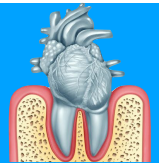
Cuida tu boca y estarás cuidando tu corazón

Dr. Antonio García Quintana, *Servicio de Cardiología del Hospital Universitario de Gran Canaria Doctor Negrín*

29 de Noviembre de 2019 - LAS PALMAS DE GRAN CANARIA



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- Sobre la enfermedad cardiovascular
- Importancia de promover la prevención en cualquier momento
- Relación en entre la enfermedad cardiovascular y la enfermedad periodontal
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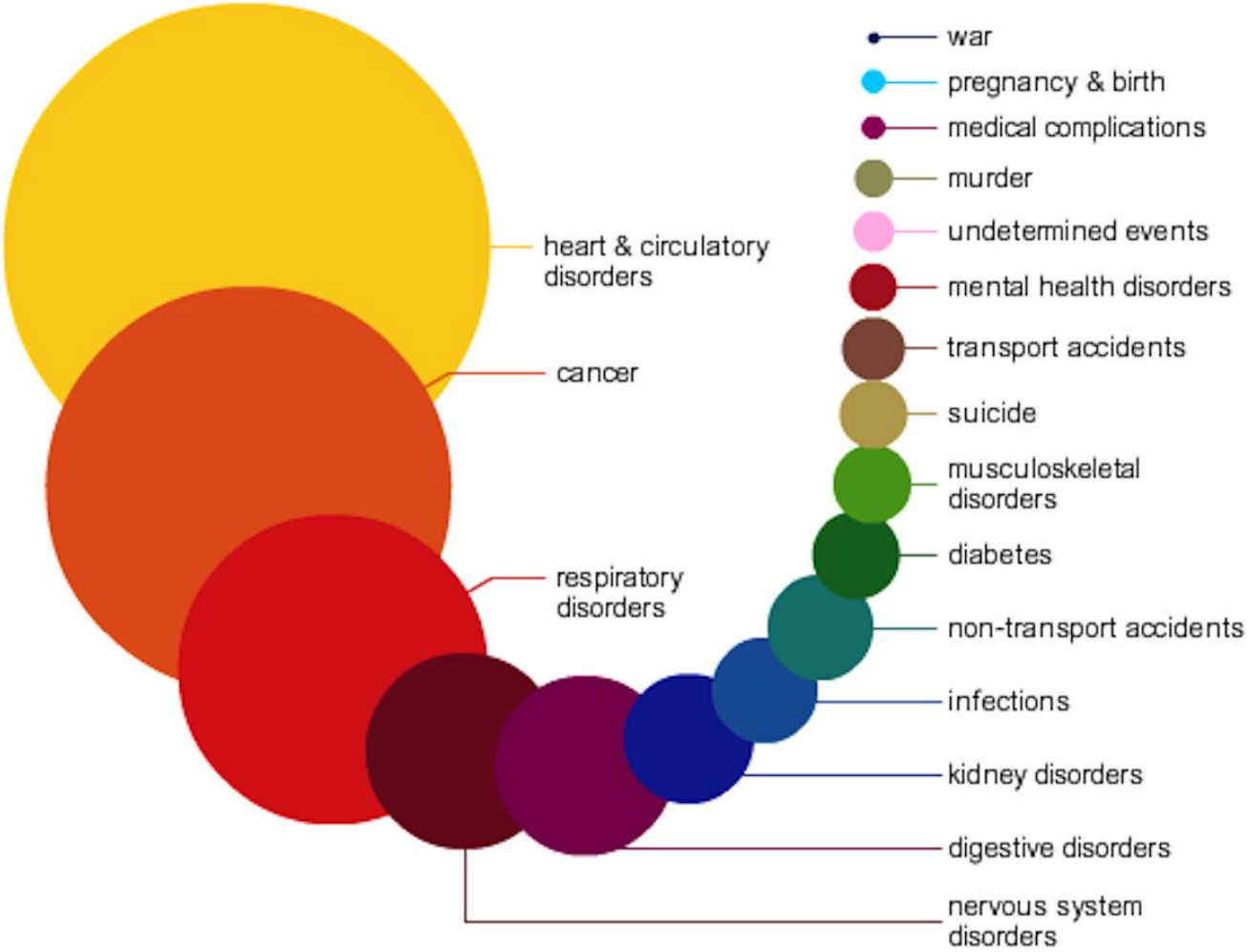
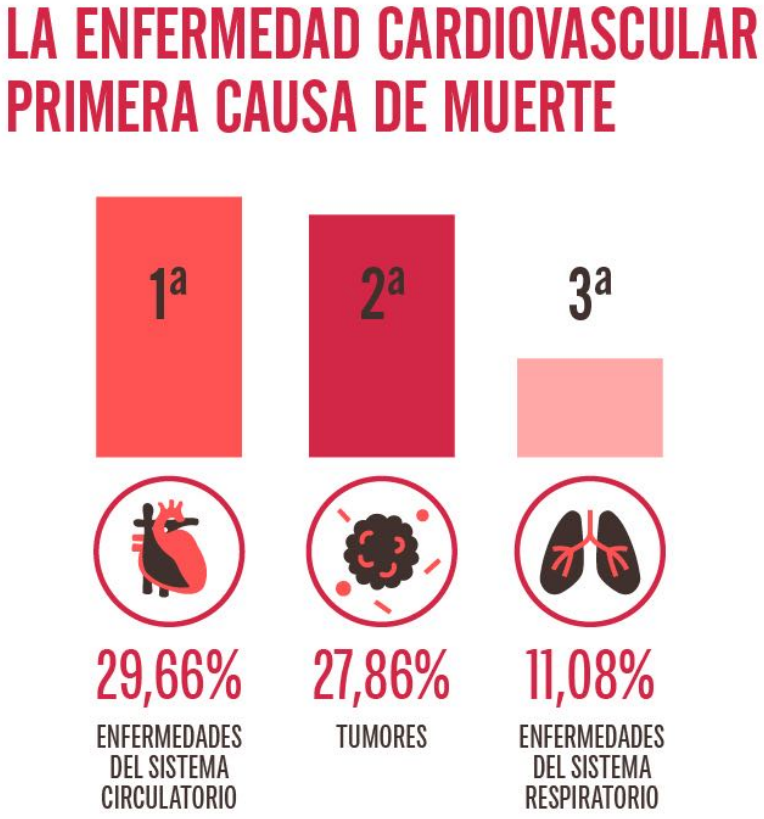
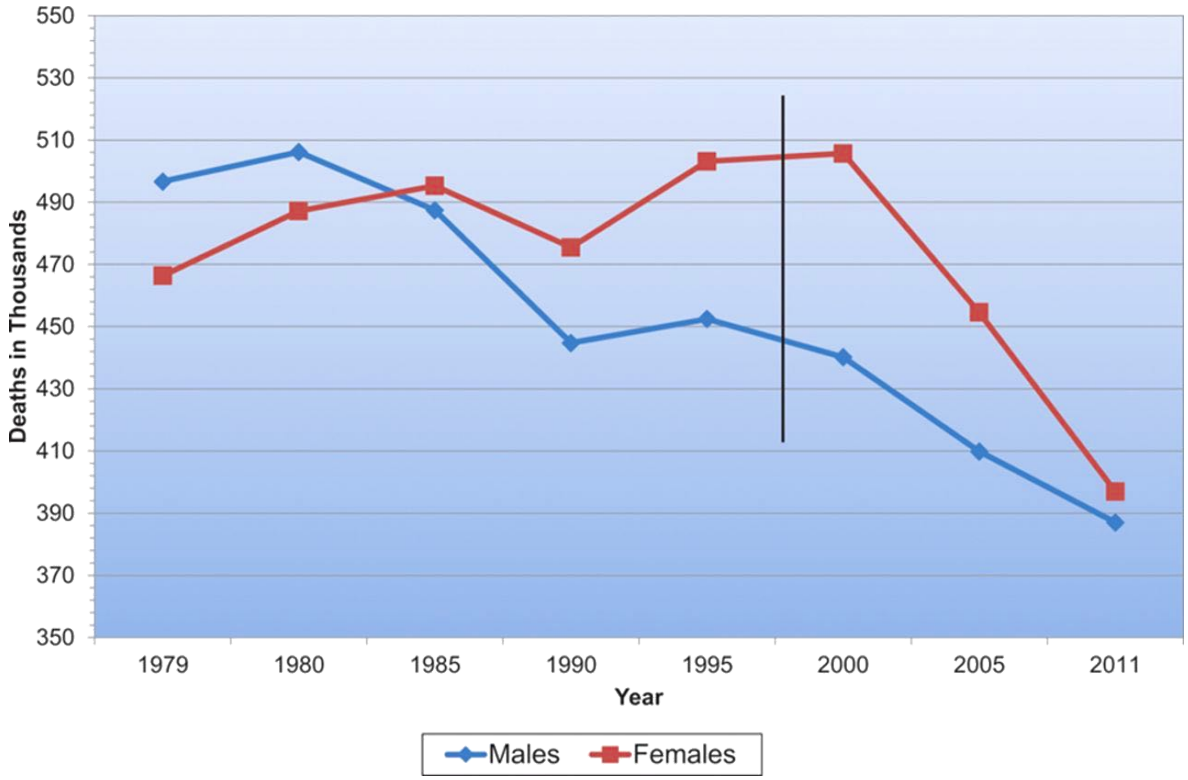
- Estrategias en marcha: Mimoencias, etc.
- Estrategias de prevención

Principales causas de muerte en perspectiva





Tendencia de la mortalidad por enfermedad cardiovascular en hombres y mujeres (1979-2011)



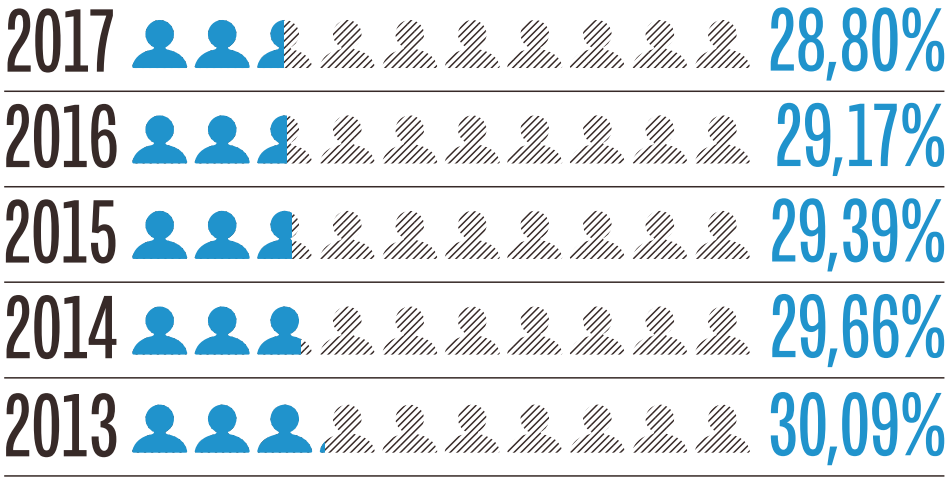
Tendencia de la mortalidad cardiovascular en hombres y mujeres entre 1979 y 2011 en EEUU. Se aprecia una disminución de la mortalidad en relación con las mejoras en la prevención y tratamiento de las complicaciones de la enfermedad cardiovascular. Al aumentar los supervivientes aumenta la prevalencia de la ECV.

American Heart Association.

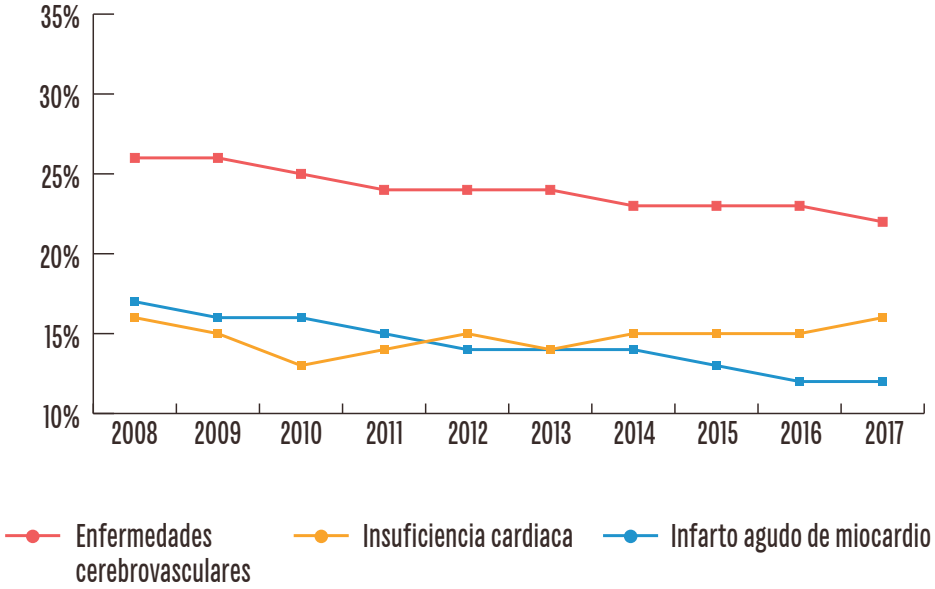
Dariush Mozaffarian et al. Circulation. 2015;131:e29-e322

ECV en España 2017 (INE)

LA TASA DE MORTALIDAD CARDIOVASCULAR DESCIENDE POR DUODÉCIMO AÑO CONSECUTIVO



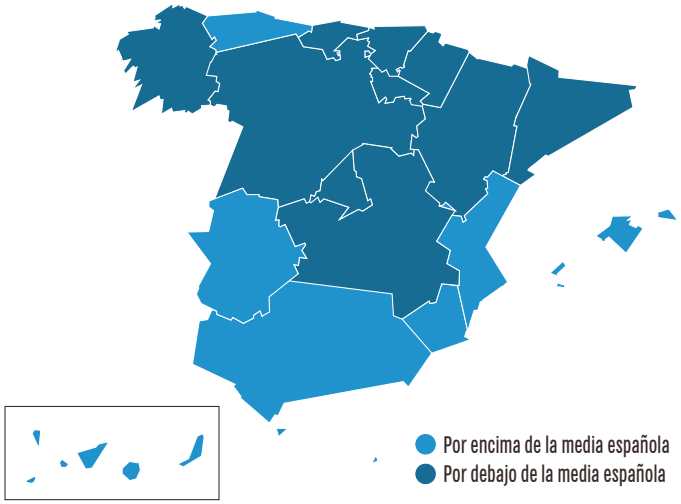
LA INSUFICIENCIA CARDIACA NO CONSIGUE DISMINUIR LA MORTALIDAD



ECV: Según áreas y sexo

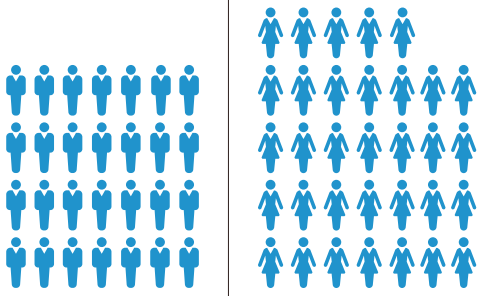
MORTALIDAD CARDIOVASCULAR POR COMUNIDADES AUTÓNOMAS

Tasa media estandarizada de mortalidad cardiovascular: 238,6 (por cada 100.000 habitantes)



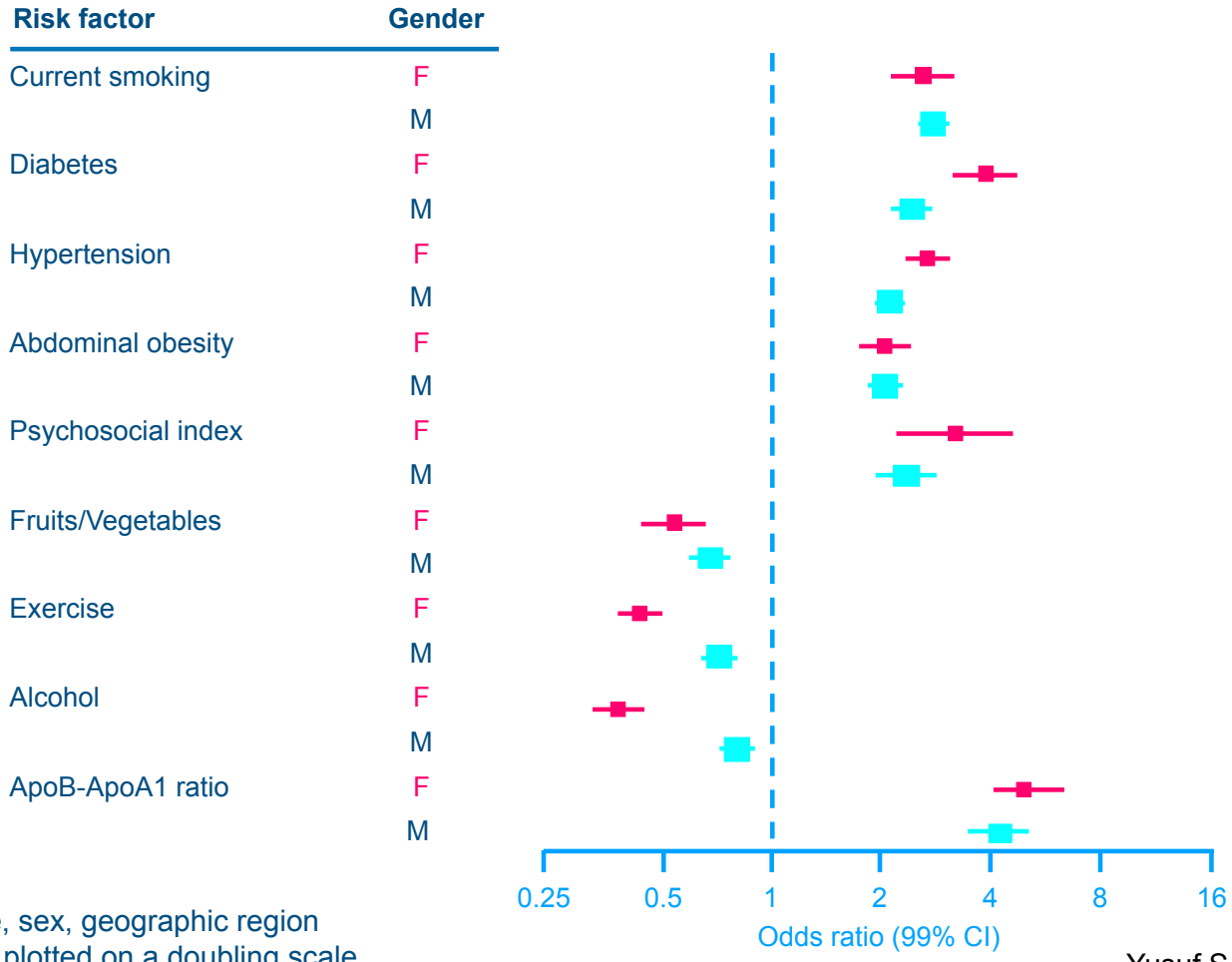
Ceuta	342,4	Aragón	236,6
Andalucía	309,9	Galicia	235,8
Región de Murcia	273,1	Castilla-La Mancha	224,7
Comunidad Valenciana	266,6	La Rioja	223,9
Extremadura	264,3	Cantabria	215,8
Principado de Asturias	252,8	Cataluña	211,3
Islas Baleares	247,2	Castilla y León	208,3
Melilla	245,1	País Vasco	205,5
Islas Canarias	243,7	Comunidad Foral de Navarra	205,1
		Comunidad de Madrid	177,6

MUEREN 10.000 MUJERES MÁS QUE HOMBRES POR ENFERMEDAD CARDIOVASCULAR AL AÑO



	HOMBRES 56.180	MUJERES 66.286
1º	11.555 ENFERMEDADES CEREBROVASCULARES	15.382 ENFERMEDADES CEREBROVASCULARES
2º	10.761 OTRAS ENFERMEDADES DEL CORAZÓN	13.553 OTRAS ENFERMEDADES DEL CORAZÓN
3º	10.221 OTRAS ENFERMEDADES ISQUÉMICAS DEL CORAZÓN	12.201 INSUFICIENCIA CARDIACA

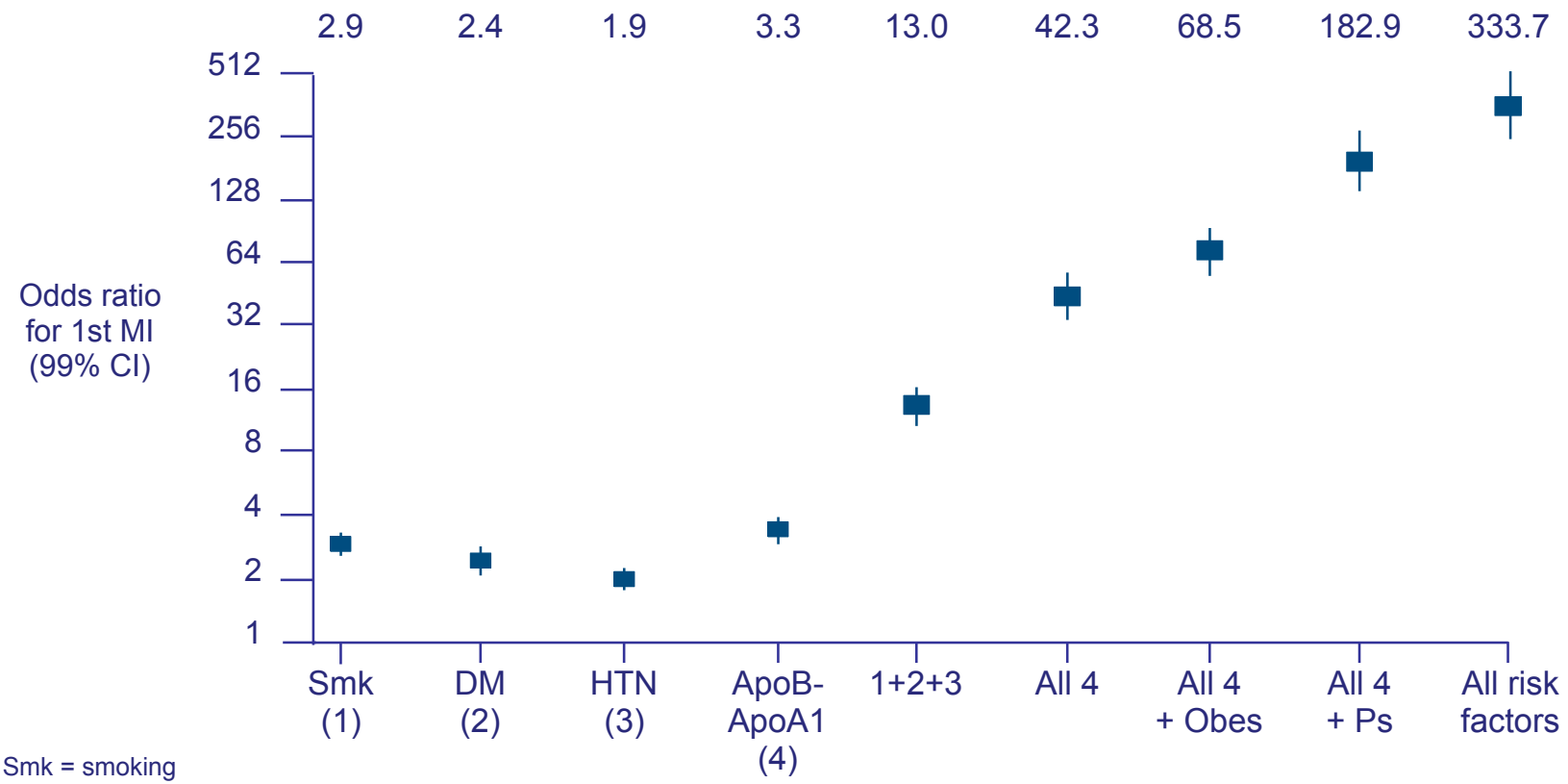
INTERHEART: Factores de riesgo CV



Adjusted for age, sex, geographic region
Note: odds ratio plotted on a doubling scale

Yusuf S et al. *Lancet*. 2004;364:937-52

INTERHEART: Cuantos más FRCV mayor riesgo

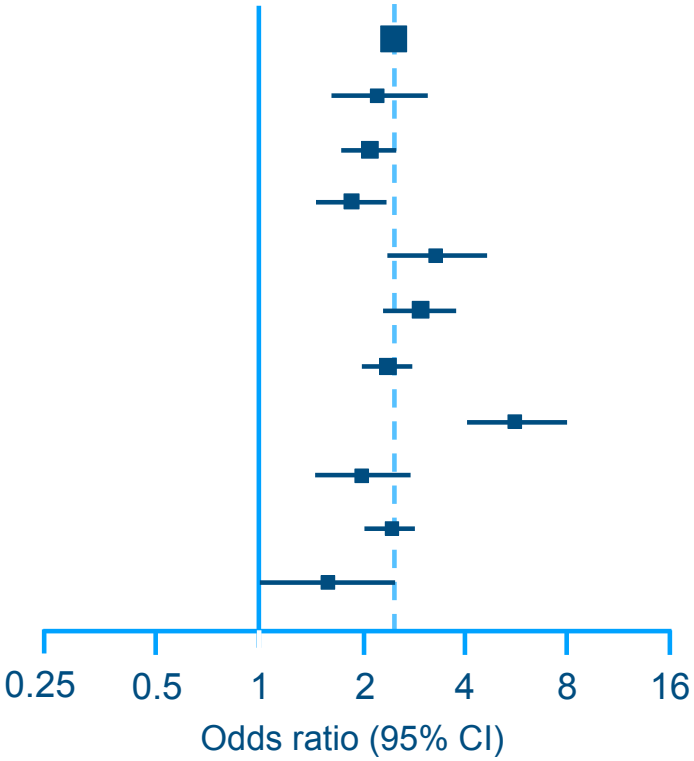


Smk = smoking
DM = diabetes
HTN = hypertension
Obes = obesity
Ps = psychosocial factors
Note: odds ratio plotted on a doubling scale

The Lancet, Volume 364, Issue 9438, 2004, 937 - 952

INTERHEART: Riesgo de MI por áreas

Region	n
Overall	26,916
Western Europe	1425
Central and Eastern Europe	3636
Middle East Crescent	3404
Africa	1355
South Asia	3881
China/Hong Kong	6075
Southeast Asia	2141
Australia and New Zealand	1269
South America	3100
North America	630



Adjusted for age, sex, smoking
Note: odds ratio plotted on a doubling scale

The Lancet, Volume 364, Issue 9438, 2004, 937 - 952

Estimación del Riesgo Cardiovascular

ESC

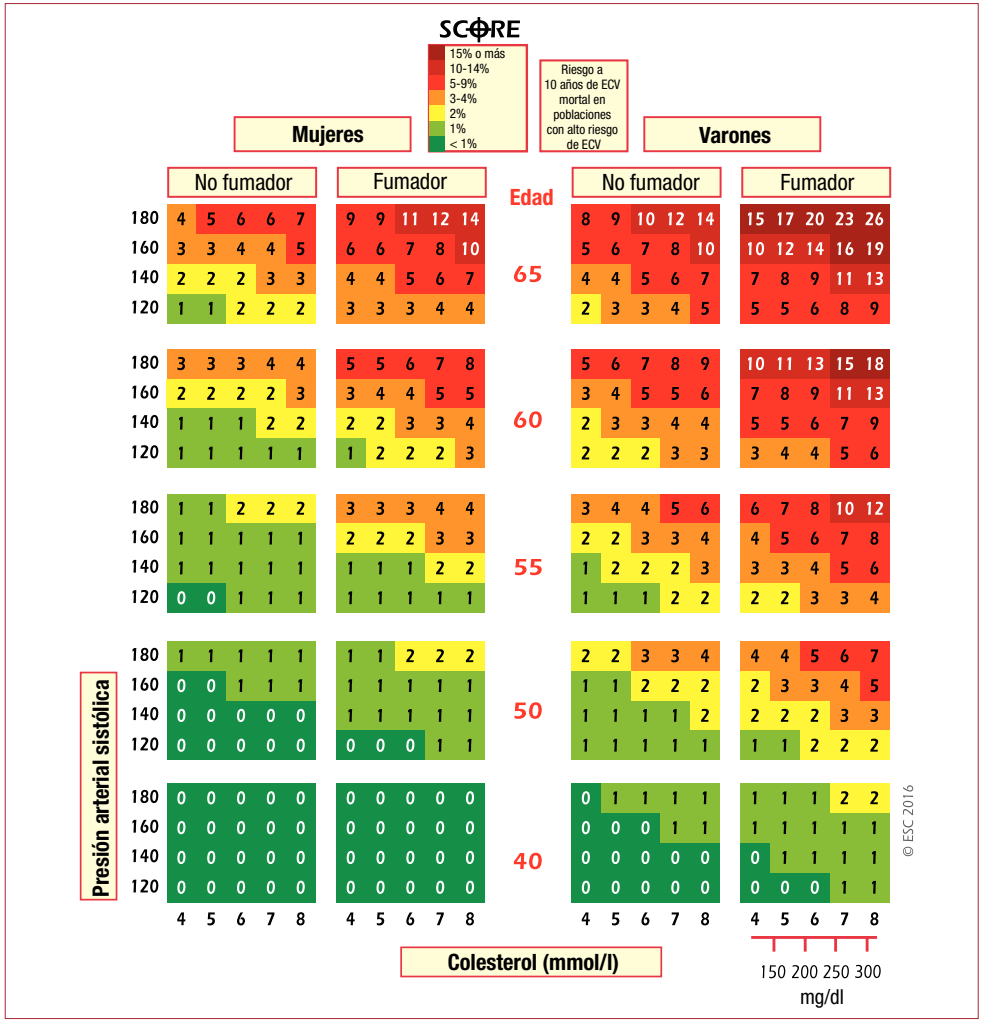
- Presión Arterial
- Sexo
- Edad
- Tabaquismo
- Colesterol

Recomendaciones sobre cómo calcular el riesgo cardiovascular

Recomendación	Clase[a]	Nivel[b]	Ref[c]
Se recomienda calcular el riesgo CV total de los adultos mayores de 40 años usando un sistema de estimación del riesgo como las tablas SCORE, excepto cuando se los clasifique automáticamente como personas en riesgo alto o muy alto basándose en una ECV documentada, DM (> 40 años), enfermedad renal o un único factor de riesgo extremadamente elevado (tabla 5)	I	C	11,25

CV: cardiovascular; DM: diabetes mellitus; SCORE: *Systematic Coronary Risk Estimation.*
[a]Clase de recomendación.
[b]Nivel de evidencia.
[c]Referencias que respaldan las recomendaciones.

Rev Esp Cardiol. 2016;69(10):939.e1-e87



Oportunidades para la intervención

Mensajes clave

- La prevención de la ECV debe practicarse en todos los contextos de atención sanitaria, incluida la atención primaria.
- Cuando sea oportuno, los profesionales de la salud deben evaluar los factores de riesgo CV para determinar el índice de riesgo CV total individual.
- Los MC y las enfermeras deben trabajar en equipo para proporcionar una atención multidisciplinaria más eficaz.

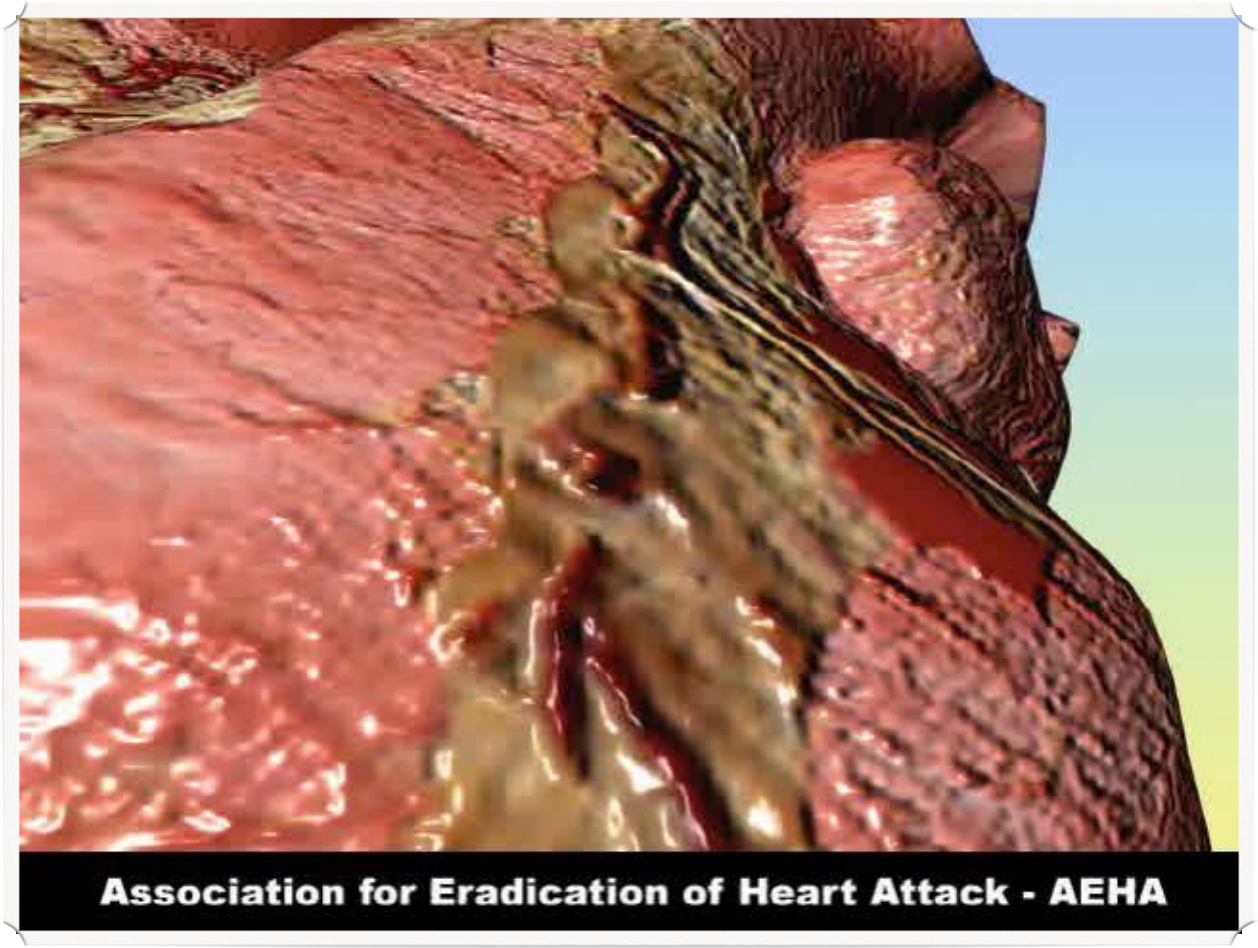
Recomendación sobre prevención de la enfermedad cardiovascular en atención primaria

Recomendación	Clase[a]	Nivel[b]
Se recomienda que los médicos de cabecera, las enfermeras y otros profesionales sanitarios de atención primaria ofrezcan prevención de la ECV a pacientes con alto riesgo	I	C

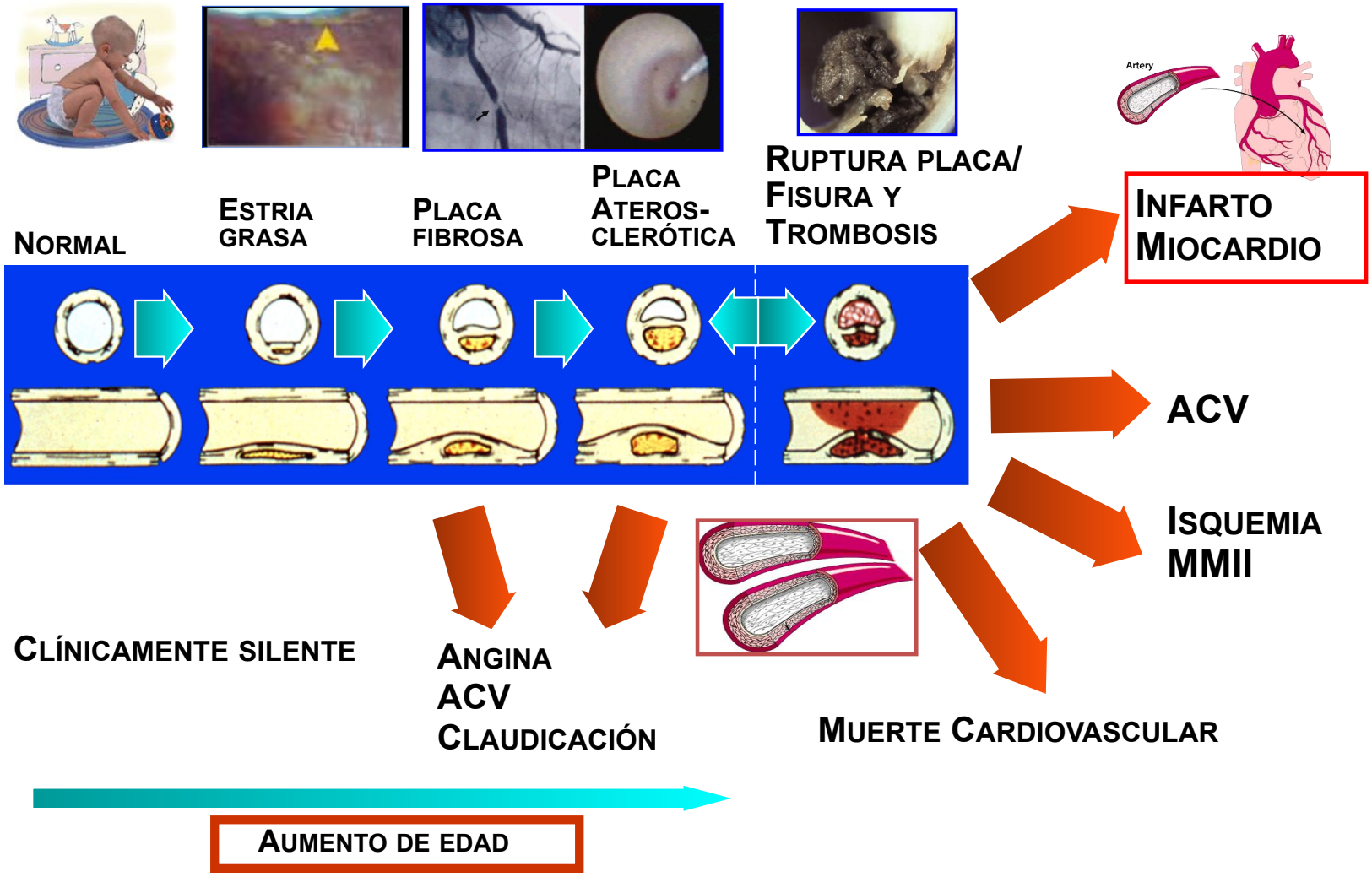
[a]Clase de recomendación.
[b]Nivel de evidencia.

Rev Esp Cardiol. 2016;69(10):939.e1-e87

Fisiopatología de la enfermedad coronaria



Aterogénesis y Aterotrombosis: Un proceso evolutivo



GINGIVITIS Y PERIODONTITIS

GIN-
GIVI-
TIS.

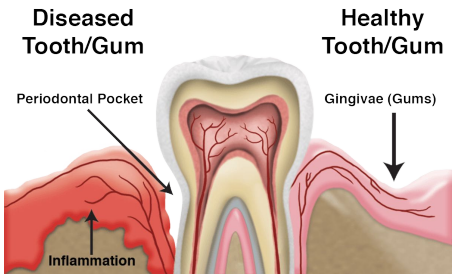
Gingivitis.

Inflamación superficial de la encía.
El sangrado es su principal señal de alerta.
Si no se trata adecuadamente
es su principal señal puede derivar en
Periodontitis.

PERIO-
DON-
TITIS.

Periodontitis.

Infección profunda de la encía
y el resto de tejidos que sujetan el diente.
Puede provocar la pérdida dental.
Repercute en la salud general: aumenta el
riesgo cardiovascular, la descompensación de
la diabetes o el parto prematuro.



FACTORES DE RIESGO ASOCIADOS A

Table 1 Clinical and demographic characteristics of the periodontitis and non-periodontitis participants

	Periodontitis n=9730 (16.2%)	Non-periodontitis n=50 444 (83.8%)	Difference (95% CI)
Age (intake)*	49.6 (8.9)	52.1 (11.2)	2.5 (2.3 to 2.7)
Age (current)*	58.2 (9.2)	59.5 (11.7)	1.3 (1.1 to 1.6)
Male sex†‡	49.2%	45.4%	3.8% (2.8% to 4.9%)
Smoking†	18.8%	5.4%	13.3% (12.5% to 14.1%)
Diabetes mellitus†	6.0%	2.2%	3.8% (3.3% to 4.3%)
Hypertension†	12.3%	4.2%	8.1% (7.4% to 8.7%)
Hypercholesterolaemia†	6.1%	1.9%	4.2% (3.7% to 4.7%)
Low SES†§	51.7%	47.1%	4.8% (3.7% to 5.9%)

*Values are mean in years (SD).
†Values are per cent of participants.
‡Missing data on sex in periodontitis group n=17 (0.2%), non-periodontitis group n=188 (0.4%).
§Missing data on SES in periodontitis group n=68 (0.7%), non-periodontitis group n=551 (1.1%).
SES, social economic status.

Beukers NGFM, et al. J Epidemiol Community Health 2016;0:1–6. doi:10.1136/jech-2015-206745

FACTORES DE RIESGO ASOCIADOS A

Table 2 Univariate and multivariate logistic regression analyses of risk variables for association with ACVD

	All (n=60 174)		≤65 years (n=53 075)		Male (n=27 591)	
	Univariate OR (95% CI)	Multivariate* OR (95% CI)	Univariate OR (95% CI)	Multivariate* OR (95% CI)	Univariate OR (95% CI)	Multivariate* OR (95% CI)
Periodontitis	2.52 (2.25 to 2.82)	1.59 (1.39 to 1.81)	2.80 (2.47 to 3.17)	1.48 (1.28 to 1.70)	2.40 (2.08 to 2.78)	1.61 (1.36 to 1.91)
Age at intake (years)	1.05 (1.04 to 1.05)	1.05 (1.04 to 1.05)	NA	NA	1.06 (1.05 to 1.06)	1.06 (1.05 to 1.06)
Male sex	1.84 (1.65 to 2.05)	1.76 (1.56 to 1.98)	1.79 (1.58 to 2.02)	1.66 (1.46 to 1.90)	NA	NA
Smoking	4.17 (3.68 to 4.73)	2.96 (2.56 to 3.42)	4.98 (4.35 to 5.69)	2.86 (2.46 to 3.32)	3.37 (2.87 to 3.95)	2.63 (2.18 to 3.17)
Diabetes mellitus	12.95 (11.33 to 14.80)	2.57 (2.18 to 3.02)	14.21 (12.22 to 16.53)	2.82 (2.34 to 3.40)	12.16 (10.26 to 14.41)	2.81 (2.28 to 3.46)
Hypertension	15.64 (13.99 to 17.49)	5.56 (4.86 to 6.37)	16.50 (15.52 to 18.75)	6.50 (5.57 to 7.58)	15.07 (13.00 to 17.47)	4.87 (4.08 to 5.81)
Hypercholesterolaemia	20.12 (17.70 to 22.89)	4.61 (3.94 to 5.39)	21.61 (18.70 to 24.99)	4.92 (4.10 to 5.88)	20.33 (17.21 to 24.02)	5.25 (4.29 to 6.41)
Low SES	1.21 (1.09 to 1.34)	1.14 (1.02 to 1.28)	1.26 (1.12 to 1.43)	1.09 (0.96 to 1.24)	1.08 (0.94 to 1.24)	1.07 (0.92 to 1.24)

For multivariate analyses all variables were included in the model.
*Owing to incomplete data for sex and SES, the number of cases included in multivariate analyses were n=59 352, n=52 335, n=27 210, respectively
ACVD, atherosclerotic cardiovascular diseases; NA, not applicable; SES, social economic status.

Beukers NGFM, et al. J Epidemiol Community Health 2016;0:1–6. doi:10.1136/jech-2015-206745

EPO en población general

Table 1. Percentage distribution of subjects classified according to the Community Periodontal Index (CPI), stratified by age, gender, occupation, country of origin, education, net income (monthly), and smoking status. Severity of periodontal disease by age strata: mean number of sextants by CPI

	n	Percentage of subjects who have as highest code (95% Confidence Interval [CI])					*p*-value
		Code 0 (%)	Code 1 (%)	Code 2 (%)	Code 3 (%)	Code 4 (%)	
All subjects	5130	5.4 (4.8–6.1)	7.6 (6.9–8.4)	48.7 (47.3–50.1)	28.3 (27.1–29.5)	10.1 (9.3–11)	
Age group (years)							
<25	459	6.3 (4.4–8.9)	12.6 (9.9–15.9)	65.6 (61.1–69.8)	14.4 (11.5–17.9)	1.0 (0.4–2.4)	<0.001
Mean sextants (95% CI)		*1.81 (1.63–1.98)*	*1.73 (1.60–1.87)*	*2.00 (1.85–2.16)*	*0.42 (0.32–0.52)*	*0.02 (0.00–0.05)*	
25–34	1498	6.5 (5.4–7.9)	10.1 (8.7–11.7)	60.8 (58.3–63.2)	20.0 (18.1–22.1)	2.6 (1.9–3.5)	
Mean sextants (95% CI)		*1.82 (1.73–1.92)*	*1.54 (1.47–1.61)*	*1.92 (1.84–2.01)*	*0.59 (0.53–0.66)*	*0.05 (0.03–0.07)*	
35–44	1455	6.0 (4.9–7.3)	7.4 (6.2–8.9)	48.8 (46.2–51.4)	29.3 (27–31.7)	8.4 (7.1–9.9)	
Mean sextants (95% CI)		*1.64 (1.55–1.74)*	*1.16 (1.10–1.23)*	*1.78 (1.70–1.86)*	*1.08 (1.00–1.16)*	*0.16 (0.13–0.19)*	
45–54	1118	3.9 (2.9–5.2)	4.6 (3.5–6)	36.1 (33.3–39)	36.8 (34–39.7)	18.6 (16.4–21)	
Mean sextants (95% CI)		*1.19 (1.09–1.29)*	*0.93 (0.85–1.01)*	*1.58 (1.48–1.67)*	*1.49 (1.38–1.59)*	*0.38 (0.33–0.44)*	
≥55	599	3.7 (2.5–5.5)	3.2 (2.1–4.9)	28.1 (24.6–31.8)	41.2 (37.3–45.2)	23.9 (20.7–27.5)	
Mean sextants (95% CI)		*0.97 (0.84–1.11)*	*0.74 (0.64–0.84)*	*1.35 (1.22–1.48)*	*1.63 (1.49–1.78)*	*0.48 (0.39–0.57)*	
Gender							
Male	2979	4.1 (3.4–4.9)	5.4 (4.6–6.3)	47.3 (45.5–49.1)	30.4 (28.8–32.1)	12.8 (11.6–14.0)	<0.001
Female	2151	7.3 (6.3–8.5)	10.6 (9.4–12)	50.5 (48.4–52.6)	25.3 (23.5–27.2)	6.3 (5.3–7.4)	
Occupation							
White-collar	2647	7.0 (6.1–8)	9.2 (8.2–10.4)	51.8 (49.9–53.7)	23.7 (22.1–25.4)	8.3 (7.3–9.4)	<0.001
Blue-collar	2483	3.8 (3.1–4.6)	5.8 (4.9–6.8)	45.3 (43.4–47.3)	33.2 (31.4–35.1)	11.9 (10.7–13.2)	
Country of origin							
Spain	4434	5.4 (4.8–6.1)	7.2 (6.5–8)	49.3 (47.8–50.8)	28 (26.7–29.3)	10.0 (9.2–10.9)	0.003
Others	517	6.3 (4.5–8.7)	11.1 (8.7–14.1)	41.9 (37.7–46.2)	29.6 (25.8–33.7)	11.1 (8.7–14.1)	
Education							
Primary school	1296	2.8 (2–3.8)	3.9 (3.0–5.1)	39.9 (37.3–42.6)	35.8 (33.2–38.4)	17.6 (15.6–19.8)	<0.001
Secondary school	2070	4.8 (4.0–5.8)	7.9 (6.8–9.1)	50 (47.8–52.2)	29.2 (27.3–31.2)	8.1 (7.0–9.4)	
University	1580	8.6 (7.3–10.1)	10.4 (9.0–12.0)	53.6 (51.1–56.0)	20.5 (18.6–22.6)	7 (5.8–8.4)	
Net income (monthly)							
<1200€	1466	4 (3.1–5.1)	6.8 (5.6–8.2)	43.9 (41.4–46.5)	32.1 (29.8–34.5)	13.1 (11.5–14.9)	<0.001
1201 to 3600€	2576	6.6 (5.7–7.6)	7.9 (6.9–9.0)	51.2 (49.3–53.1)	26.3 (24.6–28.0)	8.1 (7.1–9.2)	
>3601€	519	4.9 (3.4–7.1)	8.9 (6.7–11.7)	57.5 (53.2–61.7)	19.6 (16.4–23.2)	9.2 (7.0–12.0)	
Smoking status							
Never	2463	7.3 (6.3–8.4)	9.9 (8.8–11.1)	51.7 (49.7–53.7)	23.2 (21.6–24.9)	7.9 (6.9–9.0)	<0.001
Former smoker	652	4.1 (2.8–5.9)	5.6 (4.1–7.6)	40.8 (37.1–44.6)	34.6 (31.0–38.3)	14.9 (12.4–17.8)	
Smoker ≤10 cigarettes/day	707	3.0 (2.0–4.5)	6.3 (4.7–8.3)	51.7 (48.0–55.4)	30.3 (27.0–33.8)	8.7 (6.8–11.0)	
Smoker >10 cigarettes/day	722	1.7 (1.0–2.9)	2.8 (1.8–4.3)	41.8 (38.3–45.4)	39.5 (36.0–43.1)	14.2 (11.8–16.9)	

Code 0, Gingival health; Code 1, presence of gingival bleeding; Code 2, supra or subgingival calculus; Code 3, moderate periodontal pocket (4–5 mm); Code 4, deep periodontal pocket (≥6 mm).

- ☑ **5,4% salud periodontal**
- ☑ **56,3% gingivitis**
- ☑ **38,4% de los sujetos con periodontitis**
- ☑ **Edad >56 años 65,2% con periodontitis**

J Clin Periodontol 2016; 43: 548–556 doi: 10.1111/jcpe.12558

EPO en pacientes con enfermedad coronaria

Periodontal disease in patients with chronic coronary heart disease: Prevalence and association with cardiovascular risk factors

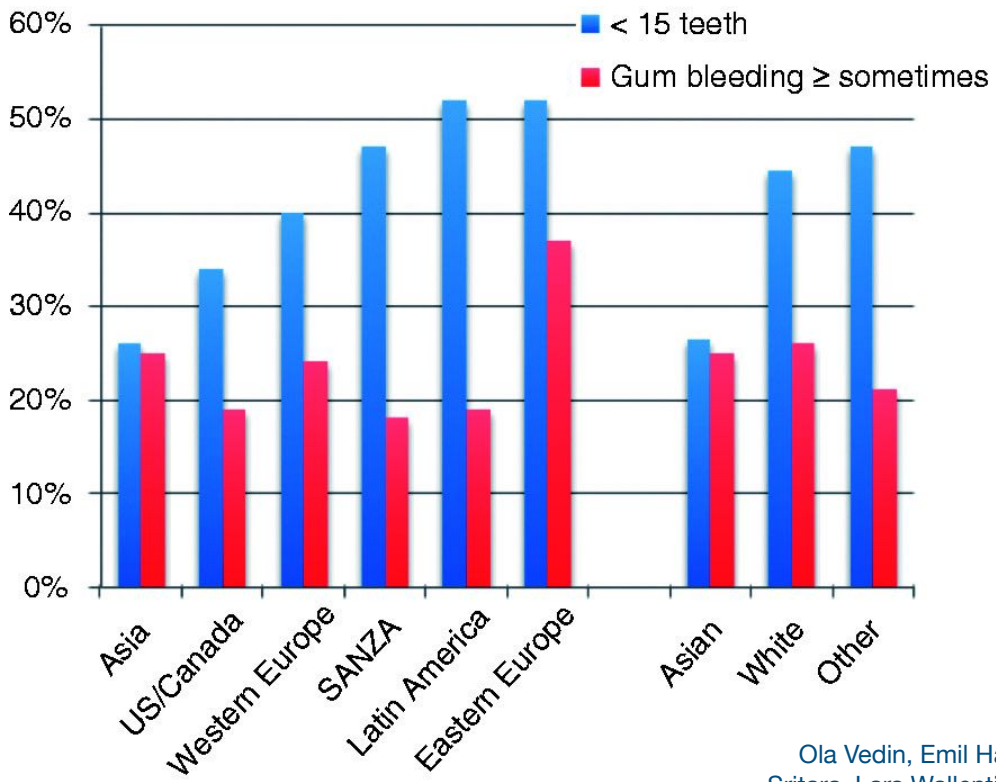


Table 2. Proportion of patients with evidence of periodontal disease by country

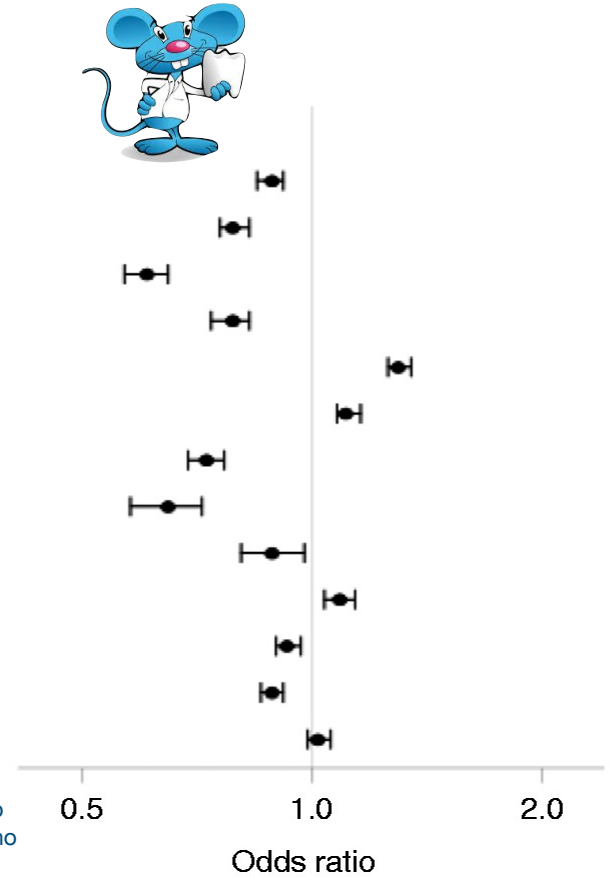
Country	Region	*n* participants (% of total)	% with <15 teeth	% with gum bleeding sometimes/often/always
All countries		15,533 (100%)	40.9%	25.6%
Slovakia	Eastern Europe	118 (0.8%)	68.6%	17.8%
Poland	Eastern Europe	490 (3.2%)	66.7%	28.5%
South Africa	SANZA	383 (2.5%)	62.7%	12.8%
Hungary	Eastern Europe	396 (2.5%)	62.4%	36.8%
Czech Republic	Eastern Europe	767 (4.9%)	60.4%	35.3%
Brazil	Latin America	376 (2.4%)	60.1%	13.6%
Netherlands	Western Europe	444 (2.9%)	59.2%	16.2%
Chile	Latin America	194 (1.2%)	54.1%	26.8%
Argentina	Latin America	537 (3.5%)	53.8%	16.2%
Estonia	Eastern Europe	77 (0.5%)	51.9%	44.2%
Romania	Eastern Europe	408 (2.6%)	49.8%	47.9%
Italy	Western Europe	196 (1.3%)	48.0%	25.0%
Belgium	Western Europe	202 (1.3%)	46.5%	19.9%
Germany	Western Europe	1,063 (6.8%)	46.4%	22.8%
Bulgaria	Eastern Europe	219 (1.4%)	46.1%	38.1%
Philippines	Asia	214 (1.4%)	45.3%	21.3%
Canada	USA/Canada	777 (5.0%)	45.0%	19.6%
New Zealand	SANZA	201 (1.3%)	43.3%	19.8%
Japan	Asia	316 (2.0%)	41.8%	30.2%
Hong Kong	Asia	116 (0.7%)	40.5%	36.8%
Spain	Western Europe	435 (2.8%)	40.5%	35.3%
United Kingdom	Western Europe	183 (1.2%)	39.9%	21.3%

40%

Ola Vedin, Emil Hagström, Dianne Gallup, Megan L Neely, Ralph Stewart, Wolfgang Koenig, Andrzej Budaj, Piyamitr Sritara, Lars Wallentin, Harvey D White and Claes Held European Journal of Preventive Cardiology published online 10 April 2014 DOI: 10.1177/2047487314530660

EPO en pacientes con enfermedad coronaria

Pérdida de dientes



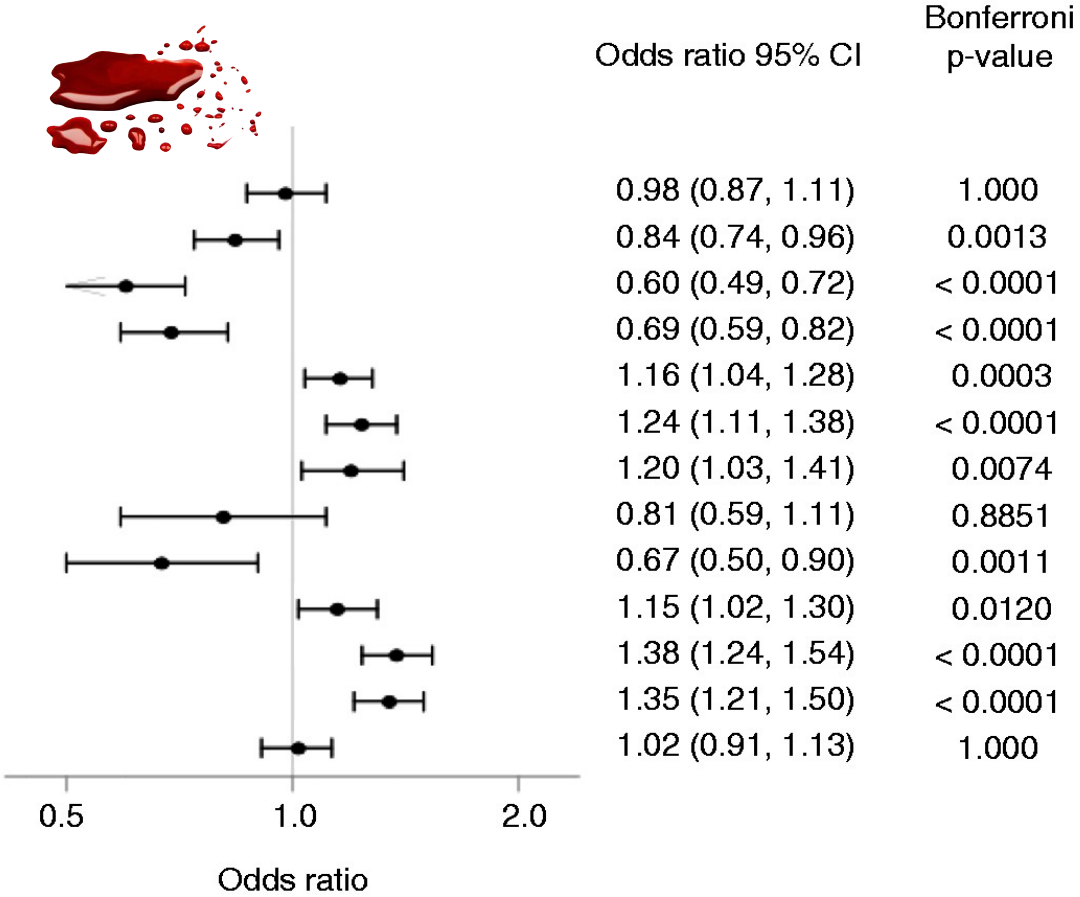
Outcome	Odds ratio 95% CI	Bonferroni p-value
Diabetes	0.89 (0.85, 0.92)	< 0.0001
Former smokers vs. Never smokers	0.79 (0.76, 0.83)	< 0.0001
Current smokers vs. Never smokers	0.61 (0.57, 0.65)	< 0.0001
Current smokers vs. Former smokers	0.79 (0.74, 0.83)	< 0.0001
Education (1)	1.30 (1.26, 1.35)	< 0.0001
Alcohal consumption (drinks/wk) (1)	1.11 (1.08, 1.16)	< 0.0001
White vs. Asian	0.73 (0.69, 0.77)	< 0.0001
Other vs. Asian	0.65 (0.58, 0.72)	< 0.0001
Other vs. White	0.89 (0.81, 0.98)	0.0058
Work stress (1)	1.09 (1.04, 1.14)	< 0.0001
Home stress (1)	0.93 (0.90, 0.97)	< 0.0001
Financial stress (1)	0.89 (0.86, 0.92)	< 0.0001
Leisure physical Activity (1)	1.02 (0.99, 1.06)	1.000

Association between tooth loss and categorical cardiovascular risk factors. Odds ratio (OR) for risk factors when moving from a higher to a lower tooth loss level, e.g. from 'no teeth' to '1-14 teeth'. All ORs adjusted for age, smoking status, educational level and diabetes, except for the following: diabetes: adjusted for age, smoking, and education level; smoking: adjusted for age, diabetes, and education level; education: adjusted for age, smoking, and diabetes. To satisfy proportional odds assumption, education levels of 'none' and '1–8 years' of education were collapsed. Work stress categories of 'do not work' and 'never/rarely work' were also collapsed prior to analysis. [1] Cumulative logit displays odds of higher value of variable vs lower value of variable. CI: confidence interval

Ola Vedin, Emil Hagström, Dianne Gallup, Megan L Neely, Ralph Stewart, Wolfgang Koenig, Andrzej Budaj, Piyamitr Sritara, Lars Wallentin, Harvey D White and Claes Held European Journal of Preventive Cardiology published online 10 April 2014 DOI: 10.1177/2047487314530660

EPO en pacientes con enfermedad coronaria

Sangrado gingival



Association between gum bleeding and categorical cardiovascular risk factors. Odds ration (OR) for risk factors for any gum bleeding vs no gum bleeding. All ORs adjusted for age, smoking status, educational level and diabetes, except for the following: diabetes: model adjusted for age, smoking status, and education level; smoking: model adjusted for age, diabetes, and education; education: model adjusted for age, smoking, and diabetes. To satisfy proportional odds assumption, education levels of 'none' and '1–8 years' of education were collapsed. Work stress categories of 'do not work' and 'never/rarely work' were also collapsed prior to analysis. [1] Cumulative logit displays odds of higher value of variable vs lower value of variable. CI: confidence interval.

Ola Vedin, Emil Hagström, Dianne Gallup, Megan L Neely, Ralph Stewart, Wolfgang Koenig, Andrzej Budaj, Piyamitr Sritara, Lars Wallentin, Harvey D White and Claes Held European Journal of Preventive Cardiology published online 10 April 2014 DOI: 10.1177/2047487314530660

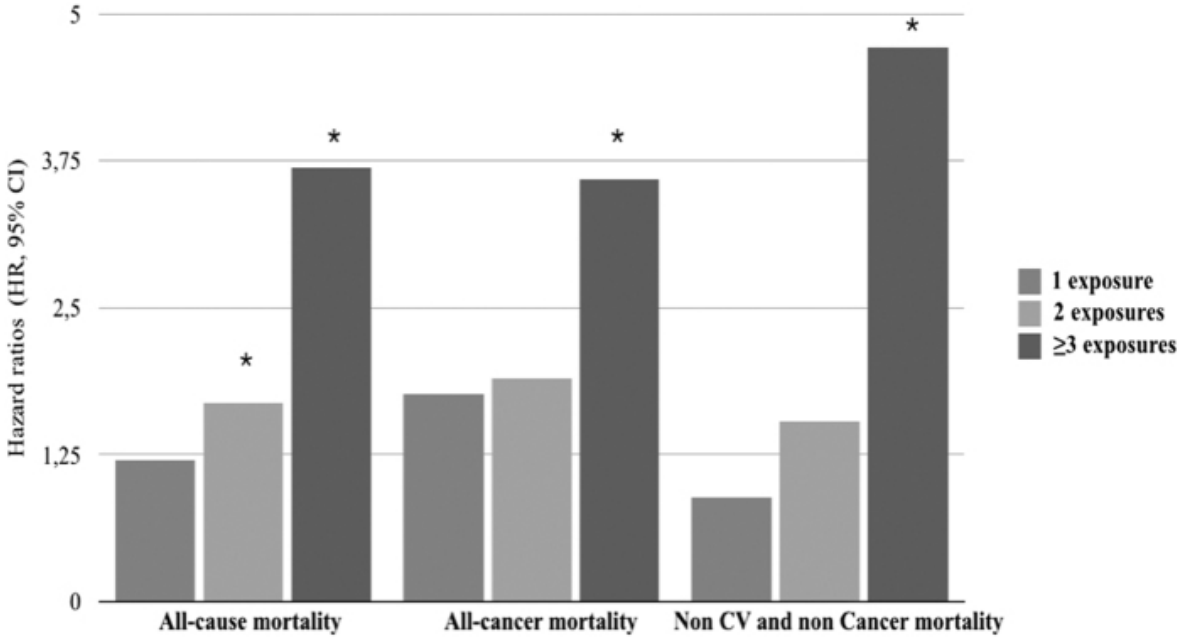
EPO y mortalidad por cualquier causa

	All-cause mortality	All-cancer mortality	Non CV and non cancer mortality
Dental Plaque	2.73 (2.19–3.40)	2.36 (1.32–4.22)	3.30 (1.76–6.17)
Dental Calculus	1.12 (0.92–1.38)	1.16 (0.70–1.93)	1.03 (0.59–1.77)
Gingival Inflammation	1.68 (1.38–2.05)	1.92 (1.18–3.12)	2.86 (1.71–4.79)
Missing teeth >10	2.02 (1.73–2.37)	1.49 (0.95–2.34)	2.31 (1.40–3.82)
Functional Masticatory Units <5	1.96 (1.68–2.29)	1.15 (0.74–1.79)	2.40 (1.55–3.73)

Table 3. Hazard Ratios (HR, 95%) for all-cause, all-cancer and non CV and non cancer mortality in case of High Amount of Dental Plaque, Dental Calculus, Gingival Inflammation and Masticatory Efficiency status (Propensity score model).

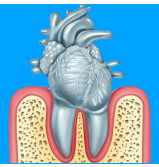
Dental exposure	All-cause mortality n = 370	All-cancer mortality n = 184	Non CV and non cancer mortality n = 129
0 n = 15222 (reference group)	1	1	1
1 n = 4242	1.21 (0.88–1.67)	1.77 (0.76–4.09)	0.89 (0.36–2.20)
2 n = 1600	1.69 (1.16–2.46)	1.90 (0.72–5.0)	1.54 (0.54–4.4)
≥3 n = 1022	3.69 (2.51–5.42)	3.59 (1.23–10.5)	4.71 (1.74–12.7)
Increase of 1 dental exposure	1.71 (1.62–1.81)[b]	1.56 (1.34–1.81)[b]	1.86 (1.49–2.16)[b]

Table 4. Hazard Ratios (HR, 95% CI) for all-cause, all-cancer, and non CV and non cancer mortality depending on cumulative dental exposure (dental plaque, dental calculus, gingival inflammation, functional Masticatory Units <5 and missing teeth >10) (Propensity score model).



Histogram representing Hazard Ratios (HR, 95% CI) for all-cause, all-cancer, and non CV and non cancer mortality depending on cumulative dental exposure (dental plaque, dental calculus, gingival inflammation, functional Masticatory Units <5 and missing teeth >10) (Propensity score model).

Adolph, M., Darnaud, C., Thomas, F., Pannier, B., Danchin, N., Batty, G. D., & Bouchard, P. (2017). Oral health in relation to all-cause mortality: the IPC cohort study. Scientific reports, 7, 44604. doi:10.1038/srep44604



Mala salud oral y riesgo de ECV

No: of natural teeth left	Events/n	Rate	HR[1](95% CI)	HR[2] (95% CI)	p_{trend}
IHD					
20	1812/117464	4.1	1	1	
10-19	708/30013	6.3	1.15 (1.06-1.26)	1.05 (0.96-1.15)	0.002
1-9	354/11423	8.2	1.38 (1.22-1.55)	1.20 (1.06-1.35)	
None	258/8797	7.7	1.27 (1.11-1.45)	1.10 (0.95-1.26)	
Heart Failure					
20	67/117464	0.2	1	1	
10-19	55/30013	0.5	2.18 (1.52-3.13)	1.50 (1.04-2.18)	
1-9	43/11423	1.0	3.67 (2.47-5.45)	2.04 (1.35-3.09)	0.0005
None	38/8797	1.1	3.88 (2.56-5.88)	1.97 (1.27-3.07)	
PVD					
20	127/117464	0.3	1	1	
10-19	98/30013	0.9	2.11 (1.62-2.76)	1.67 (1.27-2.19)	
1-9	53/11423	1.2	2.58 (1.86-3.59)	1.73 (1.23-2.44)	<.0001
None	65/8797	1.9	3.92 (2.87-5.37)	2.53 (1.81-3.52)	
Ischaemic Stroke					
20	145/117464	0.3	1	1	
10-19	72/30013	0.6	1.32 (0.99-1.77)	1.11 (0.72-1.73)	
1-9	27/11423	0.6	1.08 (0.71-1.64)	0.90 (0.59-1.40)	0.8
None	27/8797	0.8	1.31 (0.86-1.99)	1.20 (0.90-1.62)	
All Cause Mortality					
20	807/117464	1.6	1	1	
10-19	448/30013	3.5	1.58 (1.40-1.78)	1.27 (1.13-1.43)	
1-9	308/11423	6.2	2.46 (2.15-2.82)	1.67 (1.45-1.93)	<.0001
None	251/8797	6.6	2.47 (2.13-2.86)	1.60 (1.37-1.87)	

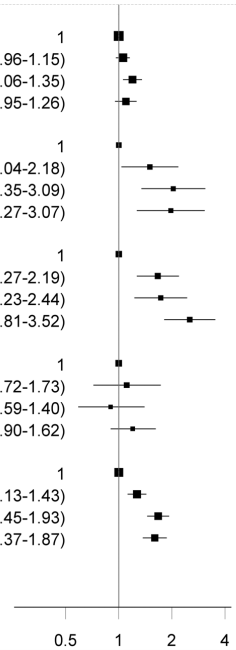


Figure 2 HR (95% CI) for incident hospitalisation for cardiovascular disease (CVD) and all-cause mortality by number of natural teeth left. Events, number of events; rate, crude rate per 1000 person years; CVD, cardiovascular disease; IHD, ischaemic heart disease; PVD, peripheral vascular disease. HR1, adjusted for age and sex; HR2, additionally adjusted for tobacco smoking, alcohol consumption, Australian born status, region of residence, education, health insurance, physical activity and body mass index, with missing values in covariates were coded as a separate categories (0.3%, 1.7%, 0%, 0.03%, 1.3%, 0%, 4% and 7%, respectively). There were no missing values in age or sex. HR2s are plotted on a log scale and are represented with squares, with areas inversely proportional to the logarithm of events; 95% CIs are indicated by horizontal lines.

BMJ Open **Is poor oral health a risk marker for incident cardiovascular disease hospitalisation and all-cause mortality? Findings from 172 630 participants from the prospective 45 and Up Study**

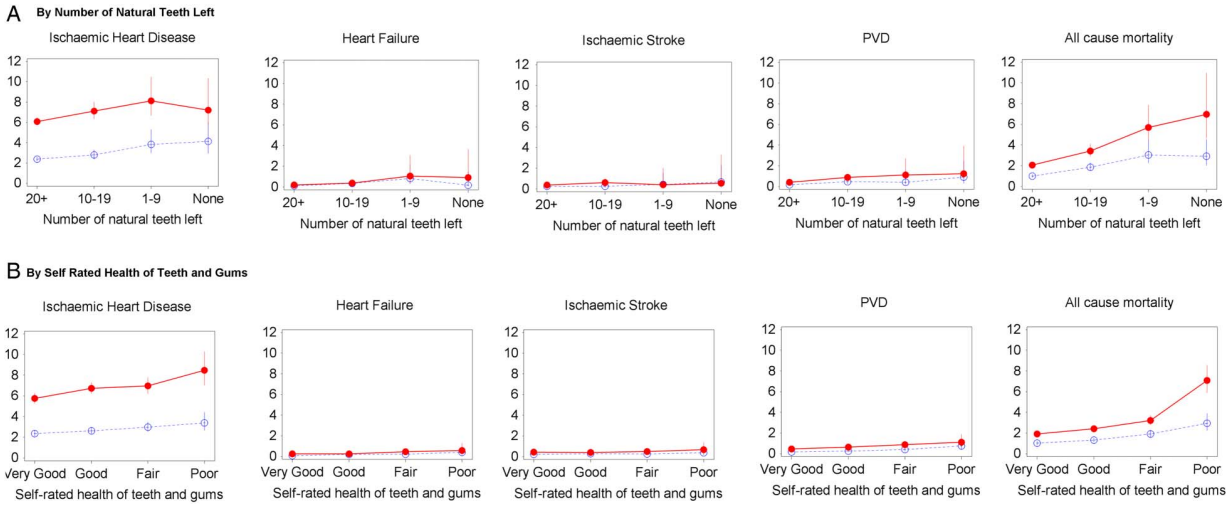


Figure 1 Age-standardised rates per 1000 person-years of all-cause mortality and incident cause-specific CVD hospitalisation since baseline, directly age-adjusted to 2006 New South Wales population. Male: ; female: .

Joshy G, Arora M, Korda RJ, et al. Is poor oral health a risk marker for incident cardiovascular disease hospitalisation and all-cause mortality? Findings from 172 630 participants from the prospective 45 and Up Study. BMJ Open 2016;6: e012386. doi:10.1136/ bmjopen-2016-012386

Periodontitis e Infarto de miocardio

Epidemiology and Prevention

Periodontitis Increases the Risk of a First Myocardial Infarction

A Report From the PAROKRANK Study

Lars Rydén, MD; Kåre Buhlin, DDS; Eva Ekstrand, DDS; Ulf de Faire, MD; Anders Gustafsson, DDS; Jacob Holmer, DDS; Barbro Kjellström, PhD; Bertil Lindahl, MD; Anna Norhammar, MD; Åke Nygren, DDS, MD; Per Näsman, PhD; Nilminie Rathnayake, DDS; Elisabet Svenungsson, MD; Björn Klinge, DDS

Background—The relationship between periodontitis (PD) and cardiovascular disease is debated. PD is common in patients with cardiovascular disease. It has been postulated that PD could be causally related to the risk for cardiovascular disease, a hypothesis tested in the Periodontitis and Its Relation to Coronary Artery Disease (PAROKRANK) study.

Methods and Results—Eight hundred five patients (<75 years of age) with a first myocardial infarction (MI) and 805 age- (mean 62±8), sex- (male 81%), and area-matched controls without MI underwent standardized dental examination including panoramic x-ray. The periodontal status was defined as healthy (≥80% remaining bone) or as mild-moderate (from 79% to 66%) or severe PD (<66%). Great efforts were made to collect information on possibly related confounders (≈100 variables). Statistical comparisons included the Student pairwise *t* test and the McNemar test in 2×2 contingency tables. Contingency tables exceeding 2×2 with ranked alternatives were tested by Wilcoxon signed rank test. Odds ratios (95% confidence intervals) were calculated by conditional logistic regression. PD was more common (43%) in patients than in controls (33%; *P*<0.001). There was an increased risk for MI among those with PD (odds ratio, 1.49; 95% confidence interval, 1.21–1.83), which remained significant (odds ratio, 1.28; 95% confidence interval, 1.03–1.60) after adjusting for variables that differed between patients and controls (smoking habits, diabetes mellitus, years of education, and marital status).

Conclusions—In this large case–control study of PD, verified by radiographic bone loss and with a careful consideration of potential confounders, the risk of a first MI was significantly increased in patients with PD even after adjustment for confounding factors. These findings strengthen the possibility of an independent relationship between PD and MI. **(*Circulation*. 2016;133:576-583. DOI: 10.1161/CIRCULATIONAHA.115.020324.)**

Key words: case-control study ■ causality ■ myocardial infarction ■ periodontitis ■ radiography, panoramic ■ risk factors

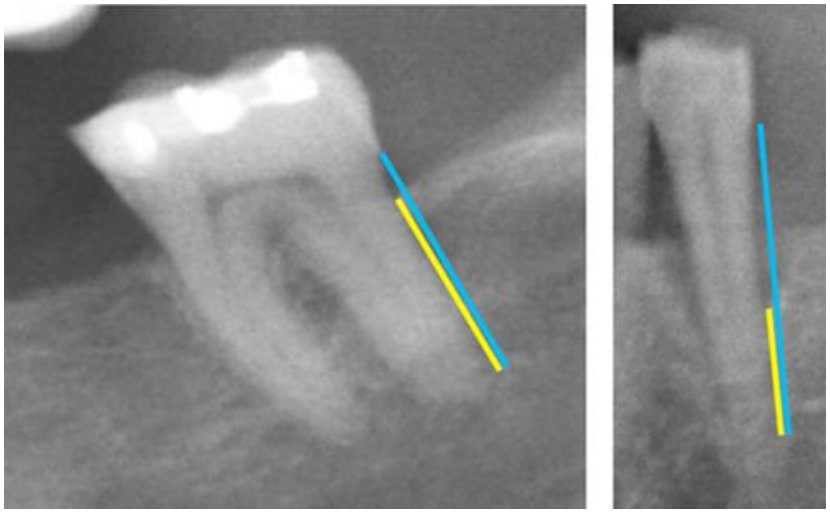


Table 3. Periodontal Status According to Panoramic X-Rays*

Periodontal Status	Patients n=796	Controls n=797	*P* Value
Healthy	458 (58)	530 (67)	
Mild-moderate periodontitis	261 (33)	231 (29)	<0.001
Severe periodontitis	78 (10)	35 (4)	

Data presented as number (%).

*X-rays not available in 9 patients and 8 controls.

Lars Rydén. Circulation. Periodontitis Increases the Risk of a First Myocardial Infarction, Volume: 133, Issue: 6, Pages: 576-583, DOI: (10.1161/CIRCULATIONAHA.115.020324)

Periodontitis e Infarto de miocardio

RESEARCH ARTICLE Open Access

The association between periodontal disease and the risk of myocardial infarction: a pooled analysis of observational studies

Shuai Xu[1], Mingbao Song[2], Yu Xiong[3], Xi Liu[2], Yongming He[2] and Zhexue Qin[2*]

Abstract

Background: Several meta-analyses have indicated that periodontal disease (PD) are related to cardiovascular diseases (CVDs). However, the association between PD and myocardial infarction (MI) remains controversial. Here we aimed to assess the association between PD and MI by meta-analysis of observational studies.

Methods: PubMed, EMBASE and the Cochrane Library were searched through July, 2016. Observational studies including cohort, cross-sectional and case–control studies reporting odds ratio (OR) or relative risk (RR) with 95% confidence intervals (CIs) were included in the analysis. Either fixed or random-effects model were applied to evaluate the pooled risk estimates. Sensitivity and subgroup analyses were also carried out to identify the sources of heterogeneity. Publication bias was assessed by the Begg's, Egger's test and funnel plot.

Results: We included 22 observational studies with 4 cohort, 6 cross-sectional and 12 case–control studies, including 129,630 participants. Patients with PD have increased risk of MI (OR 2.02; 95% CI 1.59-2.57). Substantial heterogeneity in risk estimates was revealed. Subgroup analyses showed that the higher risk of MI in PD patients exists in both cross-sectional studies (OR 1.71; 95% CI 1.07-2.73) and case–control studies (OR 2.93; 95% CI 1.95-4.39), and marginally in cohort studies (OR 1.18; 95% CI 0.98-1.42). Further, subgroup meta-analyses by location, PD exposure, participant number, and study quality showed that PD was significantly associated with elevated risk of MI.

Conclusion: Our meta-analysis suggested that PD is associated with increased risk of future MI. However, the causative relation between PD and MI remains not established based on the pooled estimates from observational studies and more studies are warranted.

Keywords: Periodontal disease, Myocardial infarction, Observational studies, Meta-analysis

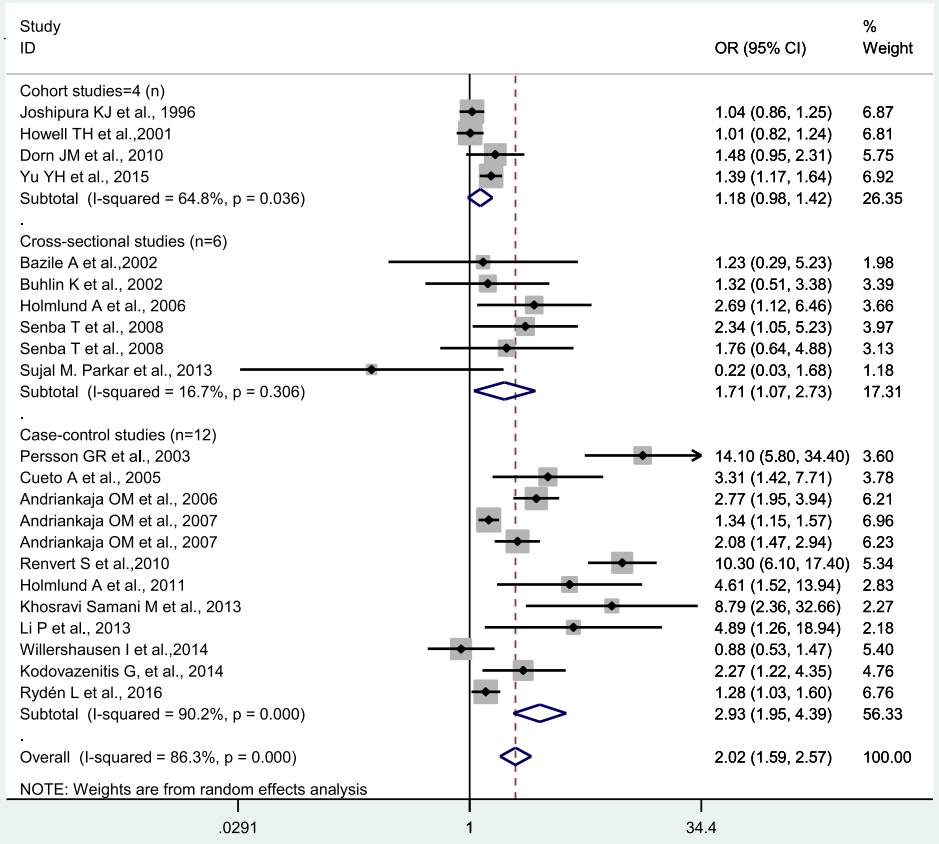
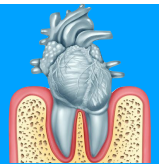


Fig. 3 Association between periodontal disease and myocardial infarction in a random-effect model meta-analyses by study design. OR, odds ratio; RR, relative ratio; CI, confidence interval

Xu et al. BMC Cardiovascular Disorders (2017) 17:50 DOI 10.1186/s12872-017-0480-y



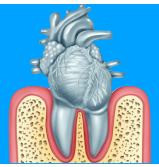
Periodontitis e Infarto de miocardio

Means and proportions of risk factors in patients with AMI and CHD

	AMI	CHD	p
Number of patients	250	250	-
Diabetic status (diabetic/non-diabetic)	99/151	68/182	0.03*
Hypertensive status (hypertensive/non-hypertensive)	189/61	181/69	415
Smoking status (current/former/non-smoker)	54/62/134	39/47/164	0.023*
Serum total cholesterol (mean ± SD)	250 ± 222.2	200.6 ± 25.7	0.00#
Serum LDL cholesterol (mean ± SD)	156.7 ± 24.3	135.5 ± 18.6	0.00#
Serum HDL cholesterol (mean ± SD)	39.9 ± 4.7	44.7 ± 6.3	0.00#
Serum triglyceride level (mean ± SD)	174.8 ± 65.3	130.7 ± 35.4	0.00#

*Statistically significant ($p < 0.05$, Pearson's chi-square test)
#Statistically significant ($p < 0.05$, Student's t-test)

Kaisare, S., Rao, J. & Dubashi, N. Periodontal disease as a risk factor for acute myocardial infarction. A case-control study in Goans highlighting a review of the literature. *Br Dent J* **203,** E5 (2007) doi:10.1038/bdj.2007.582



Periodontitis e Infarto de miocardio

Means and proportions of clinical parameters in patients with AMI and CHD

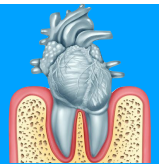
	AMI	CHD	p
Number of patients	250	250	-
Number of decayed teeth (Mean ± SD)	4 ± 3.7	2.8 ± 2.6	0.00#
Number of missing teeth (mean ± SD)	12 ± 12	7.5 ± 10	0.00#
Number of filled teeth (mean ± SD)	2 ± 3.1	1.3 ± 1.39	0.006#
Mean probing depth (± SD)	6.1 ± 1.98	3.1 ± 1.1	0.00#
Oral hygiene status (poor/fair/good)	127/65/4	18/182/20	0.00*
Percentage of sites with bleeding on probing (mean ± SD)	34.3 ± 8.8	15.1 ± 10.2	0.00#

*Statistically significant ($p < 0.05$, Pearson's chi-square test) #Statistically significant ($p < 0.05$, Student's t-test)

IN BRIEF

- The study results indicate a definite association between periodontal disease and acute myocardial infarction, a finding of significant public health importance.
- Dental infections by way of decayed teeth are also found to be significantly associated with acute myocardial infarction.
- Although stressing the relation between dental infections and acute myocardial infarction, the importance of serum lipids in the occurrence of coronary heart disease is also shown.

Kaisare, S., Rao, J. & Dubashi, N. Periodontal disease as a risk factor for acute myocardial infarction. A case-control study in Goans highlighting a review of the literature. *Br Dent J* 203, E5 (2007) doi:10.1038/bdj.2007.582



PERIODONTITIS Y ENFERMEDAD CV

PERIODONTITIS

Bacteriemia y liposacáridos sistémicos

Respuesta del hospedador: variación individual en las vías inflamatorias debido a la herencia genética.

1. Estado proinflamatorio aumentado por incremento de mediadores inflamatorios sistémicos, producidos en la lesión periodontal o mediante sintesis sistémica y hepática.

2. Estimulación del sistema inmune innato y adaptativo: generación de linfocitos Th autoreactivos, reactividad cruzada debida a mimetismo molecular.

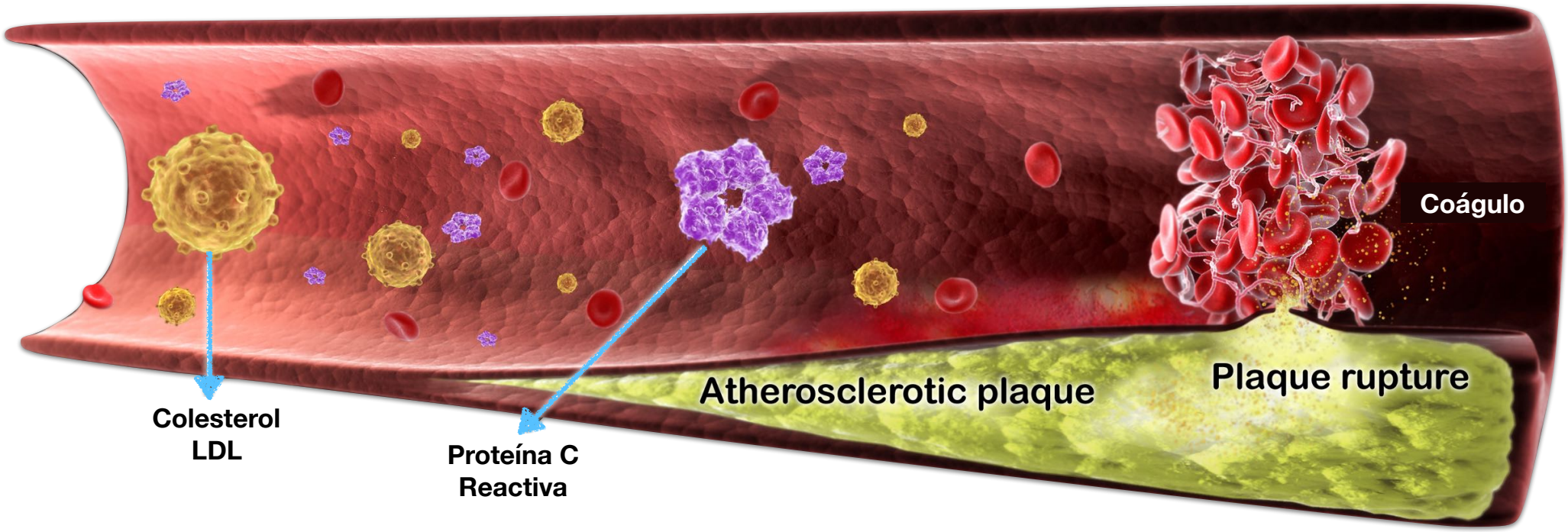
3. Estado protrombótico. Incremento de marcadores trombóticos y hemostáticos de producción sistémica y hepática.

4. Aumento de la síntesis de colesterol en el higado: displemia y modificación de los lípidos séricos inducida por las bacterias.

Alguno de estos procesos, o todos de forma simultánea pueden ocurrir en el paciente periodontal.

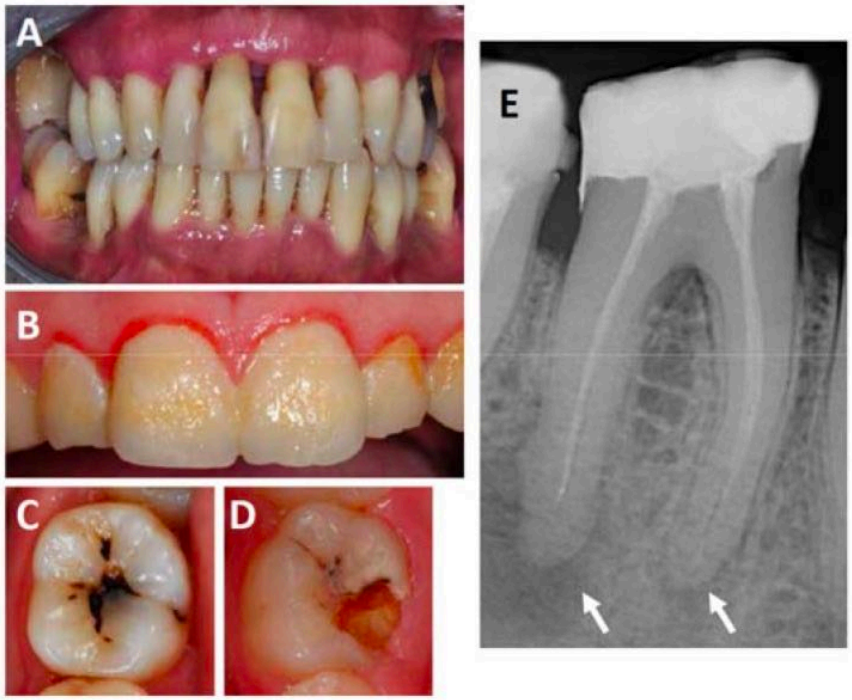
Aumento del potencial de ateregénesis o exacerbación de las lesiones ateroscletóticas, lo que da lugar a eventos cardiovasculares.

Infamación y enfermedad cardiovascular



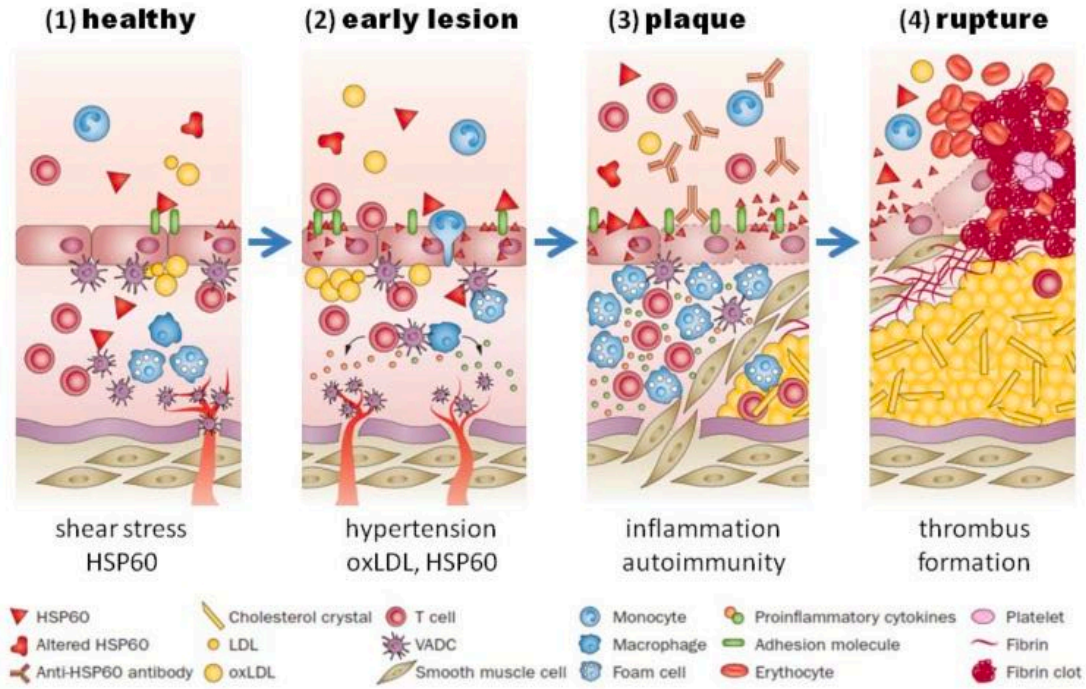
(1) Bacteriemia de bajo nivel por la cual las bacterias orales entran al torrente sanguíneo e invaden la pared arterial;
(2) Inflamación sistémica inducida por mediadores inflamatorios liberados de los sitios de la inflamación oral al torrente sanguíneo;
(3) Autoinmunidad a las proteínas del huésped causadas por la respuesta inmune del huésped a componentes específicos de los patógenos orales;
(4) Toxinas bacterianas específicas con efectos pro-aterogénicos producidas por bacterias patógenas orales.

Patología Oral y Enf. Cardiovascular



Frequent oral inflammations affecting the teeth, gingiva and the periodontium. (A) Shows an example of a severe case of periodontitis. Note the extensive loss of attachment and gingiva recession visible at most teeth; (B) shows a case of gingivitis. Note the soft plaque that covers the entire surface of the teeth and the gingiva reddened by the inflammation; panels (C,D) show examples of teeth affected by root caries, which often lead to the formation of endodontic lesions in the form of periapical abscesses, which can be detected on radiographs as shown in panel (E). Arrows mark the locations of periapical abscesses.

Int. J. Mol. Sci. 2018, 19, 1978



HSP60-induced atherosclerosis. (1) Healthy arteries are exposed to hemodynamic turbulences leading to shear stress at curves and branching points, which are prone to atherosclerosis; (2) classic risk factors (i.e., high blood pressure) may aggravate the stress response in endothelial cells, which leads to more surface expression of adhesion molecules and HSP60. This, together with secreted HSP60, attracts T cells and other proinflammatory cells to infiltrate the intima. Binding of cross-reactive antibodies to HSP60 to endothelial cells induce an autoimmune response, which promotes endothelial dysfunction and migration of mononuclear cells into the intima; (3) plaques start to develop, when macrophages and vascular smooth muscle are transformed to foam cells and produce proinflammatory cytokines. Soluble HSP60 is further released from damaged cells. If the inflammation persists, the lesion becomes more complex and a necrotic core composed of necrotic and apoptotic cells is formed. Cell debris, cholesterol crystals accumulate, and a fibrous cap is formed; (4) unstable plaques can rupture which leads to exposure of the core to the blood followed by thrombus formation. Abbreviations: HSP, heat shock protein; oxLDL, oxidized LDL; SMC, smooth-muscle cell; VADC, vascular-associated dendritic cell.

Infección bacteriana periodontal y ECV

Acute Ischemic Heart Disease

Detection of periodontal bacteria in thrombi of patients with acute myocardial infarction by polymerase chain reaction

Takahiro Ohki, MD,[a] Yuji Itabashi, MD, PhD,[a] Takashi Kohno, MD, PhD,[a] Akihiro Yoshizawa, MD,[a] Shuichi Nishikubo, DDS, PhD,[b] Shinya Watanabe, DDS, PhD,[b] Genyuki Yamane, DDS, PhD,[b] and Kazuyuki Ishihara, DDS, PhD[c] ***Chiba Prefecture, Japan***

Backgrounds Numerous reports have demonstrated that periodontal bacteria are present in plaques from atherosclerotic arteries. Although periodontitis has recently been recognized as a risk factor for coronary artery disease, the direct relationship between periodontal bacteria and coronary artery disease has not yet been clarified. It has been suggested that these bacteria might contribute to inflammation and plaque instability. We assumed that if periodontal bacteria induce inflammation of plaque, the bacteria would be released into the bloodstream when vulnerable plaque ruptures. To determine whether periodontal bacteria are present in thrombi at the site of acute myocardial infarction, we tried to detect periodontal bacteria in thrombi of patients with acute myocardial infarction by polymerase chain reaction (PCR).

Methods We studied 81 consecutive adults with ST-segment elevation acute myocardial infarction who underwent primary percutaneous coronary intervention (PCI). All patients underwent removal of thrombus with aspiration catheters at the beginning of percutaneous coronary intervention, and a small sample of thrombus was obtained for PCR.

Results The detection rates of periodontal bacteria by PCR were 19.7% for *Aggregatibacter actinomycetemcomitans*, 3.4% for *Porphyromonas gingivalis*, and 2.3% for *Treponema denticola*.

Conclusions Three species of periodontal bacteria were detected in the thrombi of patients with acute myocardial infarction. This raises the possibility that such bacteria are latently present in plaque and also suggests that these bacteria might have a role in plaque inflammation and instability. (Am Heart J 2012;163:164-7.)

Table 4. Association Among Periodontal Pathogens, CPITN, and Coronary Heart Disease Simultaneously Assessed in the Same Basic Model*

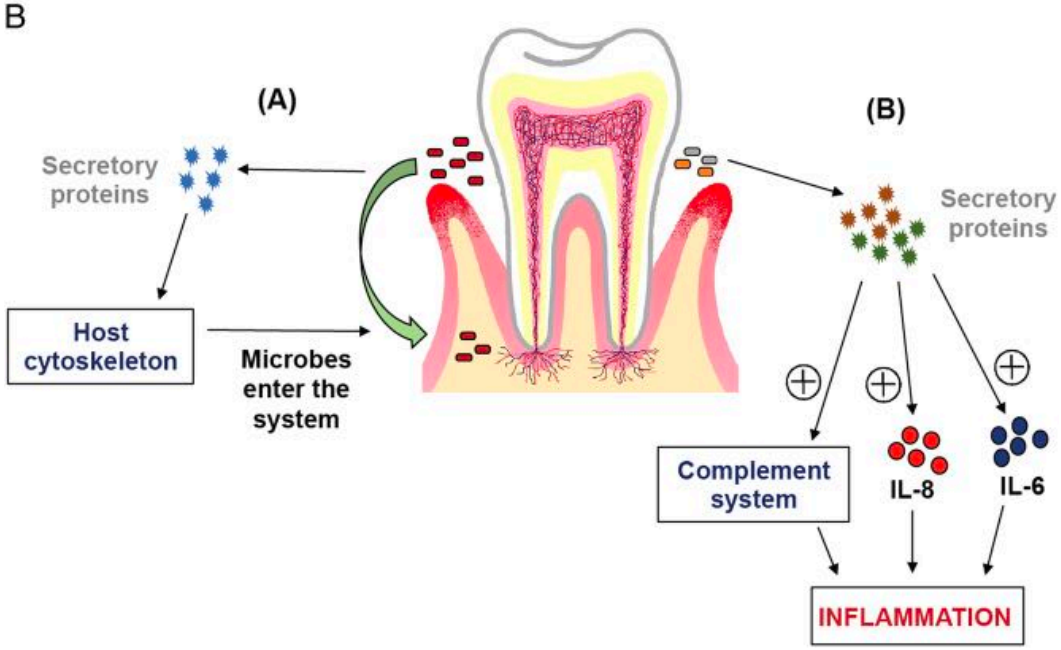
Variable	OR (95% CI)	*P* Value
Total periodontal pathogen burden, log	1.83 (1.23-2.71)	.003
CPITN score, mean	1.15 (0.70-1.89)	.58
Actinobacillus actinomycetemcomitans, log	2.68 (1.74-4.14)	<.001
CPITN score, mean	1.02 (0.62-1.70)	.93
Prevotella intermedia, log	1.25 (0.85-1.84)	.26
CPITN score, mean	1.48 (0.92-2.39)	.10

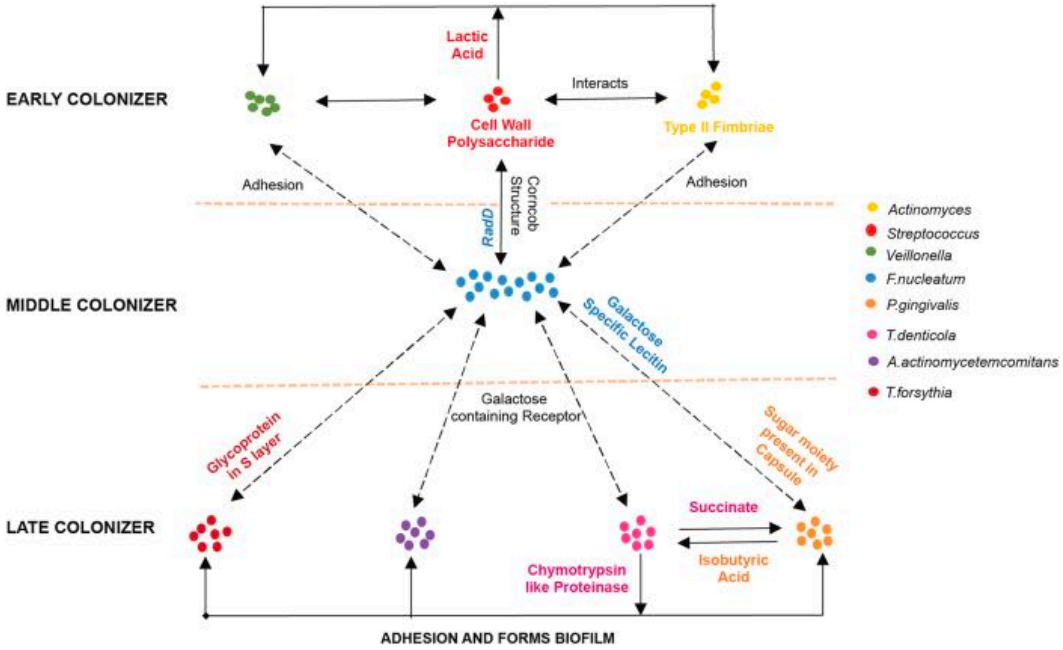
Abbreviations: CI, confidence interval; CPITN, Community Periodontal Index of Treatment Needs[19]; OR, odds ratio.

*Adjusted for age, sex, body mass index (calculated as weight in kilograms divided by the square of height in meters), smoking, alcohol consumption, diabetes mellitus, hypertension, hyperlipoproteinemia, level of education, physical activity, and statin intake. For an increase in mean CPITN score of 1 unit and an increase in periodontal pathogens of $\log_{10}$, pathogen numbers were logarithmically transformed to base 10.

Spahr, A., Klein, E., Khuseyinova, N., Boeckh, C., Muche, R., Kunze, M., Rothenbacher, D., Pezeshki, G., Hoffmeister, A., Koenig, W. (2006). Periodontal Infections and Coronary Heart Disease: Role of Periodontal Bacteria and Importance of Total Pathogen Burden in the Coronary Event and Periodontal Disease (CORODONT) Study Archives of Internal Medicine 166(5), 554-559. https://dx.doi.org/10.1001/archinte.166.5.554

Linkages between oral commensal bacteria and atherosclerotic plaques in coronary artery disease patients



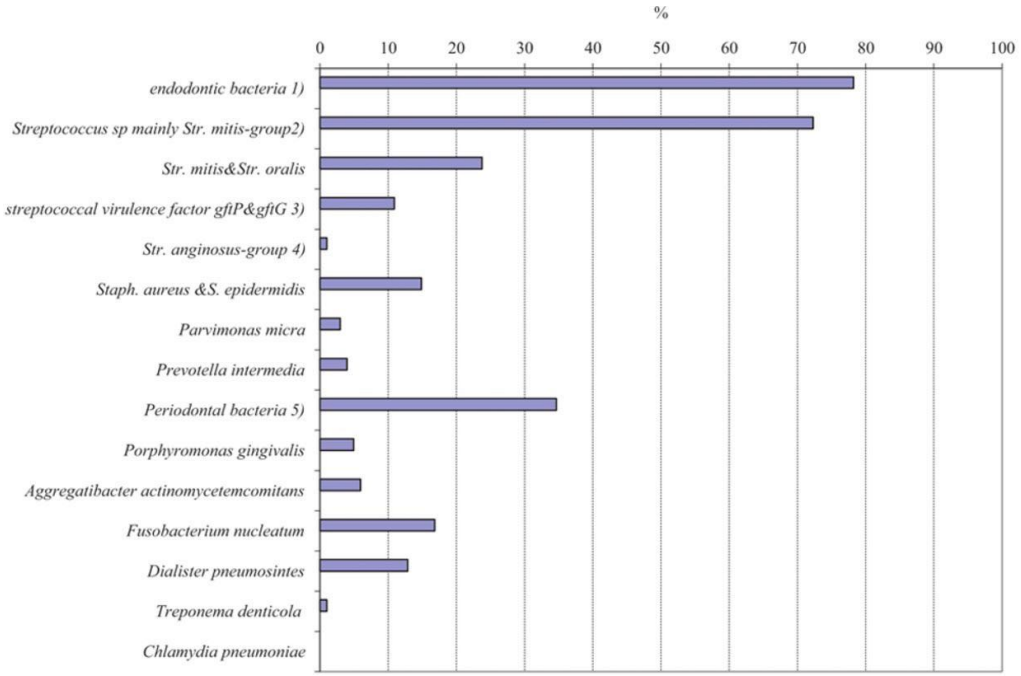


The gingival crevice is a habitat to many oral microbes that secrete proteins, peptides and proteases. (1) Secretory peptides and proteases are likely responsible for altering the host actin cytoskeleton in the gingival epithelium leading to microbial entry into the system. (2) These secreted proteins can also activate the immune system causing inflammation. Primarily, cytokine-mediated (IL-6 and IL-8) inflammation is associated with atherosclerotic plaque formation. Certain proteases cause inflammatory response by activating the complement system

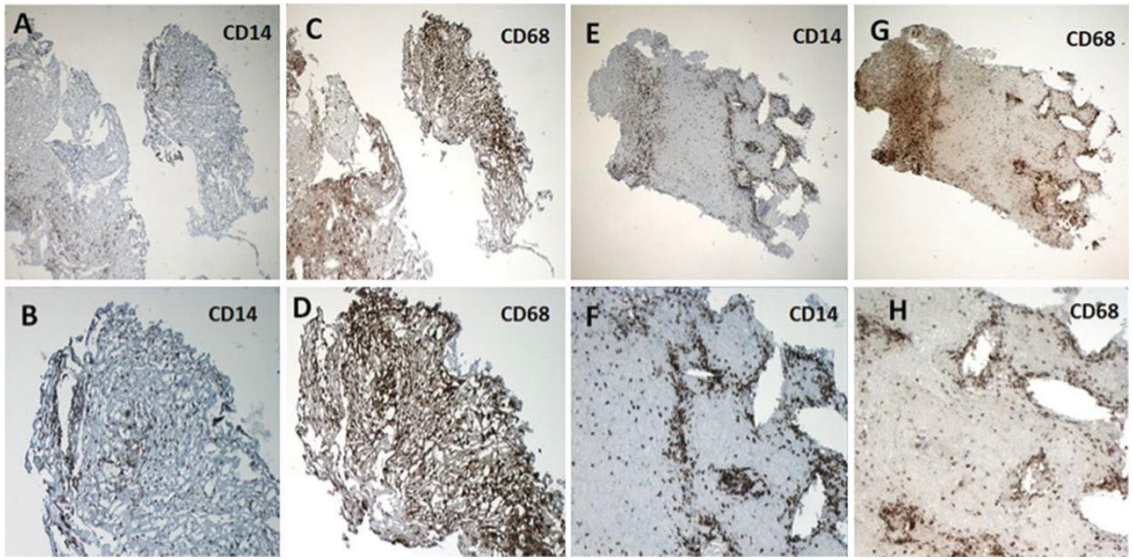
Atherosclerotic plaque-associated bacteria form biofilm structures within the atherosclerotic plaque samples. During initial phase of biofilm formation, early colonizers—Veillonella, Streptococcus, and Actinomyces—interact to establish an initial microenvironment supporting each other with the help of metabolic products. These bacteria act as a platform for the middle colonizer F. nucleatum, which then completes the biofilm formation by providing an adhering platform for the late colonizers—T. forsythia, A. actinomycetemcomitans, T. denticola, and P. gingivalis

Chhibber-Goel, J., Singhal, V., Bhowmik, D., Vivek, R., Parakh, N., Bhargava, B., & Sharma, A. (2016). Linkages between oral commensal bacteria and atherosclerotic plaques in coronary artery disease patients. NPJ biofilms and microbiomes, 2, 7. doi:10.1038/s41522-016-0009-7

Bacterial Signatures in Thrombus Aspirates of Patients With Myocardial Infarction



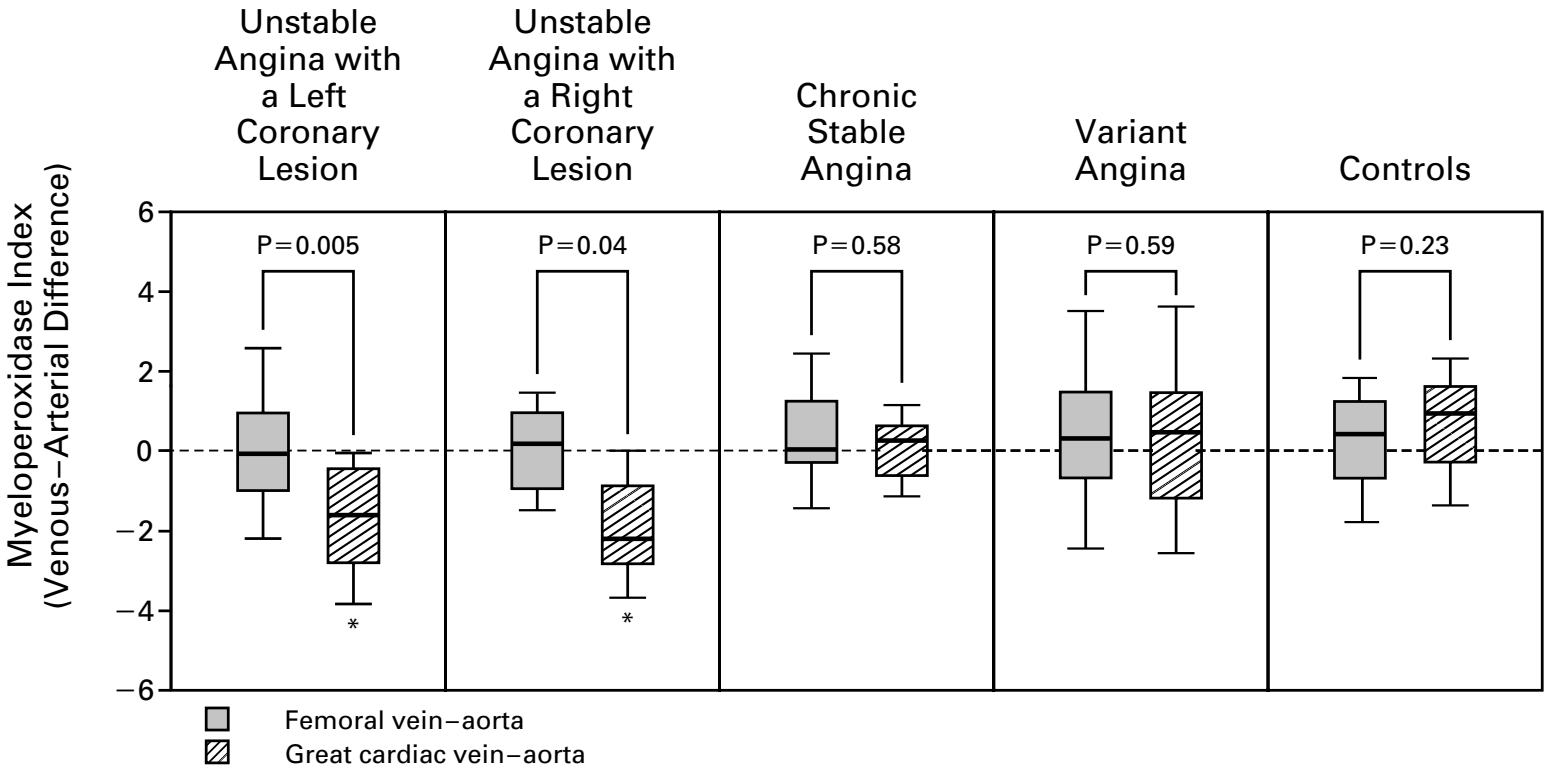
Frequencies of bacterial DNA-positive findings in thrombus aspirates of patients with ST-segment–elevation myocardial infarction using specific primers and probes in real-time quantitative polymerase chain reaction. Blood sample obtained from the arterial sheath before the procedure was used as reference.



Immunohistochemical stainings with CD14 and CD68 antibodies of 2 thrombus aspirates. A through D, Thrombus aspirate comprising tissue fragments from ruptured fibrous cap. E through H, Thrombus aspirate comprising mainly thrombotic material.

Circulation. 2013;127:1219–1228

Inflamación coronaria



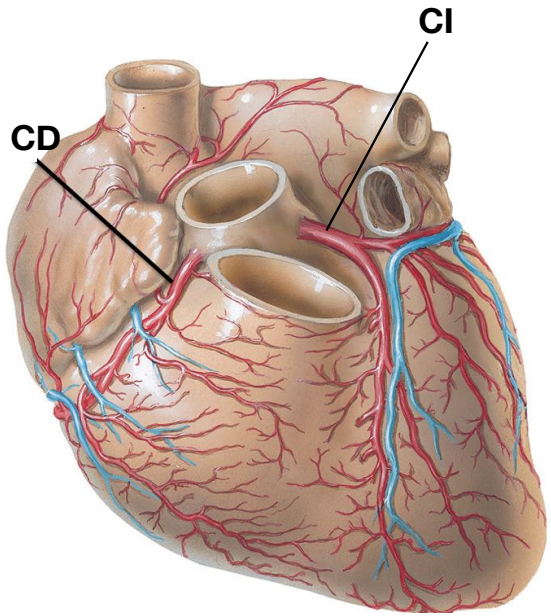
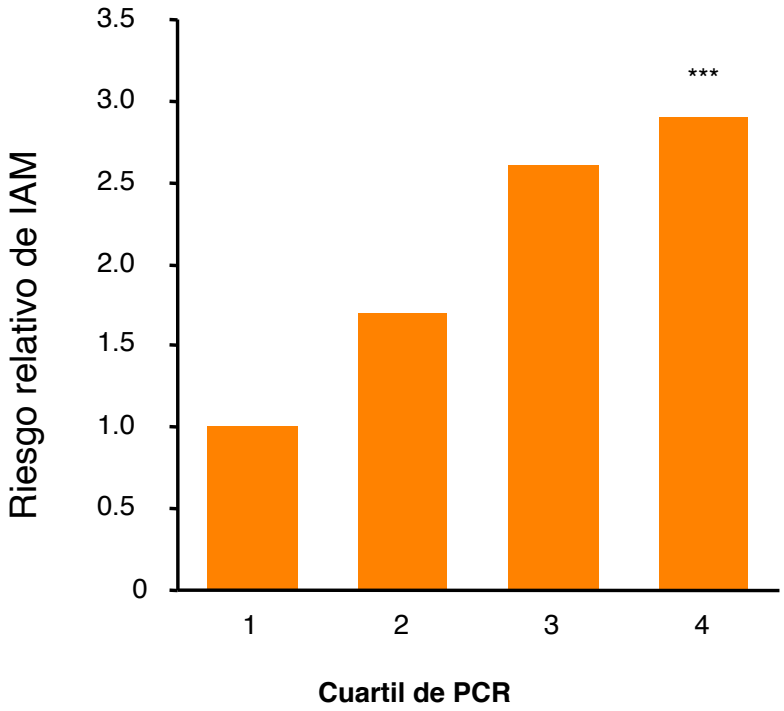
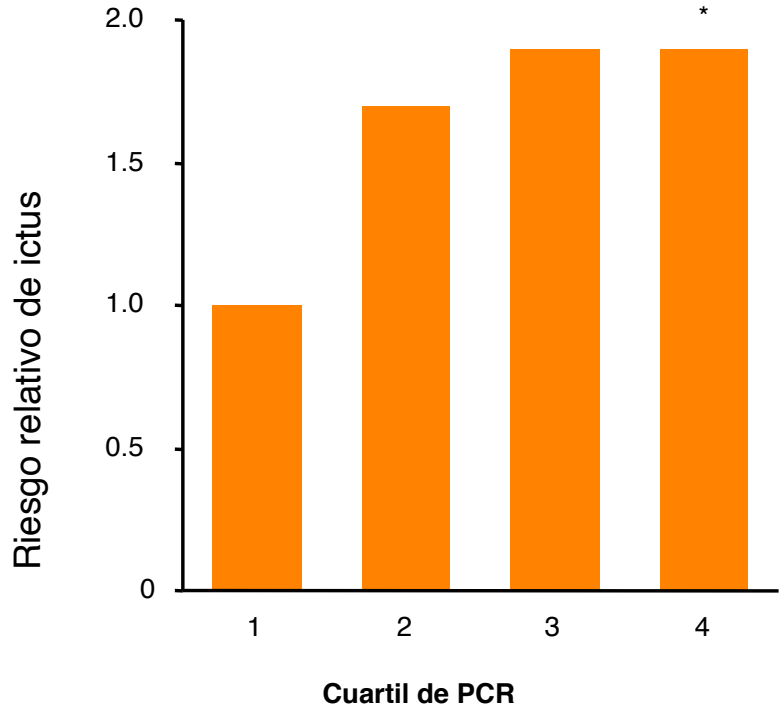


Figure 2. Venous–Arterial Differences in Myeloperoxidase Content across the Femoral and Coronary Vascular Beds.
Data are presented as medians, with 25th and 75 percentiles (boxes) and 10th and 90th percentiles (I bars). The difference in my- eloperoxidase content across the coronary circulation was significantly greater in both patients with unstable angina with a left coronary lesion and those with unstable angina with a right coronary lesion than in patients with chronic stable angina, patients with variant angina, and control patients. The difference in myeloperoxidase content across the coronary vascular bed was signif- icantly greater than that across the femoral vascular bed in both patients with unstable angina with a left coronary lesion and those with unstable angina with a right coronary lesion, but not in any of the other three groups. The asterisk indicates P<0.05 for the comparison of the group with unstable angina with a left coronary lesion and unstable angina with a right coronary lesion with the group with chronic stable angina, the group with variant angina, and controls.

N Engl J Med 2002;347:5-12

Niveles de PCR e infarto de miocardio



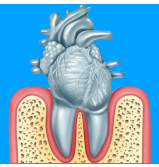


En el estudio epidemiológico Physicians' Health Study se estudiaron 543 hombres de mediana edad saludables, el riesgo relativo de un futuro IAM aumentó significativamente ($p < 0{,}001$) con el aumento de los niveles de CRP. El aumento de los niveles de PCR también se asoció con un riesgo significativamente mayor ($p = 0{,}02$) de ictus tromboembólico.

*p=0.03 vs quartile 1; ***p<0.001 vs quartile 1

Cuartil 1: ≤ 0.55 ; Cuartil 2: ≤ 0.56-1.14;
Cuartil 3: 1.15-2.10 ; Cuartil 4: ≥ 2.11.
Ridker PM *et al. N Engl J Med* 1997; 336: 973–979.

Enfermedad periodontal y disfunción eréctil



Enfermedad periodontal y disfunción eréctil

Article

Association Between Periodontal Disease and Erectile Dysfunction: A Systematic Review

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2018, Vol. 12(2) 338–346

DOI: 10.1177/1557988316639050
journals.sagepub.com/home/ajmh
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Sergio Varela Kellesarian, DDS[1], Tammy Varela Kellesarian, DDS, MPH[2], Vanessa Ros Malignaggi, DDS[3], Mansour Al-Askar, DDS[4], Alexis Ghanem, DDS[1], Hans Malmstrom, DDS[1], and Fawad Javed, DDS, PhD[1]

First International Journal of Andrology
ANDROLOGIA

ORIGINAL ARTICLE

Exploration of the association between chronic periodontal disease and erectile dysfunction from a population-based view point

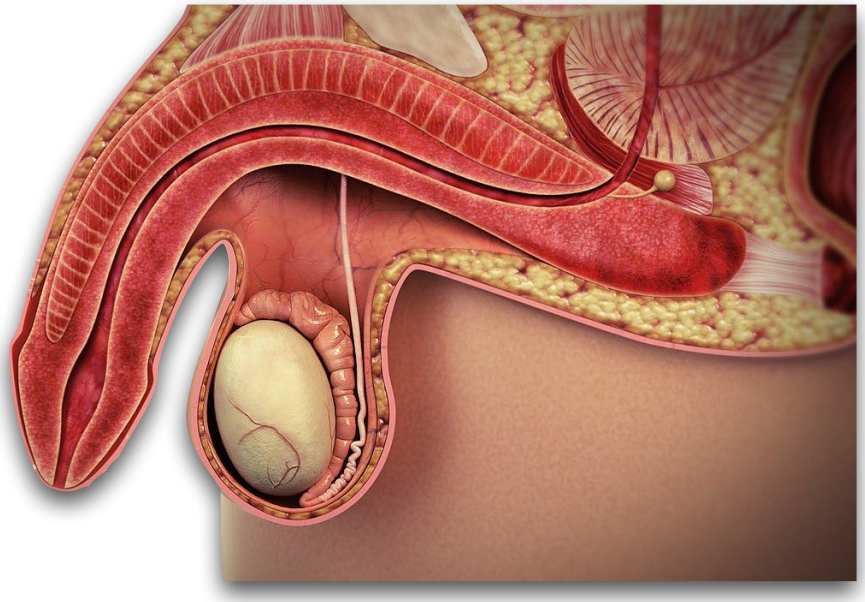
C.-W. Tsao[1,2], C.-Y. Liu[3], T.-L. Cha[1], S.-T. Wu[1], S.-C. Chen[4] & C.-Y. Hsu[2]

1 Division of Urology, Department of Surgery, Tri-Service General Hospital, National Defense Medical Center, Taipei, Taiwan;
2 Graduate Institute of Biomedical Informatics, and Center of Excellence for Cancer Research, Taipei Medical University, Taipei, Taiwan;
3 Department of Nutritional Science, Fu Jen Catholic University, New Taipei, Taiwan;
4 Graduate Institute of Patent, National Taiwan University of Science and Technology, Taipei, Taiwan

Chronic periodontitis is associated with erectile dysfunction: a case-control study in a European population

Amada Martín, Manuel Bravo, Miguel Arrabal, Antonio Magán-Fernández, Francisco Mesa
J Clin Periodontol. 2018; 45: 791-798.

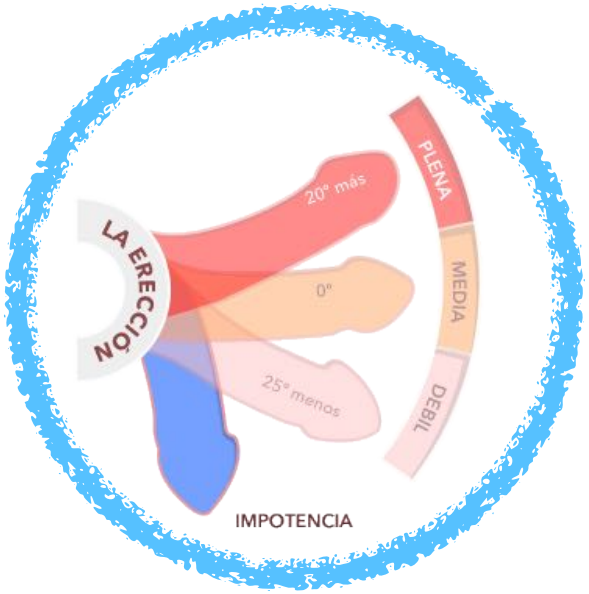
JCP Digest 07, published by the EFP in February 2019.

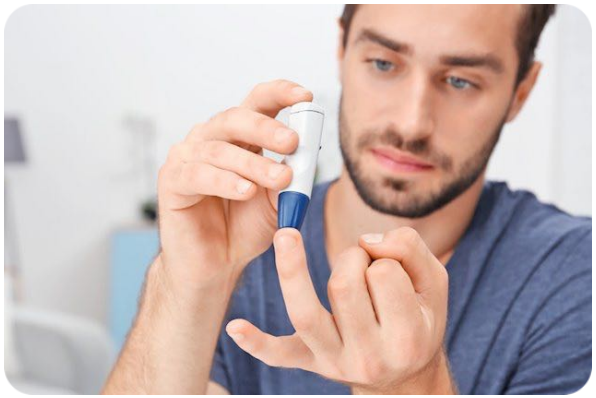


Enfermedad periodontal y disfunción eréctil

Factores de Riesgo Cardiovascular Comunes

La disfunción eréctil como signo precoz de enfermedad cardiovascular

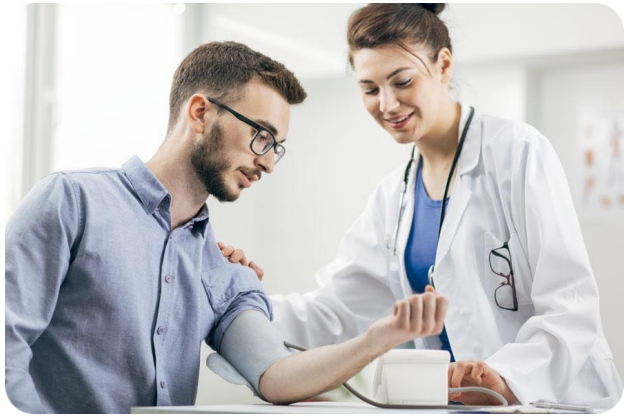




Recomendación sobre la disfunción eréctil

Recomendación	Clase[a]	Nivel[b]
Se debe considerar la evaluación de factores de riesgo CV y signos o síntomas de ECV en varones con disfunción eréctil	IIa	C

CV: cardiovascular; ECV: enfermedad cardiovascular.
[a]Clase de recomendación.
[b]Nivel de evidencia.



Tratamiento de la EPO y RCV

Treatment of Periodontitis and Endothelial Function

Maurizio S. Tonetti, D.M.D., Ph.D., Francesco D'Aiuto, D.M.D., Ph.D., Luigi Nibali, D.M.D., Ph.D., Ann Donald, Clare Storry, B.Sc., Mohamed Parkar, M.Phil., Jean Suvan, M.Sc., Aroon D. Hingorani, Ph.D., Patrick Vallance, M.D., and John Deanfield, M.B., B.Chir.

ABSTRACT

BACKGROUND

Systemic inflammation may impair vascular function, and epidemiologic data suggest a possible link between periodontitis and cardiovascular disease.

METHODS

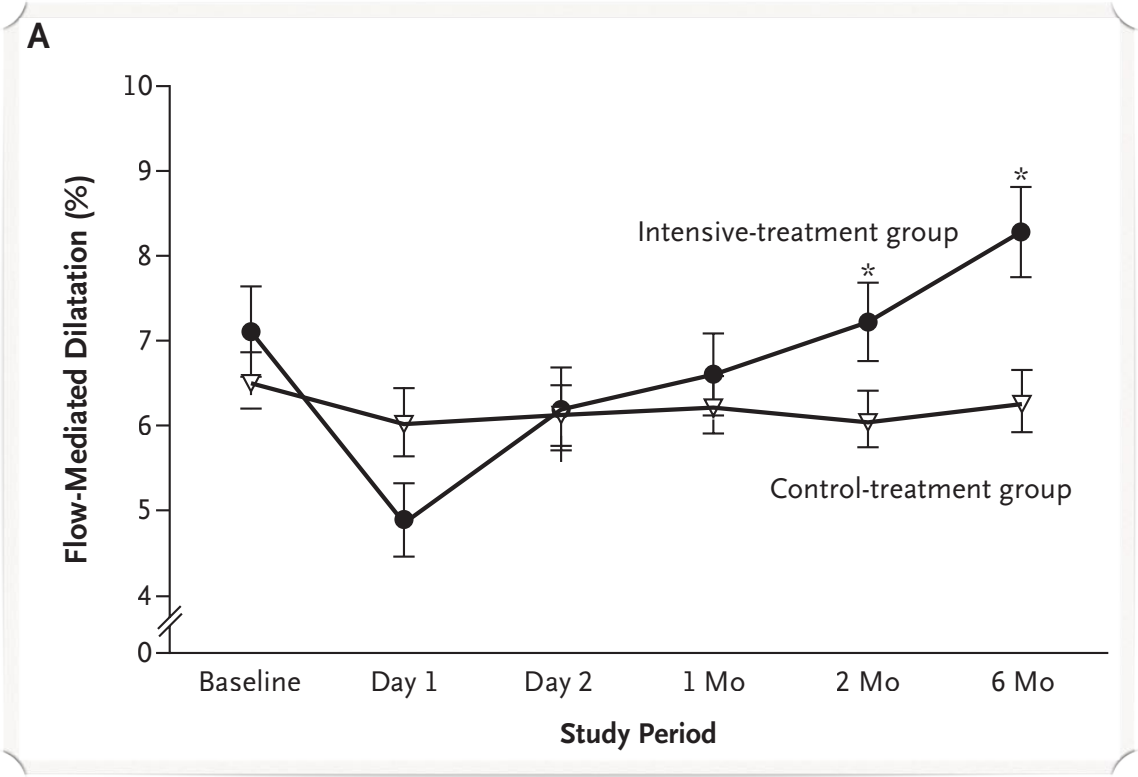
We randomly assigned 120 patients with severe periodontitis to community-based periodontal care (59 patients) or intensive periodontal treatment (61). Endothelial function, as assessed by measurement of the diameter of the brachial artery during flow (flow-mediated dilatation), and inflammatory biomarkers and markers of coagulation and endothelial activation were evaluated before treatment and 1, 7, 30, 60, and 180 days after treatment.

RESULTS

Twenty-four hours after treatment, flow-mediated dilatation was significantly lower in the intensive-treatment group than in the control-treatment group (absolute difference, 1.4%; 95% confidence interval [CI], 0.5 to 2.3; P=0.002), and levels of C-reactive protein, interleukin-6, and the endothelial-activation markers soluble E-selectin and von Willebrand factor were significantly higher (P<0.05 for all comparisons). However, flow-mediated dilatation was greater and the plasma levels of soluble E-selectin were lower in the intensive-treatment group than in the control-treatment group 60 days after therapy (absolute difference in flow-mediated dilatation, 0.9%; 95% CI, 0.1 to 1.7; P=0.02) and 180 days after therapy (difference, 2.0%; 95% CI, 1.2 to 2.8; P<0.001). The degree of improvement was associated with improvement in measures of periodontal disease (r=0.29 by Spearman rank correlation, P=0.003). There were no serious adverse effects in either of the two groups, and no cardiovascular events occurred.

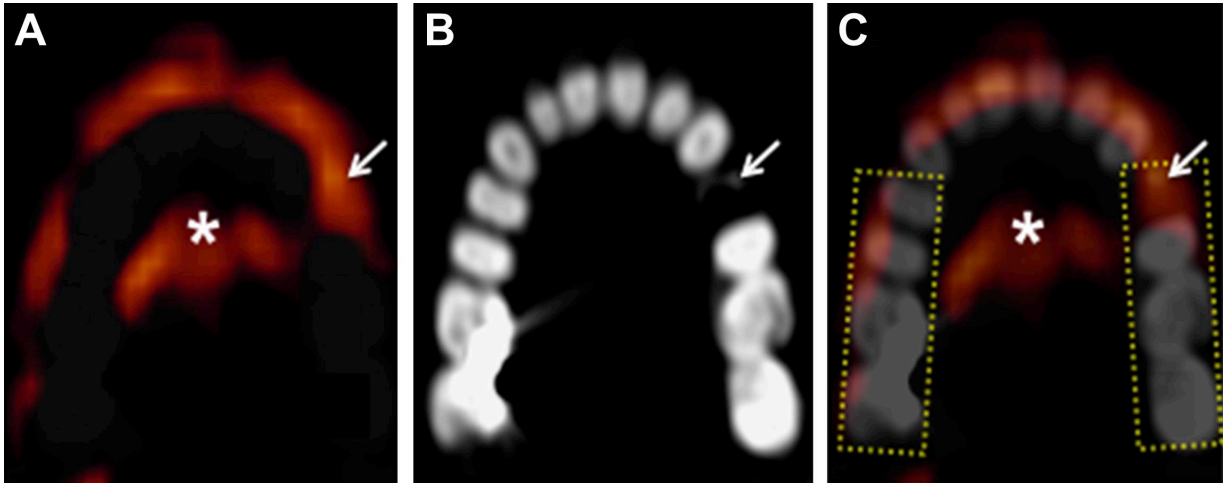
CONCLUSIONS

Intensive periodontal treatment resulted in acute, short-term systemic inflammation and endothelial dysfunction. However, 6 months after therapy, the benefits in oral health were associated with improvement in endothelial function.



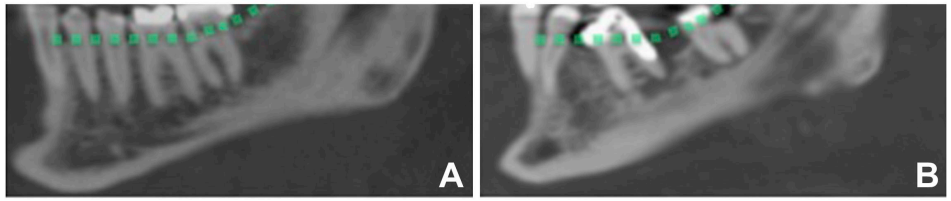
N Engl J Med 2007;356:91120

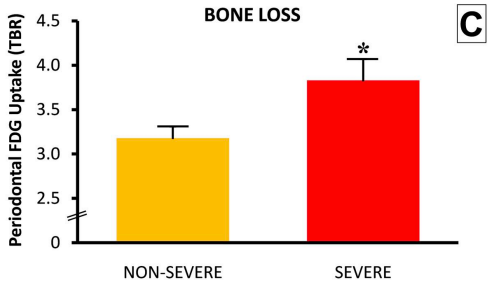
Las estatinas reducen la inflamación

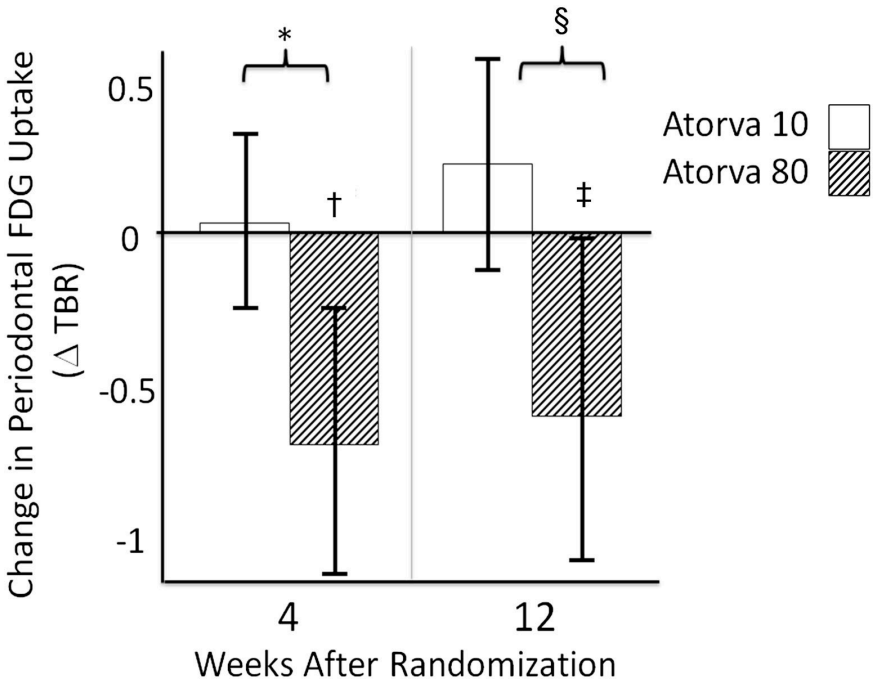


No Bone Loss

Severe Bone Loss

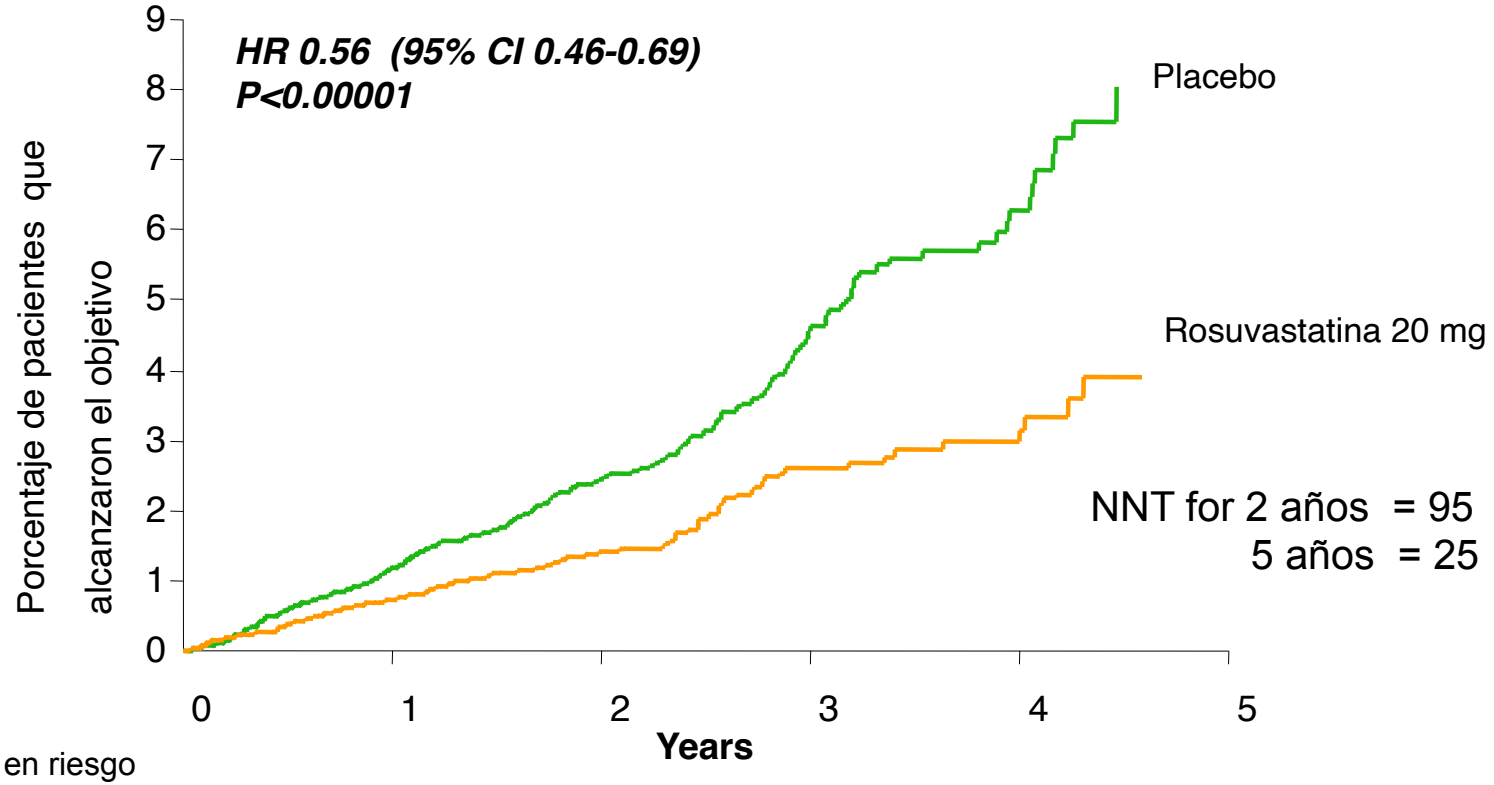






Subramanian S, Emami H, Vucic E, Singh P, Vijayakumar J, Fifer KM, Alon A, Shankar SS, Farkouh M, Rudd JHF, Fayad ZA, Van Dyke TE, Tawakol A. High-dose atorvastatin reduces periodontal inflammation: a novel pleiotropic effect of statins. J Am Coll Cardiol. 2013 Dec 24;62(25):2382-2391. doi: 10.1016/j.jacc.2013.08.1627. Epub 2013 Sep 24. PubMed PMID: 24070911.

Resultados del estudio JUPITER: Objetivo primarrio de muerte CV, ictus no fatal, IAM no fatal, angina inesatable o revascularización



Nº en riesgo						
Rosu	8901	8412	3893	1353	538	157
Placebo	8901	8353	3872	1333	531	174

17.802 individuos aparentemente sanos con LDL de menos de 130 mg/dL y niveles de PCR-hs de 2,0 mg/L o superiores asignados a rosuvastatina 20 mg al día o placebo y se les siguió para detectar la aparición combinada de infarto de miocardio, ictus, revascularización arterial, hospitalización por angina inestable o muerte por causas cardiovasculares. Los pacientes bajo tratamiento tuvieron significativamente menos eventos cardiovasculares.

Ridker P *et al. N Eng J Med* 2008;**359**: 2195-2207

Tratamiento de la EPO y reducción PCRhs

Treatment of periodontitis improves the atherosclerotic profile: a systematic review and meta-analysis

Teeuw WJ, Slot DE, Susanto H, Gerdes VEA, Abbas F, D'Aiuto F, Kastelein JJP, Loos BG. Treatment of periodontitis improves the atherosclerotic profile: a systematic review and meta-analysis. J Clin Periodontol 2014; 41: 70–79. doi: 10.1111/jcpe.12171.

Abstract

Aim: Systematic review and meta-analyses to study the robustness of observations that treatment of periodontitis improves the atherosclerotic profile.

Material and Methods: Literature was searched in Medline-PubMed, Cochrane CENTRAL and EMBASE, based on controlled periodontal intervention trials, including also a non-intervention group. Data were extracted and meta-analyses were performed.

Results: From 3928 screened studies, 25 trials met the eligibility criteria. These trials enrolled 1748 periodontitis patients. Seven trials enrolled periodontitis patients that were otherwise healthy, 18 trials recruited periodontal patients with various co-morbidities, such as CVD or diabetes. None of the trials used hard clinical endpoints of CVD. However, improvement of endothelial function has been consistently reported. Meta-analyses demonstrated significant weighted mean difference (WMD) for hsCRP (−0.50 mg/l, 95% CI:−0.78; −0.22), IL-6 (−0.48 ng/l, 95% CI: −0.90; −0.06), TNF-α (−0.75 pg/ml, 95% CI: −1.34; −0.17), fibrinogen (−0.47 g/l, 95% CI: −0.76; −0.17), total cholesterol (−0.11 mmol/l, 95% CI: −0.21; −0.01) and HDL-C (0.04 mmol/l, 95% CI: 0.03; 0.06) favouring periodontal intervention. Importantly, periodontitis patients with co-morbidity benefitted most from periodontal therapy; significant WMD were observed for levels of hsCRP (−0.71 mg/l, 95% CI: −1.05; −0.36), IL-6 (−0.87 ng/l, 95% CI: −0.97; −0.78), triglycerides (−0.24 mmol/l, 95% CI: −0.26; −0.22), total cholesterol (−0.15 mmol/l, 95% CI: −0.29; −0.01), HDL-C (0.05 mmol/l, 95% CI: 0.03; 0.06) and HbA_{1c} (−0.43%, 95% CI: −0.60; −0.25).

Conclusions: This systematic review and meta-analyses demonstrate that periodontal treatment improves endothelial function and reduces biomarkers of atherosclerotic disease, especially in those already suffering from CVD and/or diabetes.

Systematic Review

Wijnand J. Teeuw[1], Dagmar E. Slot[1], Hendri Susanto[2,3], Victor E. A. Gerdes[4,5], Frank Abbas[3], Francesco D'Aiuto[6], John J. P. Kastelein[4] and Bruno G. Loos[1]

[1]Department of Periodontology, Academic Centre for Dentistry Amsterdam (ACTA), University of Amsterdam and VU University Amsterdam, Amsterdam, The Netherlands; [2]Department of Oral Medicine, Faculty of Dentistry, Gadjah Mada University, Yogyakarta, Indonesia; [3]Center for Dentistry and Oral Hygiene, Department of Periodontology, University of Groningen and University Medical Center Groningen, Groningen, The Netherlands; [4]Department of Vascular Medicine, Academic Medical Center (AMC), University of Amsterdam, Amsterdam, The Netherlands; [5]Department of Internal Medicine, Slotervaart Hospital, Amsterdam, The Netherlands; [6]Periodontology Unit, Department of Clinical Research, UCL Eastman Dental Institute, London, UK

Key words: atherosclerosis; periodontitis; systematic review; therapy

Accepted for publication 18 September 2013

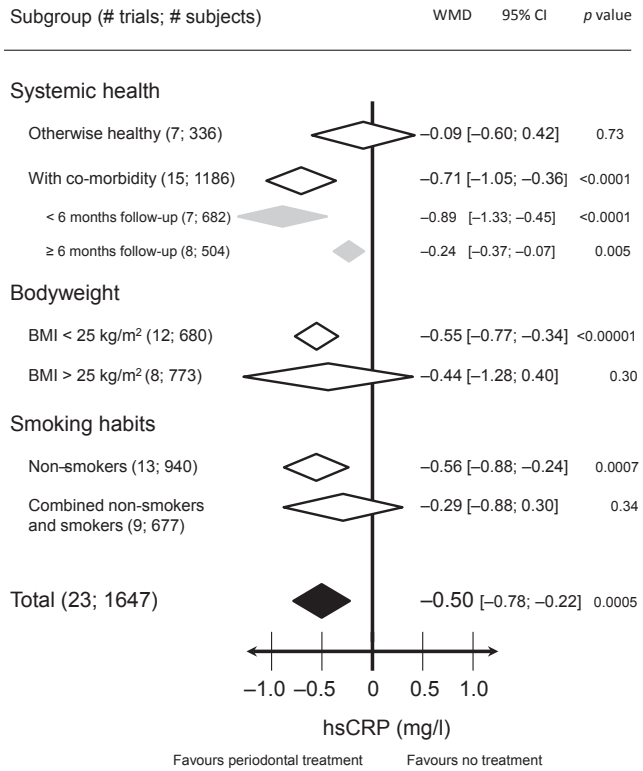
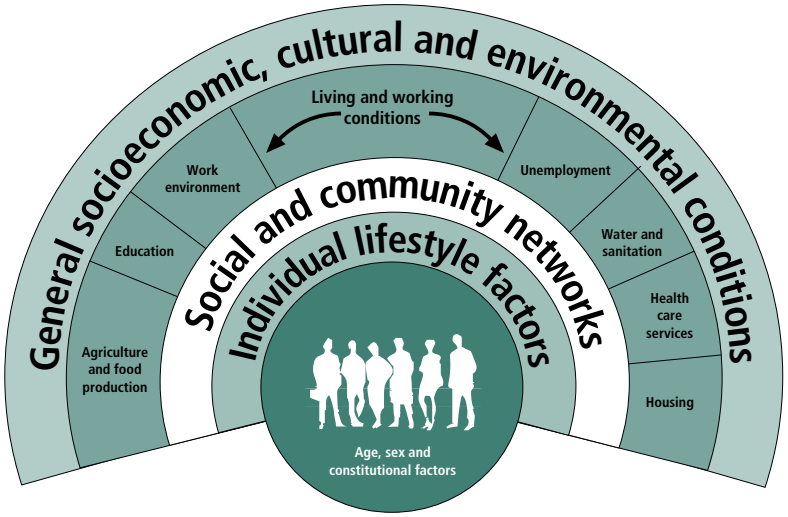


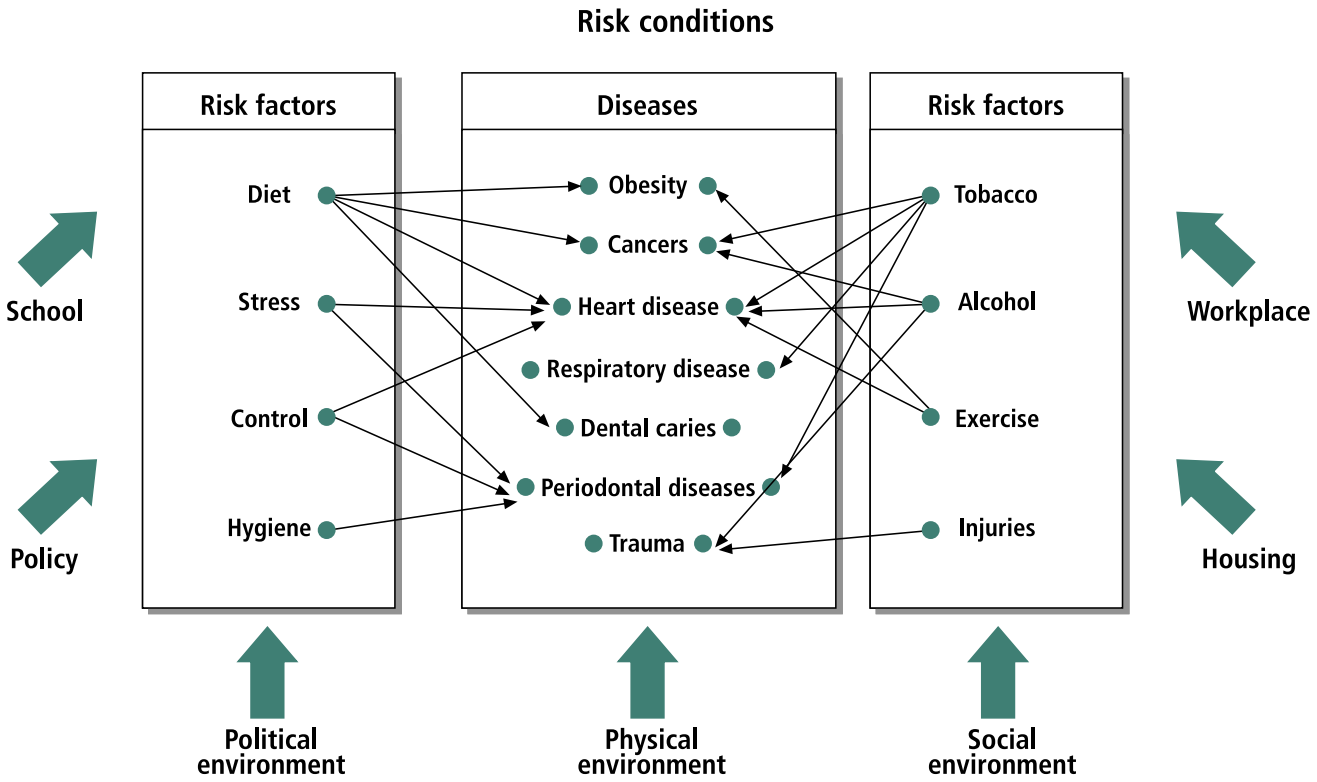
Fig. 4. Summary figure of individual meta-analyses of hsCRP levels. Weighted mean difference (WMD) and 95% confidence interval (CI) of hsCRP levels between the periodontal treatment groups and non-periodontal treatment groups (forest plots are presented in Figs 2 and 3 and Supplementary Figs 4 and 5 in Data S1). In parentheses, the number of trials and subjects evaluated.

J Clin Periodontol 2014; 41: 70–79 doi: 10.1111/jcpe.12171

Prevención de la EPO y de la ECV

Estrategias de prevención basadas en los factores de riesgo comunes





Watt. Bulletin of the World Health Organization 2005;83:711-718

Prevención de la EPO y de la ECV

https://mimocardio.org/mimoencias

GINGIVITIS Y PERIODONTITIS

Factores de Riesgo.

- **Tabaco.**
- **Estrés.**
- **Enfermedades generales** o defensas bajas: diabetes, osteoporosis, VIH, herpes, transplantados, etc...
- **Cambios hormonales:** embarazo, menopausia.
- **Antecedentes familiares.**

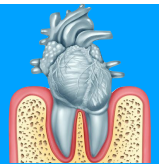
Señales de Alerta.

- **Sangrado** o enrojecimiento de encías.
- **Mal aliento.**
- **Hipersensibilidad al frío.**
- **Movilidad o separación** de dientes.
- **Dientes más largos.**
- **Pérdida de dientes.**

Tratamientos sencillos.

- **Mejora de la higiene bucal.**
- **Limpieza completa** de la placa bacteriana en la encía. Evaluación del estado Periodontal.
- **Control de placa y bacterias** por debajo de la encía (raspado).
- **En casos avanzados**, pequeñas cirugías.

Es importante el cuidado de la salud periodontal en el paciente cardiovascular y el despistaje de la patología periodontal debe formar parte de los protocolos de atención cardiológica.



Conclusiones



- A nivel mundial, las enfermedades cardiovasculares son una causa importante de morbilidad y mortalidad.
- Los factores de riesgo conocidos incluyen hipercolesterolemia, hipertensión, diabetes mellitus y tabaquismo.
- Existe relación entre enfermedades cardiovasculares y periodontitis. Las personas que han sufrido un infarto de miocardio, ACV o la aterosclerosis parecen tener una mayor prevalencia de periodontitis.
- Se proponen varios mecanismos sobre la asociación, como la presencia de bacteriemias de corta duración debido a periodontitis, niveles elevados de proteína C reactiva, establecimiento de un estado protrombótico y reacciones autoinmunes. Además, en caucásicos, hay evidencia de factores de riesgo genéticos superpuestos.
- En conjunto estas vías son plausibles y pueden provocar disfunción endotelial que hace a los individuos más susceptibles a eventos de enfermedades sufrir cardiovasculares.
- Se ha constatado la asociación independiente de la periodontitis con la enfermedad cardiovascular.
- Las medidas de prevención son fundamentales, para no sólo tener una boca sana, sino también disminuir el riesgo de sufrir un evento cardiovascular.

GRACIAS

Dr. Antonio García Quintana,
Servicio de Cardiología
Hospital Universitario de Gran Canaria Doctor Negrín
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