraged, we followed the small cohort of patients (adding cholesterol-lowering drugs in 1987) and reported results after 5 and 12 years of follow-up.[1,3] Of the 22 patients, 17 were adherent to the protocol, and their disease progression halted. In 4 of the 12, we angiographically confirmed disease reversal,[4] which can be striking (FIGURE 2).[4]

The significance of these findings. CAD remains the number one killer of women and men in western civilization despite 40 years of aggressive drug and surgical interventions.[5] These approaches can be lifesaving in the midst of a heart attack. However, the

2014

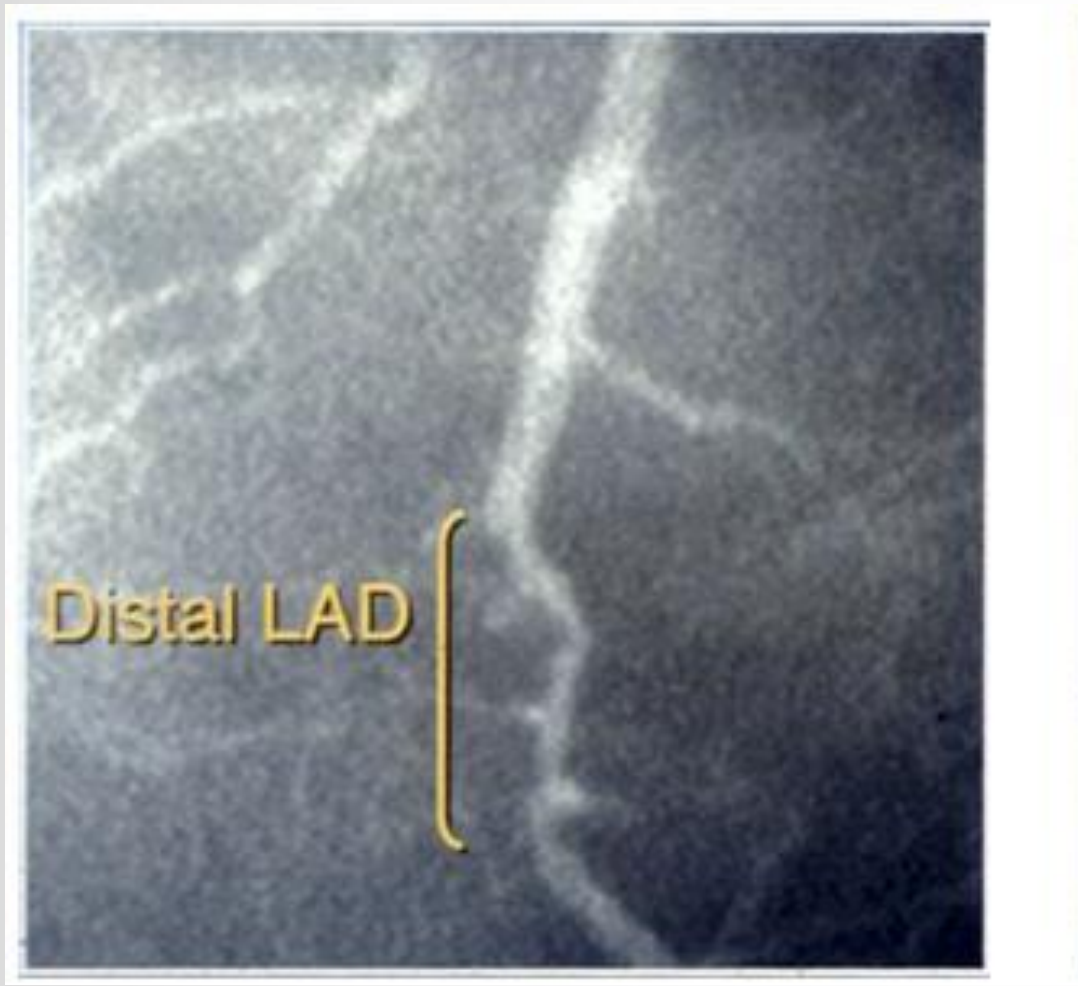
Dr. Esselstyn

198 pacientes: 3,7 años

- ✓ 177 se adhirieron a la alimentación (89%)
- ✓ 1 evento posterior (tasa de eventos del 0,6%)

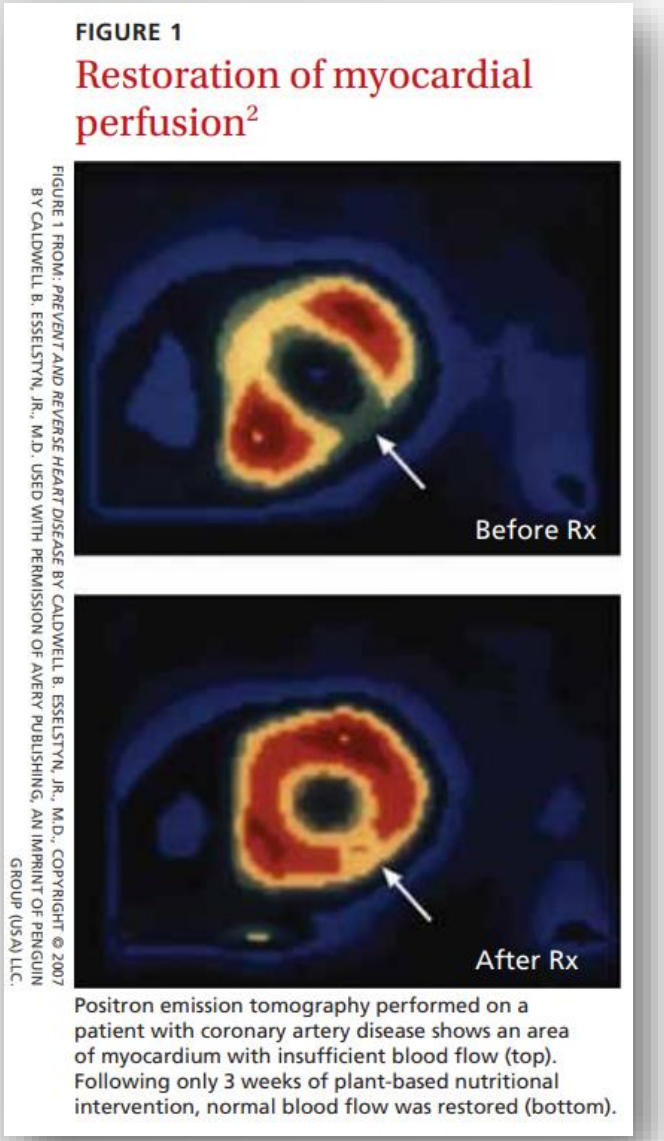
- × 21 no se adhirieron (11%)
- × 13 eventos posteriores (tasa de eventos del 62%)

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Esselstyn CB, Jr., Gendy G, Doyle J, Golubic M, Roizen M. A way to Reverse CAD? J Fam Pract 2014; 63:356-64b.

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3 semanas

2014

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198 pacientes: 3,7 años

- ✓ 177 se adhirieron a la alimentación (89%)
- ✓ 1 evento posterior (tasa de eventos del 0,6%)

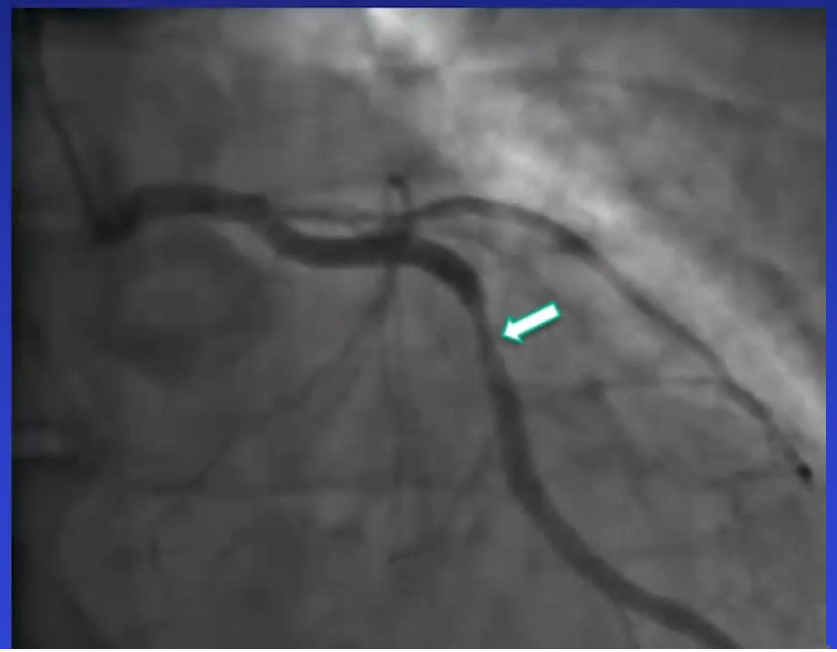
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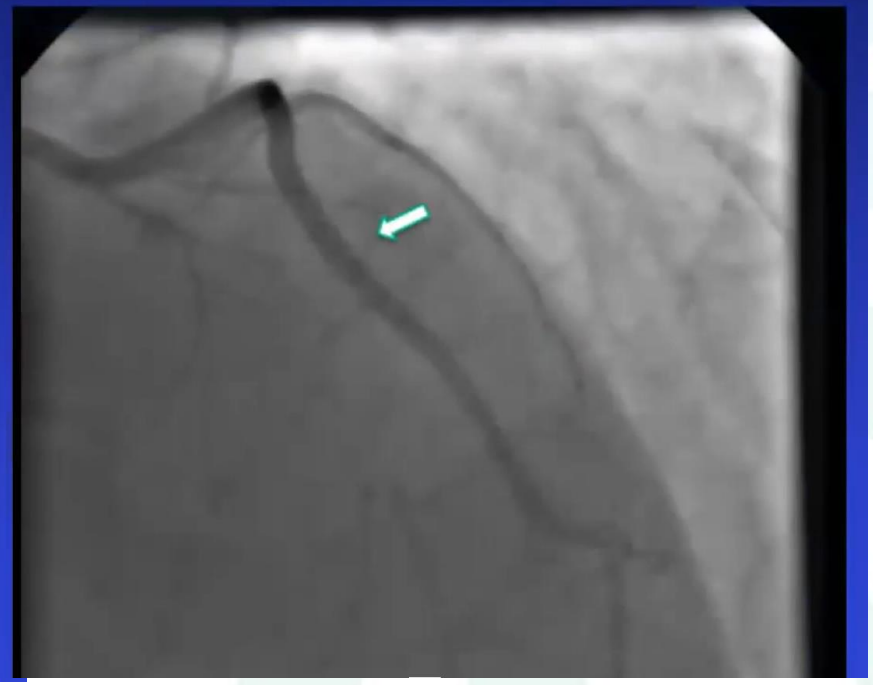
July 14, 2017



July 2, 2018

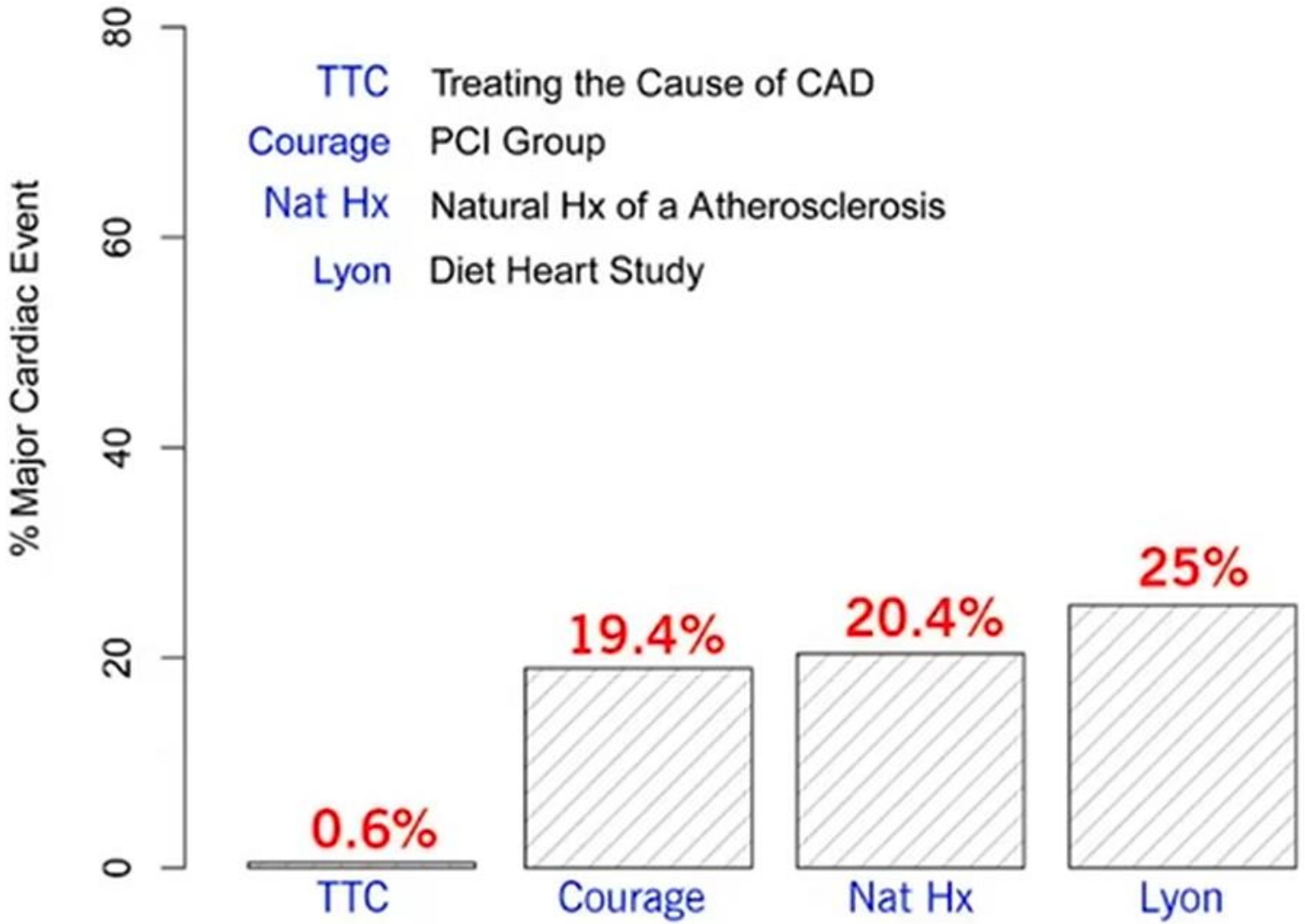


January 20, 2020



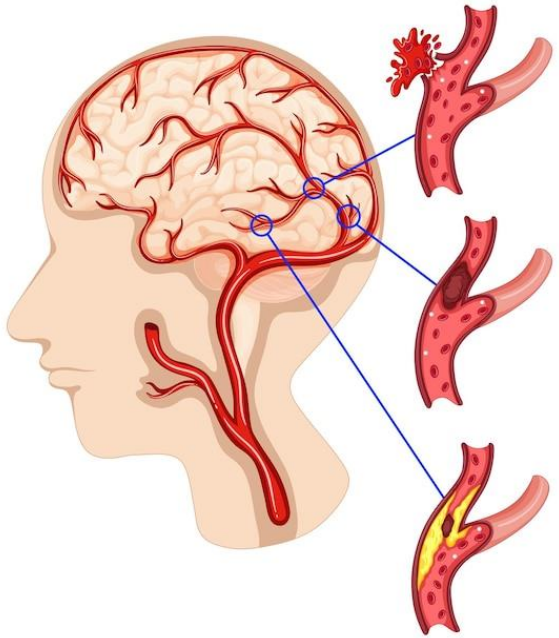
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Recurrent Major Cardiac Events Study Comparison



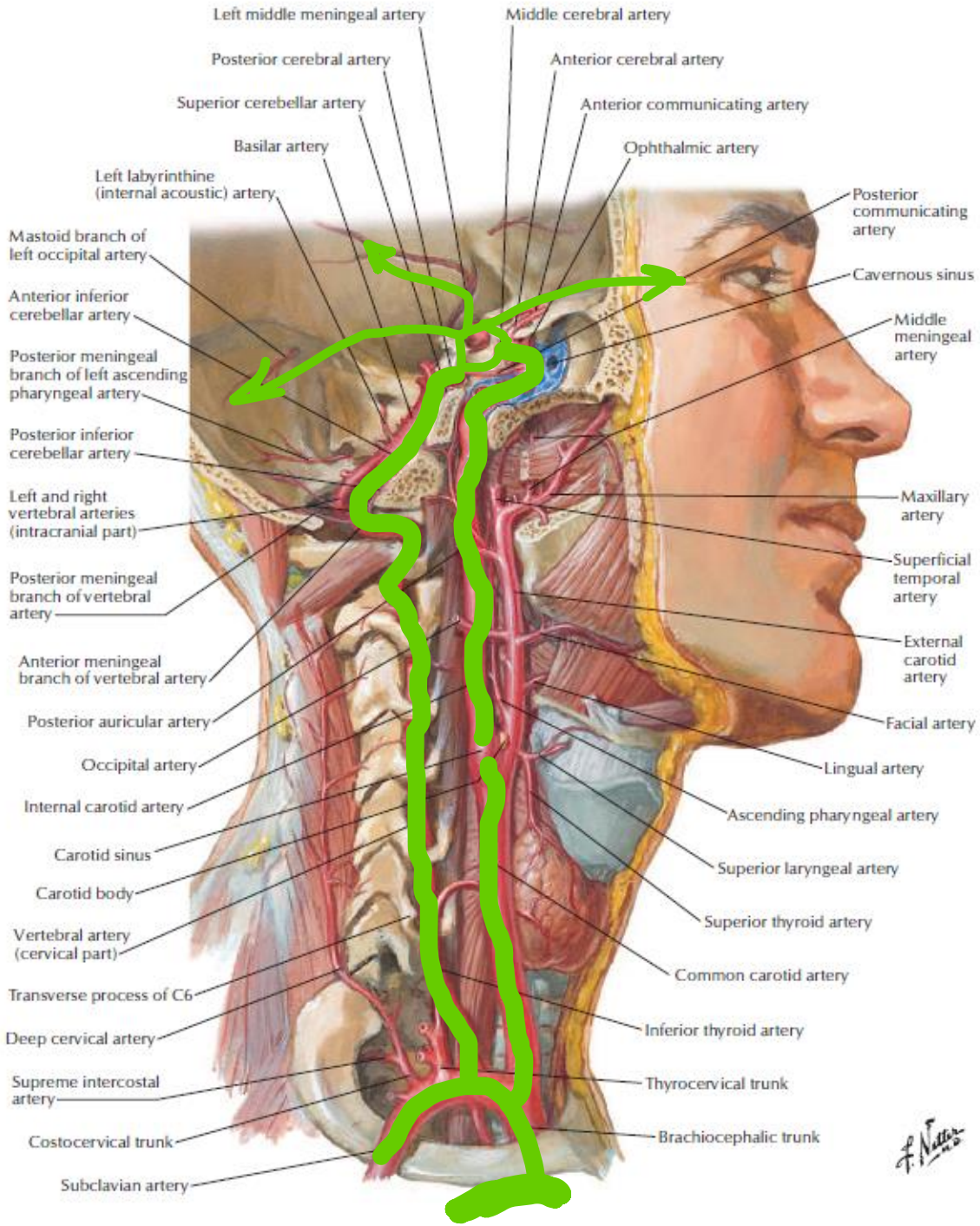
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Una mirada profunda

a las arterias de tu cerebro

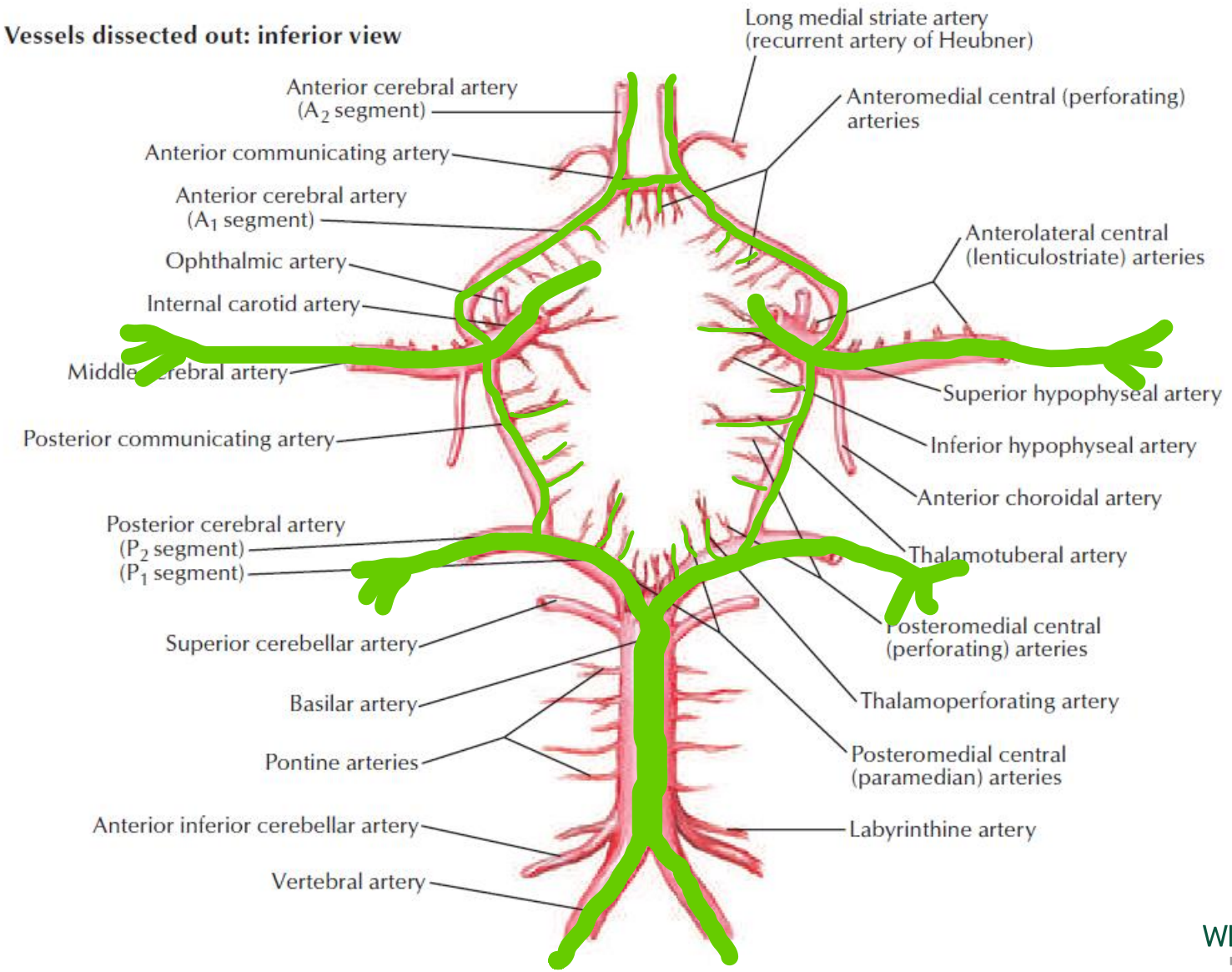


Las arterias que llevan la circulación a tu cerebro

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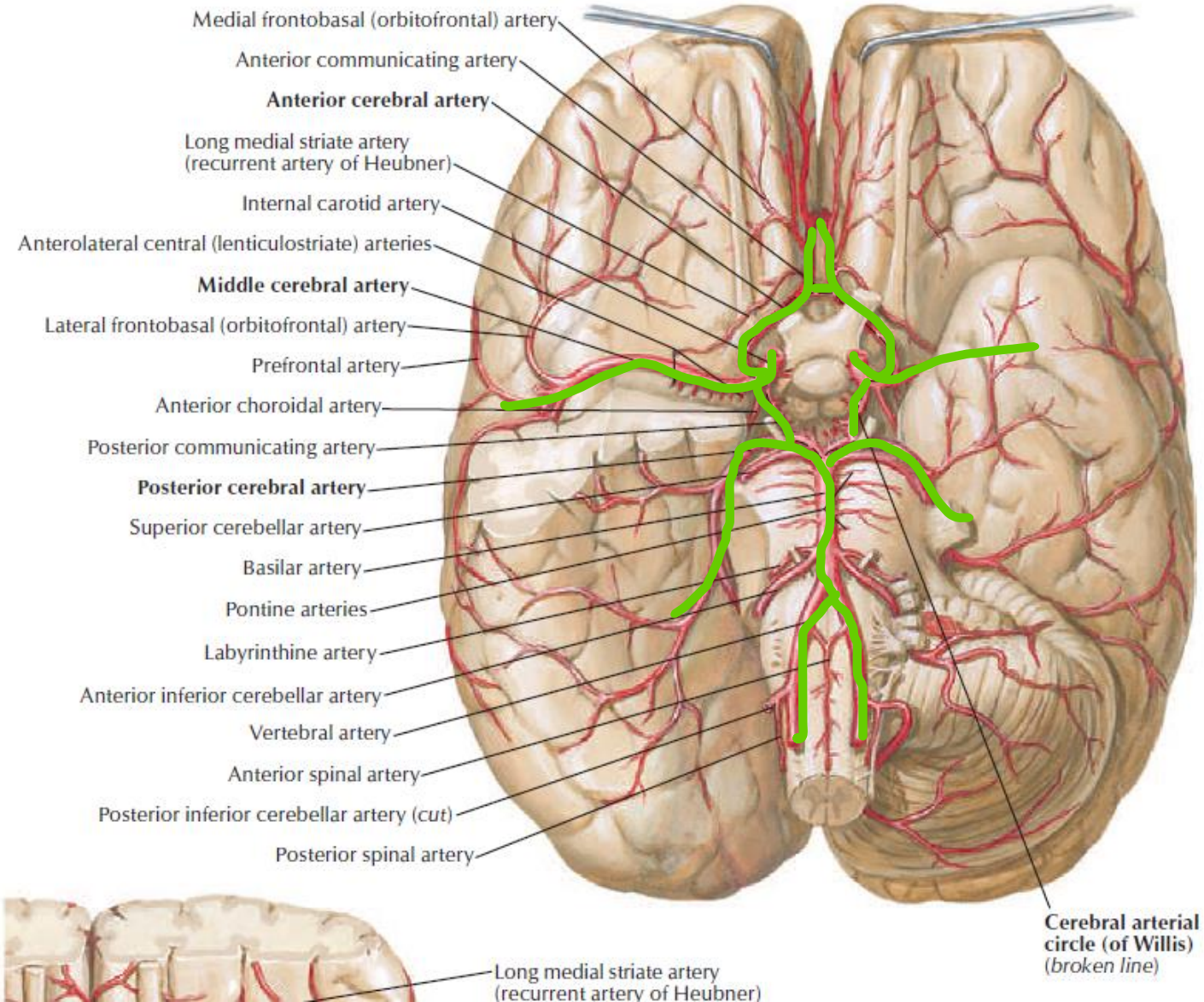
El circuito de arterias responsable de la circulación de tu cerebro

Cerebral Arterial Circle (of Willis)



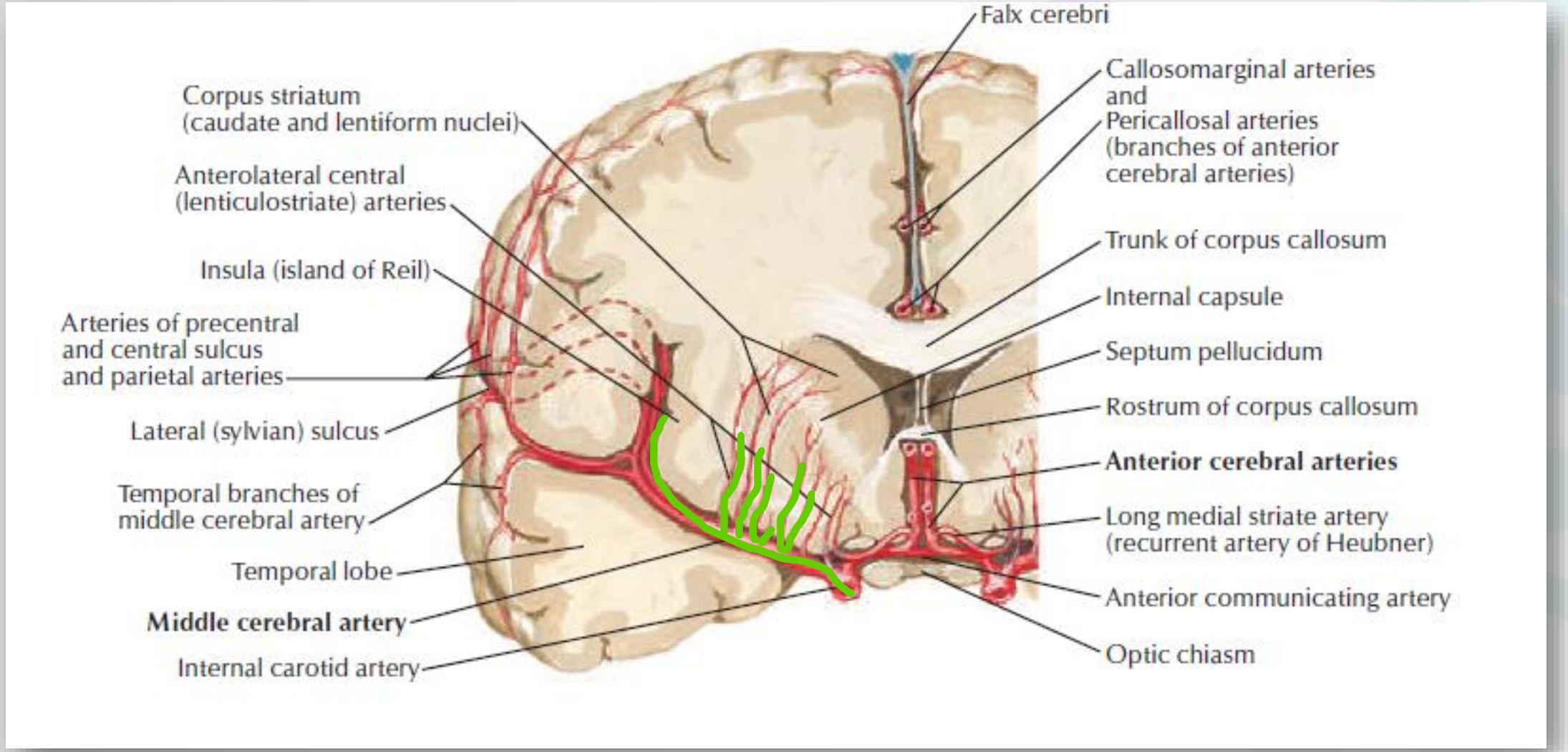
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See also Plate 148



El circuito de arterias responsable de la circulación de tu cerebro

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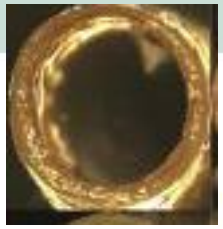


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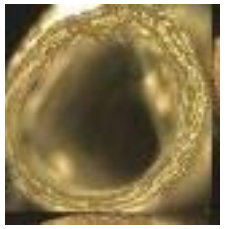


Así son las arterias SANAS del Polígono de Willis

Cerebral Arterial Circle (of Willis)



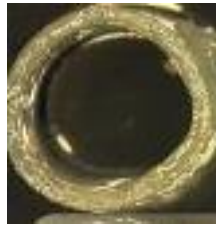
Vessels dissected out: inferior view

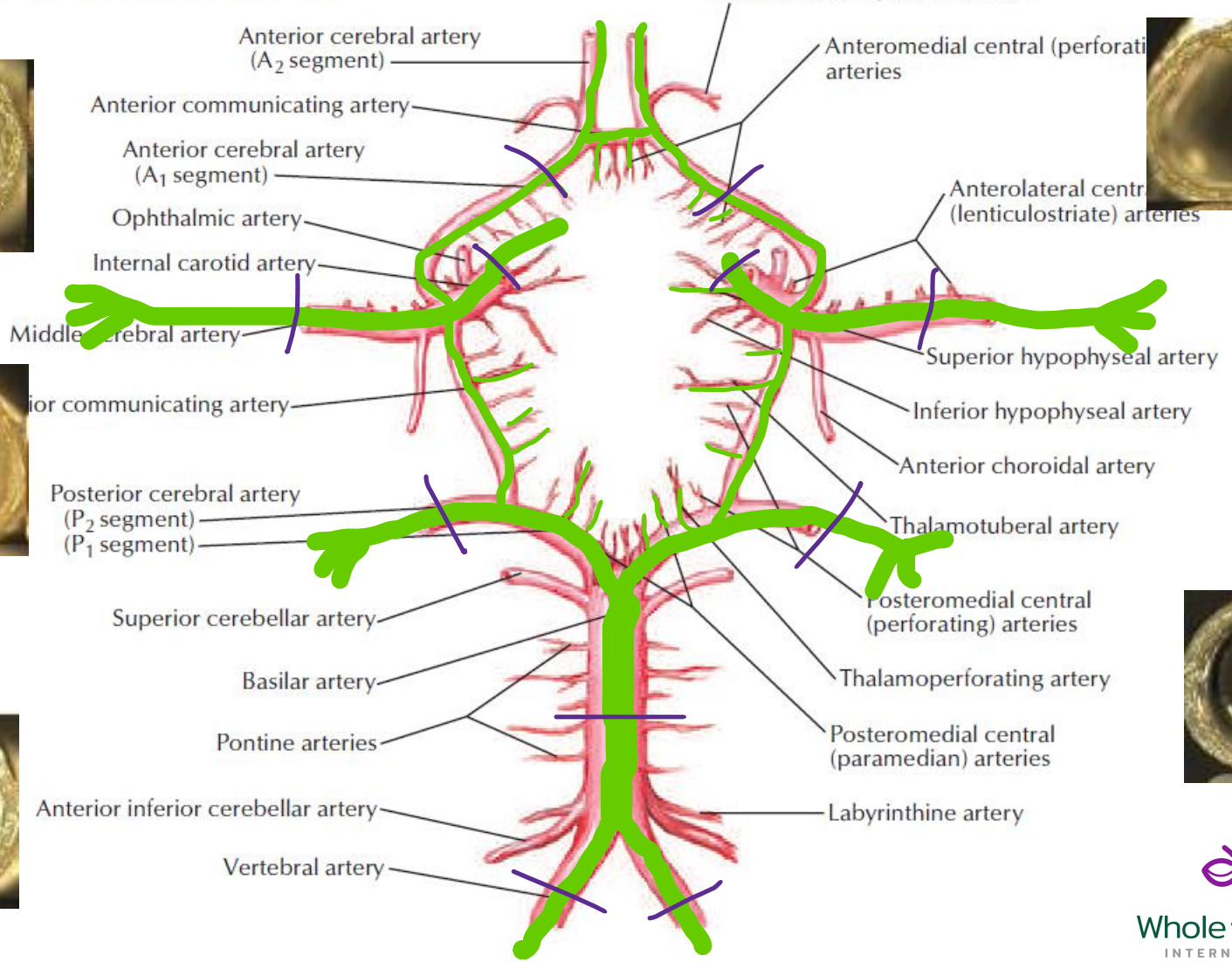












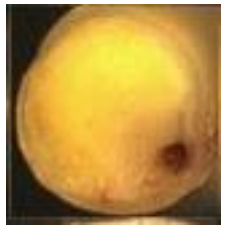
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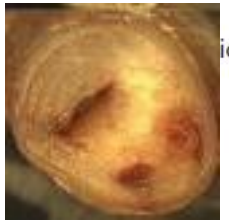


Así son las arterias enfermas del Polígono de Willis

Cerebral Arterial Circle (of Willis)

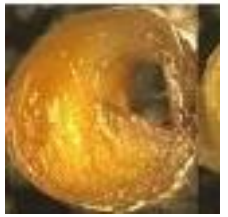




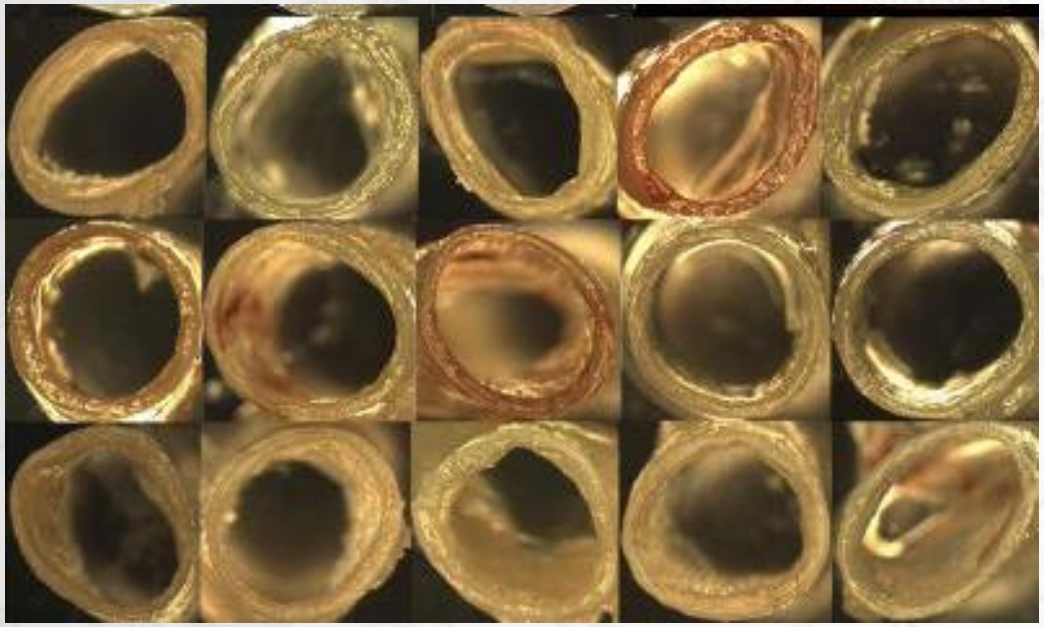








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Roher AE, Tyas SL, Maarouf CL, et al. Intracranial atherosclerosis as a contributing factor to Alzheimer's disease dementia. Alzheimers Dement. 2011;7(4):436-444.

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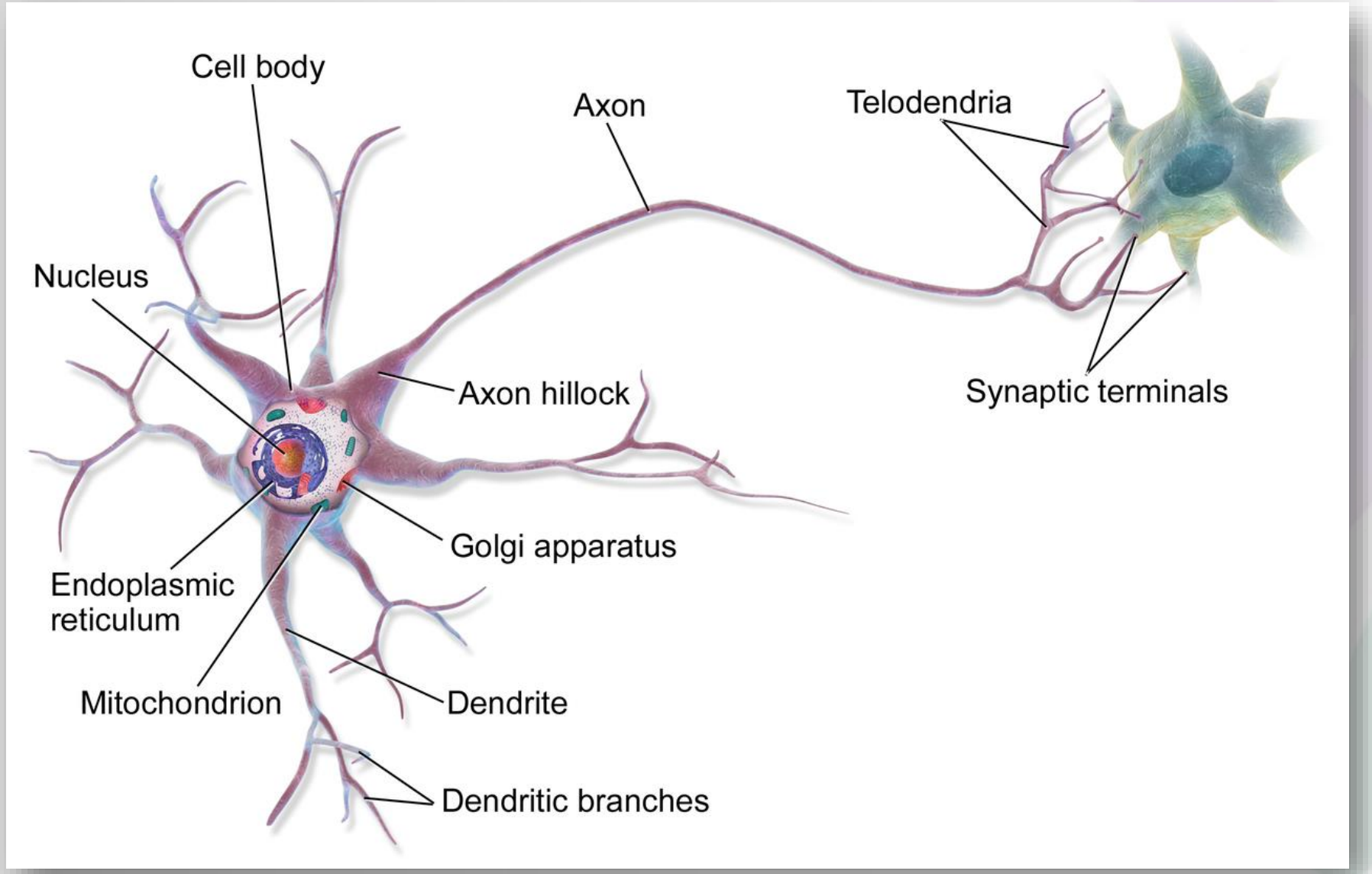
¿Cómo esta condición

causa deterioro cognitivo y demencia?

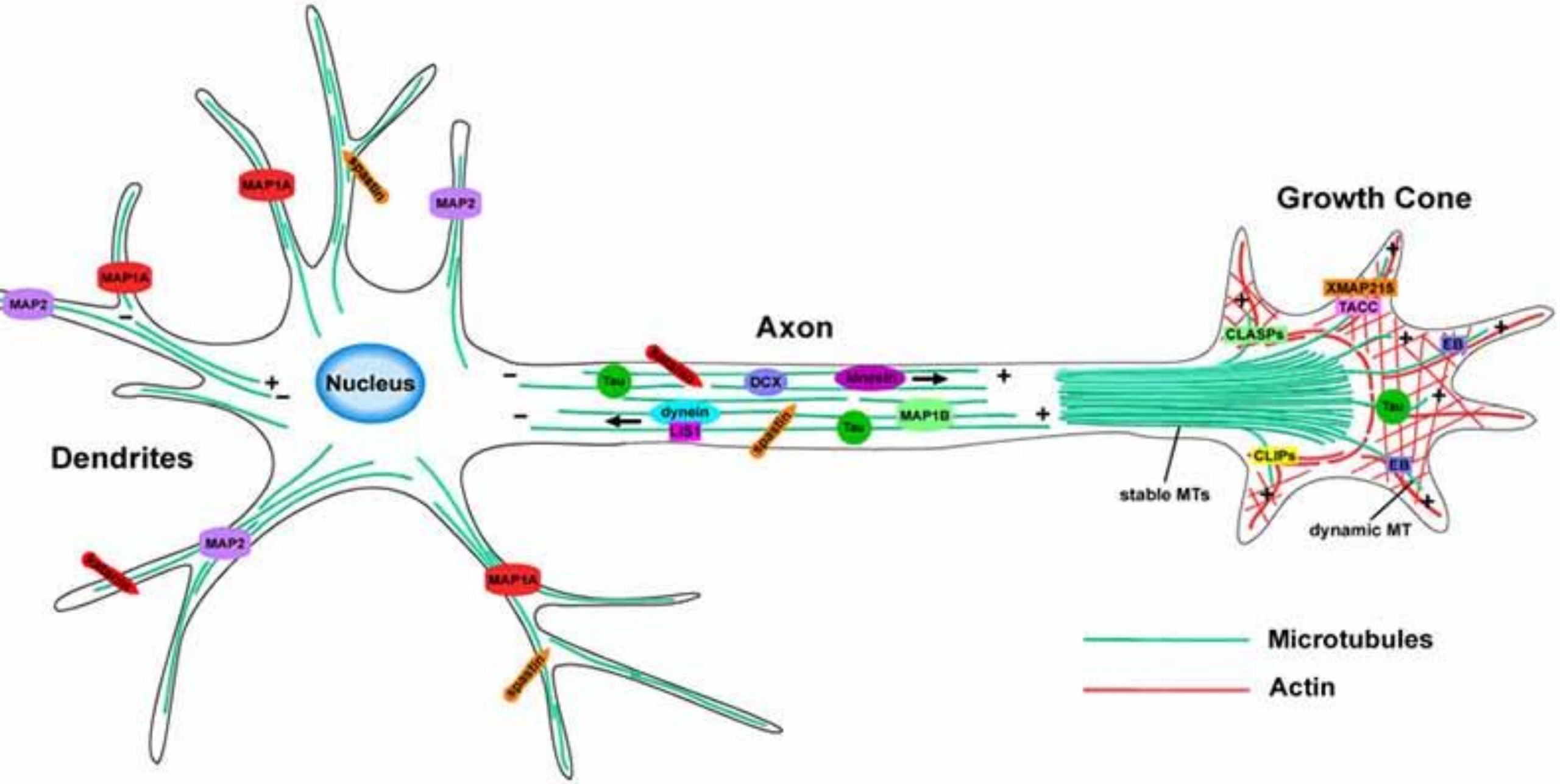


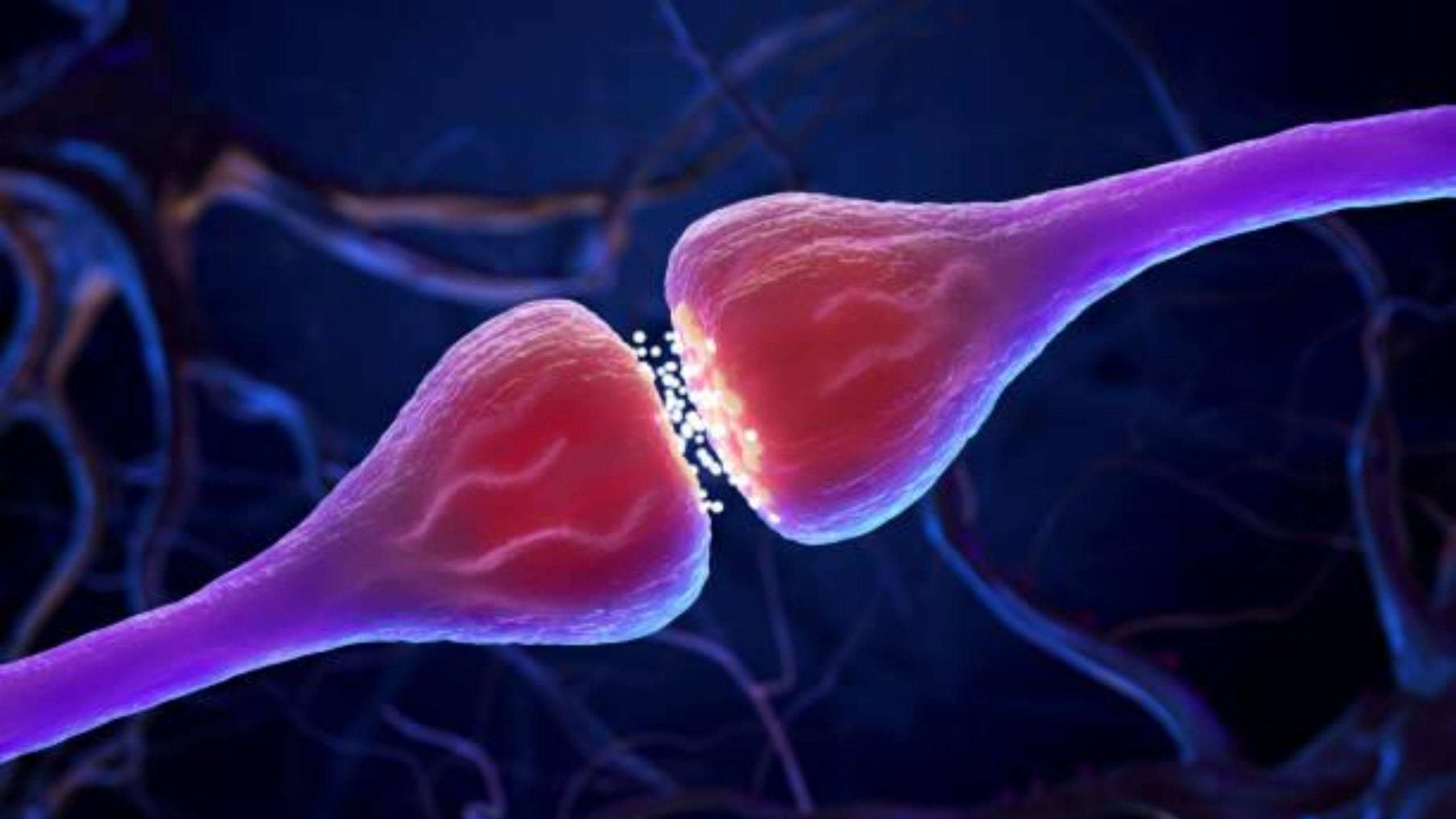


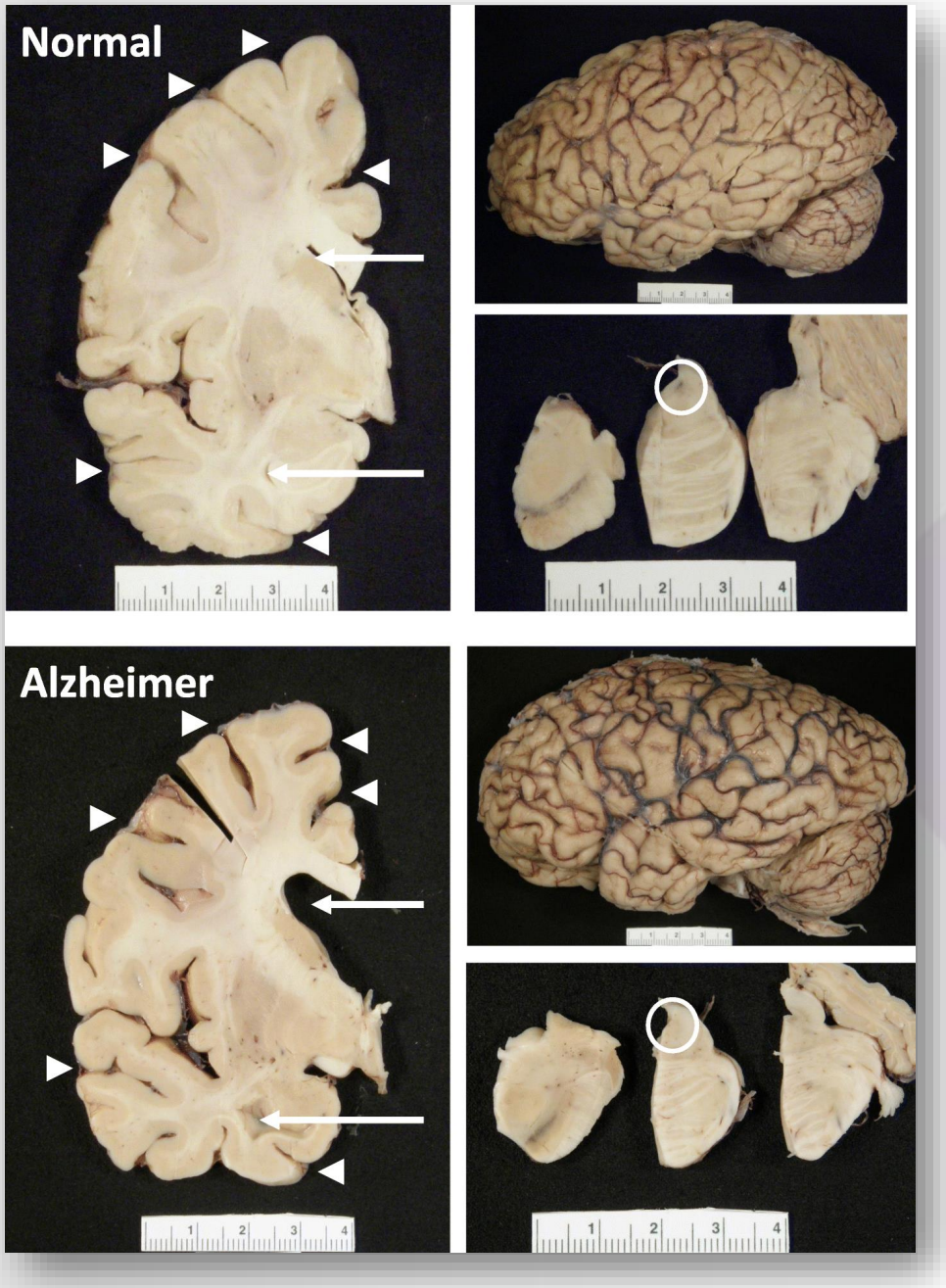
Neuronas



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Growth Cone
Axon
Dendrites
Nucleus
MAP1A
MAP2
MAP1A
MAP2
spectrin
Tau
DCX
kinesin
dynein
LIS1
spectrin
Tau
MAP1B
XMAP215
TACC
CLASPs
EB
Tau
CLIPs
EB
stable MTs
dynamic MT
MAP2
MAP1A
spectrin
Microtubules
Actin





¿QUÉ COMUNMENTE SE SABE DE LA EN ENFERMEDAD DE ALZHEIMER?

The neuropathological diagnosis of Alzheimer's disease. DeTure and Dickson Molecular Neurodegeneration (2019) 14:32

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Neuronas → Exceso de proteína beta-amiloide (Aβ)

¿Qué es la proteína beta-Amiloide (Aβ)?

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¿Qué es la proteína beta-Amiloide (Aβ)?

- ✓ Promoción de el crecimiento neuronal
- ✓ En niveles normales reduce la muerte neuronal
- ✓ Neuroprotectora
- ✓ Actividad antimicrobiana
- ✓ Bloquea filtraciones en la barrera hematoencefálica
- ✓ Aumenta la recuperación después de lesiones postraumáticas
- ✓ Probable supresora del cáncer al inhibir virus oncogenicos

J.E. MORLEY, S.A. FARR, A.D. NGUYEN, F. XU. WHAT IS THE PHYSIOLOGICAL FUNCTION OF AMYLOID-BETA PROTEIN? *J Nutr Health Aging. 2019;23(3):225-226*

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¿Qué es la proteína beta-Amiloide (Aβ)?

- ✓ Aumenta la plasticidad sináptica, memoria y aprendizaje en dosis normales
- ✓ En dosis altas altera la memoria
- ✓ Aumenta la producción de la acetilcolina y la actividad de sus receptores

- ✓ Al no ser producida: muerte neuronal
- ✓ **Su precursora: APP (Proteína Precursora de Amiloide)**

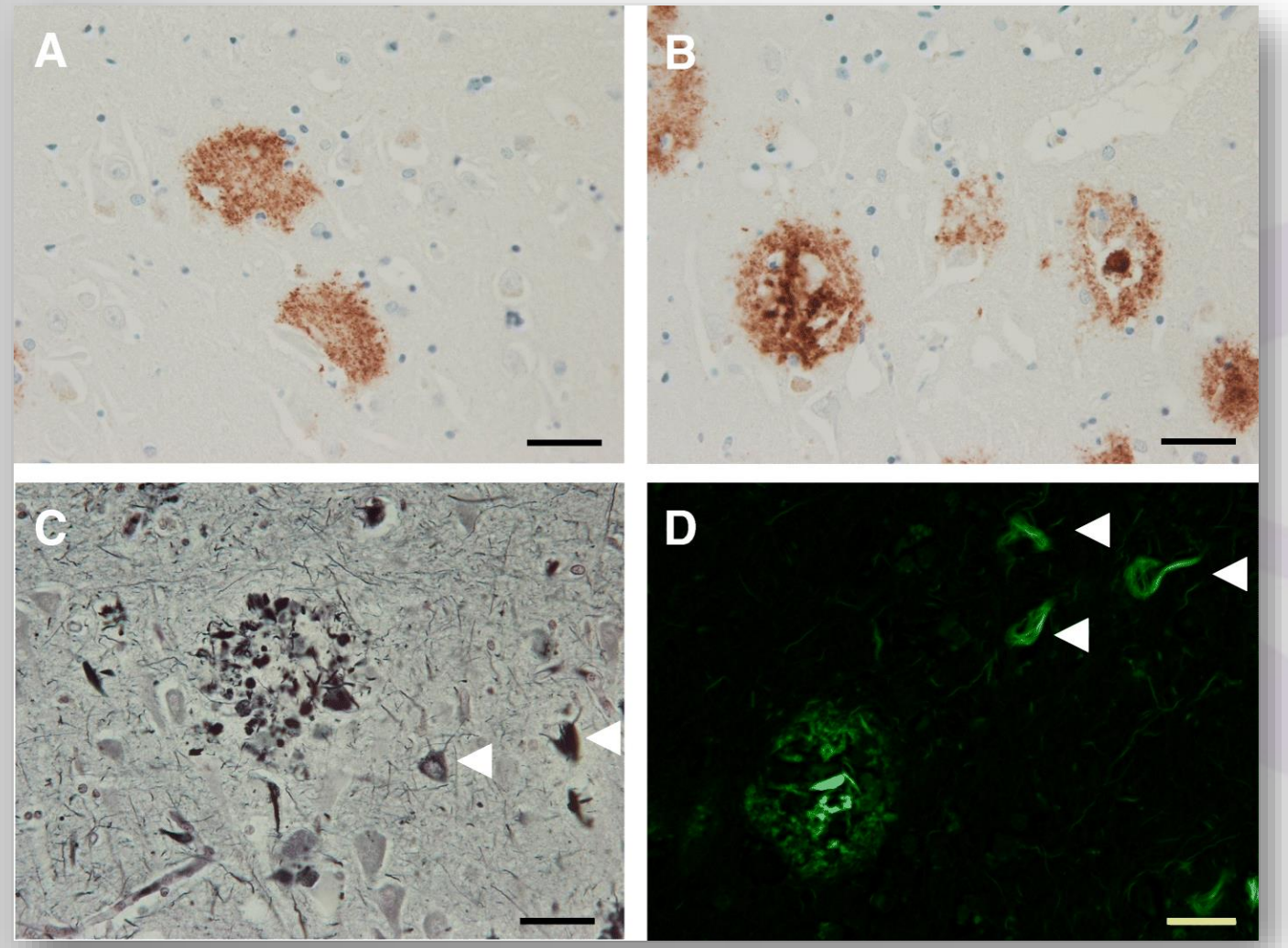
J.E. MORLEY, S.A. FARR, A.D. NGUYEN, F. XU. WHAT IS THE PHYSIOLOGICAL FUNCTION OF AMYLOID-BETA PROTEIN? *J Nutr Health Aging. 2019;23(3):225-226*

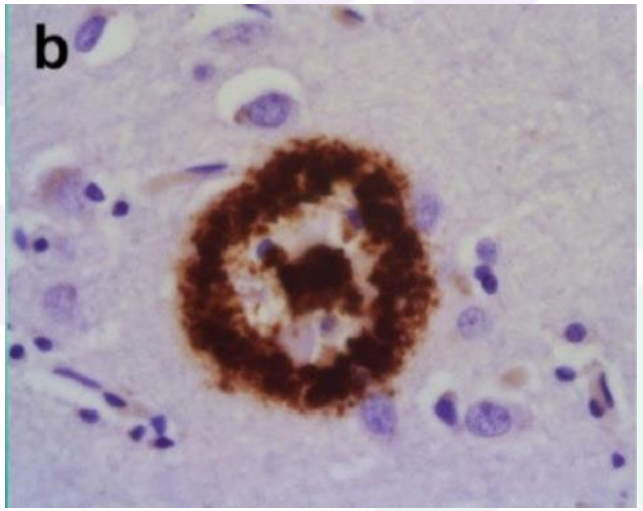
Whole to Heal INTERNATIONAL

Neuronas → **Exceso de proteína beta-amiloide (Aβ)** → **Placas beta-Amiloide (Aβ) Insolubles extracelulares**

Whole to Heal
INTERNATIONAL

Placas de péptido beta-Amiloide (Aβ)





The neuropathological diagnosis of Alzheimer's disease. DeTure and Dickson Molecular Neurodegeneration (2019) 14:32

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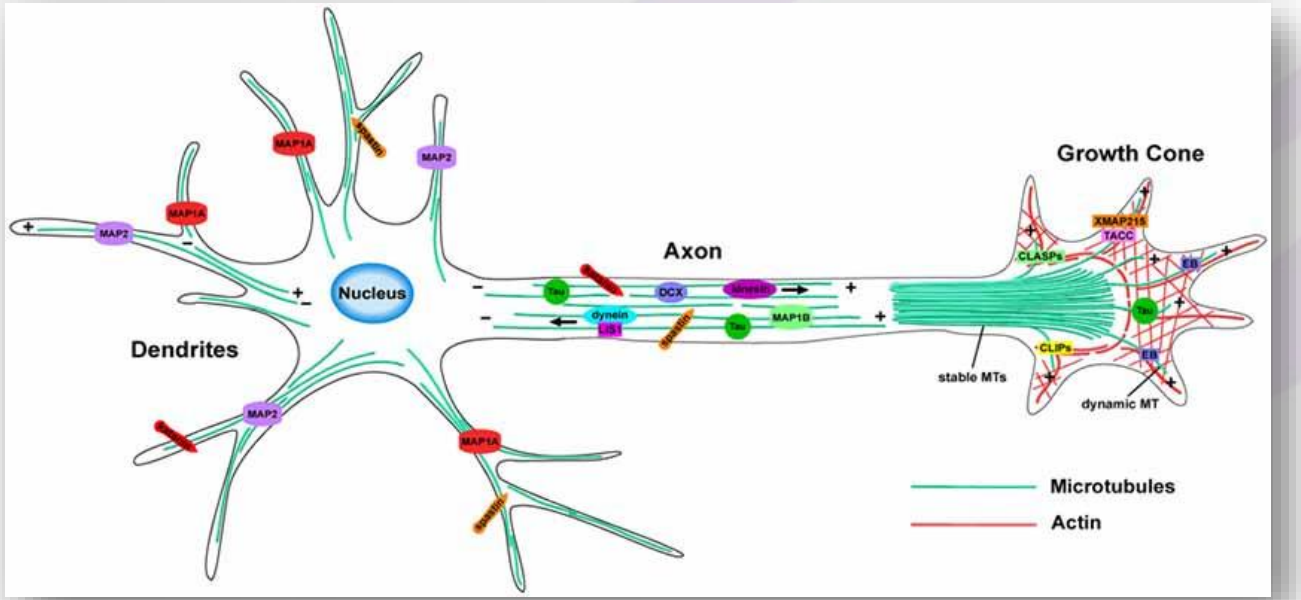
Neuronas → Exceso de proteína beta-amiloide (Aβ) → Placas beta-Amiloide (Aβ) Insolubles extracelulares → Disrupción neuronal y proceso degenerativo → Ovillos neurofibrilares
Hiperfosforilacion de proteína Tau

¿Qué es la proteína Tau?

Whole to Heal
INTERNATIONAL

¿Qué es la proteína Tau?

- ✓ Proteína asociada a microtúbulos primordialmente encontrada en neuronas

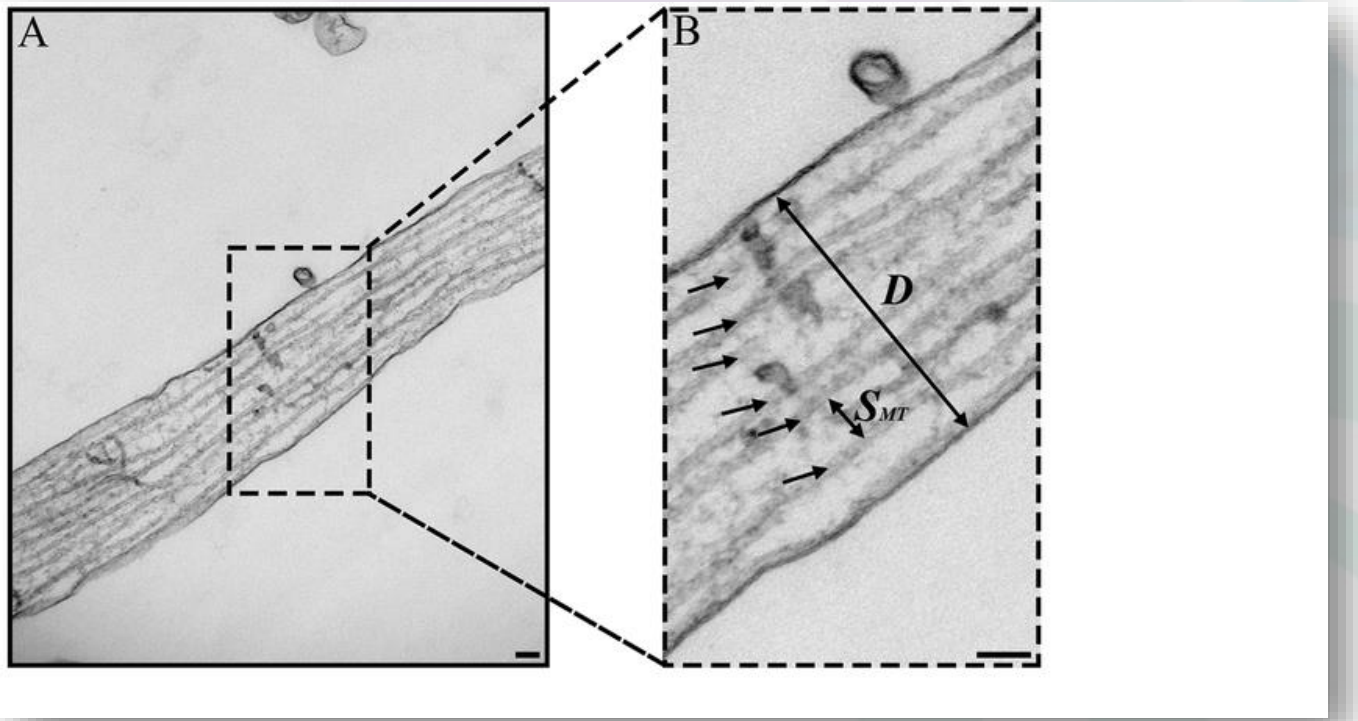


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¿Qué es la proteína Tau?

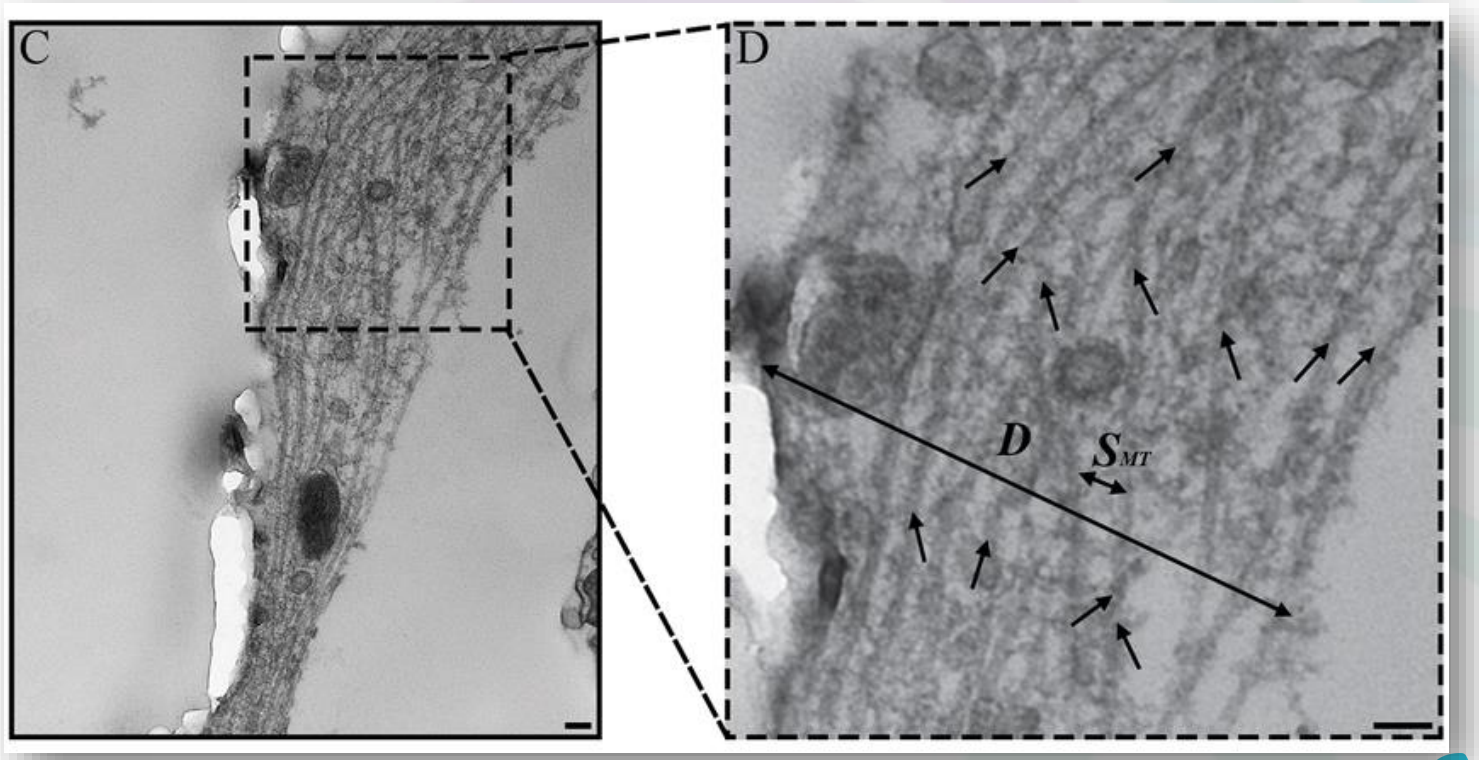
- ✓ Proteína asociada a microtúbulos primordialmente encontrada en neuronas
- ✓ Rol fundamental de estabilizar los microtúbulos: componentes esenciales del citoesqueleto
- ✓ Promueve ensamblaje y estabilidad para dar estructura a las neuronas
- ✓ Al estabilizar los microtúbulos, facilita transporte eficiente de organelos, vesículas y otros a través de los axones

J.E. MORLEY, S.A. FARR, A.D. NGUYEN, F. XU. WHAT IS THE PHYSIOLOGICAL FUNCTION OF AMYLOID-BETA PROTEIN? *J Nutr Health Aging. 2019;23(3):225-226*

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¿Qué pasa con Tau en el Alzheimer?

- ✓ Proteína Tau se hiperfosforila
- ✓ No puede adherirse a los microtúbulos. Estos no se ensamblan y se desestabilizan



J.E. MORLEY, S.A. FARR, A.D. NGUYEN, F. XU. WHAT IS THE PHYSIOLOGICAL FUNCTION OF AMYLOID-BETA PROTEIN? *J Nutr Health Aging.* *2019;23(3):225-226*

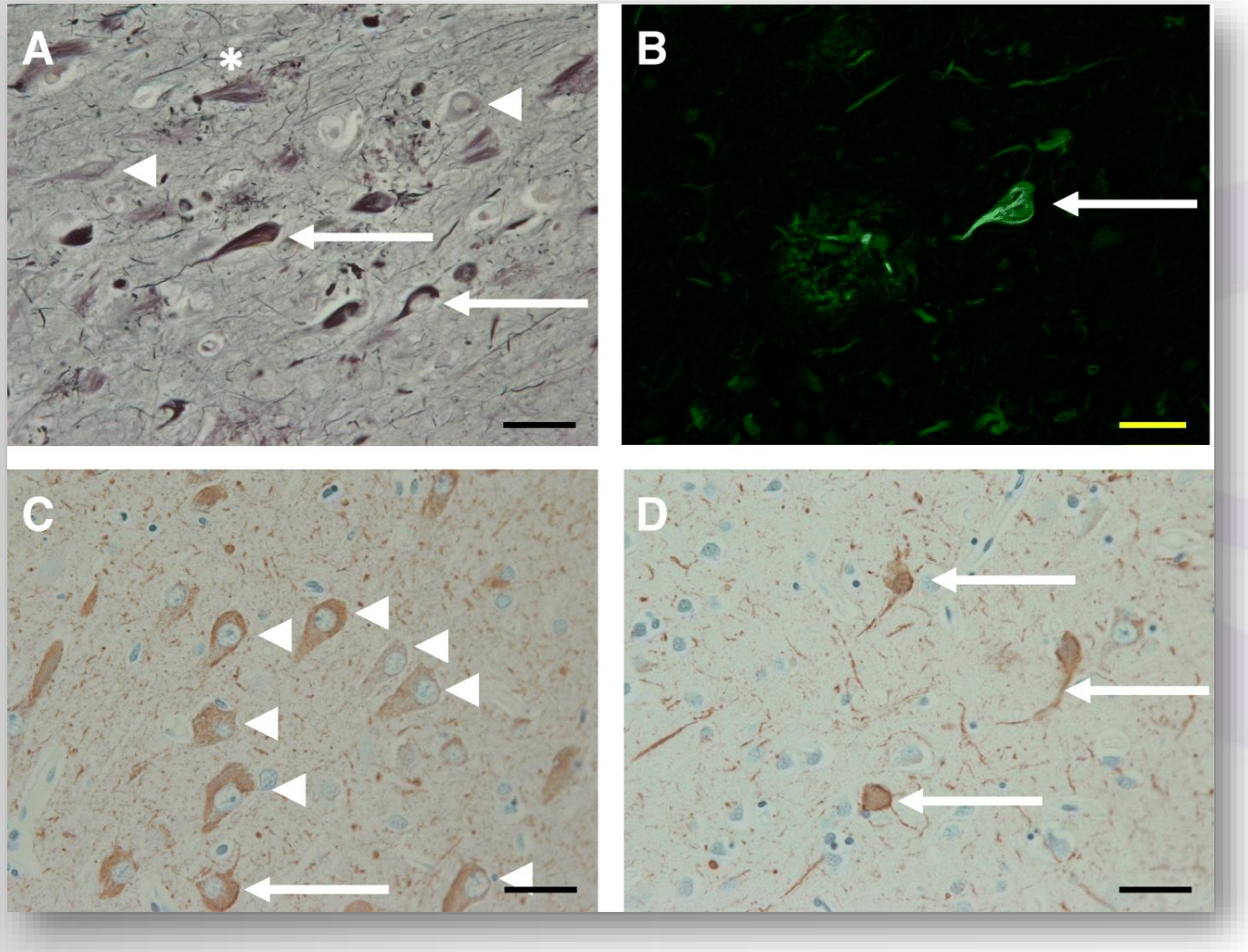
Whole to Heal INTERNATIONAL

¿Qué pasa con Tau en el Alzheimer?

- ✓ Proteína Tau se hiperfosforila
- ✓ No puede adherirse a los microtúbulos. Estos no se ensamblan y se desestabilizan
- ✓ Tau hiperfosforilada se agrega en filamentos formando ovillos neurofibrilares
- ✓ Signo típico de Enfermedad de Alzheimer
- ✓ Afecta gravemente transporte en las Axonas
- ✓ Lleva a disfunción sináptica, perdida de conexión neuronal y muerte neuronal

J.E. MORLEY, S.A. FARR, A.D. NGUYEN, F. XU. WHAT IS THE PHYSIOLOGICAL FUNCTION OF AMYLOID-BETA PROTEIN? *J Nutr Health Aging. 2019;23(3):225-226*

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Ovillos neurofibrilares – Proteína Tau

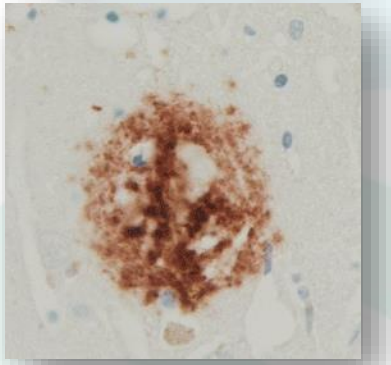
The neuropathological diagnosis of Alzheimer's disease. DeTure and Dickson Molecular Neurodegeneration (2019) 14:32

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Neuronas

Exceso de proteína beta-amiloide (Aβ)

Placas beta-Amiloide (Aβ) Insolubles extracelulares



Disfunción sináptica

Deterioro cognitivo y pérdida de memoria

Disrupción neuronal y proceso degenerativo

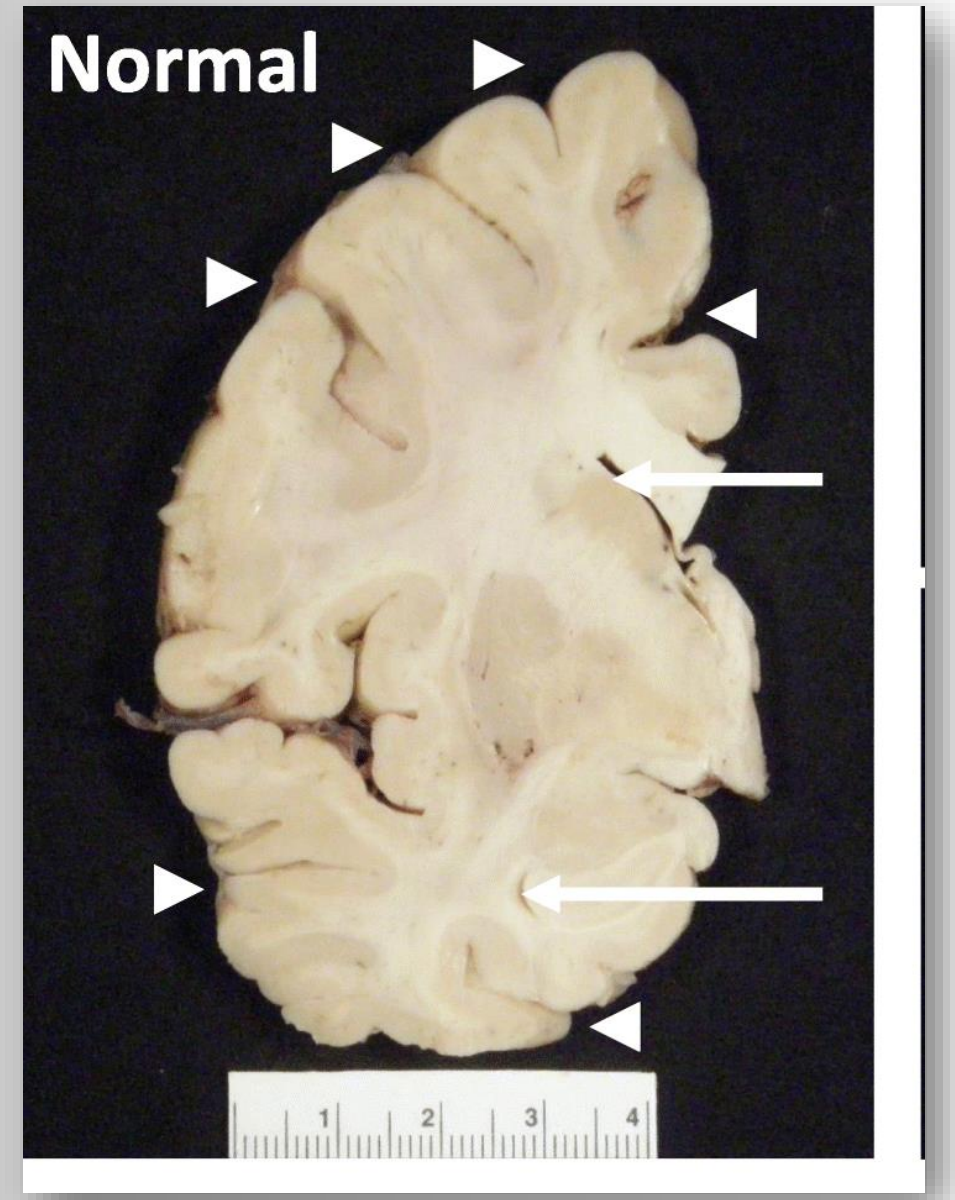
Ovillos neurofibrilares

Hiperfosforilacion de proteína Tau

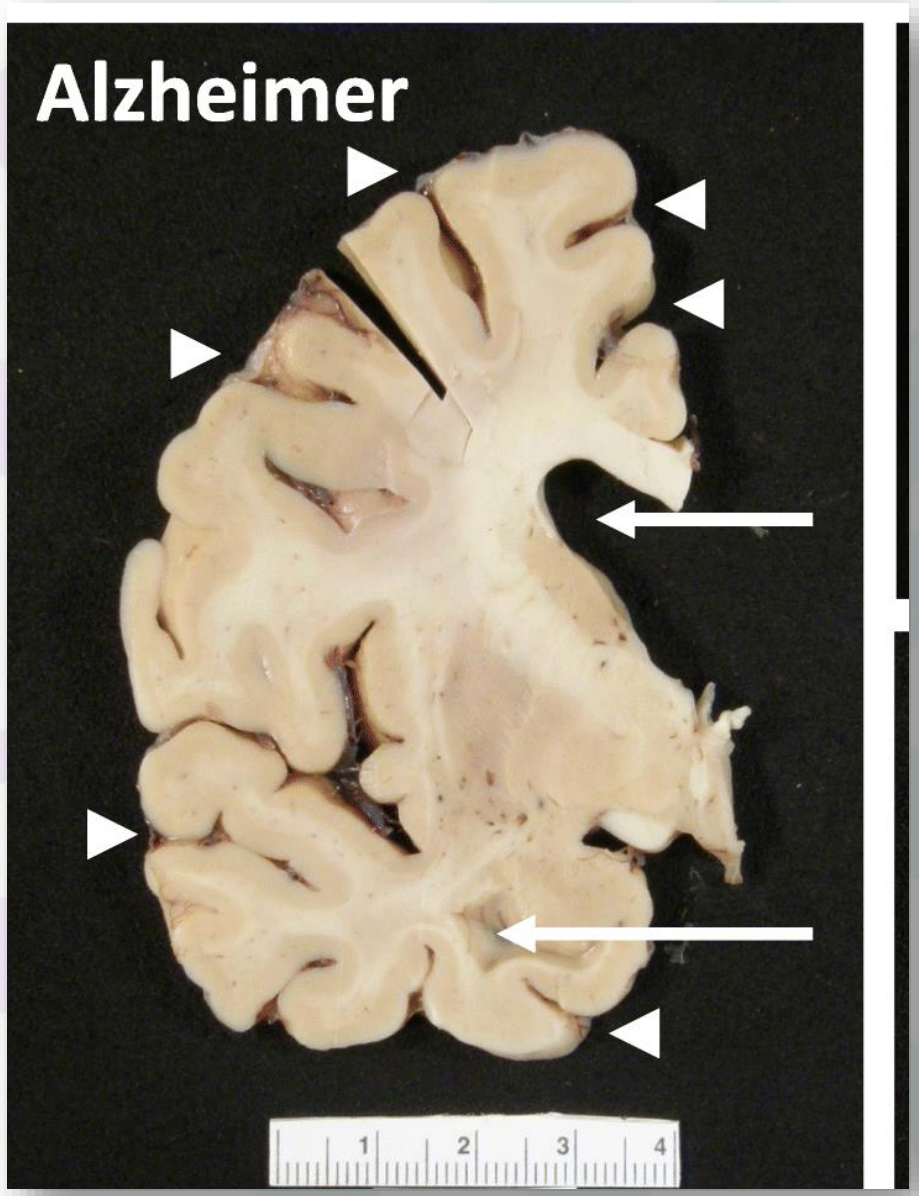
Muerte neuronal

Atrofia cerebral

Whole to Heal INTERNATIONAL



ATROFIA CEREBRAL



The neuropathological diagnosis of Alzheimer's disease. DeTure and Dickson Molecular Neurodegeneration (2019) 14:32

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Neuronas

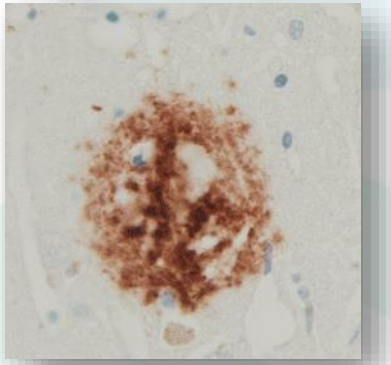
Exceso de proteína beta-amiloide (Aβ)

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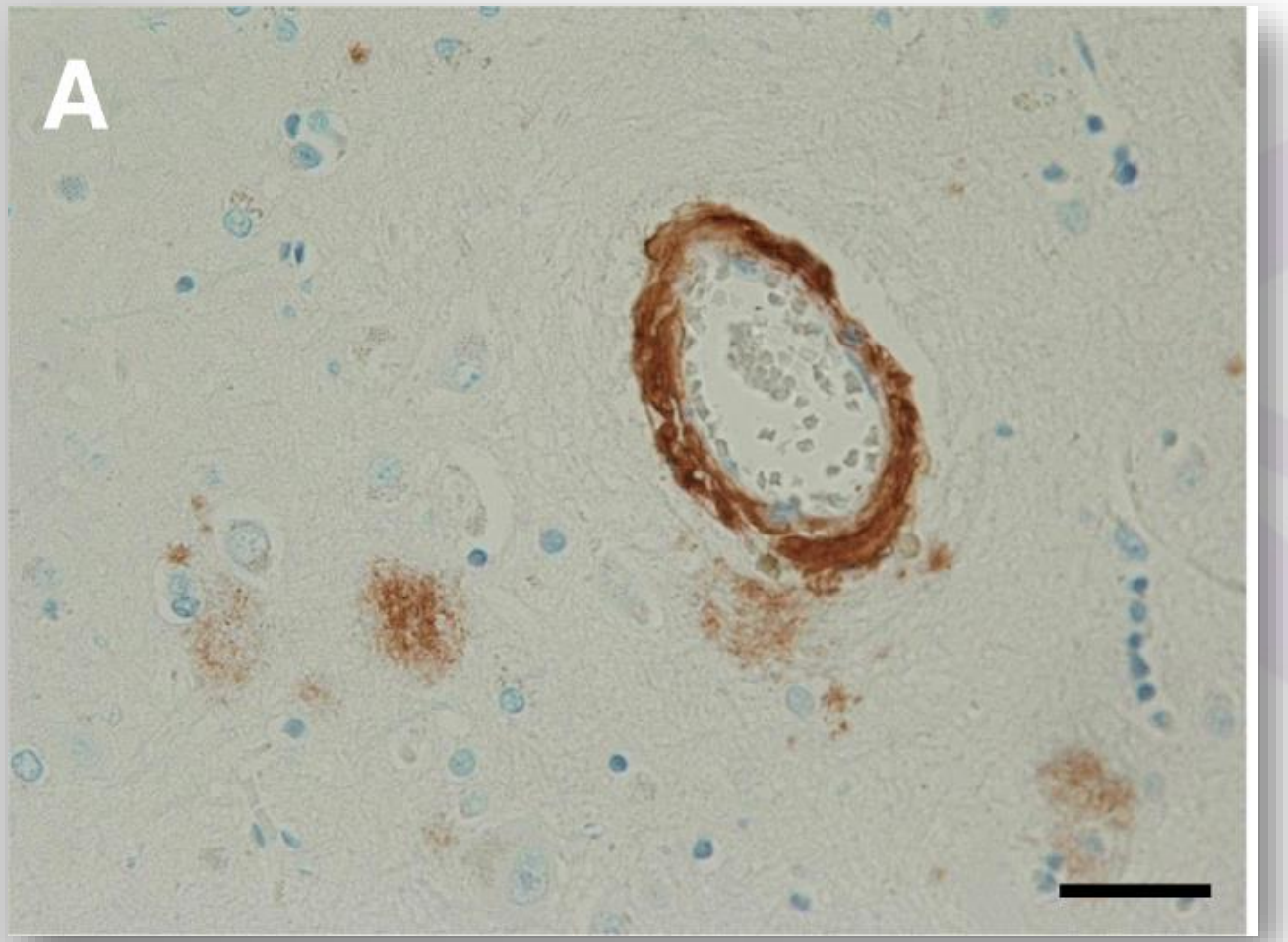
Deterioro cognitivo y pérdida de memoria

Atrofia cerebral

Muerte por Alzheimer

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INTERNATIONAL

Otros hallazgos…



Angiopatía Amiloide Cerebral

The neuropathological diagnosis of Alzheimer's disease. DeTure and Dickson Molecular Neurodegeneration (2019) 14:32

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Otros hallazgos...

Neuroinflamación crónica

Estrés oxidativo

Factores de riesgo reconocidos:

- ✓ Hipertensión, diabetes, obesidad e hipercolesterolemia.
- ✓ Inactividad física
- ✓ Tabaquismo
- ✓ Baja participación cognitiva y social
- ✓ Factores genéticos:
  - ✓ Enfermedad de Alzheimer familiar
  - ✓ Gen de la Apolipoproteina E (APOE)

Wolozin B, Brown J III, Theisler C, Silberman S. The cellular biochemistry of cholesterol and statins: insights into the pathophysiology and therapy of Alzheimer's disease. CNS Drug Rev. 2004; 10:127–46.

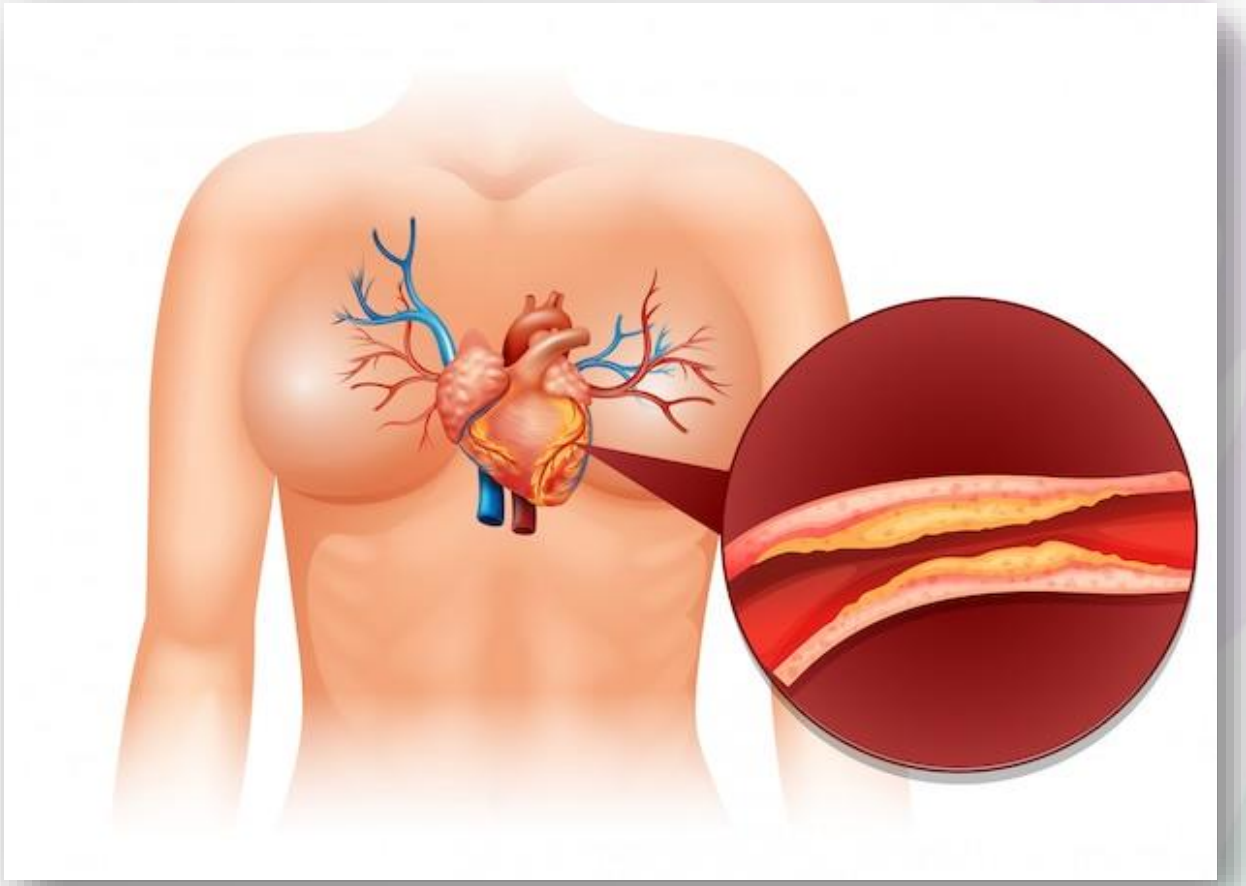
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¿Qué muestra la **evidencia científica más reciente** sobre la **Enfermedad de Alzheimer?**

COLESTEROL ALTO



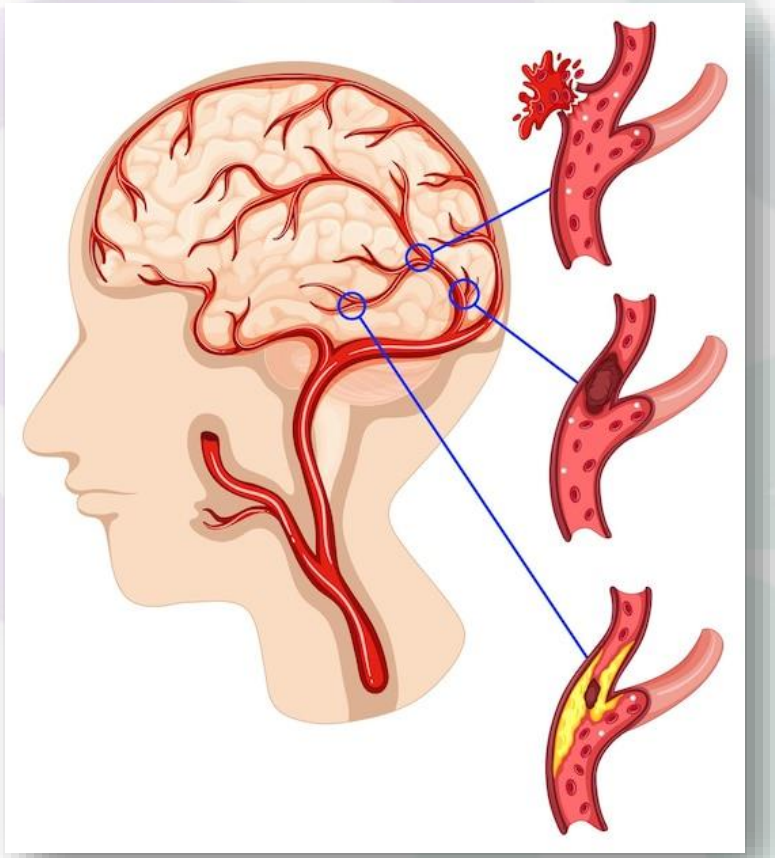
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COLESTEROL ALTO

Personas con colesterol alto en la mitad de la vida tienen más riesgo de sufrir de Enf. de Alzheimer

La producción de la proteína precursora de amiloide (APP) y el péptido beta-Amiloide (Aβ) es regulada por el colesterol



Roher AE, Tyas SL, Maarouf CL, et al. Intracranial atherosclerosis as a contributing factor to Alzheimer's disease dementia. Alzheimers Dement. 2011;7(4):436-444.

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HIPOPERFUSIÓN



Cualquier condición que impida la circulación, incluyendo la alteración de función cardiovascular relacionado con los hábitos alimenticios y estilo de vida del paciente y la consecuente y creciente incapacidad para adaptarse y repararse, es perjudicial para el cerebro.

Roher AE, Tyas SL, Maarouf CL, et al. Intracranial atherosclerosis as a contributing factor to Alzheimer's disease dementia. Alzheimers Dement. 2011;7(4):436-444.

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HIPOPERFUSIÓN

- Disminuye disponibilidad de nutrientes
- Eliminación ineficaz de CO2 y metabolitos nocivos
- Induce a atrofia cortical por lenta inanición del parénquima cerebral

Los infartos cerebrales esta presentes en 40% de pacientes con Enfermedad de Alzheimer



Roher AE, Tyas SL, Maarouf CL, et al. Intracranial atherosclerosis as a contributing factor to Alzheimer's disease dementia. Alzheimers Dement. 2011;7(4):436-444.

¿Siempre existe?

Alrededor del 50% de los pacientes con enfermedad de Alzheimer tienen Enfermedad Vascular Arterial



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Roher AE, Tyas SL, Maarouf CL, et al. Intracranial atherosclerosis as a contributing factor to Alzheimer's disease dementia. Alzheimers Dement. 2011;7(4):436-444.

¿Siempre existe?

- Estudio en Japón con 300 autopsias de hombres
- La frecuencia de demencia aumentó más del doble cuando existían lesiones cerebrovasculares



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INTERNATIONAL

Petrovitch H, Ross GW, Steinhorn SC, Abbott RD, Markesbery W, Davis D, et al. AD lesions and infarcts in demented and non-demented Japanese-American men. Ann Neurol. 2005; 57:98–103.

¿Siempre existe?



- Estudio con 180 sacerdotes
- Casi todos con patología de Enfermedad de Alzheimer
- 35% tenia infartos cerebrales

D A Bennett 1, J A Schneider, J L Bienias, D A Evans, R S Wilson Mild cognitive impairment is related to Alzheimer disease pathology and cerebral infarctions. Neurology 2005 Mar 8;64(5):834-41

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- Arterias del Polígono de Willis de 61 pacientes con Enfermedad de Alzheimer
- 2,108 cortes transversales fueron estudiados
- Índice de oclusión o estenosis fue calculado para todas las arterias

Roher AE, Tyas SL, Maarouf CL, et al. Intracranial atherosclerosis as a contributing factor to Alzheimer's disease dementia. Alzheimers Dement. 2011;7(4):436-444.

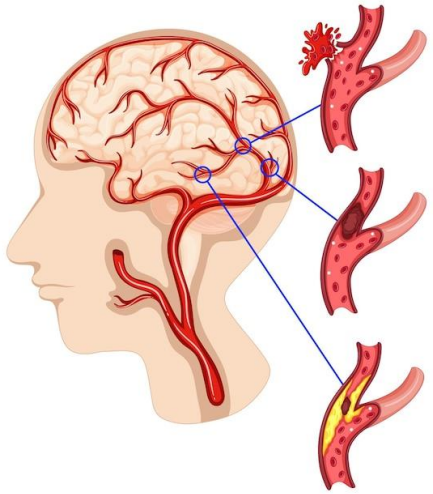


- Niveles de estenosis promedio en todo el estudio variaron entre un 33% hasta 84%
- Hubo un 300% de incremento en probabilidad de sufrir de Alzheimer entre los niveles más bajos y más altos de estenosis

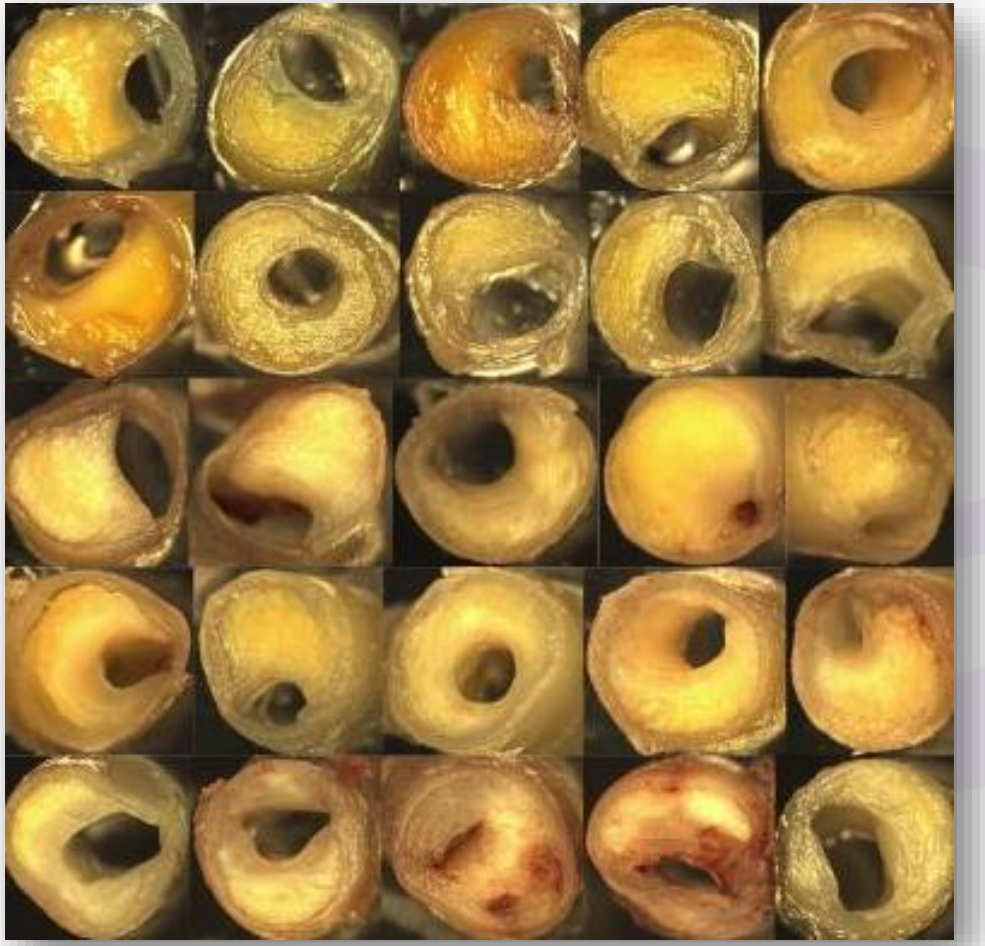
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Teoría vascular de la **Enfermedad de Alzheimer**



Colesterol, grasas saturadas y exceso de proteína → **Arterioesclerosis**

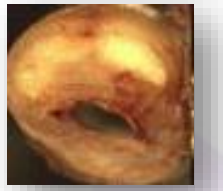


Cullen KM, Kocsi Z, Stone J. Microvascular pathology in the aging human brain: evidence that senile plaques are sites of microhaemorrhages. Neurobiol Aging. 2006; 27:1786–96.

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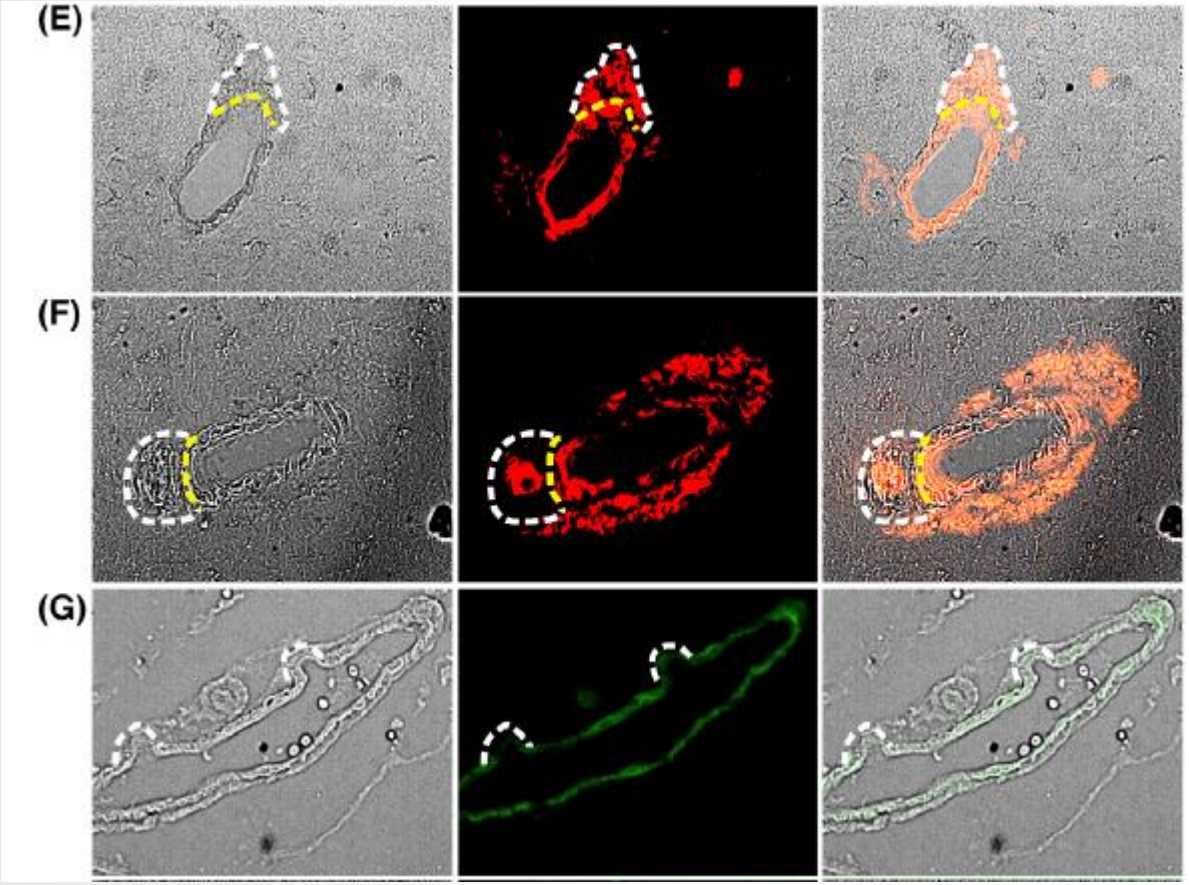
Colesterol, grasas saturadas y exceso de proteína

Arterioesclerosis



Daño del endotelio

microaneurismas



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Colesterol, grasas saturadas y exceso de proteína

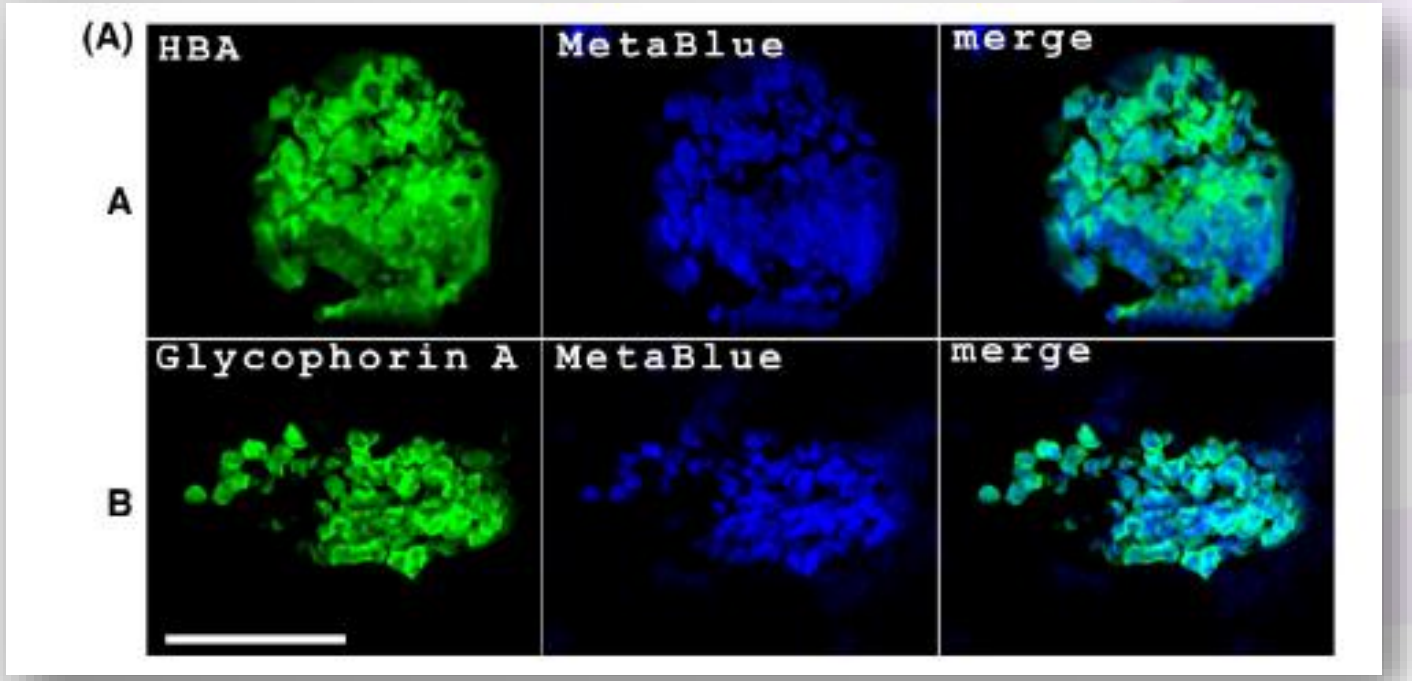
Arterioesclerosis



Daño del endotelio

microaneurismas

microsangrados



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Colesterol, grasas saturadas y exceso de proteína

Arterioesclerosis

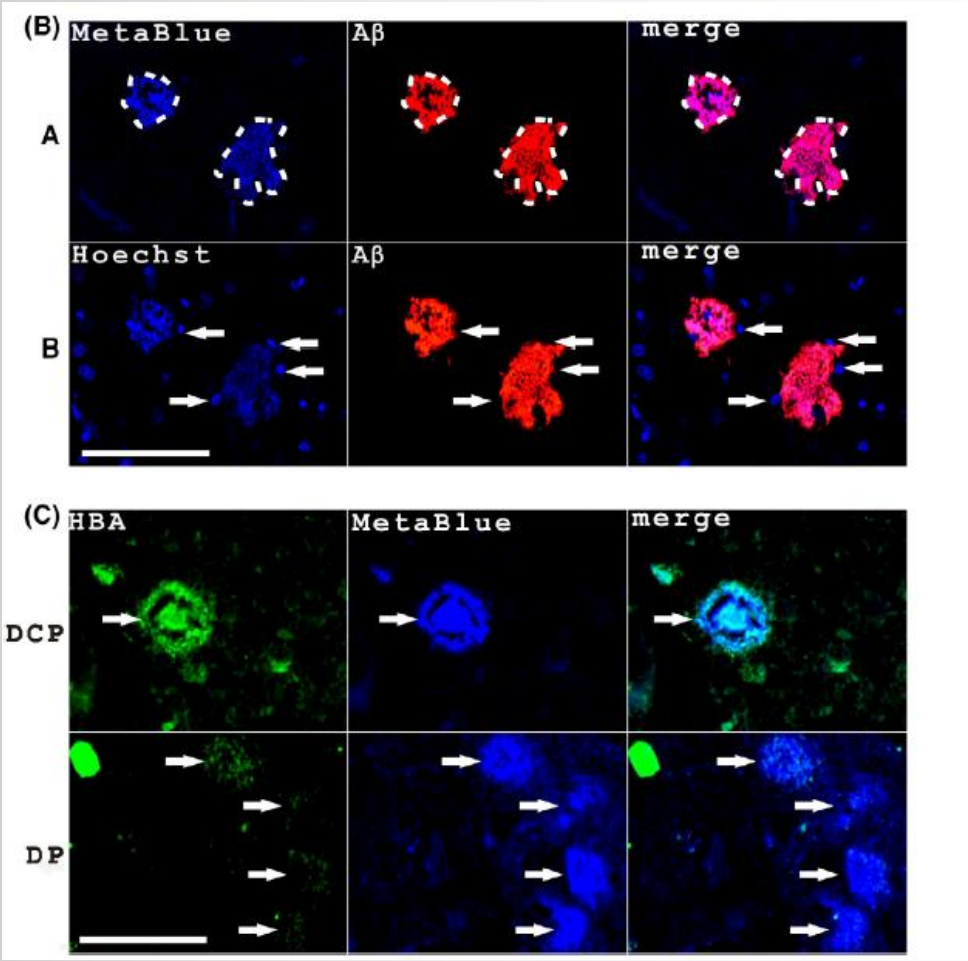


Daño del endotelio

microaneurismas

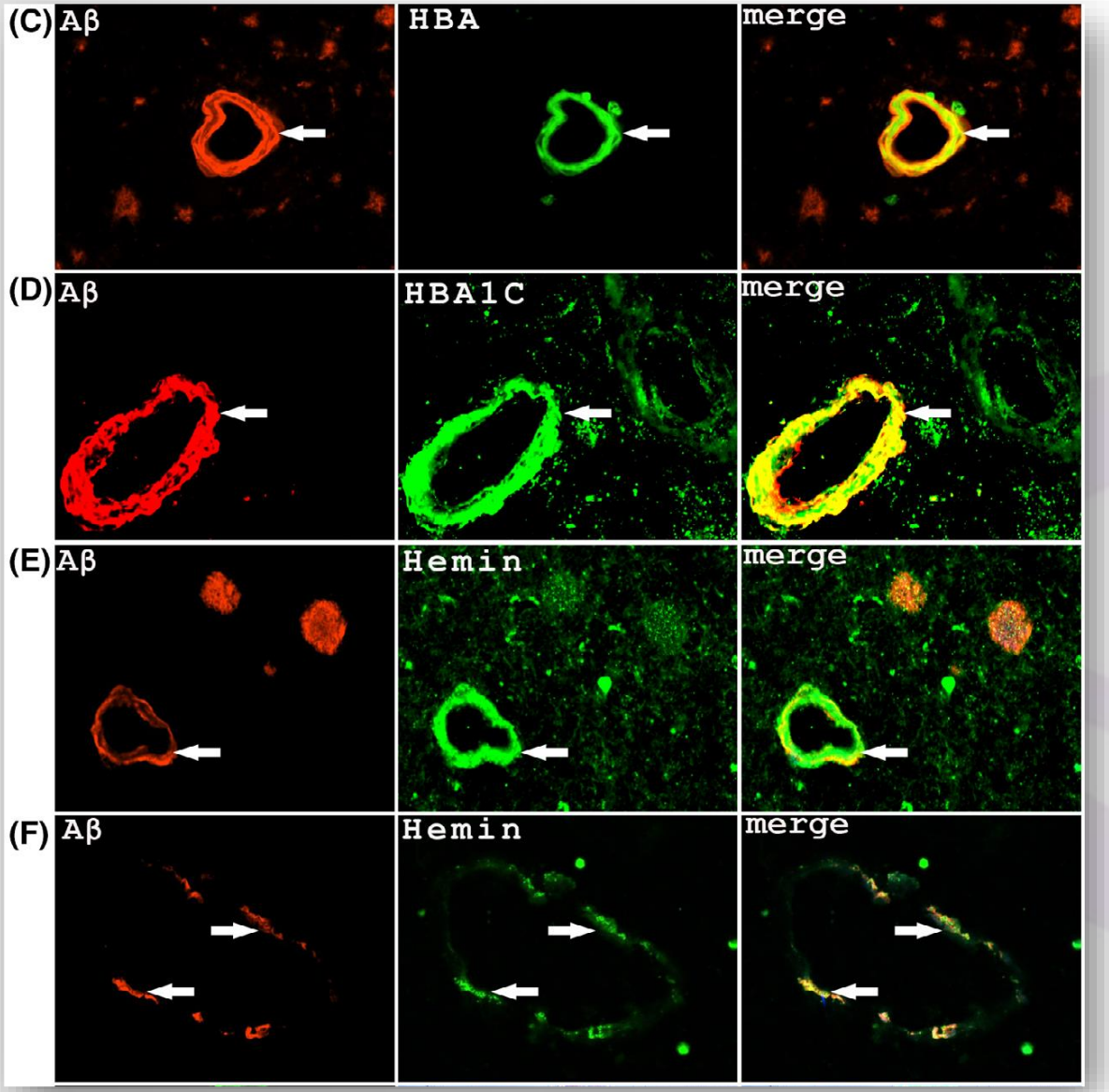
microsangrados

Beta-Amiloide (Aβ) al rescate



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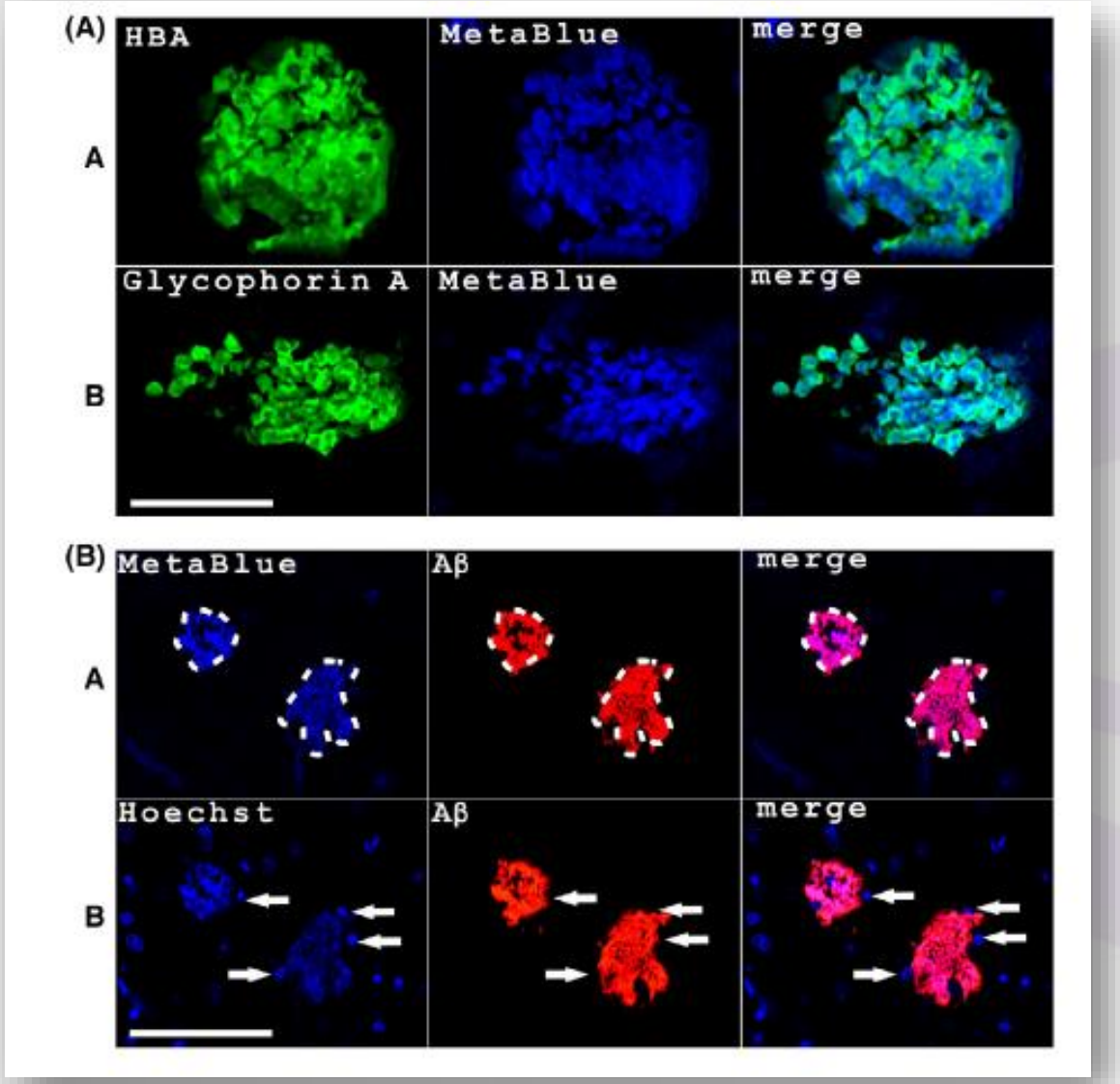
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La placa de beta-Amiloide (Aβ) coinciden con hemoglobina

Senile plaques are sites of micHualin Fu , Jilong Li, Peng D, Weilin Jin, Guo Gao and Daxiang Cui. Senile plaques in Alzheimer's disease arise from Ab- and Cathepsin D-enriched mixtures leaking out during intravascular haemolysis and microaneurysm rupture. FEBS Letters 597 (2023) 1007–1040 2022 Federation of European Biochemical Societies.

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La placa de beta-Amiloide (Aβ) coinciden con sangrado

Senile plaques are sites of micHualin Fu , Jilong Li, Peng D, Weilin Jin, Guo Gao and Daxiang Cui. Senile plaques in Alzheimer's disease arise from Ab- and Cathepsin D-enriched mixtures leaking out during intravascular haemolysis and microaneurysm rupture. FEBS Letters 597 (2023) 1007–1040 2022 Federation of European Biochemical Societies.

Colesterol, grasas saturadas y exceso de proteína

Arterioesclerosis



Hipoperfusión

Daño del endotelio

microaneurismas

microsangrados

Beta-Amiloide (Aβ) al rescate

Angiopatia cerebral amiloide

Disfunción neuronal

Ovillos neurofibrilares

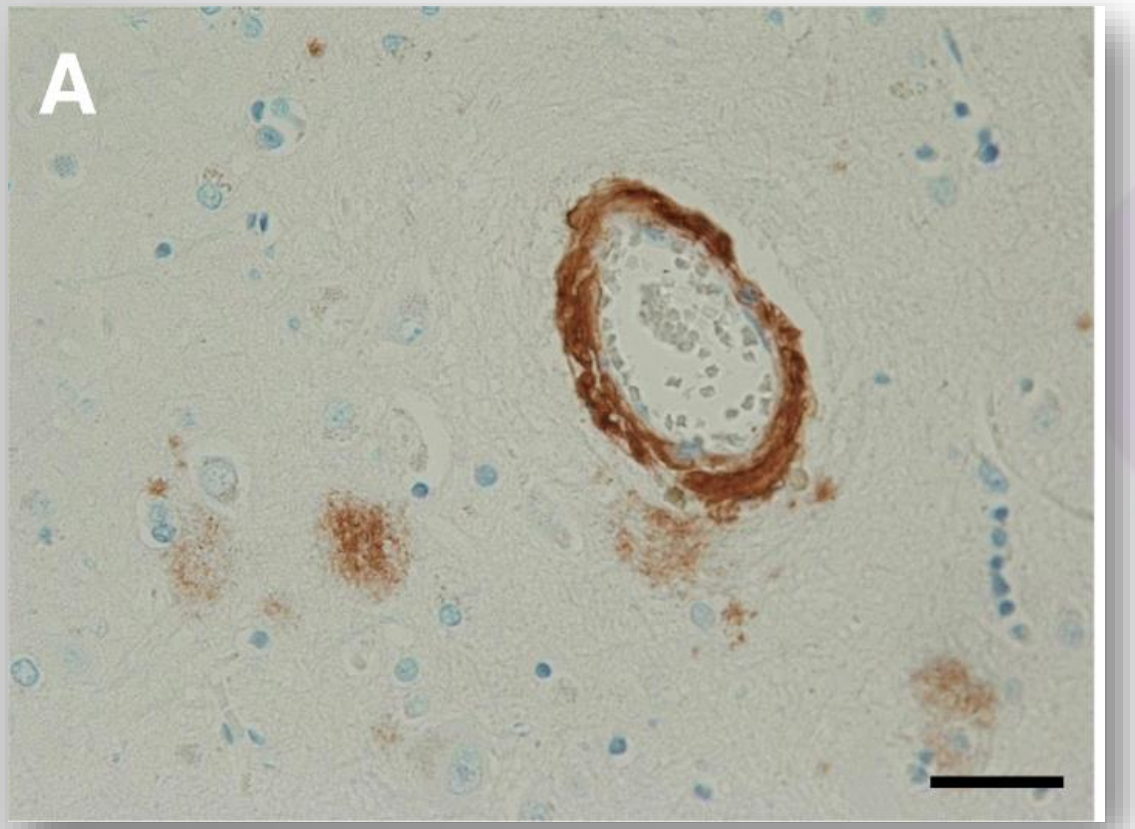
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Deterioro cognitivo y pérdida de memoria

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Angiopatía cerebral amiloide

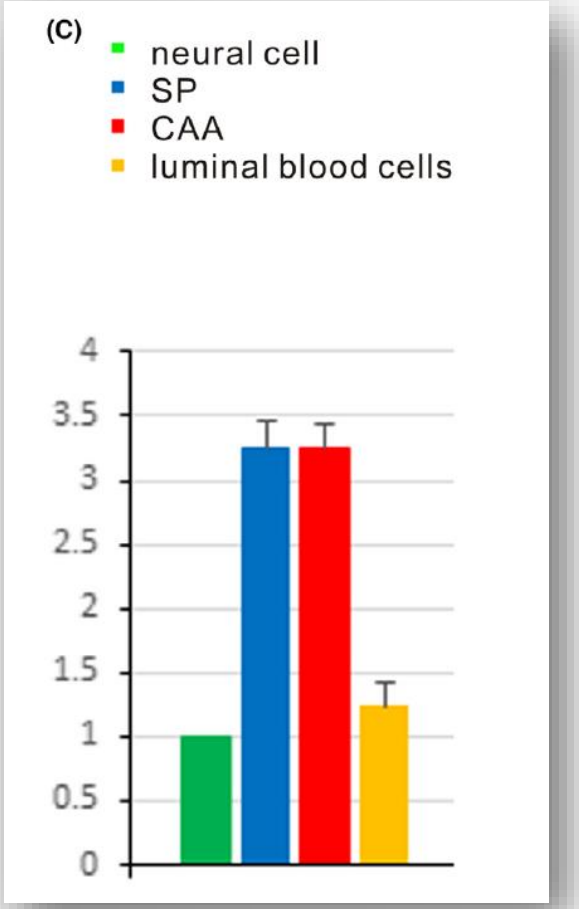


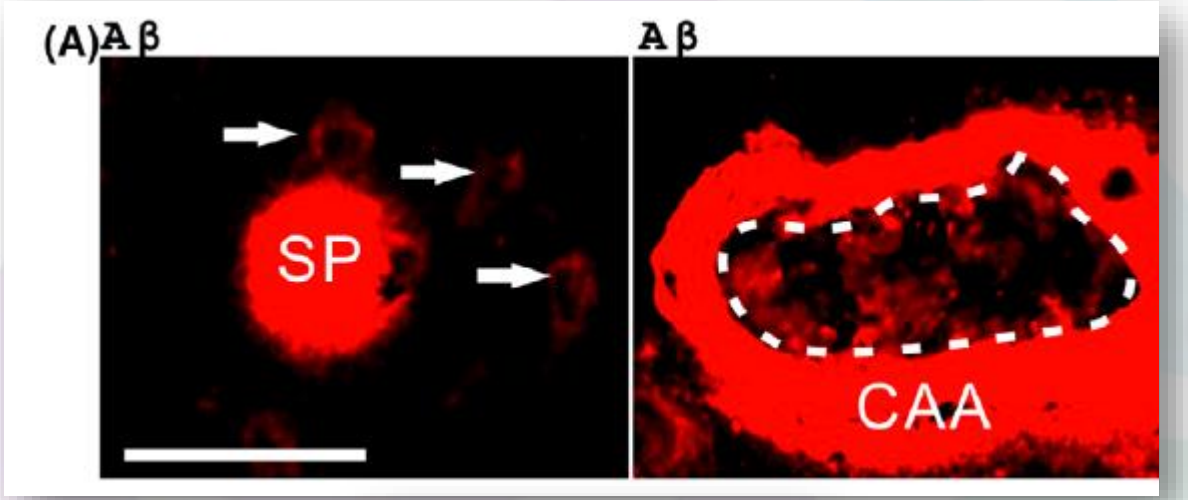
Los microsangrados pueden inducir una amiloidosis cerebrovascular beneficiosa como intento de tapar las rupturas de capilares y arteriolas en el cerebro

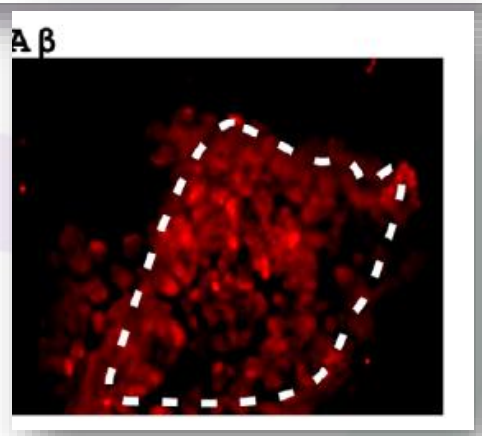
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Promedio de la intensidad de beta-Amiloide (Aβ)

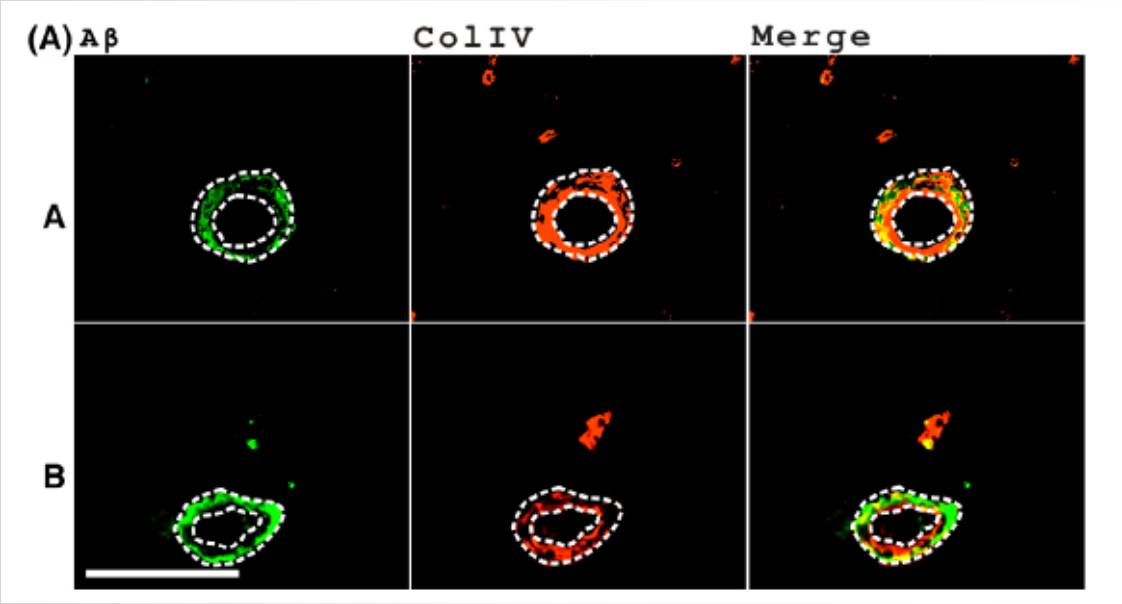


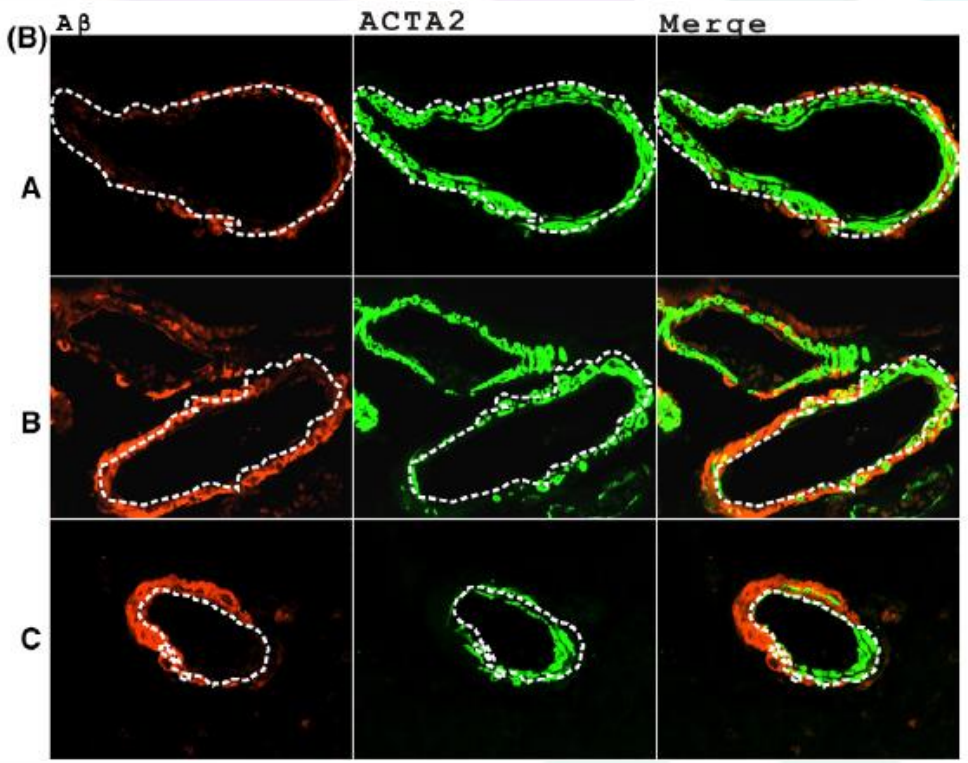




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Deposición de beta-Amiloide (Aβ) disrupte el colágeno de los capilares





Senile plaques are sites of micHualin Fu , Jilong Li, Peng D, Weilin Jin, Guo Gao and Daxiang Cui. Senile plaques in Alzheimer's disease arise from Ab- and Cathepsin D-enriched mixtures leaking out during intravascular haemolysis and microaneurysm rupture. FEBS Letters 597 (2023) 1007–1040 2022 Federation of European Biochemical Societies.

Colesterol, grasas saturadas y exceso de proteína

Arterioesclerosis



Hipoperfusión

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microaneurismas

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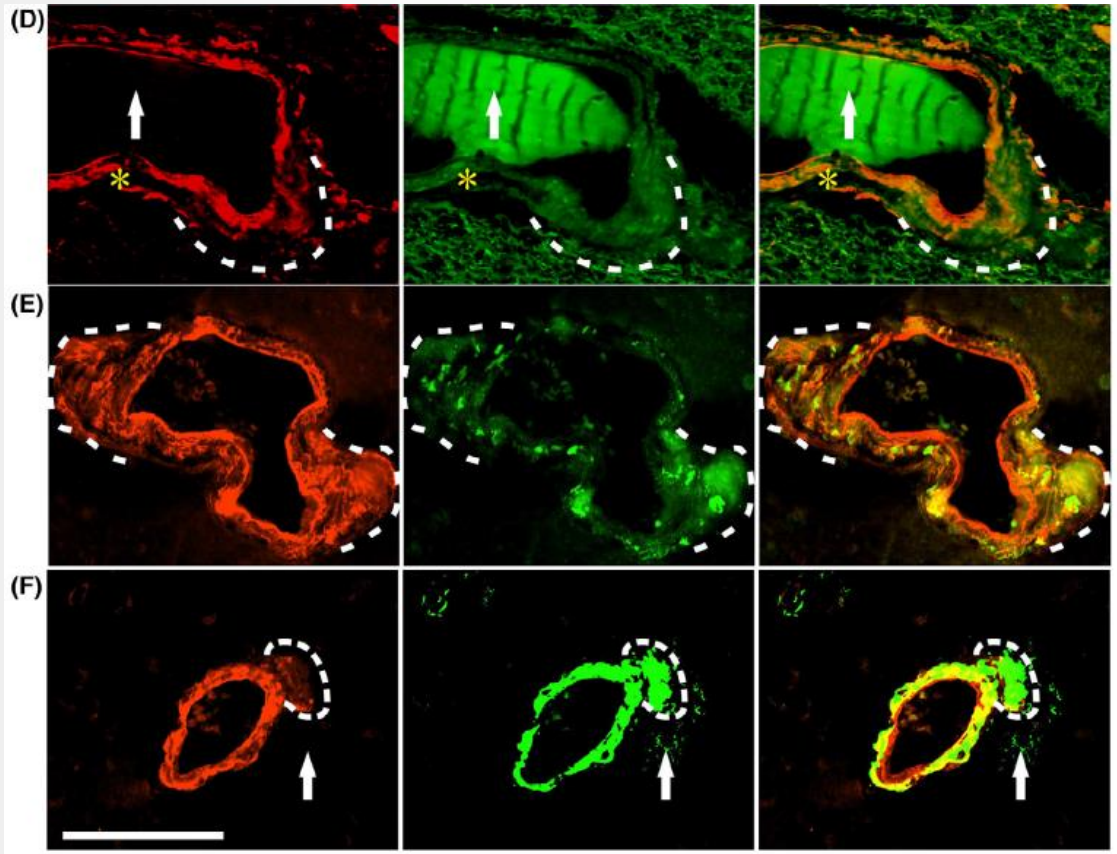
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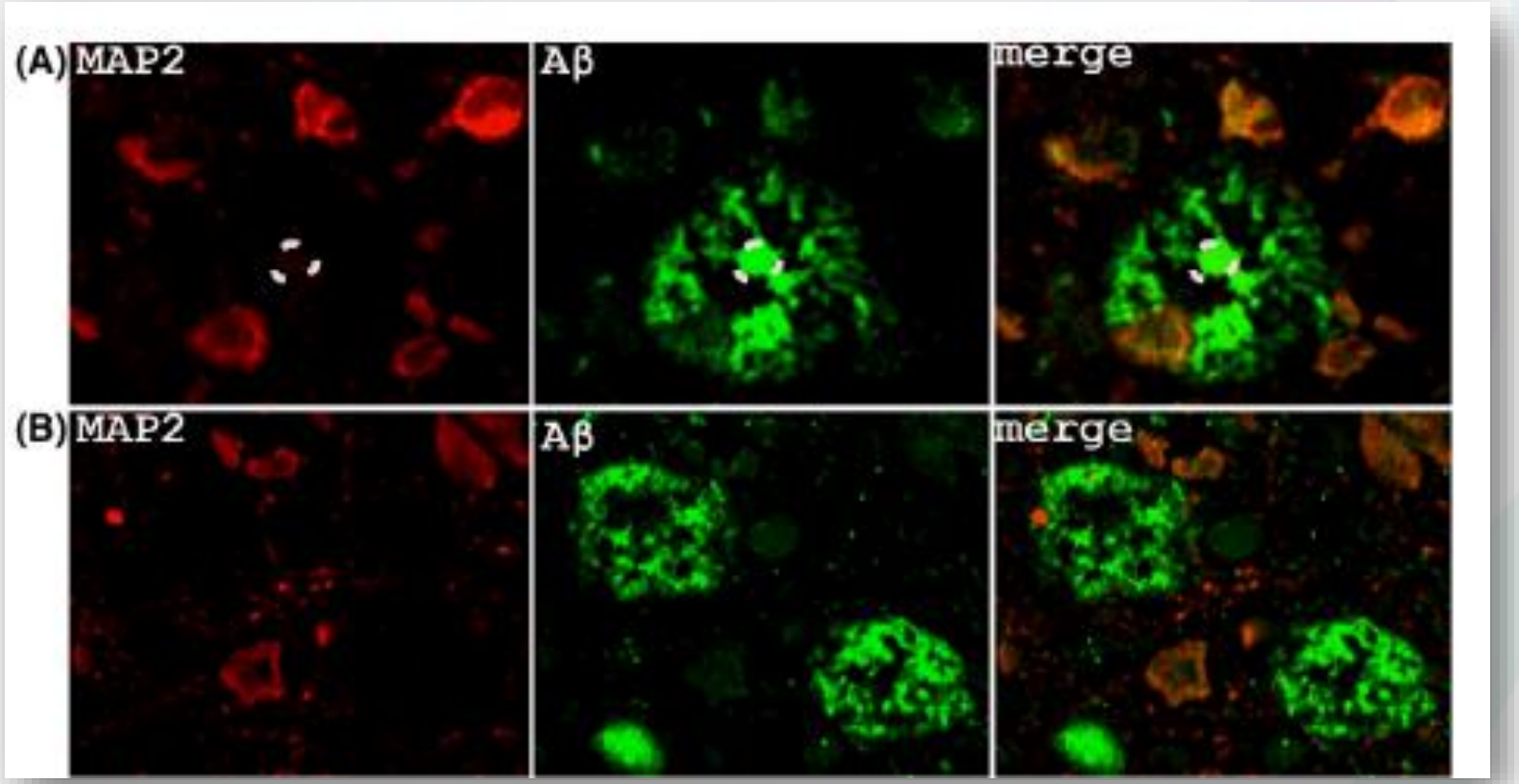
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Ruptura de microaneurismas



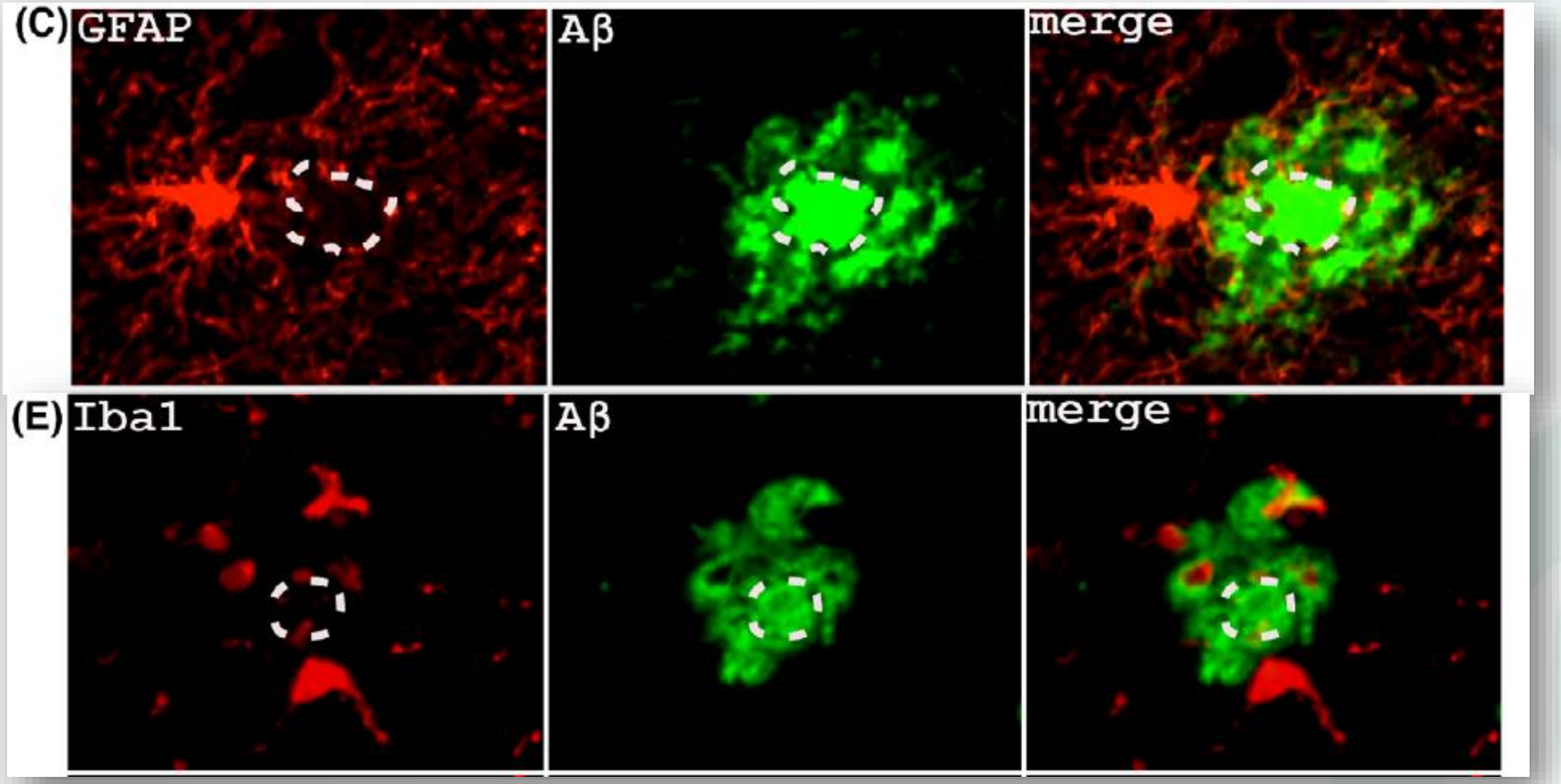
Senile plaques are sites of micHualin Fu , Jilong Li, Peng D, Weilin Jin, Guo Gao and Daxiang Cui. Senile plaques in Alzheimer's disease arise from Ab- and Cathepsin D-enriched mixtures leaking out during intravascular haemolysis and microaneurysm rupture. FEBS Letters 597 (2023) 1007–1040 2022 Federation of European Biochemical Societies.

La placa de beta-Amiloide (Aβ) no coincide con las neuronas



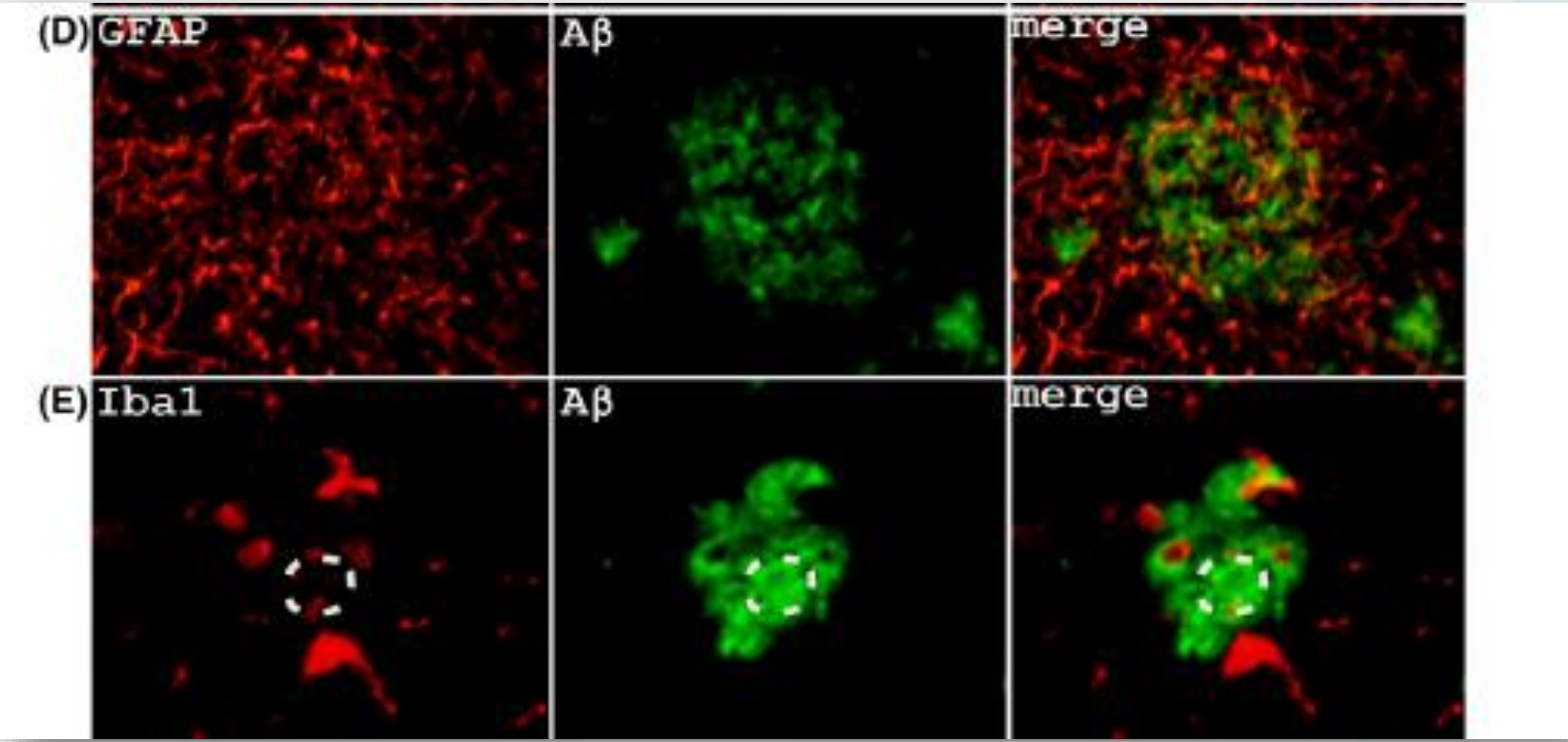
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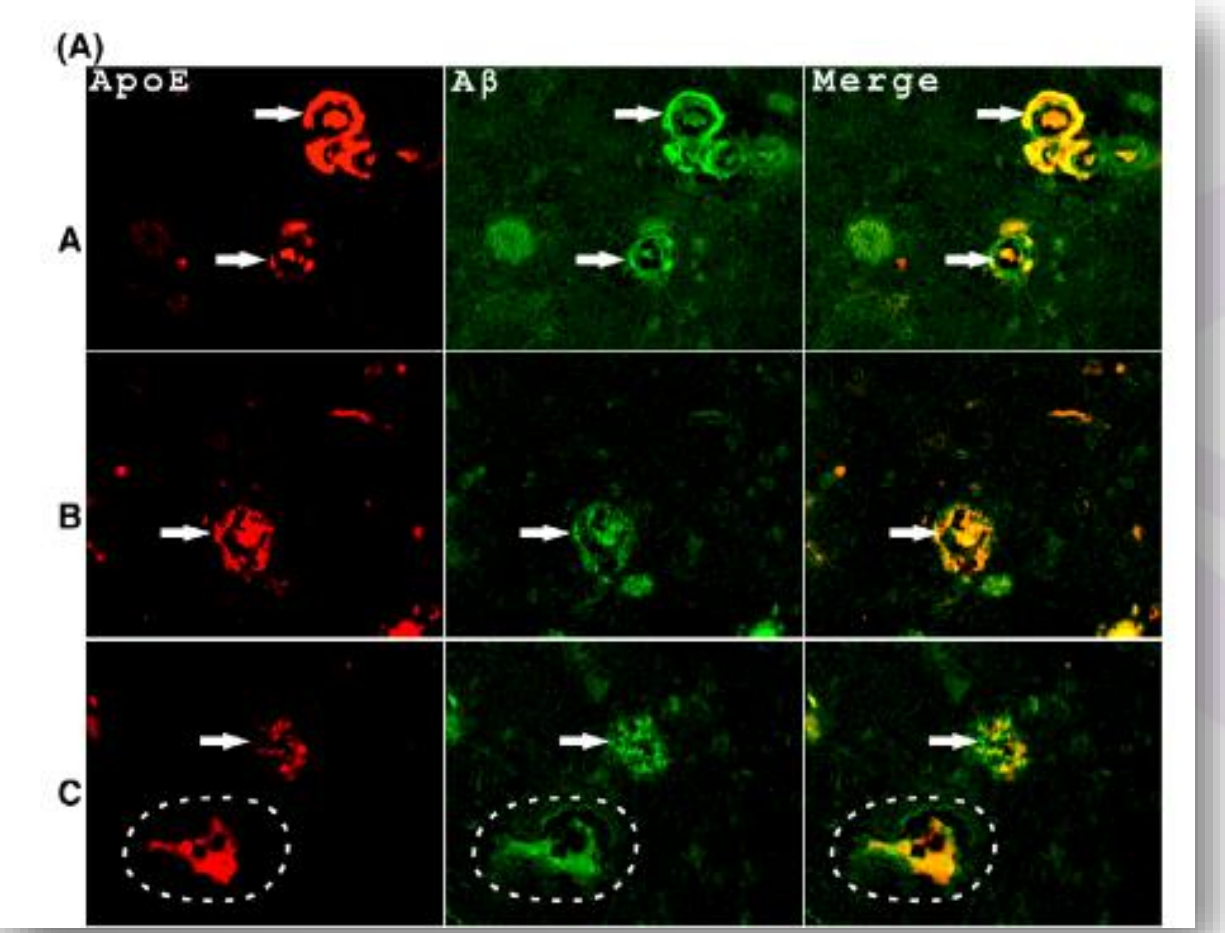
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La placa de beta-Amiloide (Aβ) coinciden con ApoE (proteína que transporta el colesterol)

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Colesterol, grasas saturadas y exceso de proteína

Arterioesclerosis



Hipoperfusión

Daño del endotelio

microaneurismas

microsangrados

Disfunción neuronal

Ovillos neurofibrilares

Disfunción sináptica y Proceso degenerativo

Deterioro cognitivo y pérdida de memoria

Muerte neuronal

Atrofia cerebral

Beta-Amiloide (Aβ) al rescate

Angiopatía cerebral amiloide

Placas beta-Amiloide (Aβ)

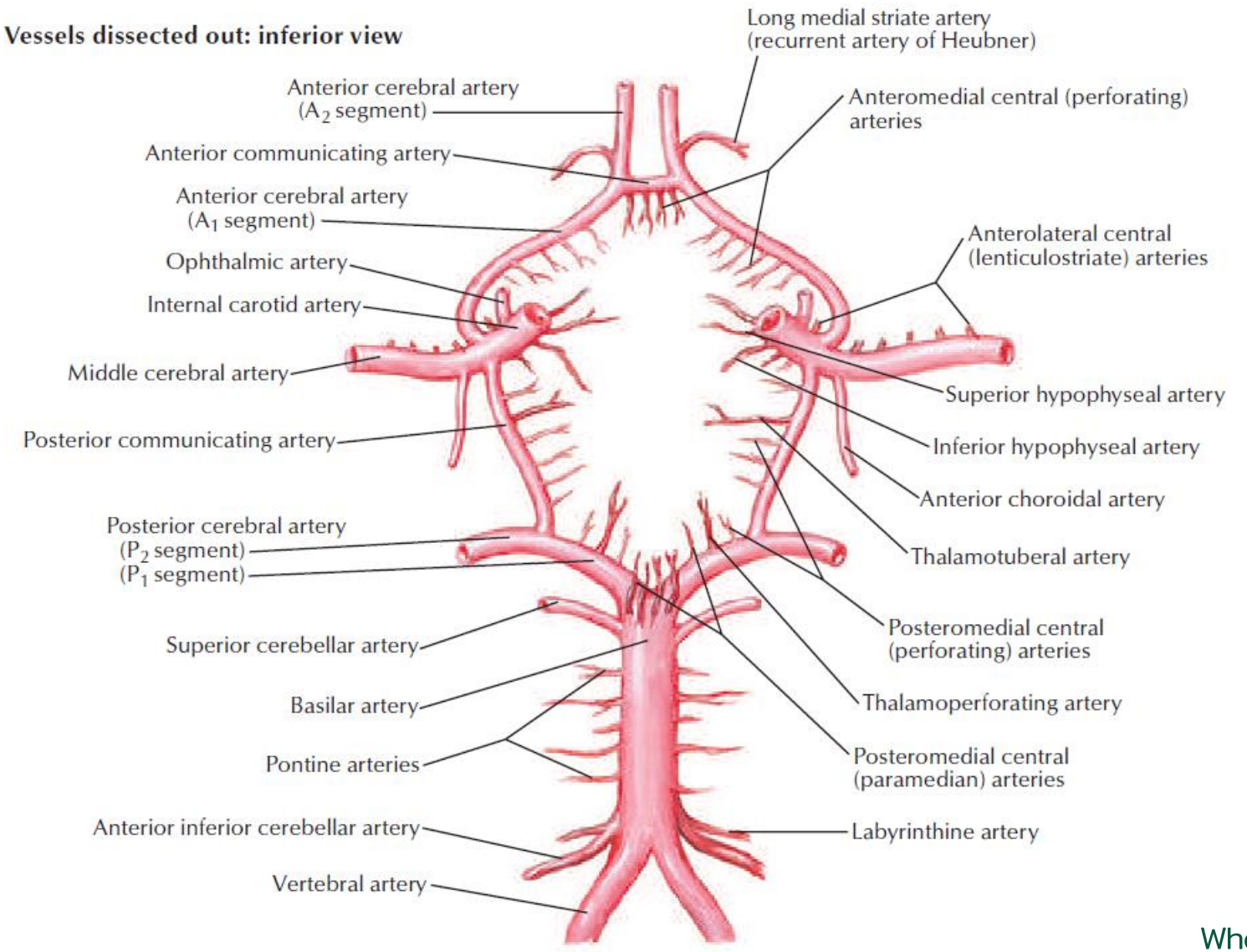
Muerte por Alzheimer

Cullen KM, Kocsi Z, Stone J. Microvascular pathology in the aging human brain: evidence that senile plaques are sites of microhaemorrhages. Neurobiol Aging. 2006; 27:1786–96.

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Polígono de Willis

Cerebral Arterial Circle (of Willis)

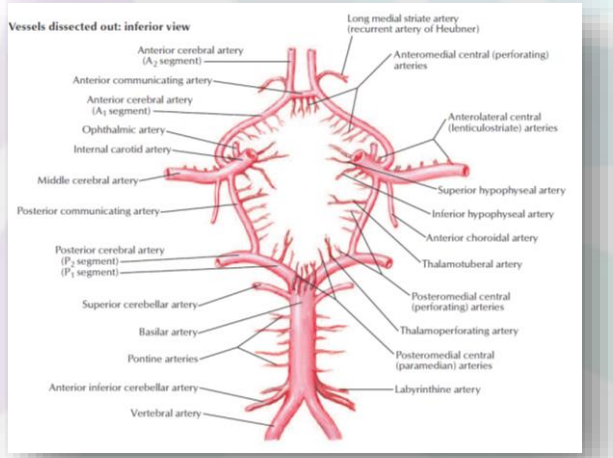


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Efecto Amortiguador

- El árbol arterial normal esta diseñado como un amortiguador.

- El endurecimiento de las paredes arteriales aumenta la amplitud del latido intracraneal y daña la microvasculatura

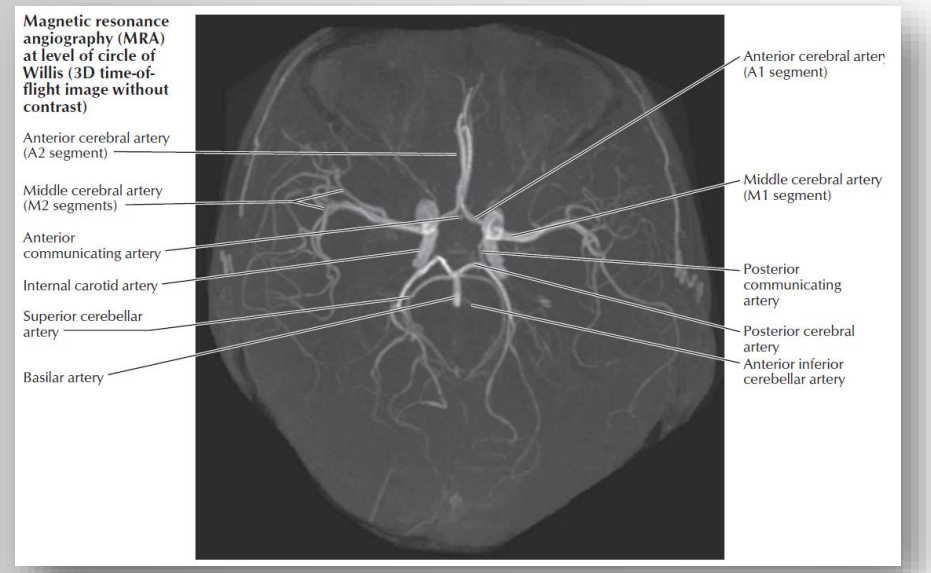


- Desprende células del endotelio, daña células musculares lisas, produce fallo en la barrera hematoencefálica y rompe vasos.

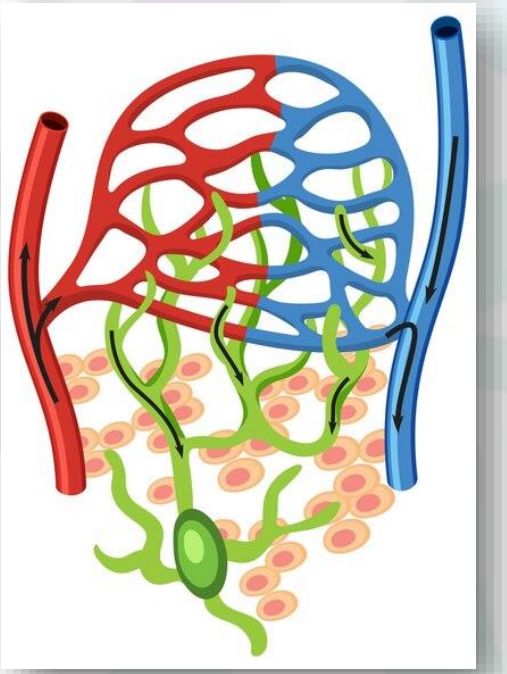
Henry-Feugeas MC, Onen F, Claeys ES. Classifying late-onset dementia with MRI: is arteriosclerotic brain degeneration the most common cause of Alzheimer's syndrome? Clin Interv Aging. 2008; 3:187–99.

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Pérdida de elasticidad arterial



- Consecuencias hemodinámicas del líquido cefalorraquídeo para mantener la homeostasis cerebral



- Mayor resistencia en vasos cerebrales afecta el drenaje venoso y del líquido cefalorraquídeo en el sistema linfático

Henry-Feugeas MC, Onen F, Claeys ES. Classifying late-onset dementia with MRI: is arteriosclerotic brain degeneration the most common cause of Alzheimer's syndrome? Clin Interv Aging. 2008; 3:187–99.

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En conclusión...

Daño del endotelio de capilares

microsangrado

Hipoperfusión crónica cerebral

Amiloidosis

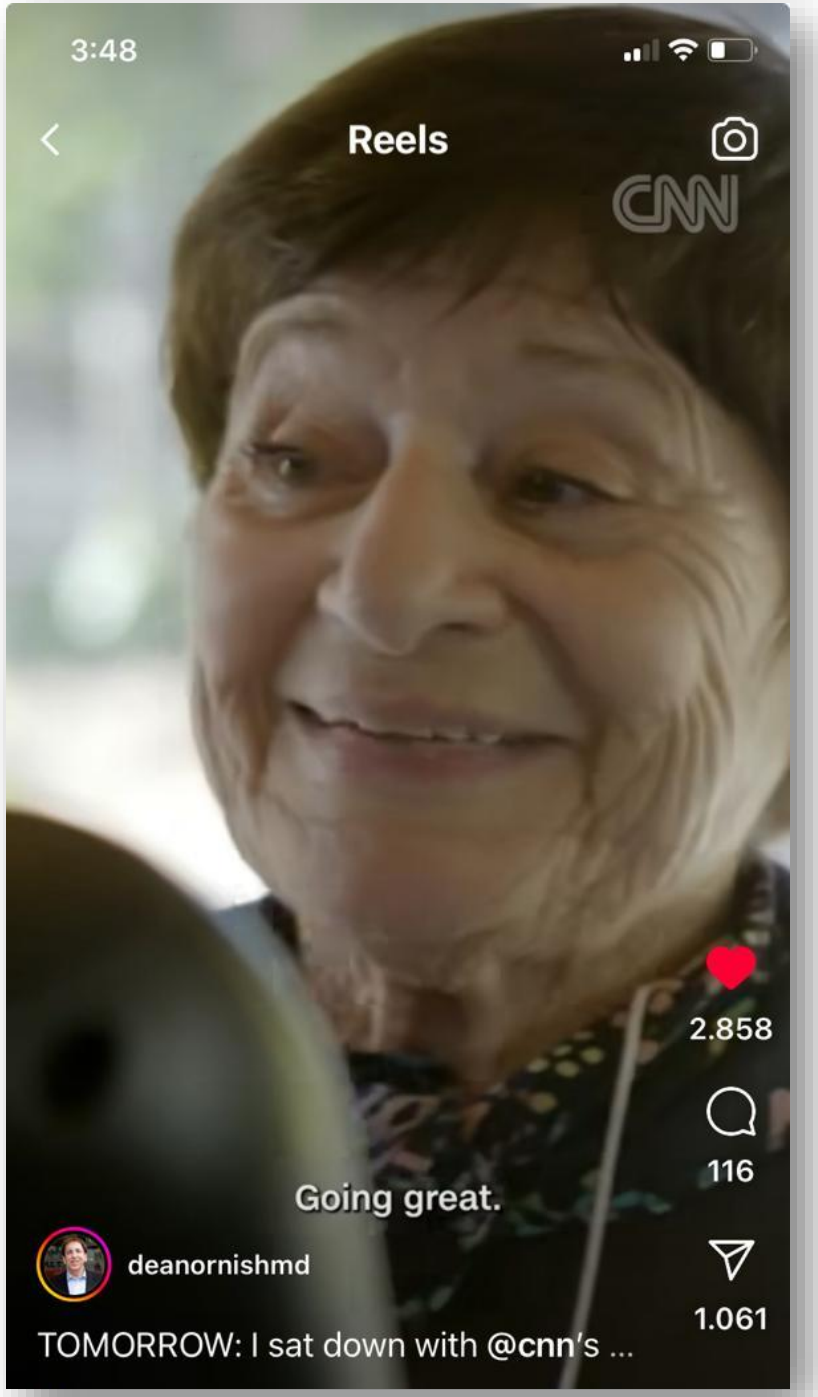
Atrofia cerebral

Infartos cerebrales

Drenaje linfático disminuido

Acumulación de sustratos nocivos

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Primer estudio sobre

reversión de Enfermedad de Alzheimer en etapa temprana

Ornish et al. Alzheimer's Research & Therapy (2024) 16:122
https://doi.org/10.1186/s13195-024-01482-z

Alzheimer's
Research & Therapy

RESEARCH Open Access

Effects of intensive lifestyle changes on the progression of mild cognitive impairment or early dementia due to Alzheimer's disease: a randomized, controlled clinical trial

Dean Ornish[1,2*], Catherine Madison[1,3], Miia Kivipelto[4,5,6,7], Colleen Kemp[8], Charles E. McCulloch[9], Douglas Galasko[10], Jon Artz[11,12], Dorene Rentz[13,14,15], Jue Lin[16], Kim Norman[17], Anne Ornish[1], Sarah Tranter[8], Nancy DeLamarter[1], Noel Wingers[1], Carra Richling[1], Rima Kaddurah-Daouk[18], Rob Knight[19], Daniel McDonald[20], Lucas Patel[21], Eric Verdin[22,23], Rudolph E. Tanzi[13,24,25,26] and Steven E. Arnold[13,27]

Abstract

Background Evidence links lifestyle factors with Alzheimer's disease (AD). We report the first randomized, controlled clinical trial to determine if intensive lifestyle changes may beneficially affect the progression of mild cognitive impairment (MCI) or early dementia due to AD.

Methods A 1:1 multicenter randomized controlled phase 2 trial, ages 45-90 with MCI or early dementia due to AD and a Montreal Cognitive Assessment (MoCA) score of 18 or higher. The primary outcome measures were changes in cognition and function tests: Clinical Global Impression of Change (CGIC), Alzheimer's Disease Assessment Scale (ADAS-Cog), Clinical Dementia Rating–Sum of Boxes (CDR-SB), and Clinical Dementia Rating Global (CDR-G) after 20 weeks of an intensive multidomain lifestyle intervention compared to a wait-list usual care control group. ADAS-Cog, CDR-SB, and CDR-Global scales were compared using a Mann-Whitney-Wilcoxon rank-sum test, and CGIC was compared using Fisher's exact test. Secondary outcomes included plasma Aβ42/40 ratio, other biomarkers, and correlating lifestyle with the degree of change in these measures.

Results Fifty-one AD patients enrolled, mean age 73.5. No significant differences in any measures at baseline. Only two patients withdrew. All patients had plasma Aβ42/40 ratios <0.0672 at baseline, strongly supporting AD diagnosis. After 20 weeks, significant between-group differences in the CGIC (p= 0.001), CDR-SB (p= 0.032), and CDR Global (p= 0.037) tests and borderline significance in the ADAS-Cog test (p= 0.053). CGIC, CDR Global, and ADAS-Cog showed

Ornish D, Madison C, Kivipelto M, et al. Effects of intensive lifestyle changes on the progression of mild cognitive impairment or early dementia due to Alzheimer's disease: a randomized, controlled clinical trial. Alzheimers Res Ther. 2024;16(1):122.

Reversión de Enfermedad de Alzheimer en etapa temprana

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Impresión Clínica Global de Cambio (CGIC)

	INTERVENCIÓN	CONTROL
Mejoraron	10	0
Sin cambios	7	8
Empeoraron	7	14

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Reversión de Enfermedad de Alzheimer en etapa temprana

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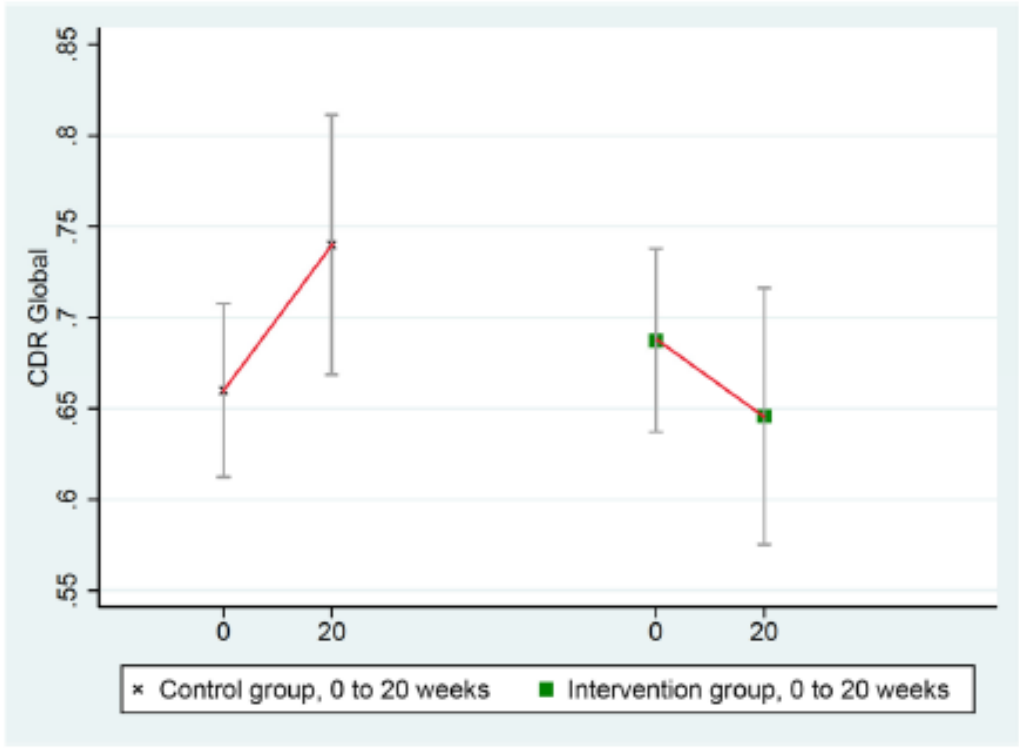


Fig. 2 Changes in CDR-Global (lower = improved)

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Reversión de Enfermedad de Alzheimer en etapa temprana

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Biomarcadores que mejoraron:

- La proporción de Aβ42/40 en plasma aumentó en el grupo de intervención pero disminuyó en el grupo de control aleatorizado
- Colesterol LDL
- Proteína Tau 181
- Microbioma

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En conclusión...

"En resumen, en personas con deterioro cognitivo leve o demencia temprana debido a la enfermedad de Alzheimer, los cambios integrales en el estilo de vida pueden mejorar la cognición y la función en varias medidas estándar después de 20 semanas. En contraste, los pacientes en el grupo de control aleatorizado mostraron un empeoramiento general en las cuatro medidas de cognición y función durante este tiempo."

Ornish D, Madison C, Kivipelto M, et al. Effects of intensive lifestyle changes on the progression of mild cognitive impairment or early dementia due to Alzheimer's disease: a randomized, controlled clinical trial. Alzheimers Res Ther. 2024;16(1):122.

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