4º CONGRESO AMAREVA

2025

27 y 28 de febrero

Auditorio Caja de Música
del Palacio de Cibeles

AMAREVA
ASOCIACIÓN MADRILEÑA DE RIESGO Y ENFERMEDAD VASCULAR

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Lp(a): De la teoría a la práctica: puntos claves

Almudena Castro
CARDIÓLOGA
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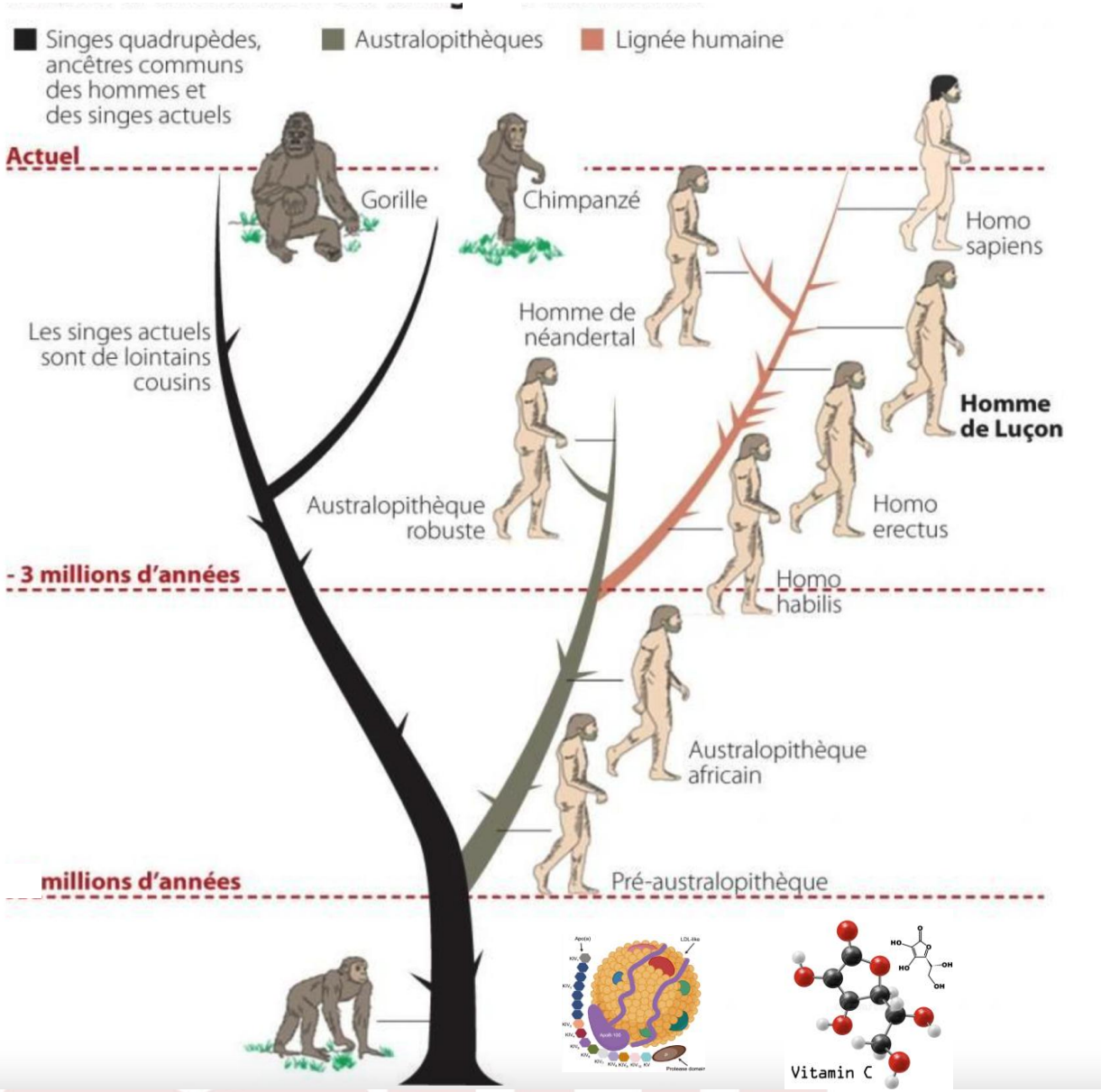
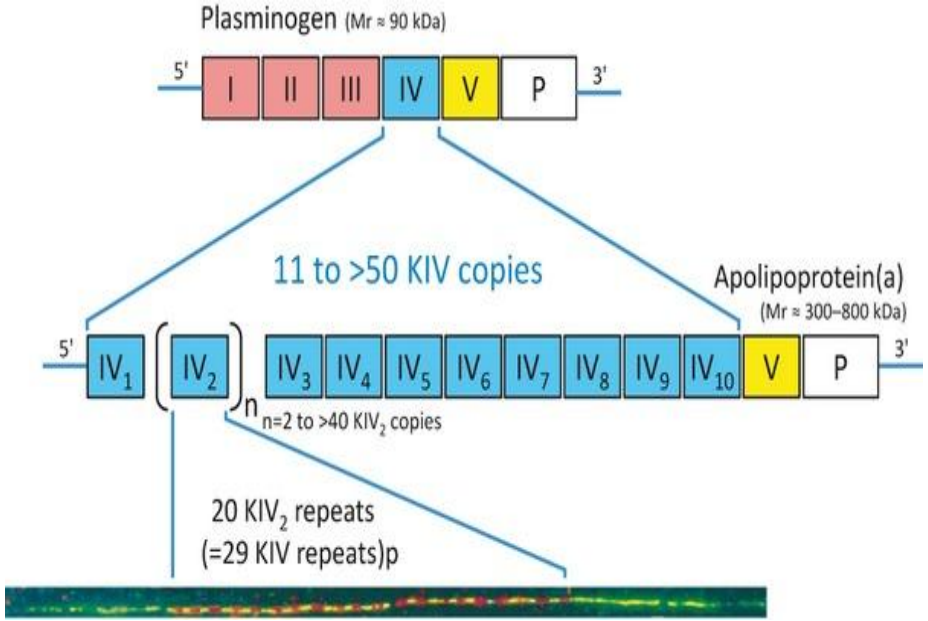
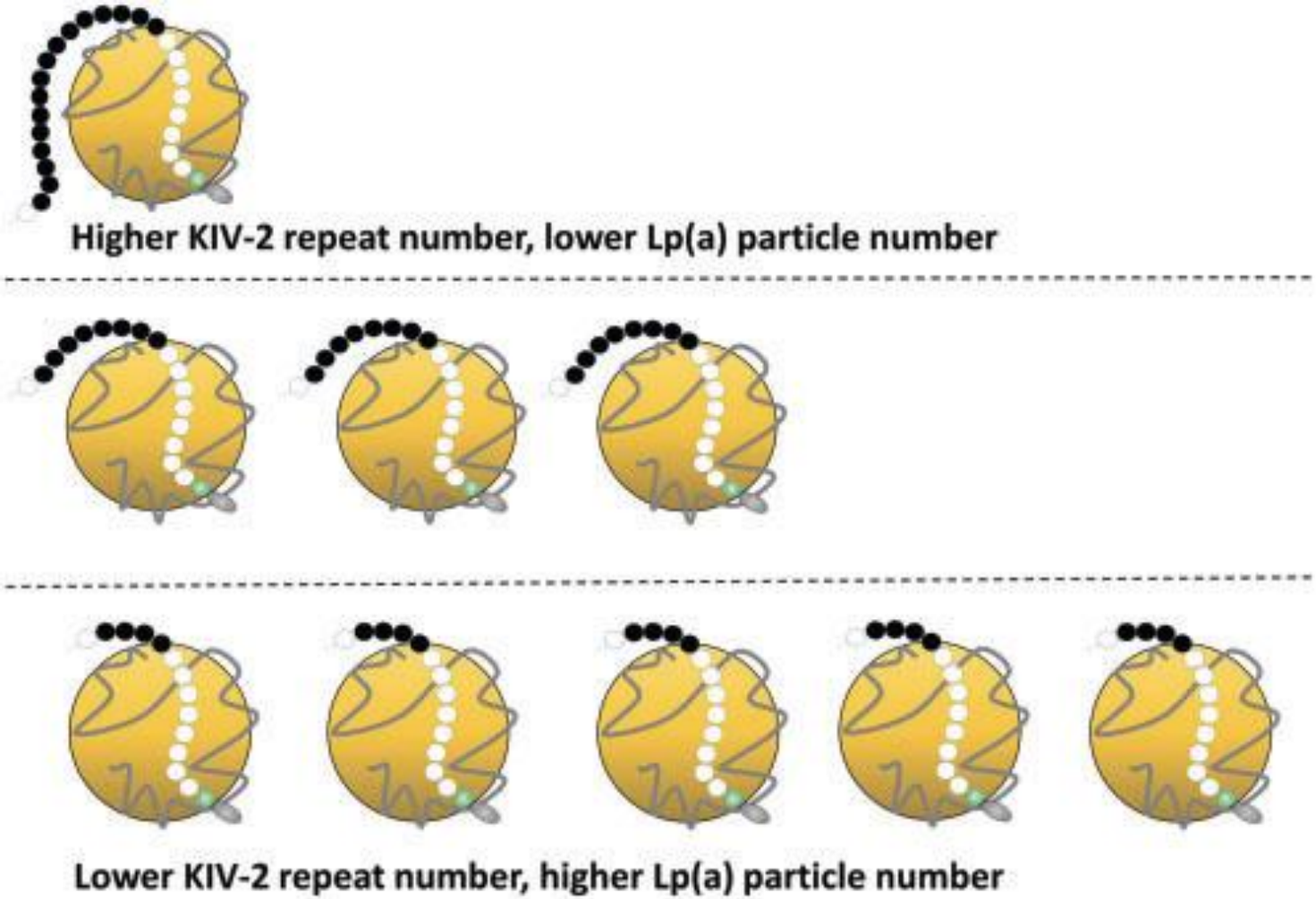


Rath M, Pauling L. Hypothesis: lipoprotein(a) is a surrogate for ascorbate. Proc Natl Acad Sci U S A. 1990 Aug;87(16):6204-7

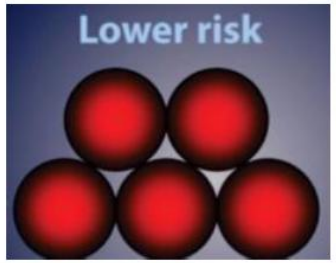

Plasminogen (Mr ≈ 90 kDa)
5'
I
II
III
IV
V
P
3'
11 to >50 KIV copies
Apolipoprotein(a)
(Mr ≈ 300–800 kDa)
5'
IV1
IV2
n
IV3
IV4
IV5
IV6
IV7
IV8
IV9
IV10
V
P
3'
n=2 to >40 KIV2 copies
20 KIV2 repeats
(=29 KIV repeats)p


Higher KIV-2 repeat number, lower Lp(a) particle number
Lower KIV-2 repeat number, higher Lp(a) particle number

A mayor número de repeticiones KIV-2

↓

Partículas más grandes



Menor concentración

↓

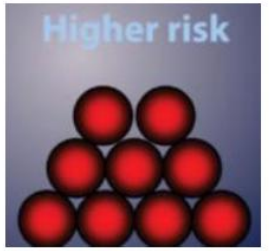
Menor RCV

Pequeñas y numerosas Lp(a) pueden infiltrarse más fácilmente en las paredes arteriales, promoviendo la acumulación de placas y aumentando la probabilidad de eventos cardiovasculares

A menor número de repeticiones KIV-2

↓

Partículas más pequeñas

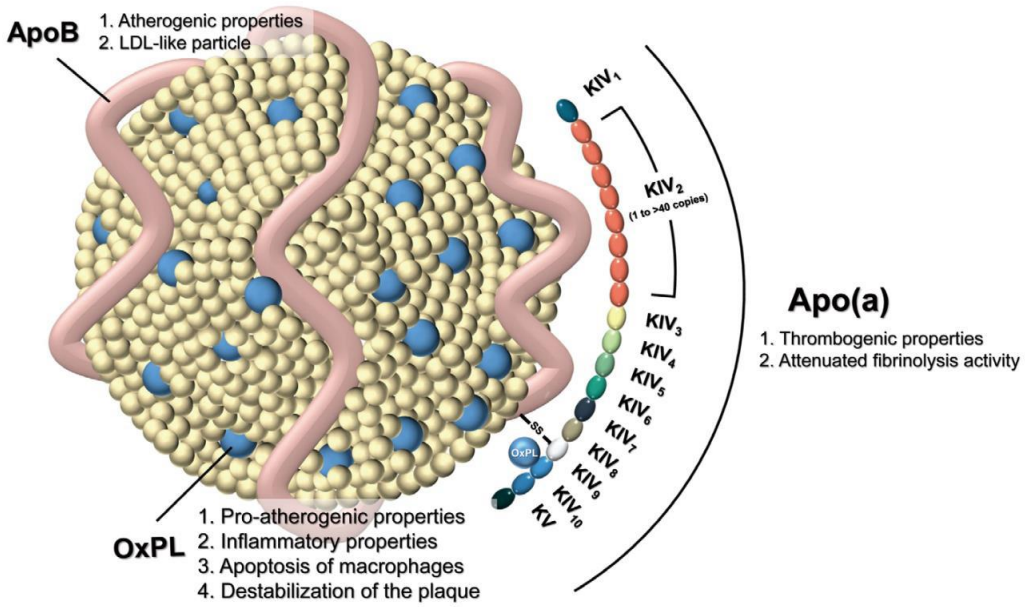


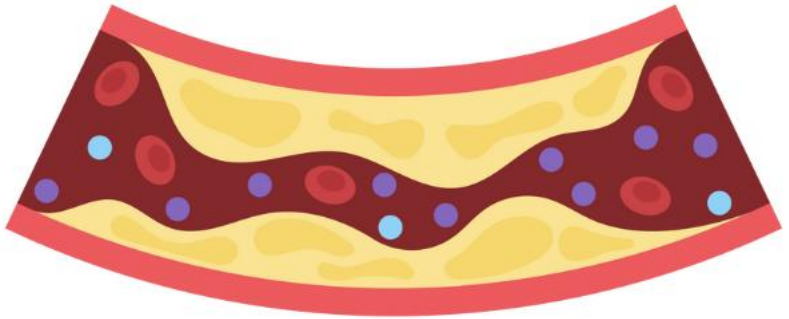
Más concentración

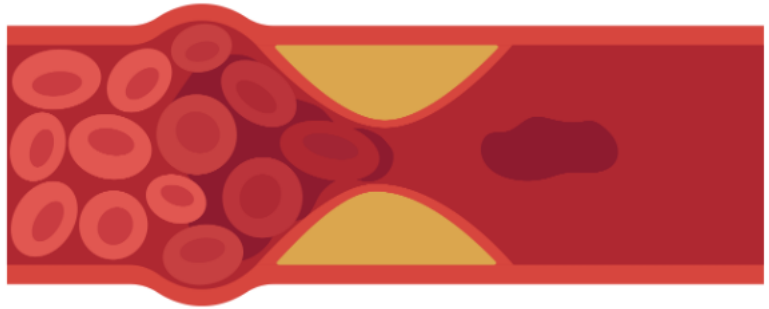
↓

Mayor RCV

1. ¿La elevación de la Lp(a) es un factor independiente, genético y causal de la enfermedad ateroesclerótica?







Apo(a): apolipoproteína a; CV: cardiovascular; Lp(a): lipoproteína a; OxPL: fosfolípidos oxidados.

1. Tsimikas S. J Am Coll Cardiol. 2017; 69(6): 692-711; 2. Kronenberg F et al. Eur Heart J. 2022;43:3925-3946; 3. Jang A.Y. et al. Circ J. 2020; 84: 867-874.

Influencia genética y modificación del riesgo CV según los niveles de Lp(a)

90%

Genético[1-4]

Modelo de herencia: autosómico codominante multialélico.

- Entre el 40 y 50 % de la variabilidad viene determinada por el **número de repeticiones del gen Kringle IV$_2$.**
- El resto de la variabilidad se debe a **SNPs** y **variantes del gen *LPA*[5,6].**

Variaciones genéticas asociadas con

Niveles elevados de Lp(a) → **Menor esperanza de vida[7]**

Niveles bajos de Lp(a) → **Reducción del riesgo** de manifestaciones de enfermedades ateroscleróticas CV[7]

- Estudios de aleatorización mendeliana han demostrado que la Lp(a) elevada es una **condición genética causal de aumento del riesgo CV[8].**
- **Efecto sumatorio e independiente** sobre los niveles de LDL y sobre el riesgo de padecer ECV[4]

Apo(a): apolipoproteína a; CV: cardiovascular; Lp(a): lipoproteína a.

1. Tsioulos Q et al. Int J Mol Sci. 2024;25: 3537. .2. Kronenberg F et al. Eur Heart J. 2022; 43: 3925-3946. 3. Nordestgaard BG et al. Lancet. 2024; 404(10459): 1255-1264; 4. Schmidt K et al. J Lipid Res. 2016; 56: 1339-1359; 5. Clarke R et al. *N Engl J Med*. 2009;361(26):2518–2528. 6. GudbjartssonDF et al. *J Am Coll Cardiol*. 2019:74(24):2982–2994. 7. Kronenberg F et al. Eur Heart J. 2022: 0: 1-22. 8. Kamstrup PR et al. *JAMA*.

Prevalencia de niveles elevados de Lp(a)

>1,5 billones de personas a nivel mundial[1]

En un estudio (n= 532.359) se observó[2]

> 20 % tiene valores de Lp(a) >50 mg/dL
> 5 % tiene valores de Lp(a) >100 mg/dL
1 % tiene valores de Lp(a) >180 mg/dL

Aquellas personas que han sufrido un evento CV presentan una prevalencia **duplicada** frente a la población normal[3]

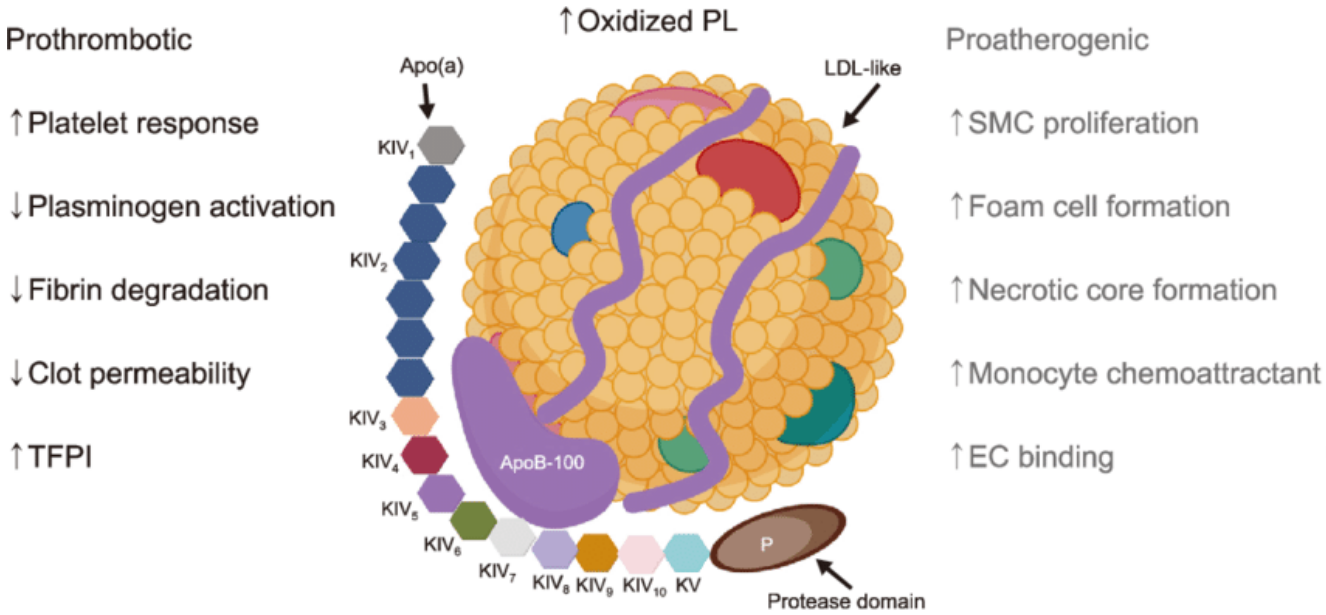
- Los pacientes con niveles **Lp(a) >200mg/dL** presentan el mismo riesgo de ECV que aquellos que presentan **hipercolesterolemia familiar**[4]
- **Lp(a) >200mg/dL** es el doble de prevalente que la hipercolesterolemia familiar[4]

Condición genética más frecuente asociada a ECV con gran impacto en el aumento del riesgo

Apo(a): apolipoproteína a; CV: cardiovascular; ECV: enfermedad cardiovascular; Lp(a): lipoproteína a

1. Tsimikas S et al.J Am Coll Cardiol. 2018; 71(2): 177-192; 2Varvel S et al. Arterioscler Thromb Vasc Biol. 2016; 36: 2239-2245; 3. Delgado-Lista J et al. Lancet. 2022; 399(10338): 1876-1885. 4. Burgess S et al. JAMA Cardiol. 2018; 3(7): 619-627.

Lp(a)



Serie RhC Hospital La Paz

2023: (n=209)
> 50 mg/dL (n=72) **(34%)**
> 100 mg/dL (n=36) **(17%)**

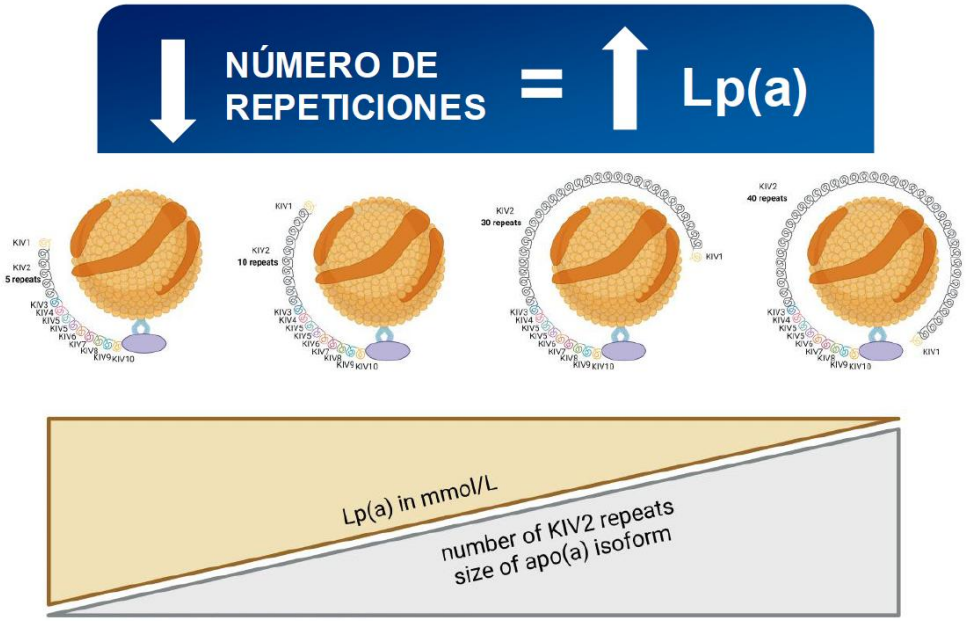
2024: (n=239)
> 50 mg/dl (n=82) **(34%)**
>100 mg/dL (n=36) **(15%).**

2023(n=55)
>50 mg/dL (n=26) **(47%)**
>100 mg/dL (n=15) **(27%)**

2024 (n=61)
>50 mg/dL (n= 29) **(48%)**
>100 mg/dl (n=18) **(30%).**

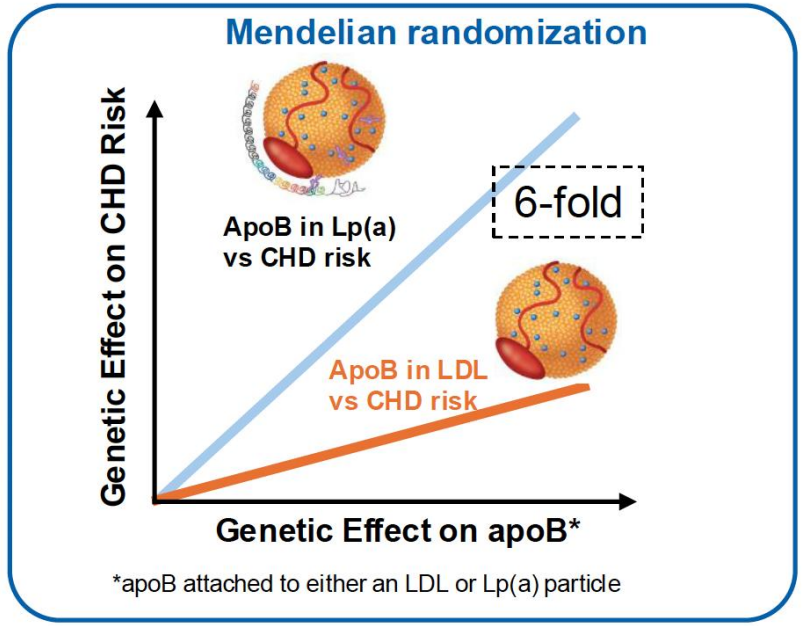
La Lp(a) es una partícula 6 veces más aterogénica que el c-LDL con niveles interindividuales variables[1,2]

2. Diferencias aterogénicas entre la Lp(a) y el cLDL



Broncel et al Explor Cardiol. 2023;1:180–92

El **KIV-2** puede presentar desde **1 a más de 40 copias**, condicionando la longitud y el peso molecular total de la apo(a)[1].



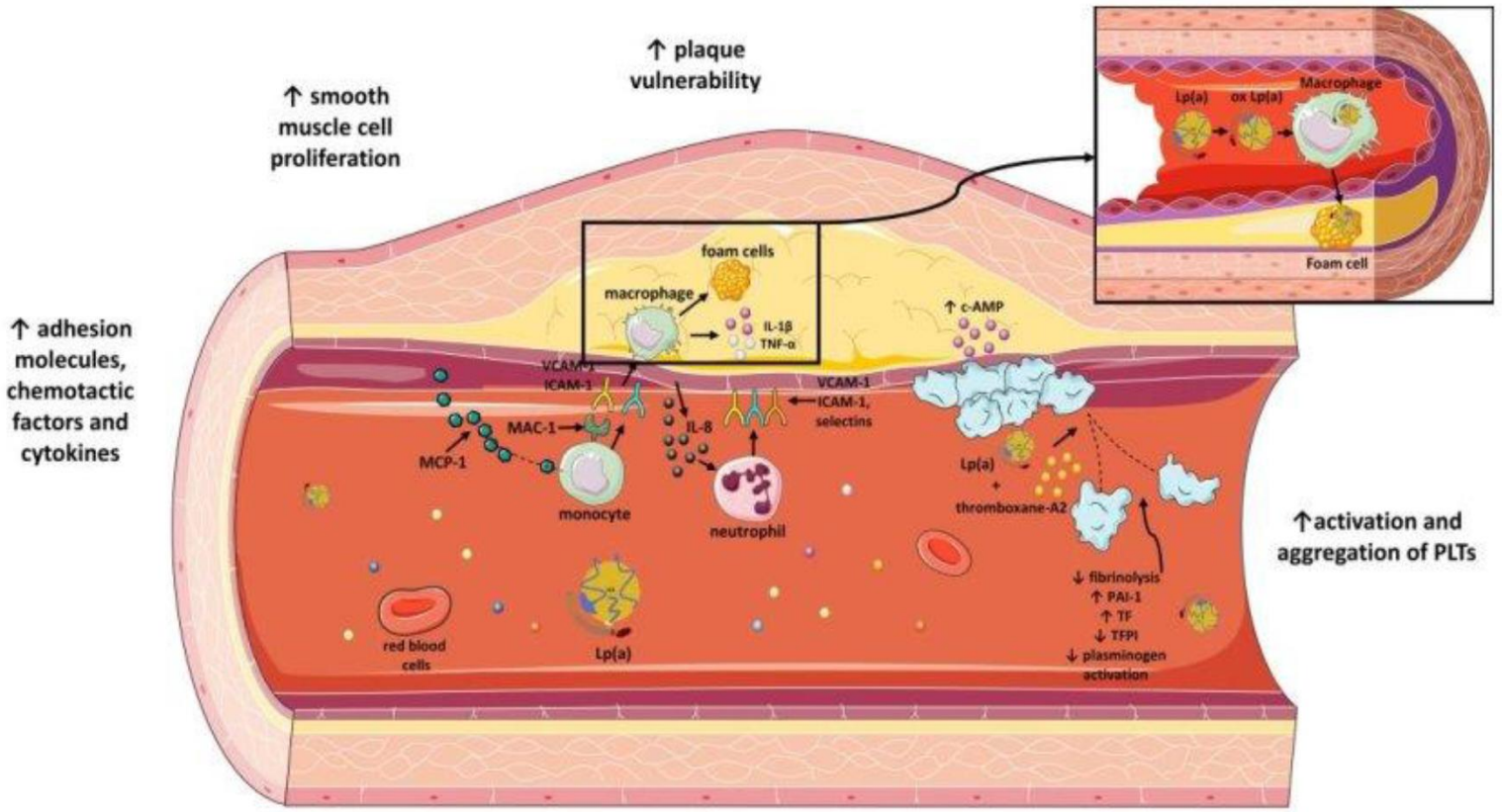
En la mayor parte de la población, las partículas de c-LDL con más abundantes y su contribución al riesgo CV superior[2].
Sin embargo, **la Lp(a) es**

6

veces **más aterógena** por partícula **que el LDL**, especialmente relevante en individuos con Lp(a) elevada[2]

KIV: Kringle IV; Lp(a): lipoproteína a.
1. Tsimikas S et al. J Am Coll Cardiol. 2017; 69(6): 692-711. 2. Björnson E et al. J Am Coll Cardiol. 2024; 83(3): 385-395.

Rol de la Lp(a) en la aterosclerosis



Circ J. 2020; 84: 867-874; Molecules. 2023; 28: 969.

- Interviene en **todas** las fases de aterogénesis. Aquellos pacientes con **mayores niveles** de Lp(a) presentan **mayores depósitos** arteriales de Lp(a)[1]
- Está involucrada en **mecanismos proaterogénicos, protrombóticos y proinflamatorios**[2]

Uno de los elementos fundamentales es la alta acumulación de **fosfolípidos oxidados**, los cuales favorecen la **progresión de ateroesclerosis** y aumentan el **riesgo de eventos CV**[2]

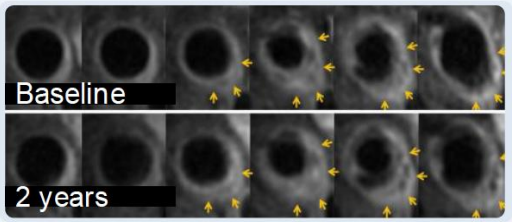
Apo(a): apolipoproteína a; CV: cardiovascular; Lp(a): lipoproteína a; OxPL: fosfolípidos oxidados.

1. Jang A.Y. At al. Circ J. 2020; 84: 867-874; 2. Tsimikas S et al. J Am Coll Cardiol. 2017; 69(6): 692-711

La Lp(a) elevada tiene impacto en características clave que determinan la vulnerabilidad de la placa aterosclerótica[1-4]

Progresión de la placa[1]

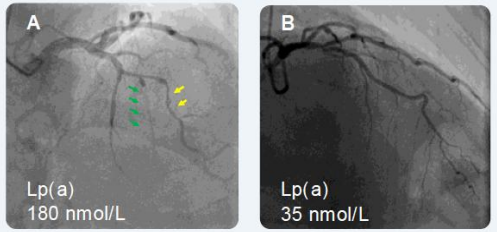
La Lp(a) elevada impulsa la **progresión de la placa**, incluso en pacientes con niveles controlados de cLDL (<70mg/dL)



Progresión de la placa carotídea evaluada mediante resonancia magnética en un paciente con Lp(a) elevada

Fenotipo y gravedad[2]

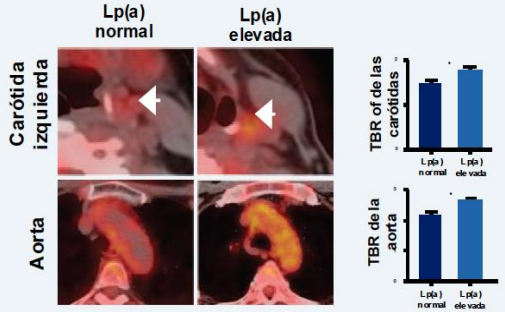
La Lp(a) elevada se asocia con una **presentación más grave** de la enfermedad y una forma de enfermedad coronaria **compleja de tratar**



Angiografías representativas de pacientes con diferentes niveles de Lp(a)

Inflamación arterial[3]

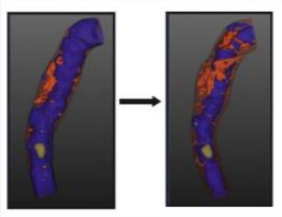
La Lp(a) elevada se asocia con un **aumento de la inflamación** de la pared arterial que no se atenúa con la reducción del C-LDL



Cross-sectional PET/CT images and quantification of 18F-FDG uptake

Placas vulnerables[4]

La Lp(a) elevada se asocia de forma independiente con el **desarrollo de placas vulnerables** (núcleos necróticos más grandes y cubiertas fibrosas más delgadas) que son propensas a romperse.

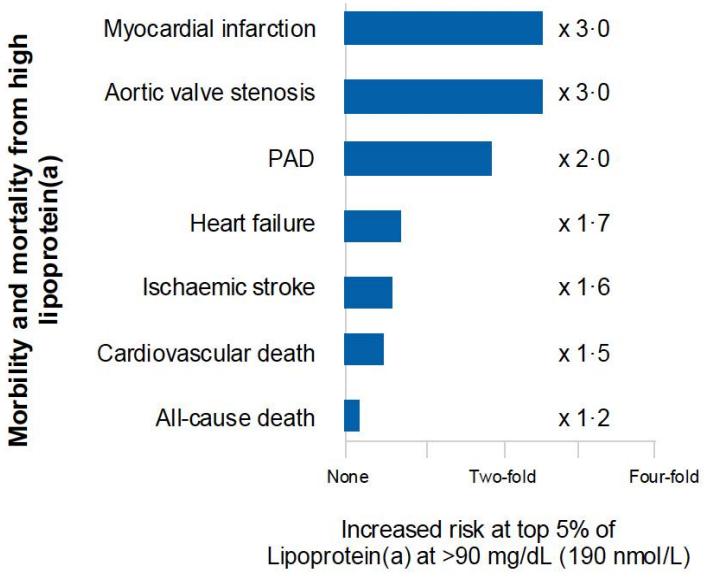


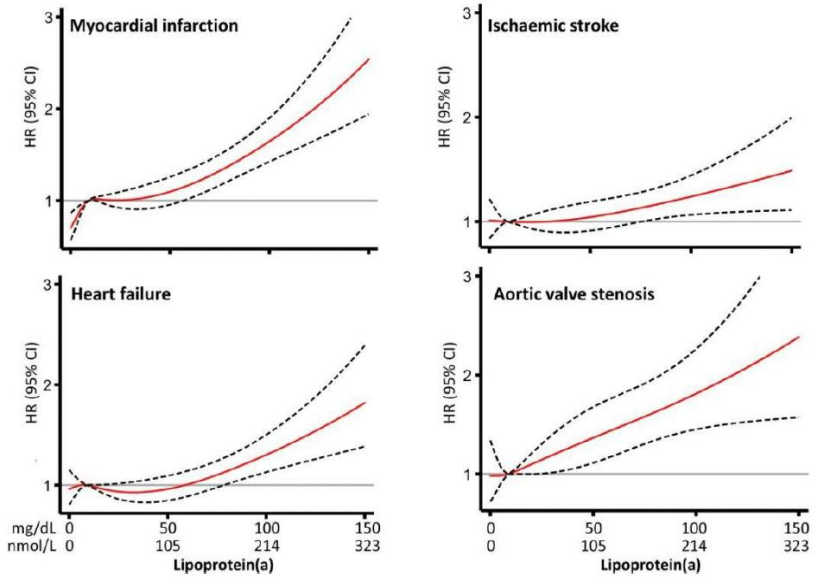
Repeat CCTA assessment of vulnerable plaque characteristics in patients with advanced CAD and elevated Lp(a) levels

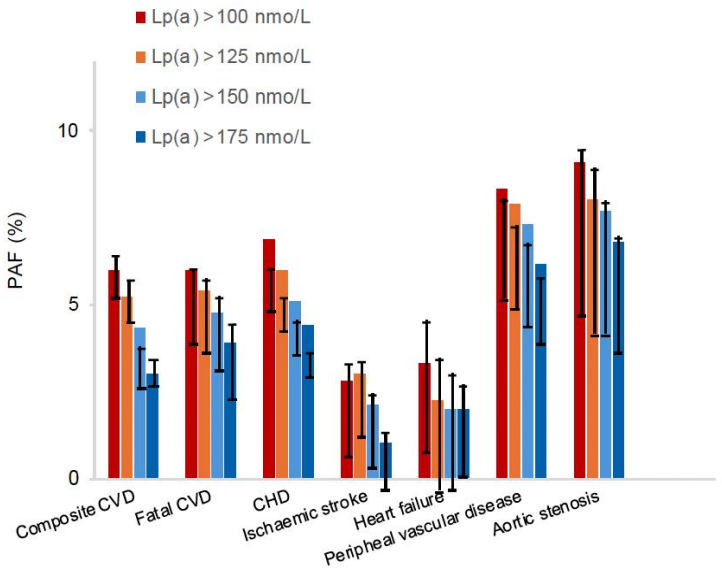
c-LDL, colesterol unido a lipoproteínas de baja densidad;CT, computed tomography; 8F-FDG, fluorine-18 fluorodeoxyglucose; Lp(a), lipoproteina(a); PET,positron emission tomography,

Hippe DS, et al. Arterioscler Thromb Vasc Biol. 2018;38(3):673–678; 2. Leistner D & Laguna-Fernandez A, et al. Eur J Prevent Cardiol. 2024.; 3. n der Valk FM, et al. Circulation. 2016;134(8):611–624. 4. Kaiser Y, et al. J Am Coll Cardiol. 2022;79(3):223–233.

Lp(a) elevada como condición genética causal en el aumento del riesgo de enfermedad CV[1-3]







Los niveles elevados de Lp(a) tienen **implicaciones** en patologías muy prevalentes y en la **mortalidad** por cualquier causa[1]

Aumenta un 50 % el riesgo de mortalidad CV[1]

A medida que **aumenta la Lp(a)** se aumenta el riesgo asociado a diferentes patologías[2]

Aumenta el riesgo de **infarto de miocardio, EVP, ictus, valvulopatías y otras enfermedades**[2]

La **proporción de ECV atribuible a Lp(a) >175nmol/L** es mayor de lo que cabría esperar por su baja prevalencia[3]

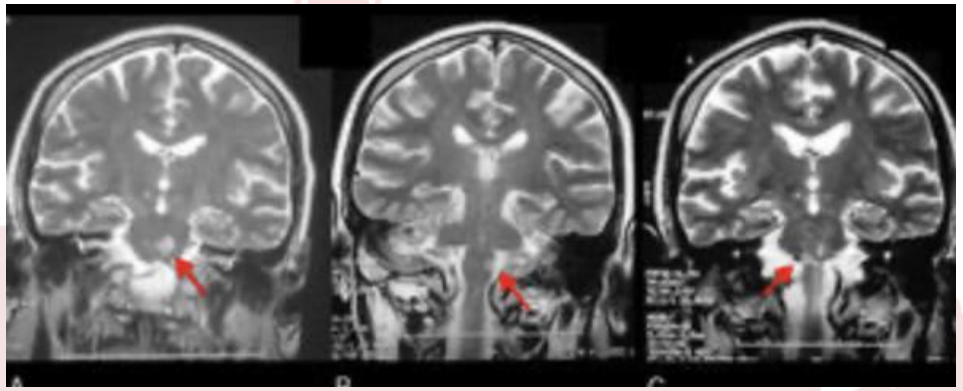
↑ Lp(a) = ↑ impacto en el pronóstico[3]

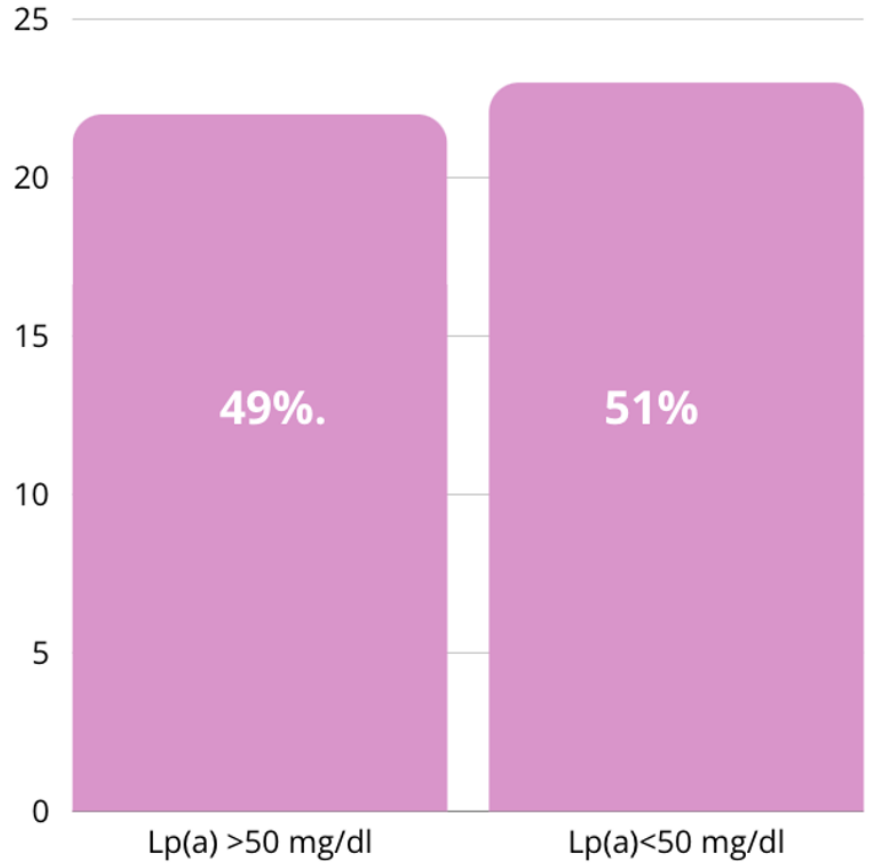
p(a): lipoproteína a; CV: cardiovascular; EVP: enfermedad vascular periférica; PAF, Population attributable fractions; ECV (CVD), enfermedad cardiovadcular; CHD, nfermedad coronaria

. Nordestgaard BG, et al. Lancet. 2024; 404: 1255-1264; 2. Kronenberg K, et al. Eur Heart J. 2022; 43: 3925-3946;
. Welsh P, et al. Eur J Prev Cardiol. 2021; 28: 1991-2000.

- ¿Medirla al menos **una vez en la vida** en adultos?

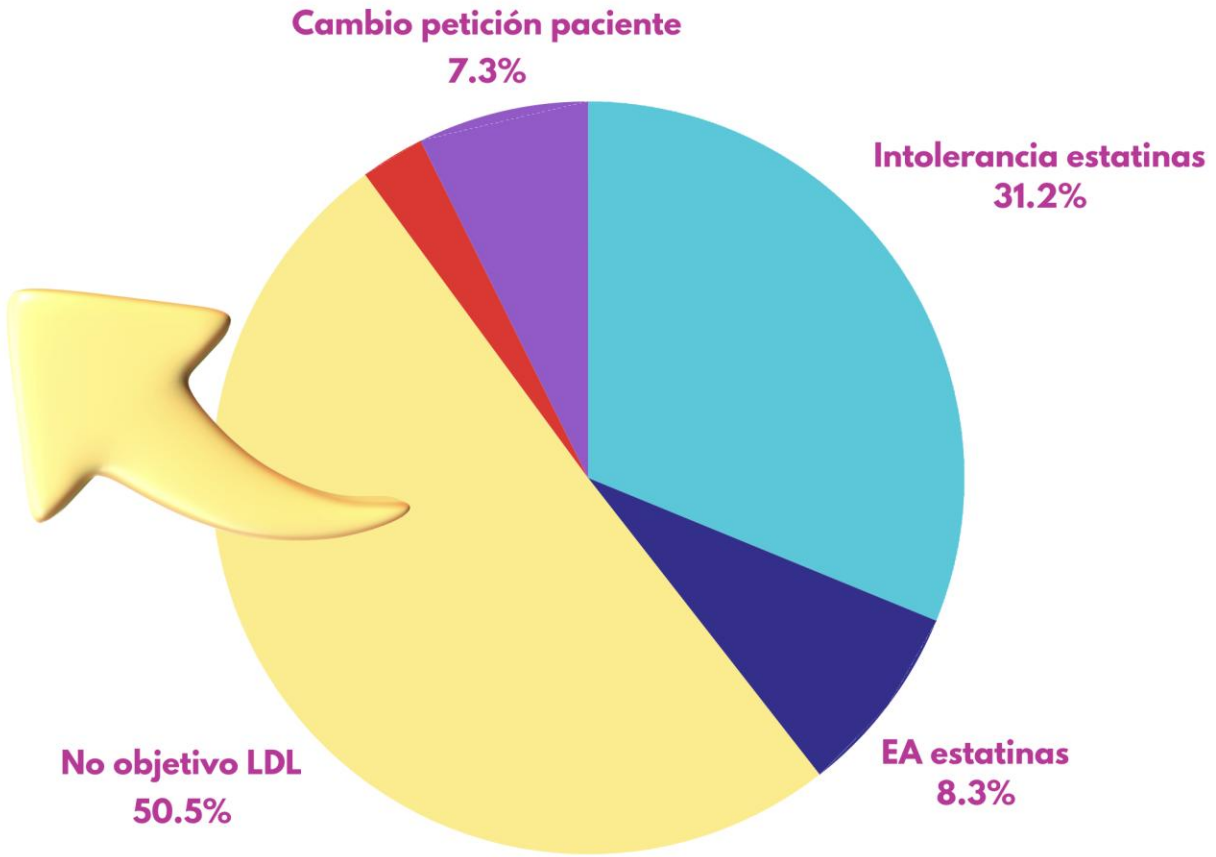
3. ¿Cuándo se debe medir la Lp(a) y a qué pacientes?



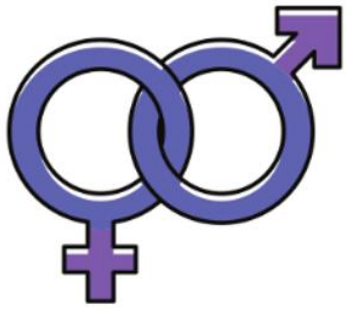


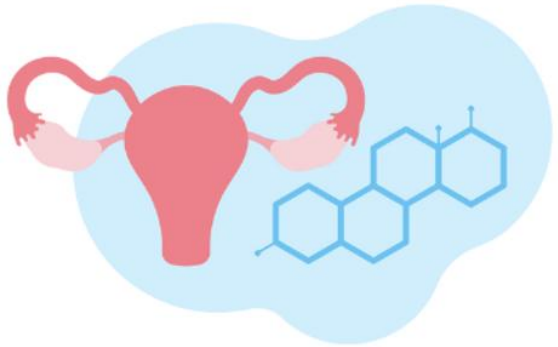
No objetivo LDL

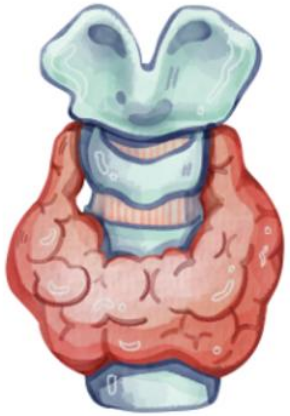
n=114 pacientes

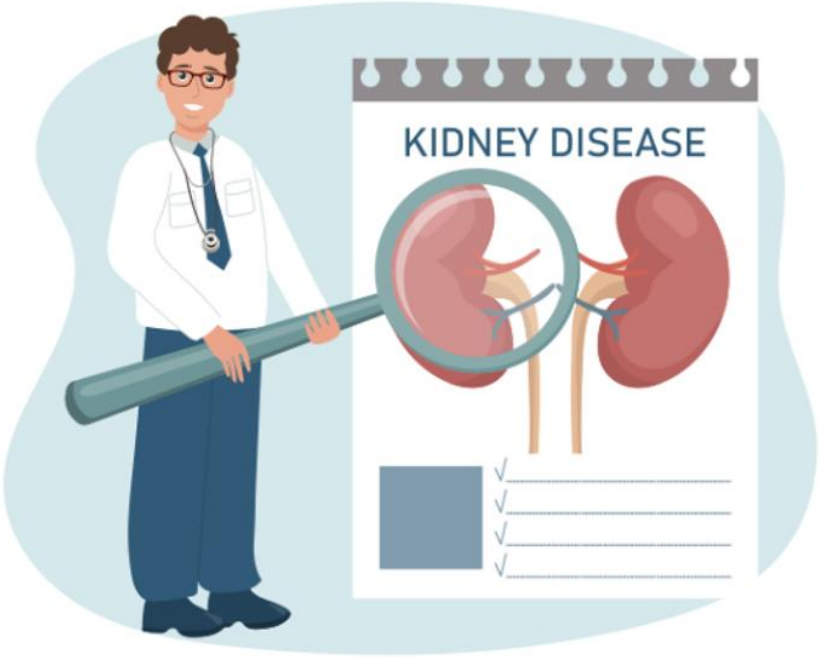


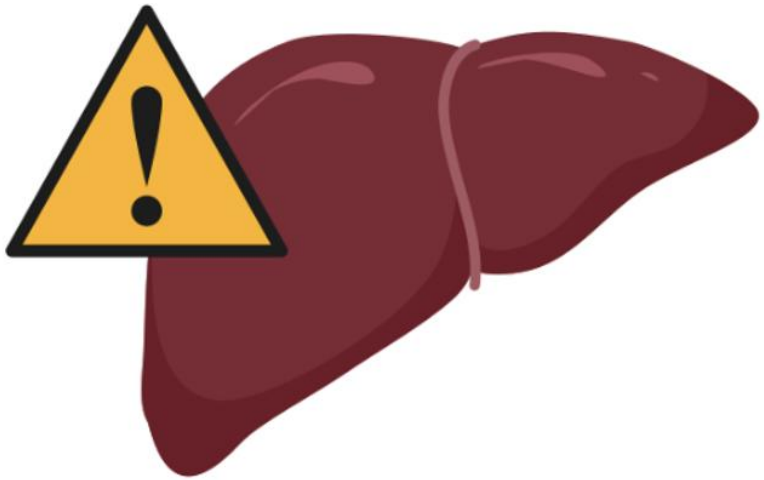
3. ¿Cuándo se debe medir la Lp(a) y a qué pacientes? ¿... una vez en la vida?











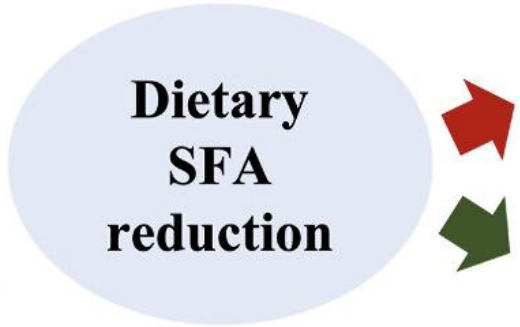
Enkhmaa B. *Atherosclerosis 349 (2022) 53–62*

4. ¿Los hábitos alimenticios y la actividad física modifican los niveles de Lp(a)?

1 Diet

a. Replacement of dietary saturate
b. Low-carbohydrate, high-saturate
c. Diets enriched with walnuts or p
d. Alcohol consumption: No associ

Opposit



Patients	Int
Healthy obese (n = 62)	6-week low
Obese and Type 2 diabetes (n = 131) Obese (n = 30) Type 2 diabetes (n = 26) Obese managed with bariatric surgery (n = 26)	4-month lo bariatric su
Overweight/Obese patients (n = 293)	7-week low followed by bypass) and follow-up ((n = 82) 59-week lo ex
40 obese women	and ex
702 obese patients (372 without metabolic syndrome)	1-year follo sleeve gastr
Single case report of a normoweight subject	Very low ca during phy
164 overweight/obese subjects with mixed dyslipidaemia	20-week we containing different an carbohydra fatty acids (vs. 60–7%)
91 overweight subjects	High-inten training pr high-intens training pr low-carb/h vs. very lov (n = 22) vs. managemen

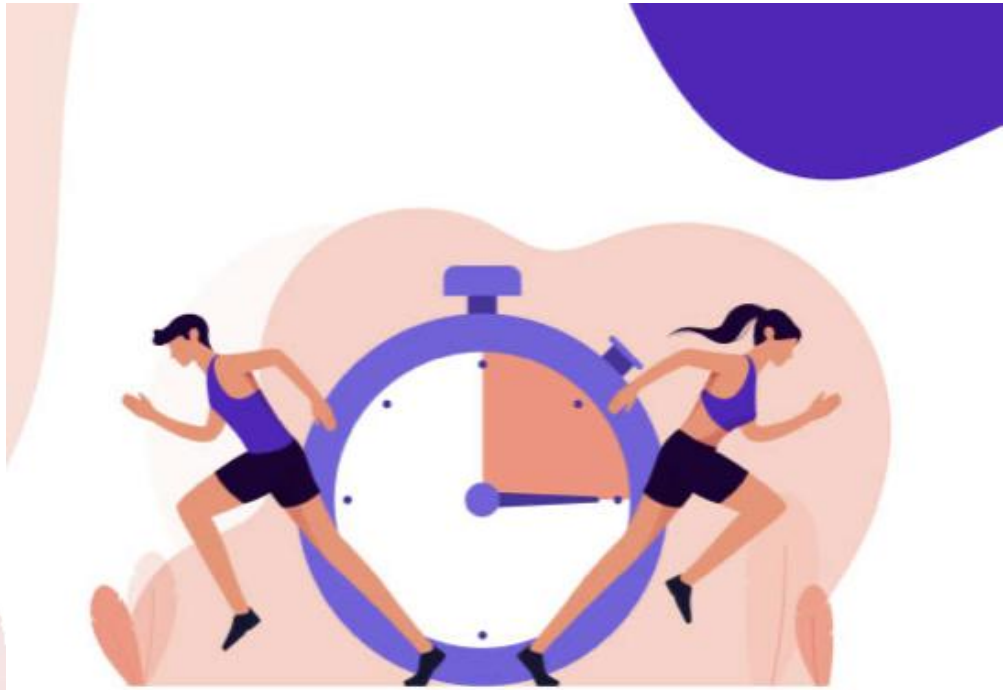
Patients	Interventions	Main Result
31 Overweight/obese with LDL-C > 100 mg/dL	4-week plant-based diet	16% Lp(a) plasma level reduction
194 healthy subjects	8-week regimen of 43 g of walnuts daily vs. standard diet	No change in Lp(a) plasma level in any group
29 overweight or obese individuals	16-week consumption of either 42.5 g/day of mixed nuts (cashew, onds, macadamia, pecans, pist uts) or	No change in Lp(a) plasma level in any group
	fat diet vs. . diet rich atty acids	Lp(a) reduced by 17 mg/L after coconut-oil intake, but increased by 25 mg/dL after intake of unsaturated long-chain fatty acids
	parison of the effect of palm , cocoa butter, extra virgin olive oil as the main oil	No change in Lp(a) plasma level in any group
58 healthy subjects	parison of the effect of high-fat energy)	8–11% decrease in Lp(a) plasma levels with high saturated fatty s; 5% Lp(a) levels in with higher Lp(a) at the with the high trans-diet
31 young men	oil, and butterfat	asma level increase with , but larger with enated fats-enriched diets.
49 hypercholesterolemic subjects	6-week effect of butter or an unsaturated margarine used for cooking or spreading in a reduced fat diet	No change in Lp(a) plasma level in any group
16 young healthy men	5 test fats dominated by (approximately 43% g/kg) stearic, palmitic, oleic, C18:1 trans, or linoleic acid incorporated into meals (1 g fat/kg body weight) after a 12-h fast in random order on different days, separated by 3-week washout periods	Lp(a) plasma levels increased after each fat except oleic and C18:1 trans; oleic, C18:1 trans intake was associated with less area under the plasma Lp(a) concentration curve

Coenzyme Q10

L-Carnitine

4. ¿Los hábitos alimenticios y la actividad física modifican los niveles de Lp(a)?

2 Physical activity and & exercise: No or minimal association



Overall, there is **inconclusive evidence** that standard physical activity improves Lp(a) plasma concentrations,

but some intensive training could be suggested in patients able to afford it.

However, this evidence also is mainly based on the results of small and short-term trials.

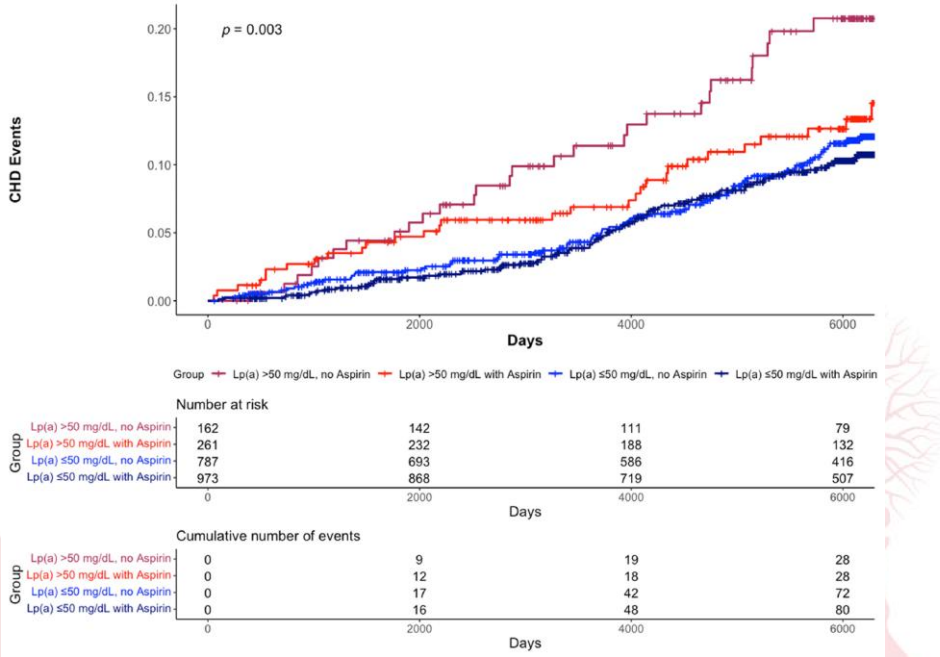
Fogacci F. J. Clin. Med. 2024, 13, 751

5. ¿Es útil la administración de AAS para el tratamiento de la Lp(a) elevada?

Prevención 1ª

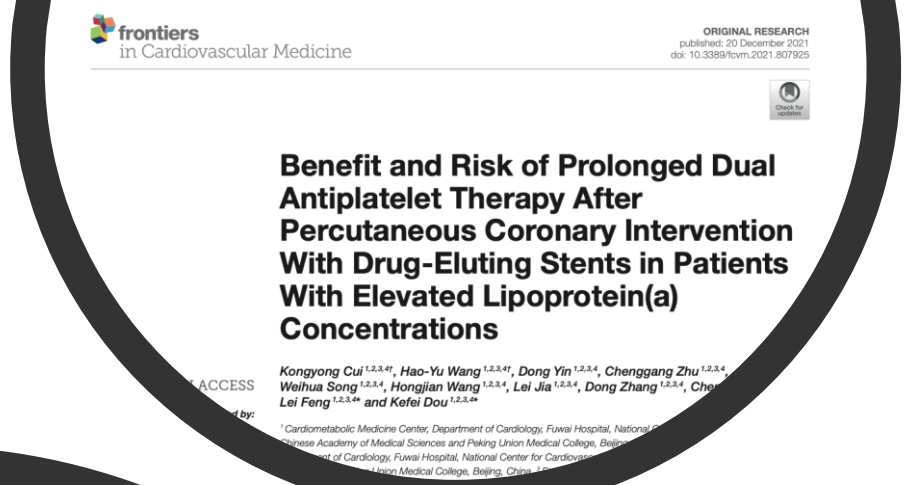
Aspirin and Cardiovascular Risk in Individuals With Elevated Lipoprotein(a): The Multi-Ethnic Study of Atherosclerosis

Author (Year)	Population	Total Participants	Lp(a) measurement	Outcomes with aspirin use	Limitations
Chasman, et al. (2009)[4]	WHS	25,131	rs3798220-C carrier status	HR 0.44 (95% CI 0.20-0.94) for MACE among carriers	Only Caucasian participants, SNP present in 3.7% of individuals, use of genetic instruments
Lacaze, et al. (2022)[5]	ASPREE	12,815	rs3798220-C carrier status and LPA genomic risk score Lp(a)	Net benefit of 8.1 events with aspirin among SNP carriers; net benefit of 1.7 events in highest quintile of GRS	European ancestry, elderly individuals, use of genetic instruments
Bhatia, et al. (2024)[6]	MESA	2,183	Lp(a) >50 mg/dL vs. ≤50 mg/dL	HR 0.54 (95% CI 0.32-0.94) for CHD in Lp(a) >50 mg/dL	Observational, subject to residual confounding and confounding by indication
Razavi, et al. (2024)[7]	NHANES III	2,990	Lp(a) ≥50 mg/dL vs. <50 mg/dL	HR 0.48 (95% CI 0.28-0.83) for ASCVD mortality in Lp(a) ≥50 mg/dL	Observational, subject to residual confounding and confounding by indication



4. Chasman DI. Atherosclerosis 2009;203:371-6.
5. Lacaze P. J Am Coll Cardiol 2022;80:1287-
6. Bhatia HS.. J Am Heart Assoc 2024;13:e033562.
7. Razavi AC. Am J Prev Cardiol 2024;18:100674.

1. Bhatia H.S et al. J Am Heart Assoc. 2024;13:e033562.


frontiers in Cardiovascular Medicine

ORIGINAL RESEARCH
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Benefit and Risk of Prolonged Dual Antiplatelet Therapy After Percutaneous Coronary Intervention With Drug-Eluting Stents in Patients With Elevated Lipoprotein(a) Concentrations

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...ery Disease in Patients
...us Coronary Intervention

...[1]; Chunlan Ma, BS[1]; Yingying Huang, BS[1]; Jingyue Hu, BS[2]; Chunmei Xu, BS[1]; Zhuxin Li, BS[1]; ...D, PhD[3]; Chunyu Zeng, MD, PhD[1,3,4,5,6,7,8]

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Abstract

Background: Elevated lipoprotein(a) (Lp[a]) is a risk factor for first atherosclerotic thrombosis events, but the role of elevated Lp(a) in secondary prevention is controversial. This study aimed to retrospectively investigate the influence of elevated Lp(a) levels on the prognosis of patients with coronary artery disease.

Methods: The team collected and compared clinical information of patients hospitalized during percutaneous coronary intervention (PCI). This study used a multivariate logistic regression model to evaluate the relationships between Lp(a) levels, cardiovascular risk factors, and the prognosis of coronary artery disease in patients undergoing PCI.

Results: There were no statistically significant differences between patients grouped according to Lp(a) level in terms of sex; age; body mass index and obesity; hyperuricemia; smoking; cardiac insufficiency; acute myocardial infarction; multivessel lesion; in-stent restenosis; secondary PCI; apolipoprotein AI level; incidence of high total cholesterol or high low-density lipoprotein cholesterol; or family history of hypertension, diabetes, or coronary artery disease. The average Lp(a) concentration did not statistically significantly decrease after 1 year of statin treatment after PCI. One year after patients began statins, there were no significant differences between Lp(a) groups in the incidence of high triglycerides ($P = .13$), high total cholesterol ($P = .52$), or high ...density lipoprotein cholesterol ($P = .051$). Multivariate logistic regression analysis indicated that diabetes ...2) was associated with in-stent restenosis, whereas diabetes ($P = .02$) and multivessel lesions ($P < .001$ ...sociated with secondary PCI in patients who underwent coronary angiography 1 year after P... ...with normal Lp(a) levels, high Lp(a) levels did not significantly increase the incidence of in... ...secondary PCI in patients who underwent coronary angiography 1 year after PCI.

...ained high concentrations of Lp(a) did not significantly increase the inciden... ...PCI in patients who underwent coronary angiography 1 year after ...

...ary restenosis; lipoprotein(a); percutaneous coro...


Prolonged dual antiplatelet t...
treated acute coronary syndr...
with different lipoprotein(a) conc...

Kongyong Cui[1, 2, 3*], Shaoyu Wu[1, 2, 3*], Dong Yin[1, 2, 3], Weihua ...
Hongjian Wang[1, 2, 3], Chenggang Zhu[1, 2, 3], Lei Feng[1, 2, 3], Yuejin Yan...
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Abstract

Background: *Lipoprotein(a) [Lp(a)] was positively associated with recurrent ischemic events in patients with acute coronary syndrome (ACS). This study was performed to investigate the effect of Lp(a) levels on outcomes of dual antiplatelet therapy (DAPT) > 1 year versus DAPT ≤ 1 year after percutaneous coronary intervention (PCI) in this population.*

Methods: *A total of 4,357 ACS patients who were event-free at 1 year after PCI were selected from the Fuwai PCI Registry, and patients were stratified into four groups according to DAPT duration (≤ 1 year vs. > 1 year) and Lp(a) levels (≤ 30 mg/dL vs. > 30 mg/dL). The primary endpoint was major adverse cardiovascular and cerebrovascular event (MACCE), defined as a composite of cardiac death, myocardial infarction or stroke.*

Results: *After 2.4-year follow-up, the incidence of MACCE (hazard ratio [HR]$_{adjusted}$ 0.284, 95% confidence interval [CI] 0.115–0.700; HR$_{grw}$ 0.351, 95% CI 0.164–0.751) were significantly reduced in DAPT > 1 year group than that in DAPT ≤ 1 year group in individuals with elevated Lp(a) levels. However, in individuals with normal Lp(a) levels, no statistically difference was found between these two groups in terms of MACCE, although the risks of all-cause death and definite/probable stent thrombosis were lower in DAPT > 1 year group. Notably, the risk of clinically relevant bleeding did not statistically differ between these two groups in individuals with different Lp(a) levels.*

Conclusions: *This study firstly demonstrated that extended DAPT (> 1 year) was statistically associated with lower risk of ischemic events in ACS patients with elevated Lp(a) levels after PCI, whereas this association was not found in individuals with normal Lp(a) levels.* (Cardiol J 2024; 31, 1: 32–44)

Keywords: lipoprotein(a), acute coronary syndrome, percutaneous coronary intervention, drug-eluting stent, dual antiplatelet therapy, prognosis

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#Drs. Rui Fu and Kefei Dou contributed equally to this article.


With ST-Segment Elevation Myocardial Infarction

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Little is known about the predictors recurrent ischemic events in patients with ST-segment elevation myocardial infarction (STEMI). This study aimed at investigating the predictors of recurrent myocardial infarction (MI) at long-term follow-up in a real-world STEMI cohort. All consecutive STEMI patients who underwent emergent coronary angiography and primary percutaneous coronary intervention between February 2013 and June 2019 at our institution were included. The primary outcome was recurrent MI; secondary outcomes were all-cause death, target vessel revascularization (TVR), in-stent restenosis, definite stent thrombosis (ST) and non-TVR. The study population included 724 STEMI patients; at median follow-up of 803 (324 to 1,394) days, the primary outcome was reported in 70 patients (10.1%). All-cause death occurred in 6.8%, TVR in 4.2%, in-stent restenosis in 2.5%, and ST in 1.9% of cases. At multivariable analysis, diabetes (hazard ratio [HR] = 1.18), serum level of lipoprotein(a) [Lp(a), HR = 1.01], and angiographic evidence of restenotic lesion (HR = 2.98) resulted independent predictors of recurrent MI. Kaplan-Meier analysis confirmed that diabetes, restenotic lesion, and differential Lp(a) risk range values, identified patients with lower long-term survival free from recurrent MI. Lp(a) level ≥ 30 mg/dL had an incremental prognostic stratification capability in patients with diabetes (HR = 5.34), and in patients with both diabetes and restenotic lesion (HR = 17.07). In conclusion, in this contemporary cohort of STEMI patients, diabetes, Lp (a) serum levels and restenotic lesions were independently associated with recurrent MI at long term. The coexistence of Lp(a) level ≥ 30 mg/dL showed an incremental risk stratification capability, supporting its implementation for long-term prognostic assessment in this high-risk clinical setting. © 2021 Elsevier Inc. All rights reserved. (Am J Cardiol 2021;159:44–51)

...e advances in preventive strategies, antithrom... ...primary percutaneous coronary intervention ... ST-segment elevation myocardial infarc... ...ll associated with a high risk of death ...ing and at long term.[1,2] In previous ...ve had a myocardial infarction (MI) ...r recurrent ischemic events and ...or better prognostic stratifica... ...after the index event.[3,4] ...involved in acute coro... ...novel predictors or ...atify the progno... ...es in patients ...oratory,

angiographical, and PCI parameters are routinely collected by real-world registries, but their prognostic performance at long term is still poorly known. Against this background, we aimed at investigating the predictors of recurrent MI in a contemporary population with STEMI who received primary PCI treatment.

Methods

This was an observational, single-center, cohort study including consecutive patients who underwent primary PCI at our Institution. From February 2014 to June 2019, all consecutive patients with STEMI who underwent urgent/emergent coronary angiography at the University Hospital of Salerno, Italy, were prospectively collected in the institu... ...al ACS register. Only drug-eluting stent (DES) was ...ing the study period. Patients who underwent

5. ¿Es útil la administración de AAS para el tratamiento de la Lp(a) elevada?

Prevención 2ª


...rm outcome after percutaneous
...era of new generation drug-eluting stents

...i Akahori (MD, PhD), Takamasa Tanaka (MD), Koji Yanaka (MD, PhD), ...Kojiro Miki (MD, PhD), Takahiro Imanaka (MD, PhD), ...PhD, FJCC), Masaharu Ishihara (MD, PhD, FJCC) *

...Renal Medicine, Hyogo College of Medicine, Nishinomiya, Japan

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...eceived form 18 February 2022
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...online 28 March 2022

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ABSTRACT

Background: High lipoprotein (a) [Lp (a)] levels are associated with worse long-term outcomes in patients undergoing percutaneous coronary intervention (PCI). However, there are limited studies investigating association between Lp (a) levels and long-term outcomes in the era of new generation drug-eluting stents (DES).
Methods: A total of 495 patients with available data on Lp (a) who underwent PCI for de novo lesions with new generation DES were enrolled between 2013 and 2017. The primary endpoint was the major adverse cardiovascular event (MACE), which was defined as a composite of cardiac death, myocardial infarction, stent thrombosis, clinically driven target lesion revascularization, and revascularization for new lesions during 3 years. Patients were divided into 2 groups according to the Lp (a) level: high Lp (a) group (≥30 mg/dL: n = 109) and low Lp (a) group (30 mg/dL>: n = 386). Multivariate Cox regression analysis was performed to identify the predictors for 3-year MACE.
Results: The incidence of 3-year MACE was significantly higher in high Lp (a) group than low Lp (a) group (33.0% vs. 15.9%, $p < 0.001$). Multivariable analysis showed that Lp (a) level of ≥30 mg/dL was an independent predictor for 3-year MACE (HR 2.01, 95%CI 1.30–3.11, $p = 0.002$).
Conclusion: High Lp (a) level was associated with worse long-term outcome even in the era of new generation DES.

Introduction

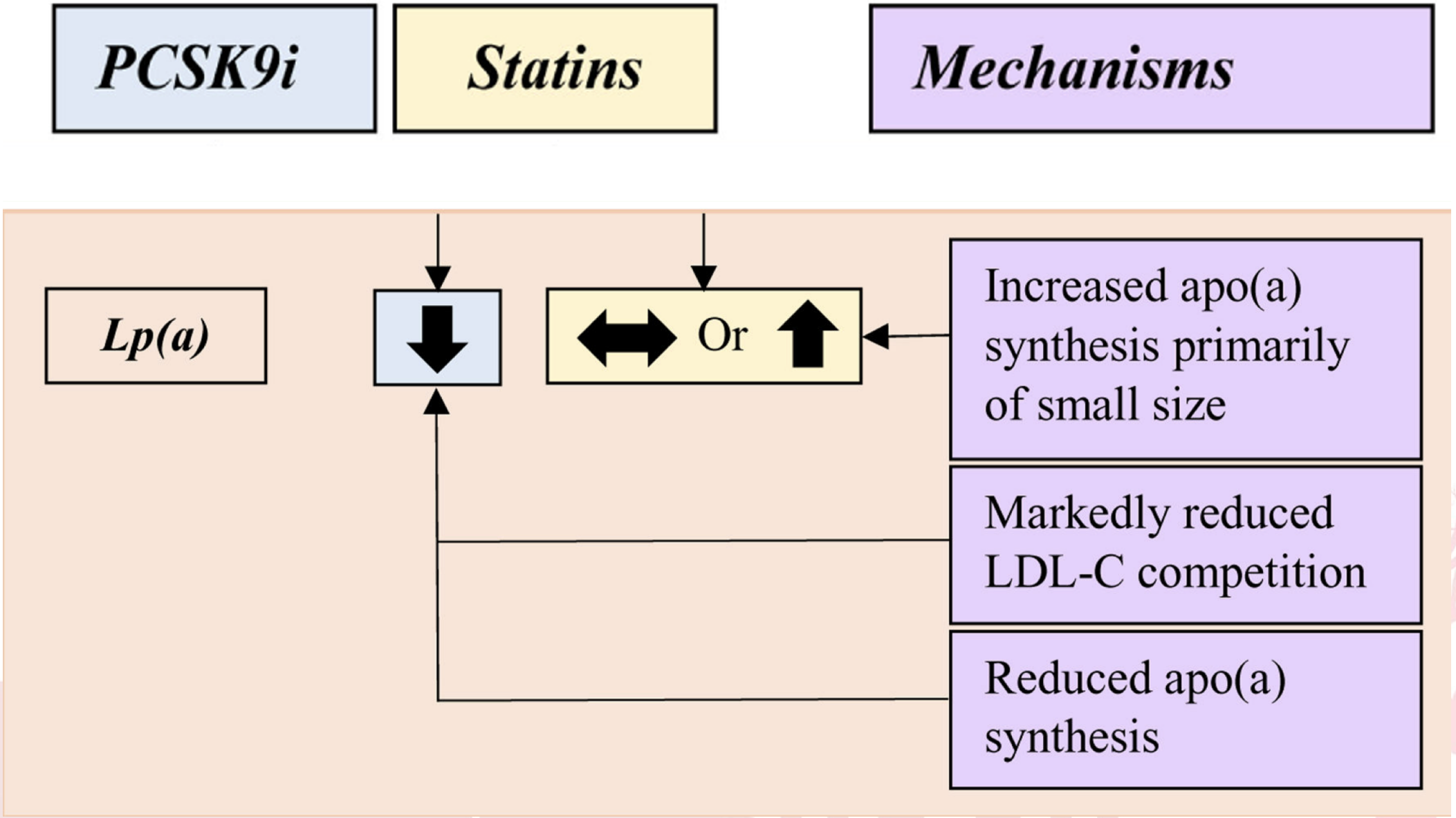
New generation drug-eluting stents (DES) provide better clinical outcomes, including lower incidence of in-stent restenosis (ISR) or stent thrombosis (ST), than balloon angioplasty, bare metal stents (BMS), or first-generation DES because of their improved stent technologies [1–4]. Management of low-density lipoprotein cholesterol (LDL-C), mainly with statin therapy, also provides better long-term outcomes in the era of new generation DES. However, cardiovascular events occur frequently even after lowering LDL-C level. Lipoprotein (a) [Lp (a)] is a modified form of low-density lipoprotein in which the large plasminogen-like glycoprotein apo (a) is covalently bound to apolipoprotein B containing low-density lipoprotein-like segment by a disulfide bridge [5]. Several clinical studies have demonstrated that high Lp (a) levels are associated with ...or long-term outcomes in patients undergoing percutaneous coronary ...ervention (PCI) with balloon angioplasty, BMS, or first-generation DES [6–11] and Lp (a) is one of the residual risk factors [12–15]. However, there are limited data on long-term outcomes in new generation DES era. High Lp (a) level may be associated with worse long-term outcome even for patients with new generation DES implantation. The present study was undertaken to assess whether the Lp (a) level could predict cardiovascular events after PCI with new generation DES.

Methods

This study was a retrospective, observational single-center study. Among consecutive patients with coronary artery disease who underwent PCI at Hyogo College of Medicine from January 2013 to December 2017, 495 patients were enrolled in this study. Inclusion criteria were as follows: 1) patients undergoing PCI for de novo lesions with new generation DES; 2) patients with available data on Lp (a); and 3) surviving patients at discharge. Patients were divided into two groups according to Lp (a) level: high Lp (a) group (≥30 mg/dL: n = 109) and low Lp (a) group (30 mg/dL>: n = 386). Because the ...

...esponding author at: Department of Cardiovascular and Renal Medicine, Hyogo ...

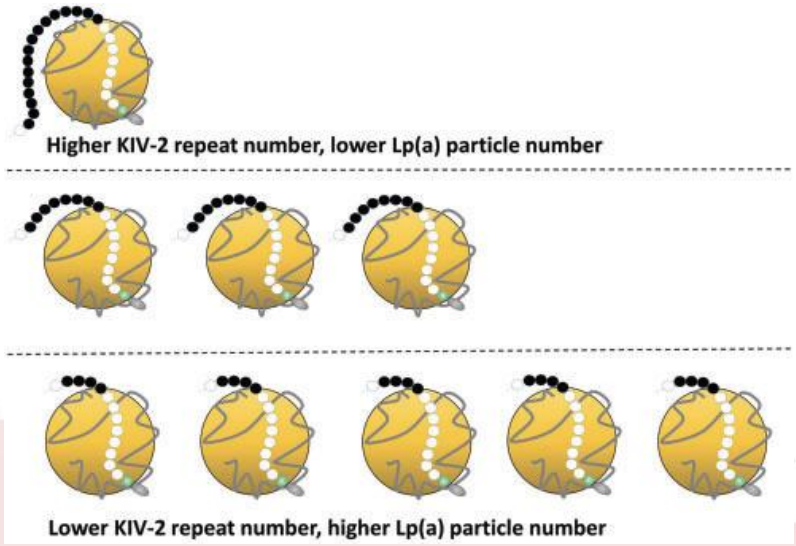
6. ¿Qué ocurre con las estatinas y la elevación de la Lp(a)?

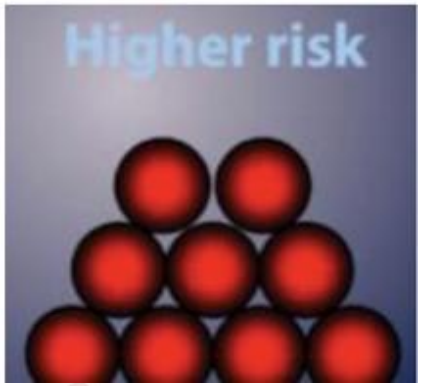


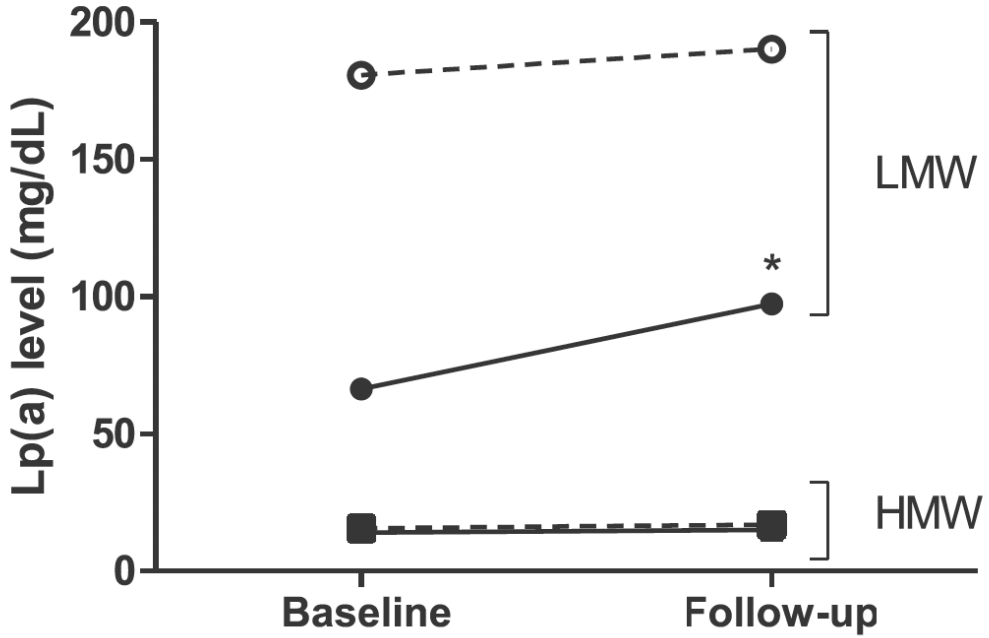
Enkhmaa B. Atherosclerosis. 2019 October ; 289: 173–175.

6. ¿Qué ocurre con las estatinas y la elevación de la Lp(a)?

Statin treatment increases lipoprotein(a) levels in subjects with **low molecular weight apolipoprotein(a)** phenotype







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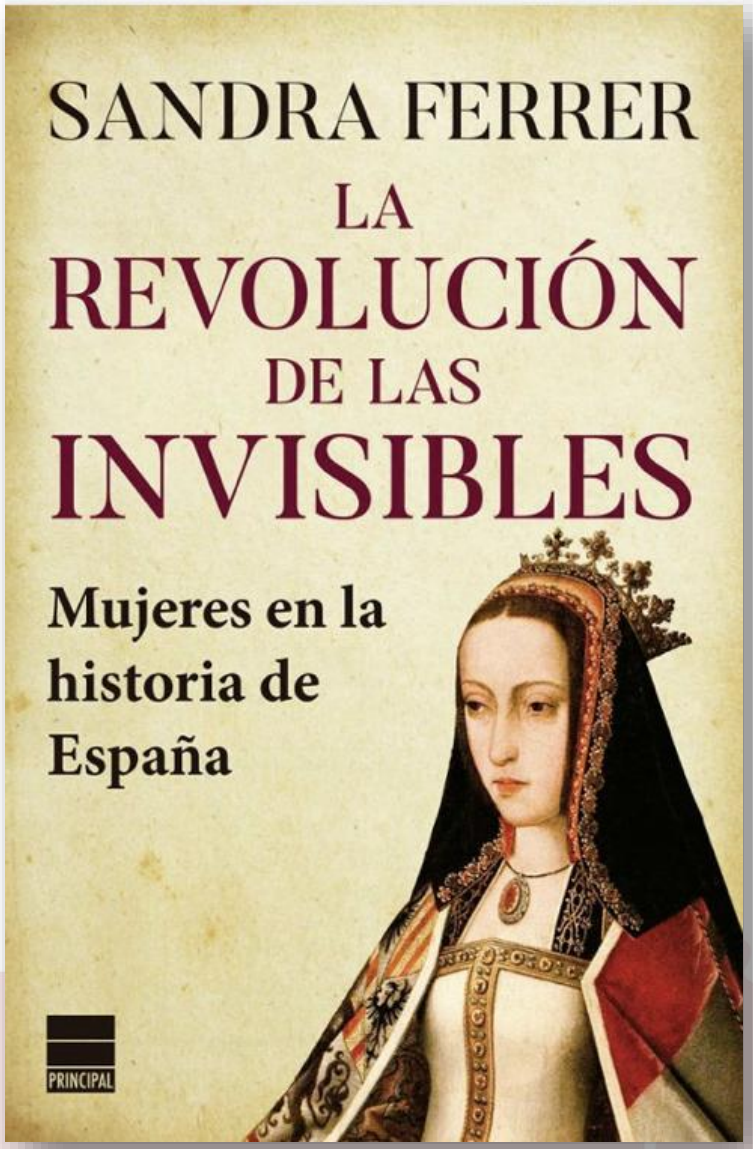
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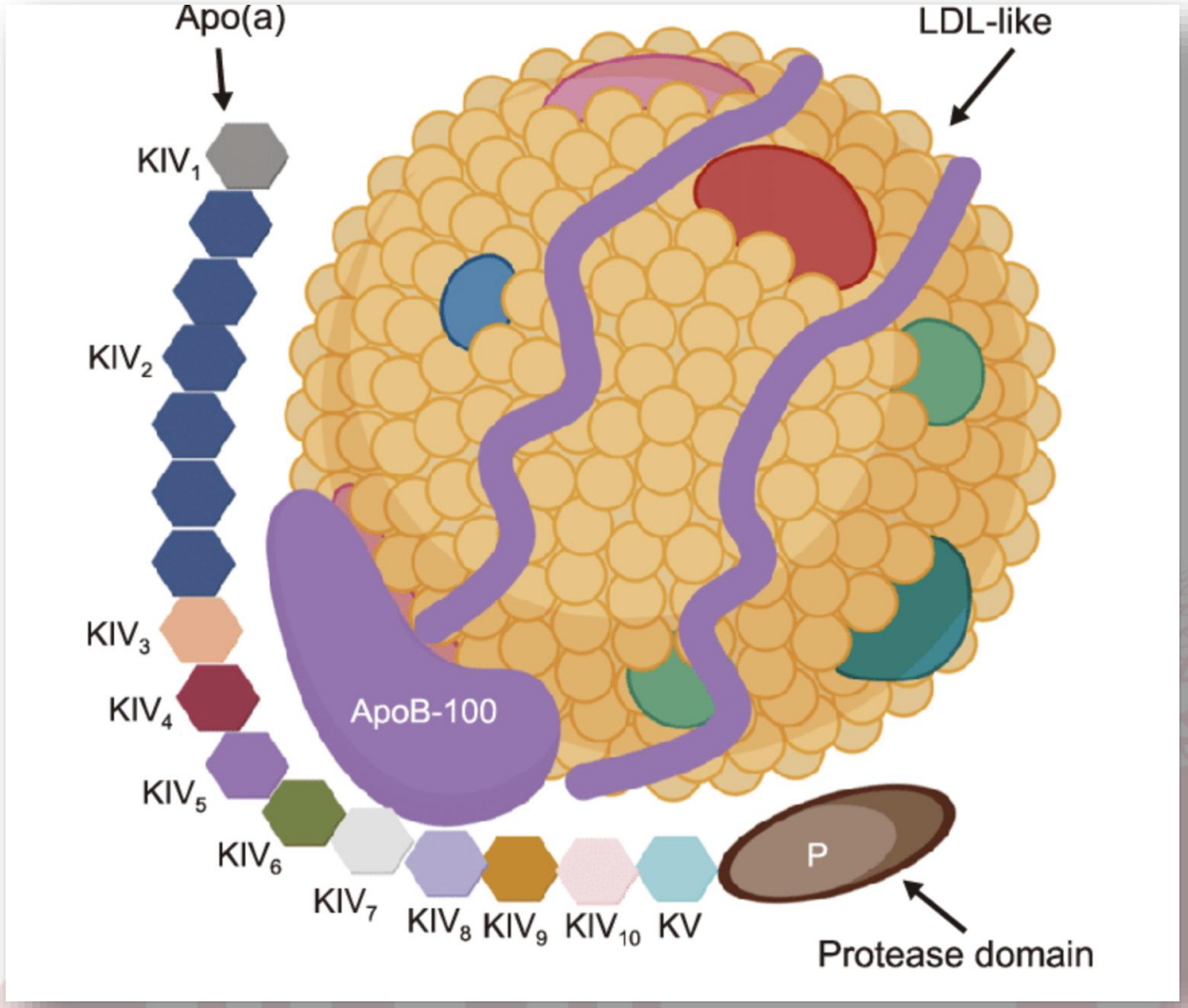
DE LAS
INVISIBLES
Mujeres en la historia de España
PRINCIPAL


Apo(a)
LDL-like
KIV1
KIV2
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KIV5
KIV6
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KV
ApoB-100
P
Protease domain