



Diabetes Tipo 2

9ª JORNADA DE ACTUALIZACIÓN TERAPÉUTICA DE LA redGDPS
CASOS CLÍNICOS E INVESTIGACIÓN

@redGDPS



PROGRAMA

Madrid, 21-22 de octubre 2016

Diabetes e Inflamación

Dr. José Manuel Fernández-Real

Madrid, 21 de octubre de 2016



Inflamación y DM-2:

1. La inflamación siempre impacta negativamente en la fisiopatología de la DM-2.
2. La inflamación es la respuesta. Lo importante es el estímulo que la desencadena.
3. Algo de inflamación es bueno. Lo perjudicial es su cronificación.
4. ¿Podemos utilizarla como base para tratar la DM-2?

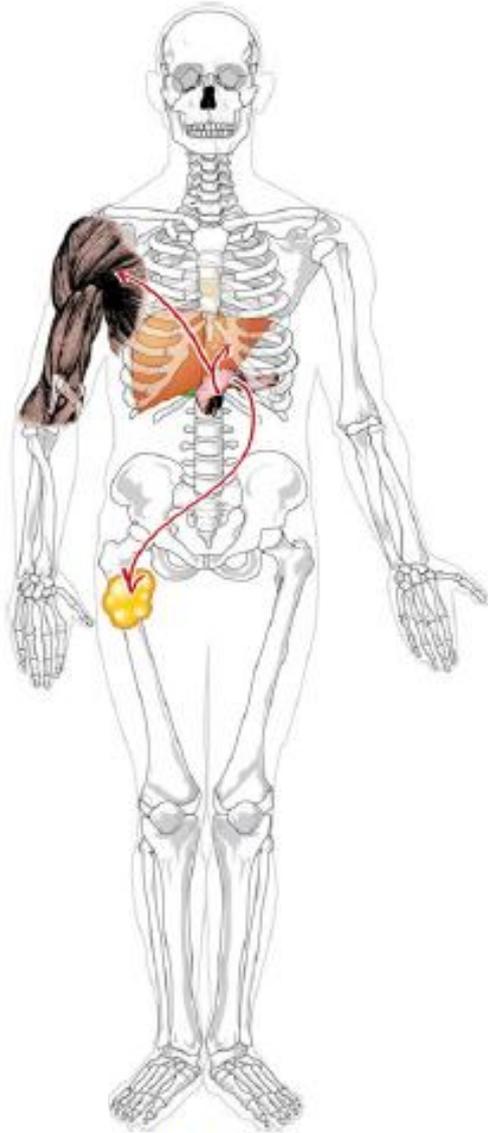


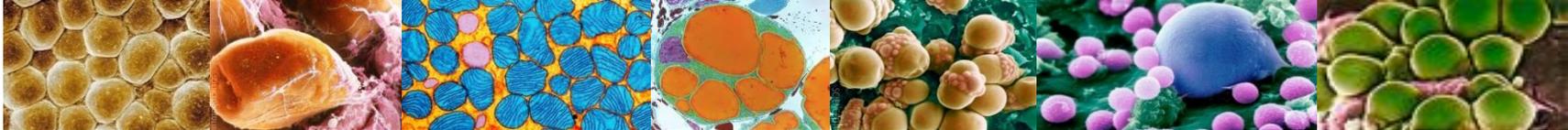
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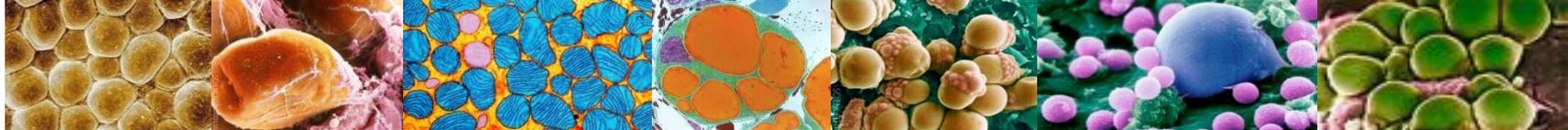
Inflamación y DM2





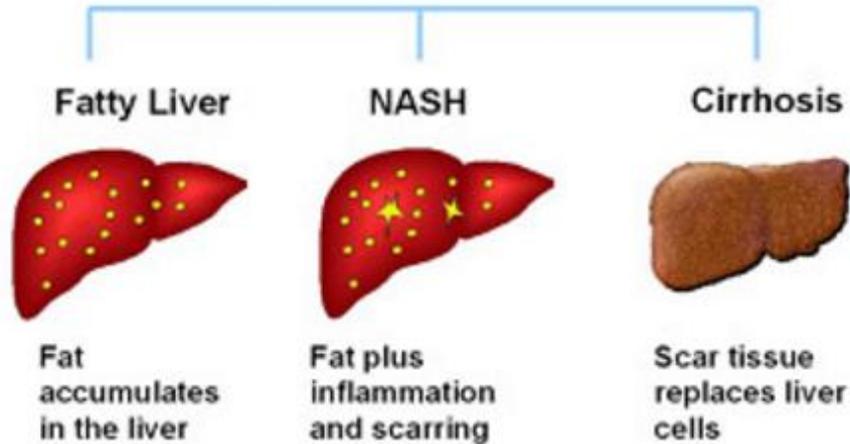
Inflamación y DM2





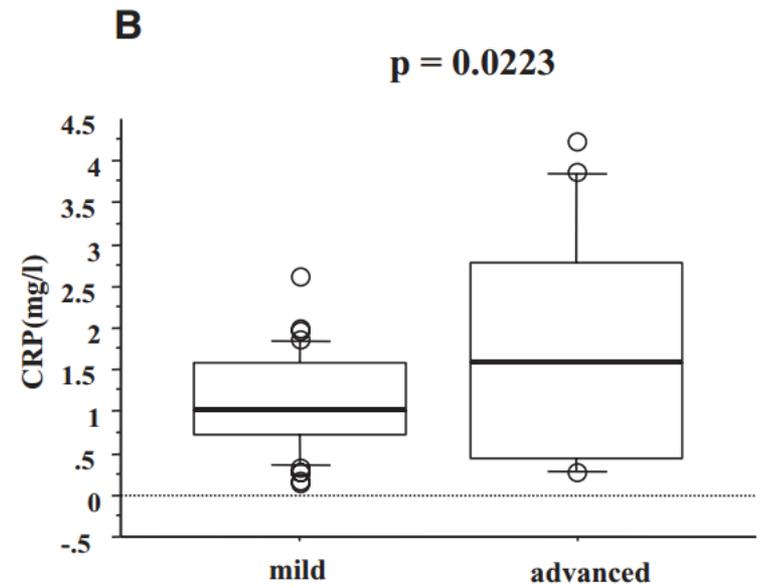
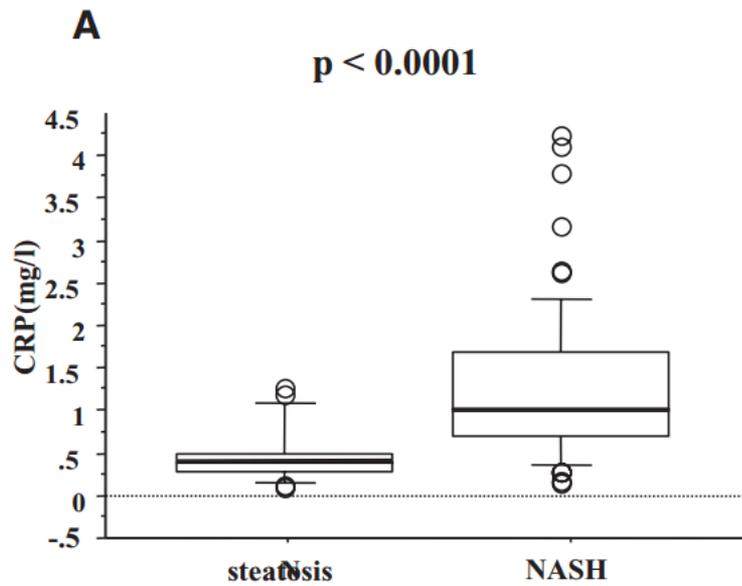
Inflamación y DM2

The Spectrum of NAFLD



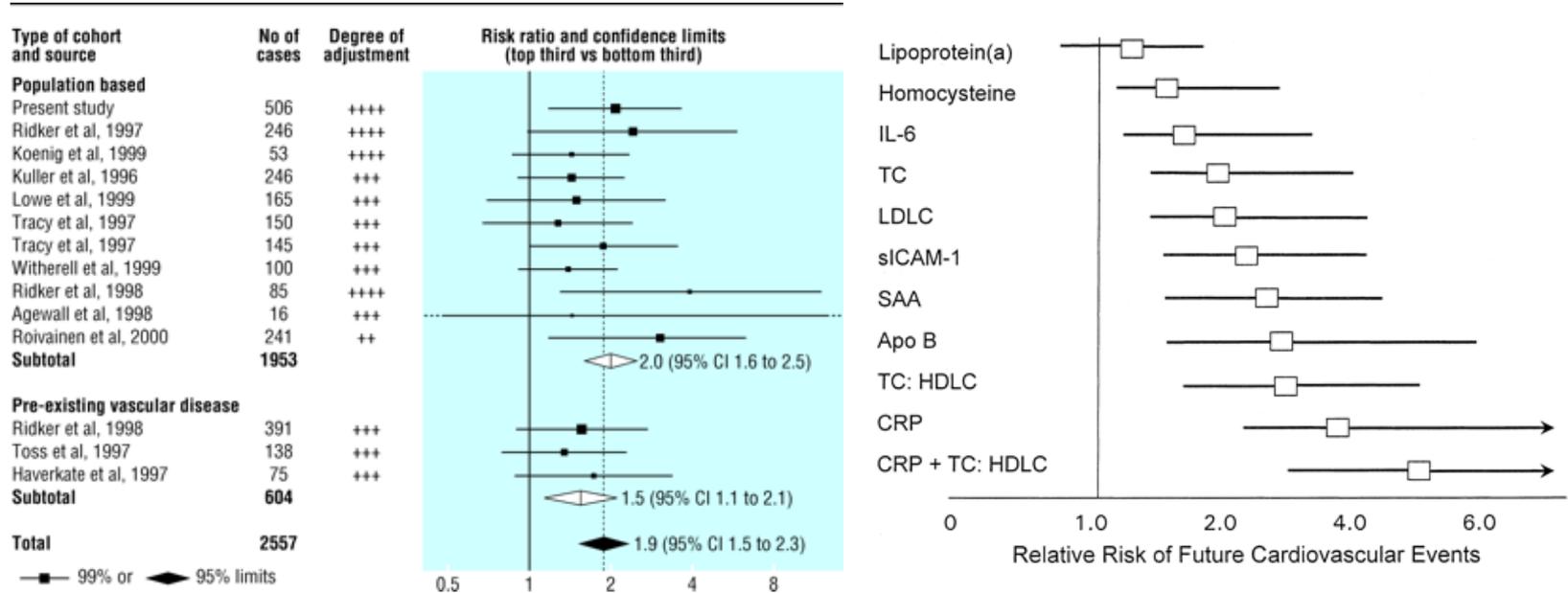


Inflamación y DM2





Inflamación y DM2

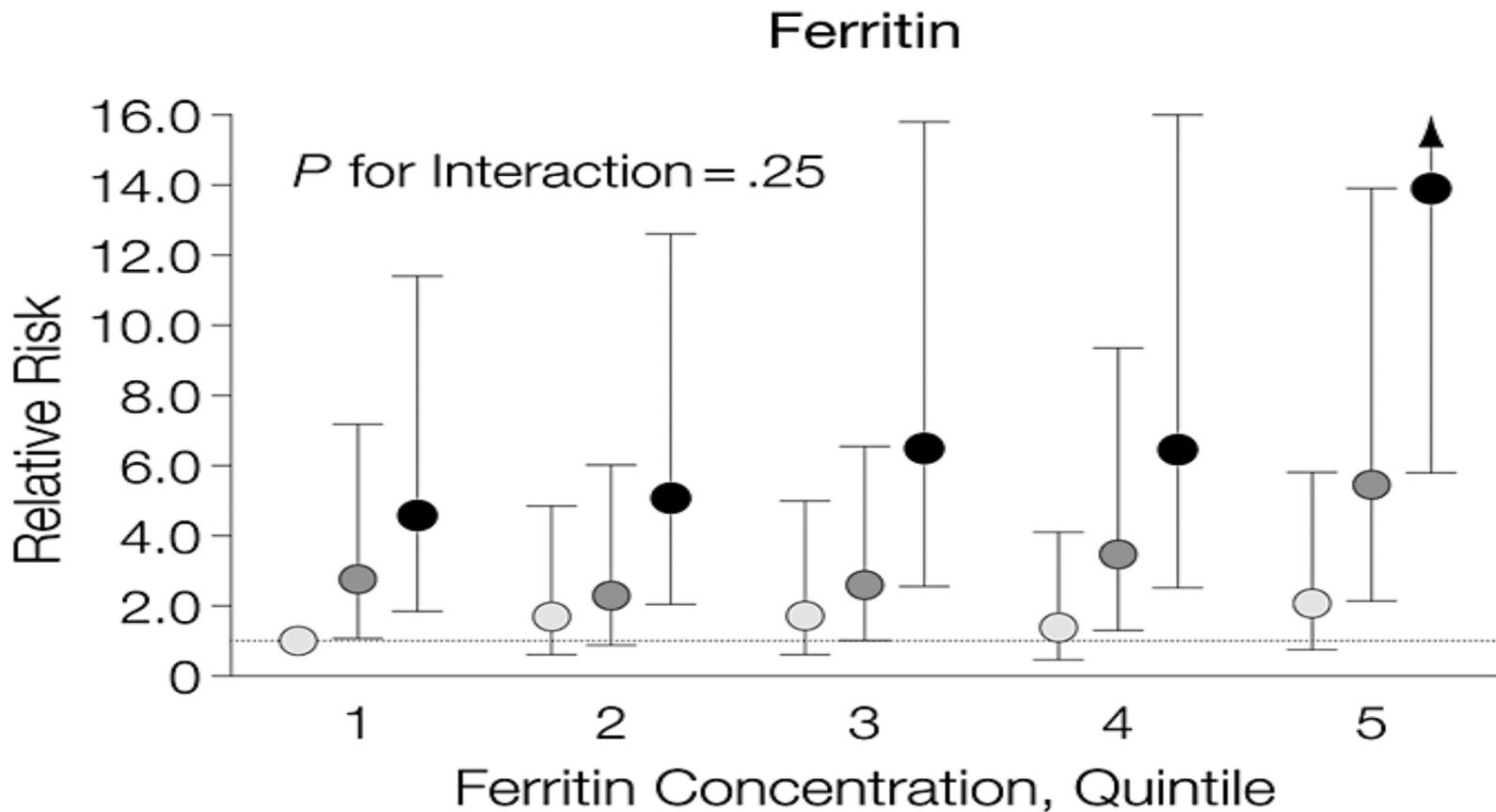




Inflamación y DM2

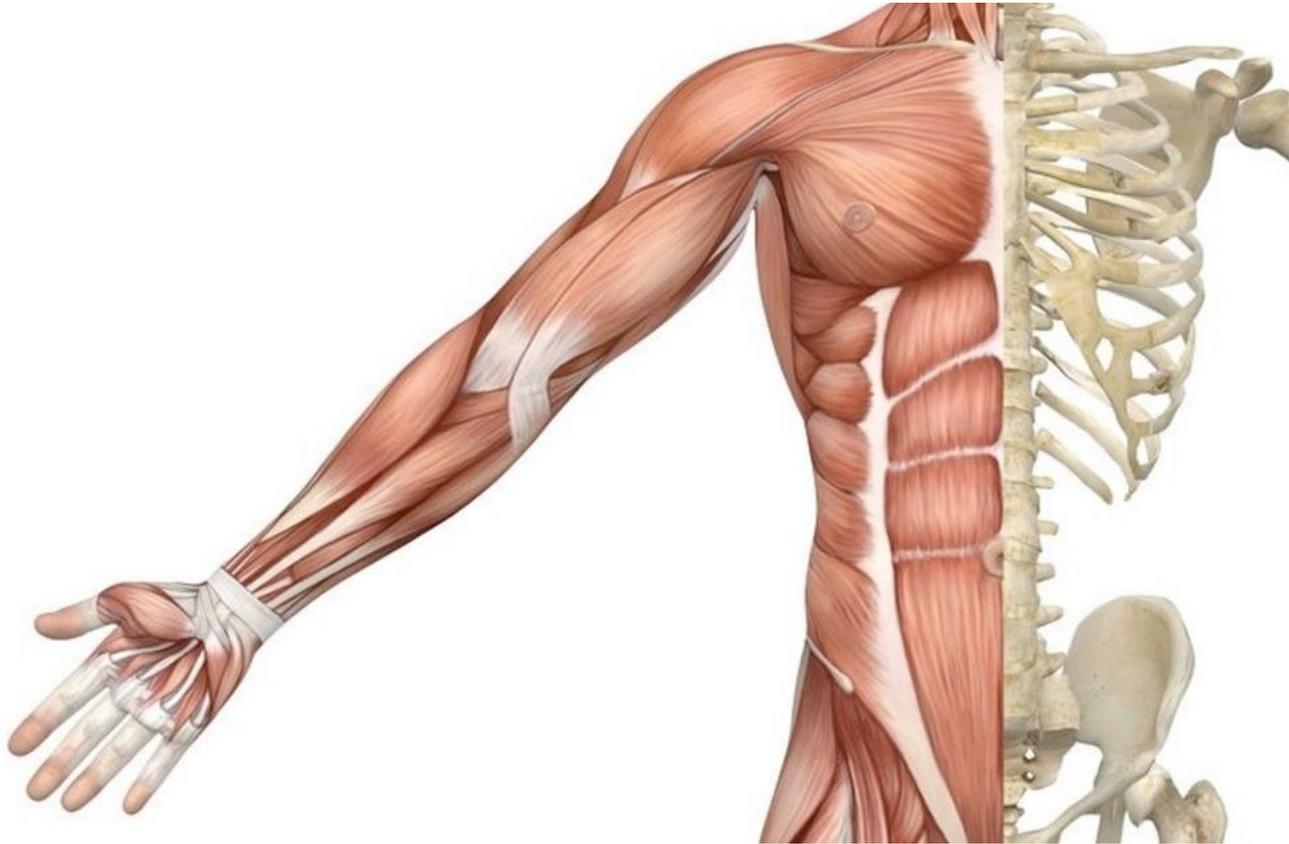
C-Reactive Protein Concentration, Tertile, mg/dL

○ (<0.095) ● (0.095-0.267) ● (≥0.267)



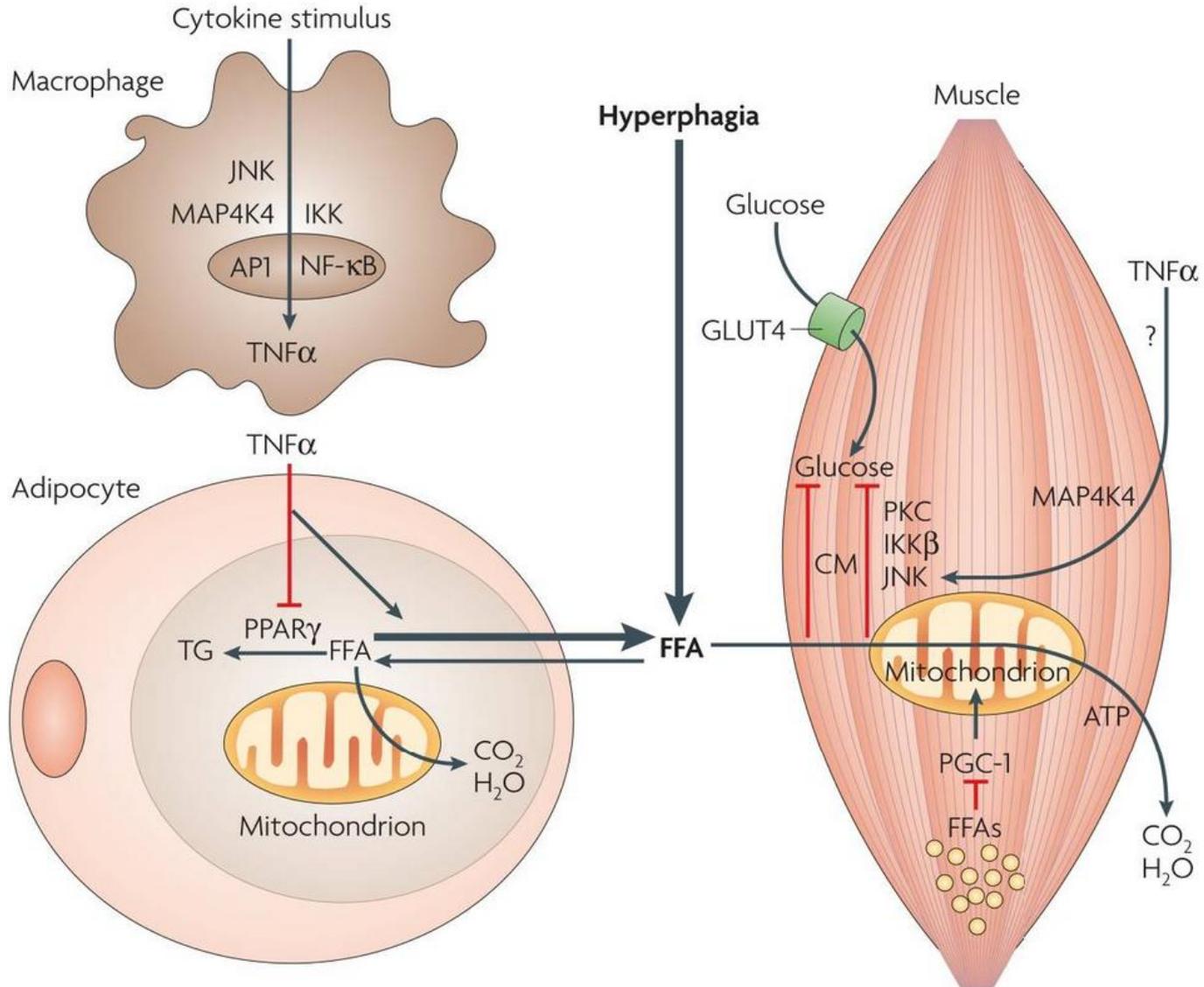


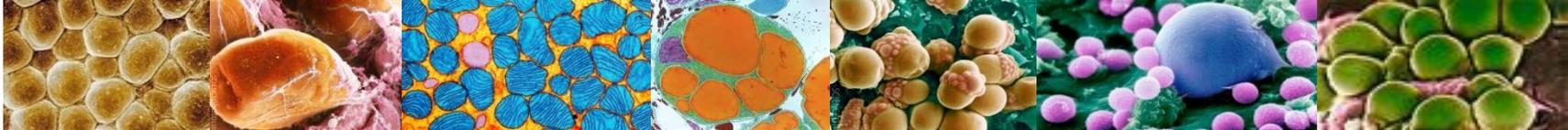
Inflamación y DM2





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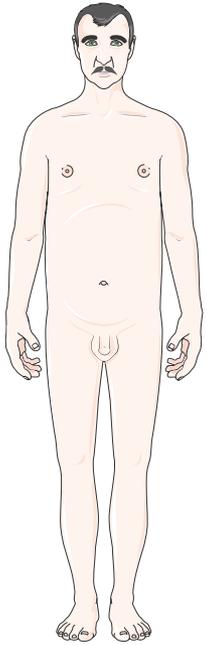




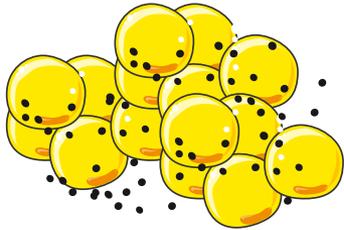
Inflamación y DM2



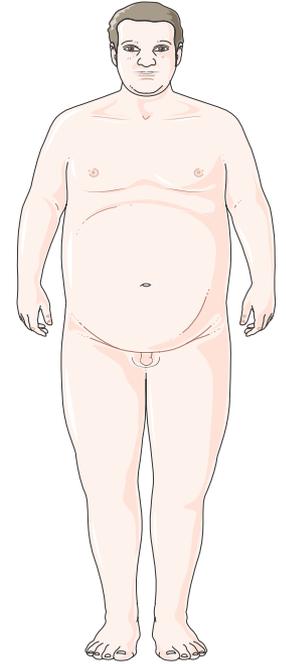
Lean



ADIPOCYTES

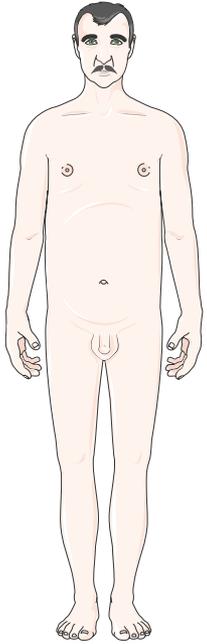


Overweight/Obese



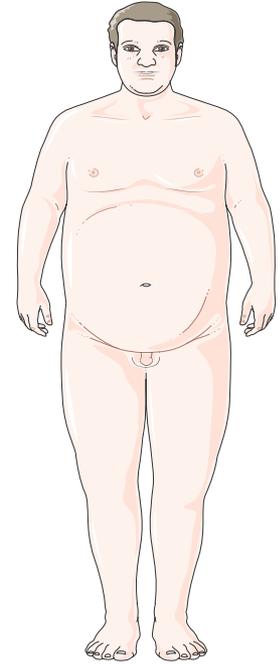
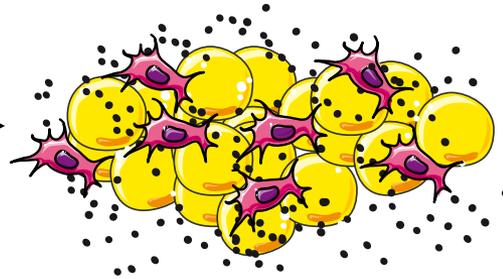
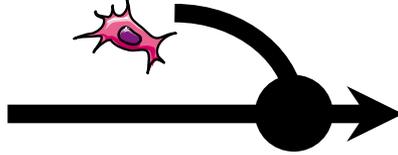
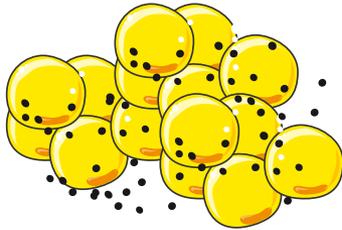
Lean

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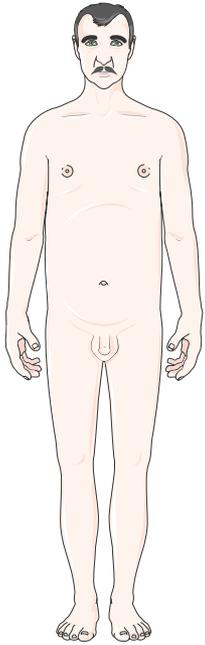
ADIPOCYTES

Macrophage infiltration



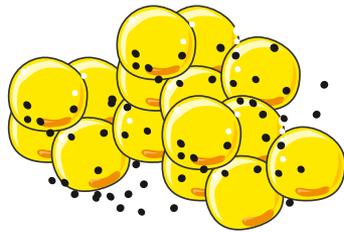
Lean

Overweight/Obese

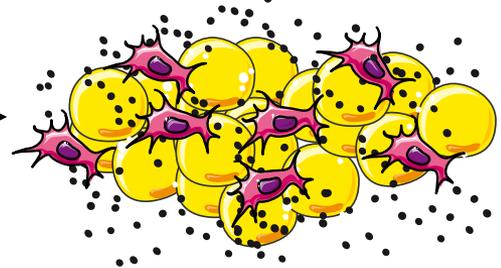
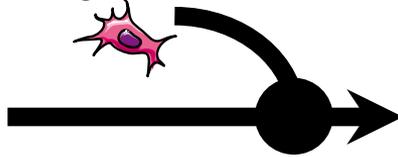


ADIPOCYTES

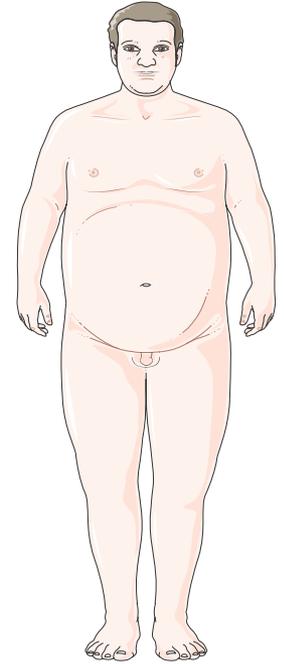
Macrophage infiltration



NON-INFLAMMATORY state



INFLAMMATORY state

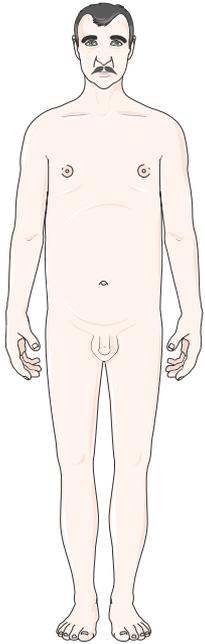


Lean

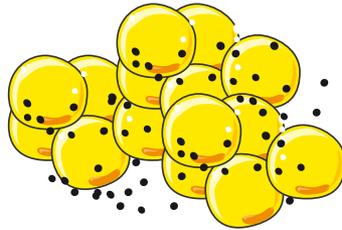
Overweight/Obese

INSULIN-SENSITIVE state

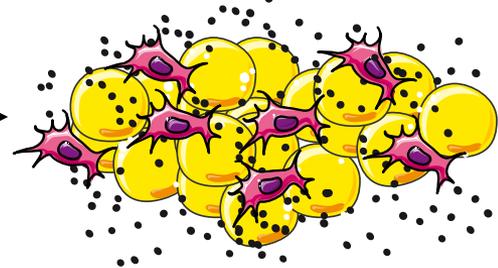
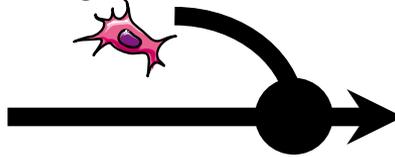
INSULIN-RESISTANT state



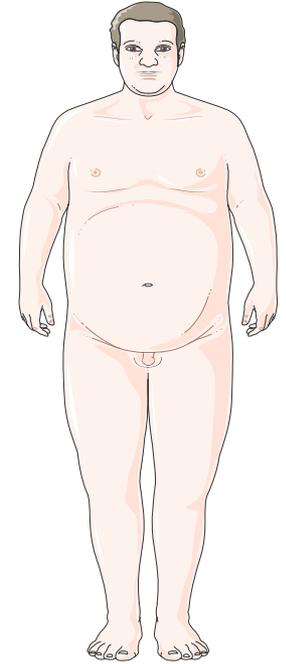
ADIPOCYTES Macrophage infiltration



NON-INFLAMMATORY state

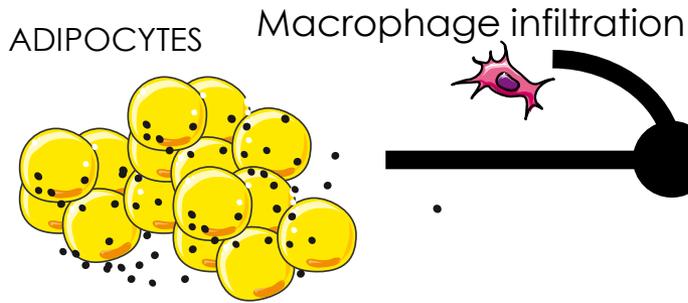
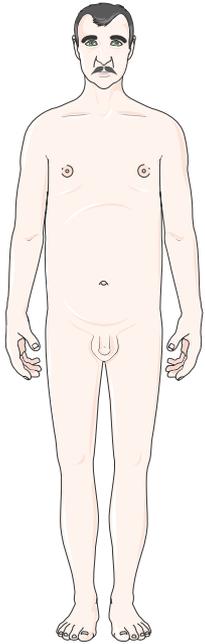


INFLAMMATORY state

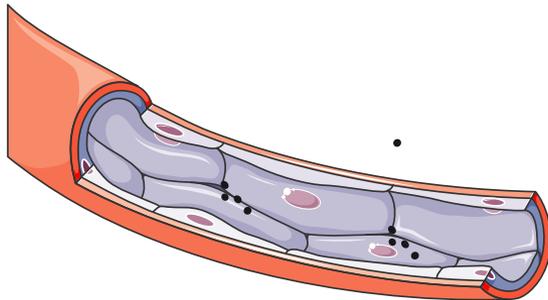


Lean

INSULIN-SENSITIVE state

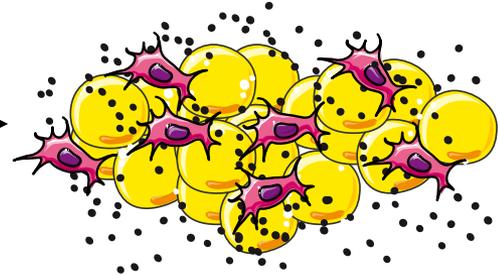
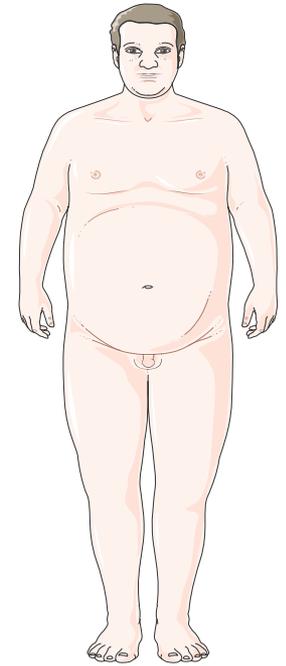


NON-INFLAMMATORY state

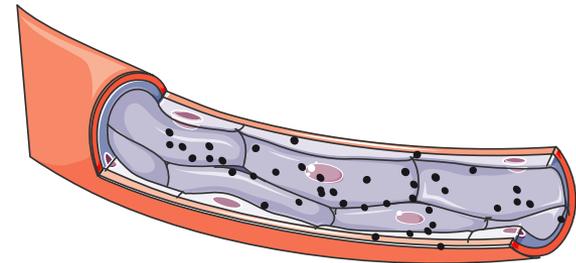


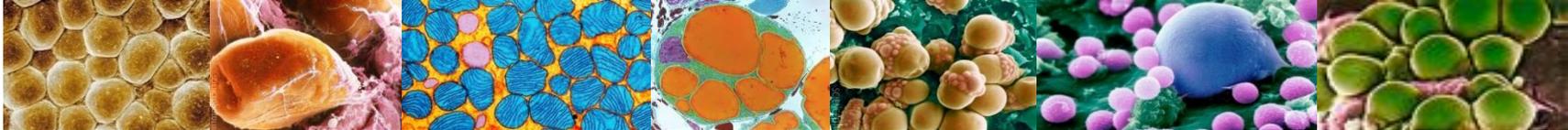
Overweight/Obese

INSULIN-RESISTANT state

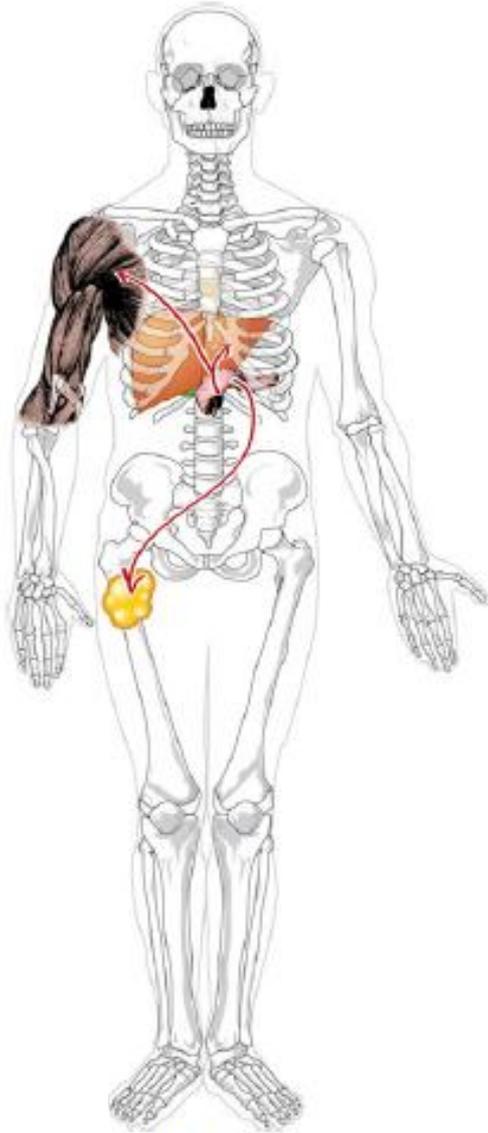


INFLAMMATORY state



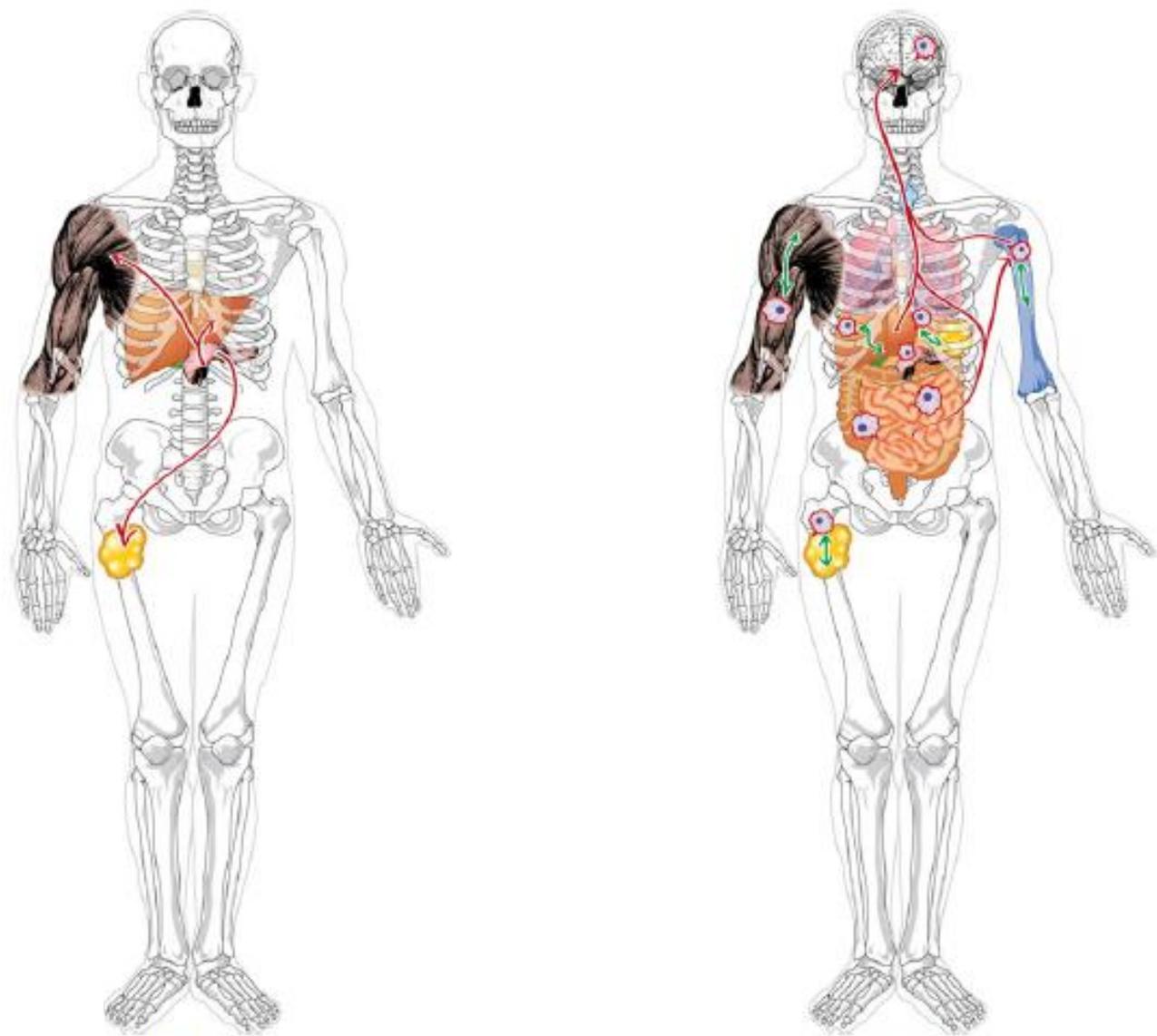


Inflamación y DM2





Inflamación y DM2

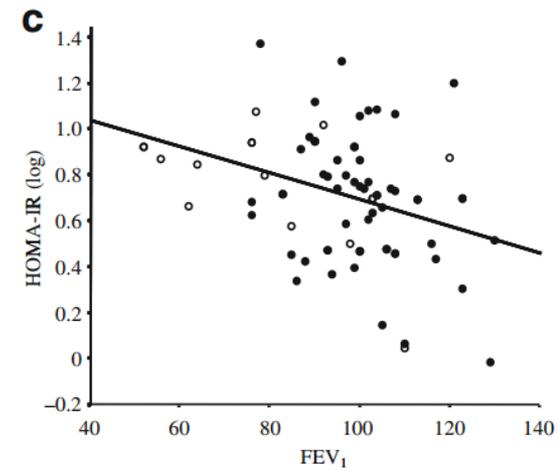
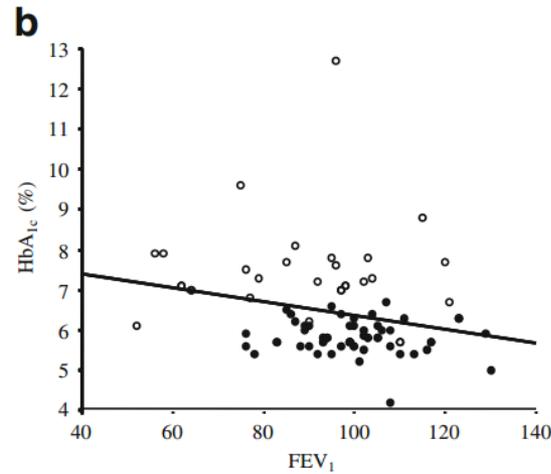
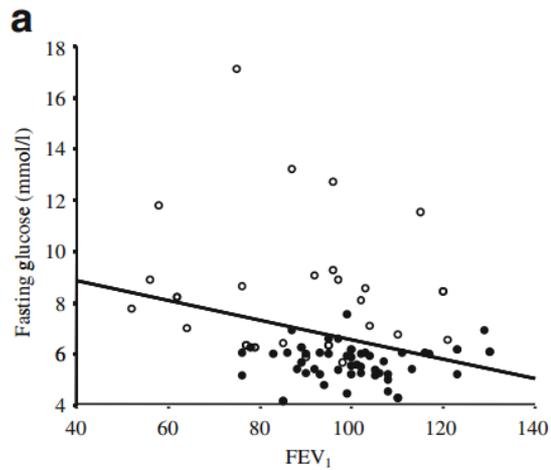
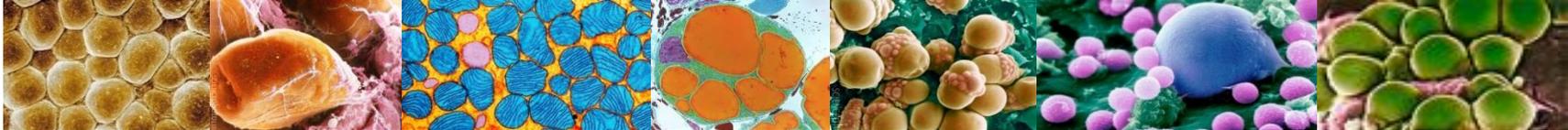


Now



Inflamación y DM2



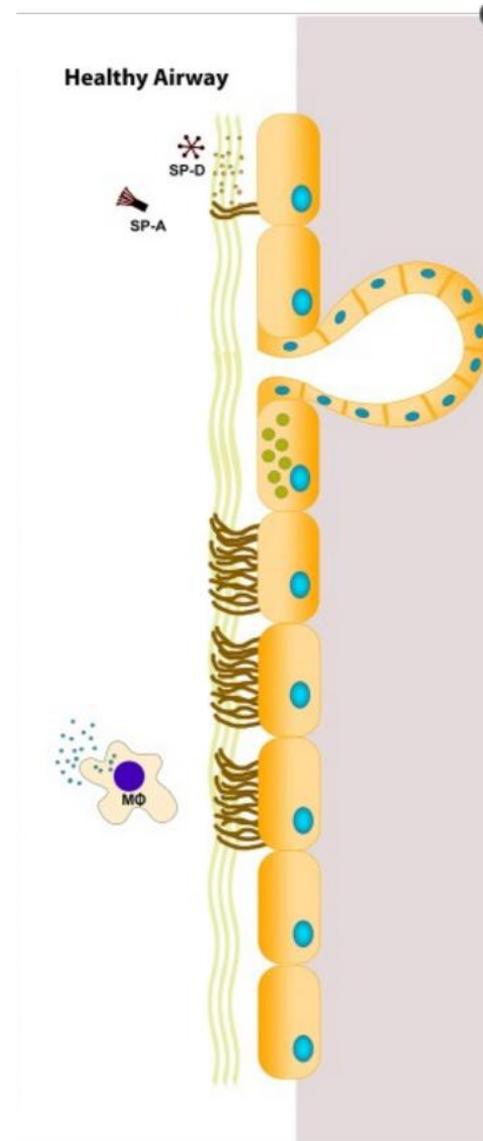


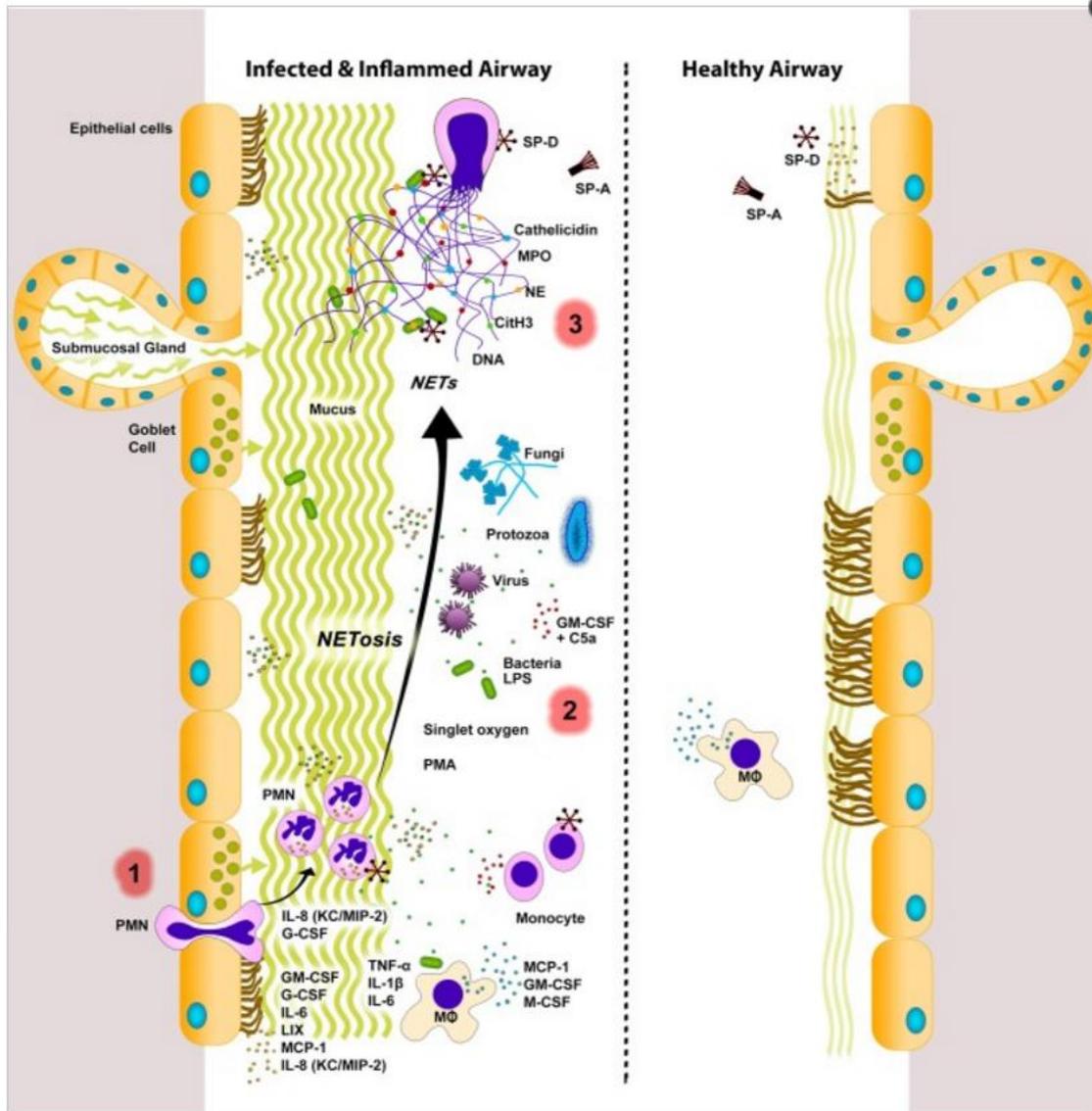
Diabetologia (2010) 53:1210–1216
DOI 10.1007/s00125-010-1700-5

ARTICLE

Type 2 diabetes impairs pulmonary function in morbidly obese women: a case–control study

A. Lecube • G. Sampol • X. Muñoz • C. Hernández •
J. Mesa • R. Simó





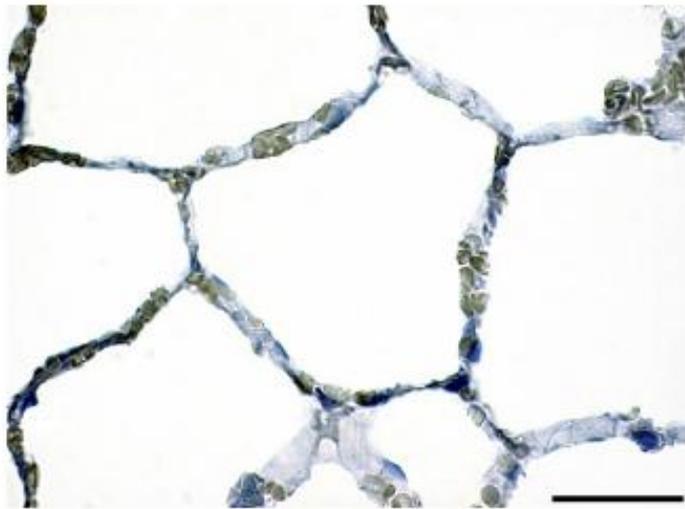
Fatty diabetic lung: altered alveolar structure and surfactant protein expression

David J. Foster,^{1*} Priya Ravikumar,^{1*} Dennis J. Bellotto,¹ Roger H. Unger,² and Connie C. W. Hsia¹

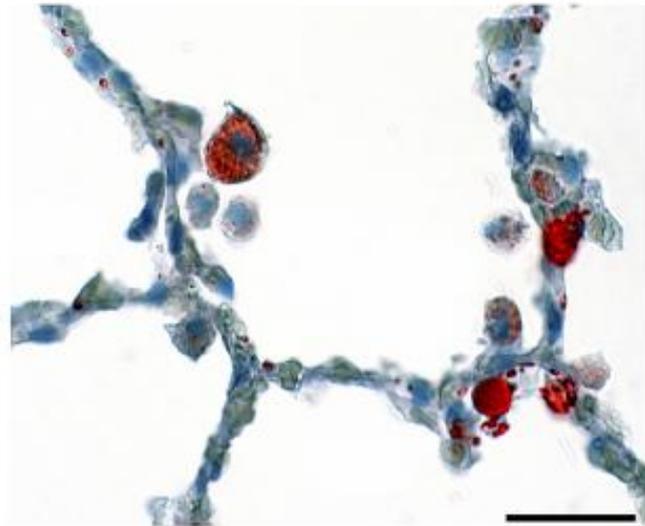
¹*Pulmonary and Critical Care Medicine, and* ²*Touchstone Diabetes Center, Department of Internal Medicine, University of Texas Southwestern Medical Center, Dallas, Texas*

Submitted 10 February 2009; accepted in final form 1 January 2010

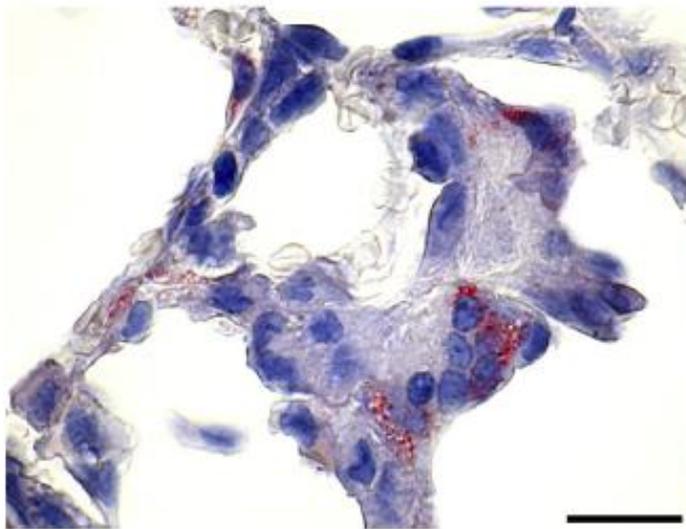
+/+



fa/fa



fa/fa



fa/fa



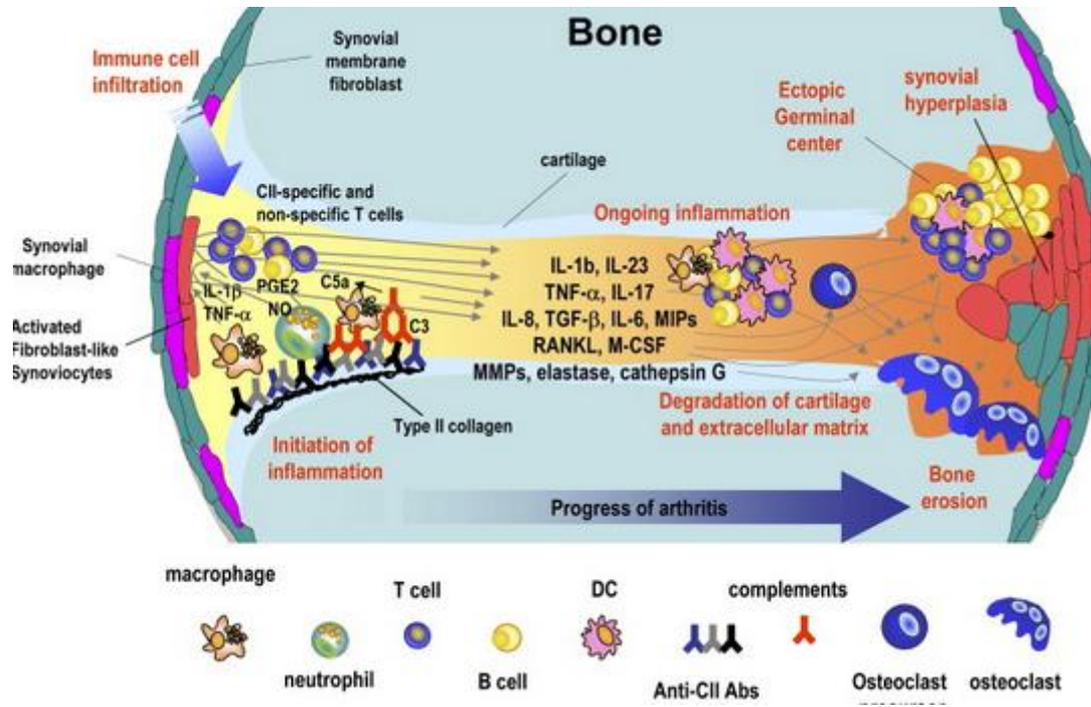


Inflamación y DM2





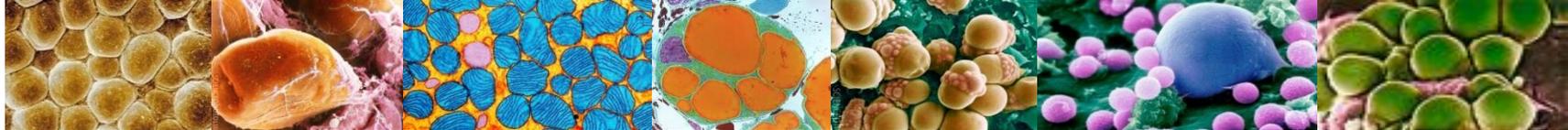
Inflamación y DM2





Inflamación y DM2

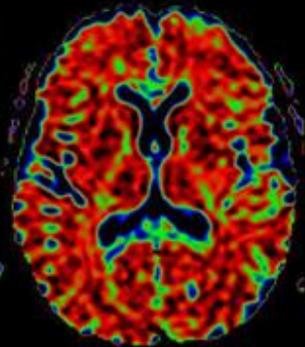




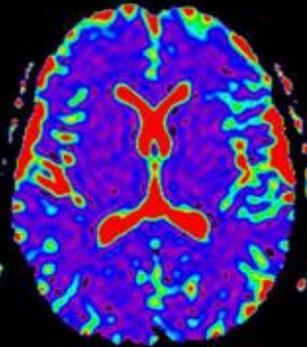
3.- Tensor de Difusión

- RM Philips Gyroscan 1.5T
- TR = 5393ms
- TE = 79ms
- N^o direcciones = 6
- Factor B = 800s/mm²
- TA = 1:26 sec
- Voxel Adq. = 3x3x3mm
- Voxel Rec. = 1.2x1.2x3mm
- CDV = 230x230mm

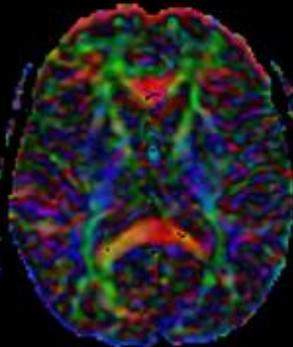
Olea Sphere 2.3



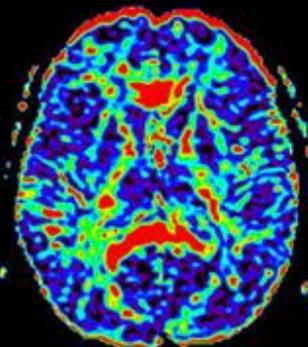
DIFUSIVIDAD
AXIAL



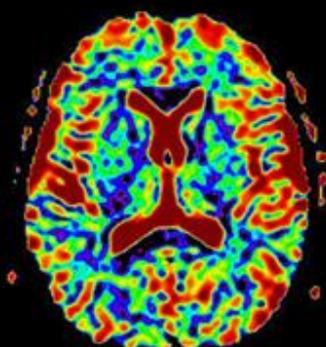
DIFUSIVIDAD
MEDIA



ANISOTROPÍA
FRACCIONAL



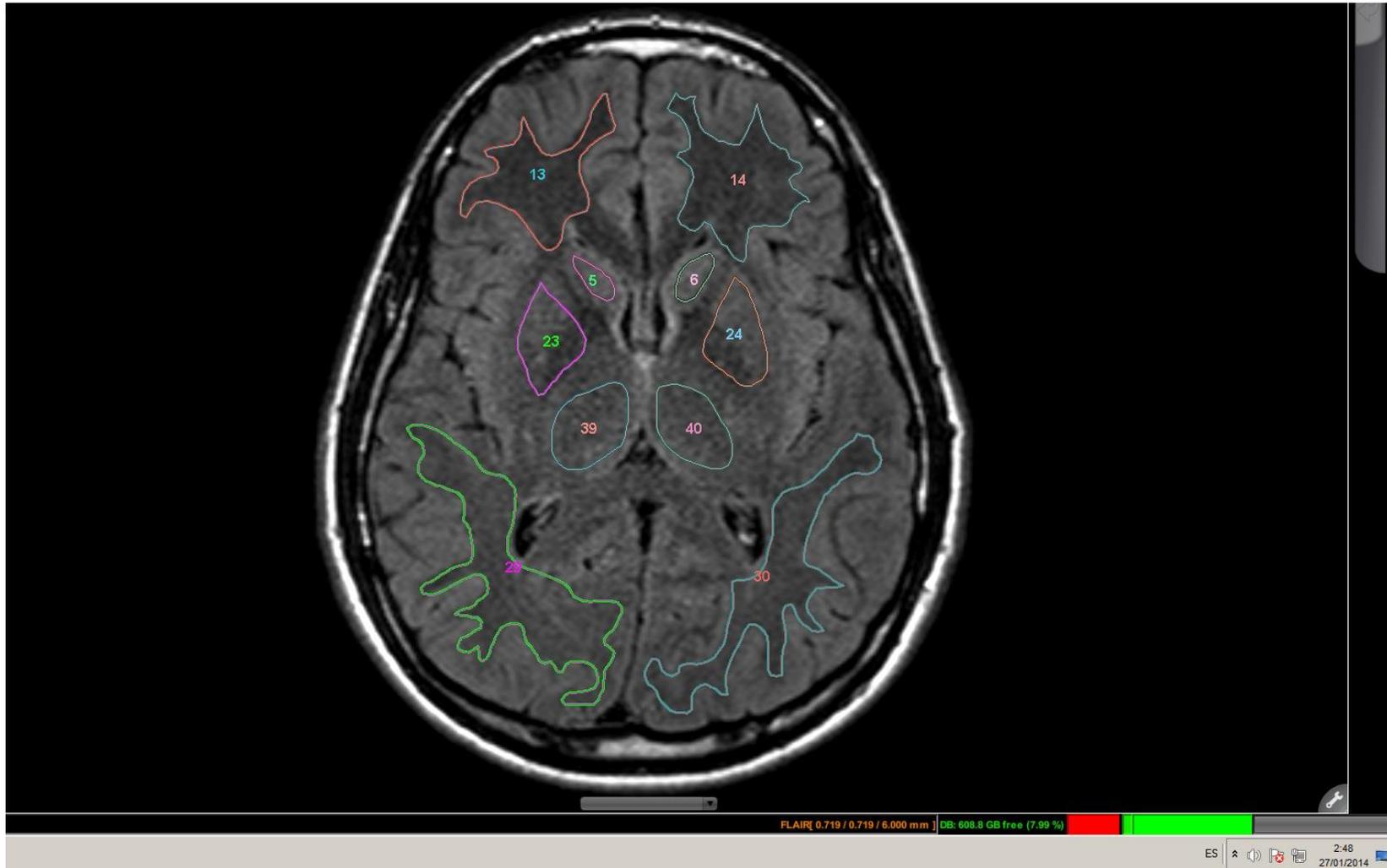
ANISOTROPÍA
RELATIVA

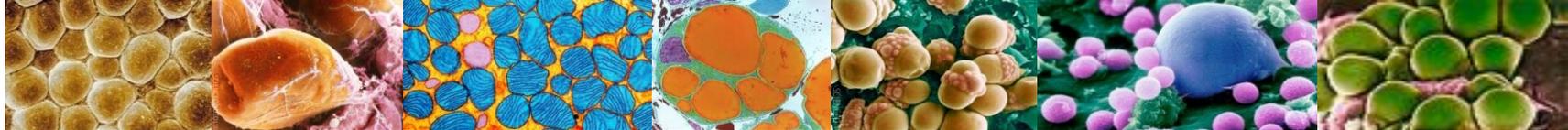


DIFUSIVIDAD
RADIAL



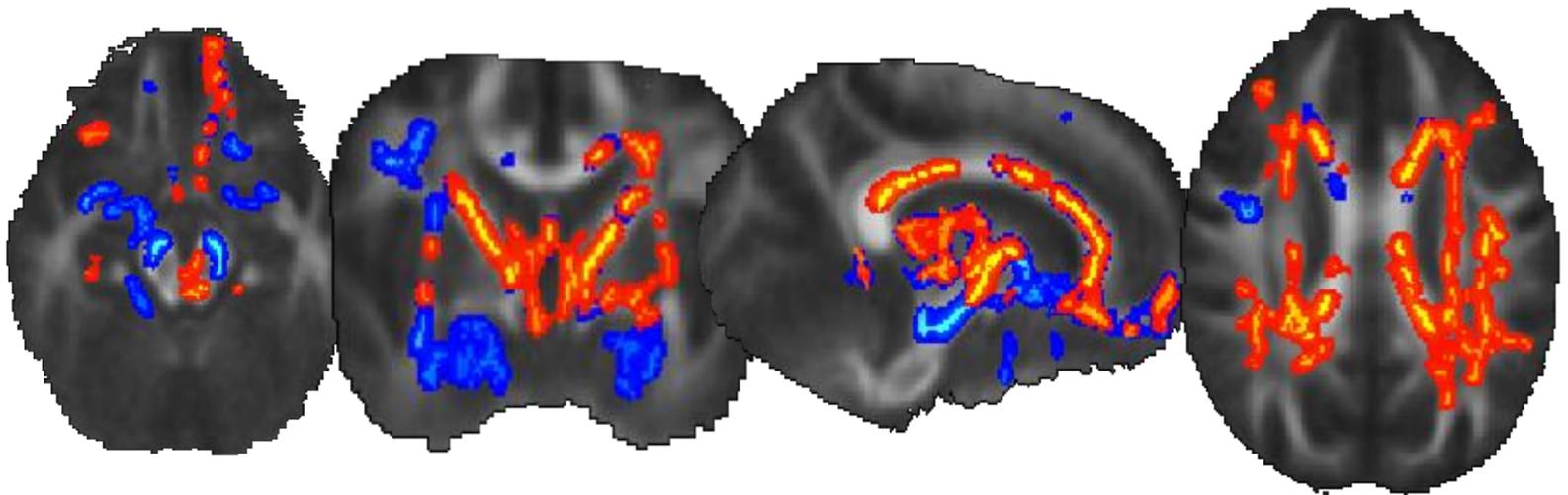
Inflamación y DM2





Cambios Estructurales

Relacionados con el Síndrome Metabólico

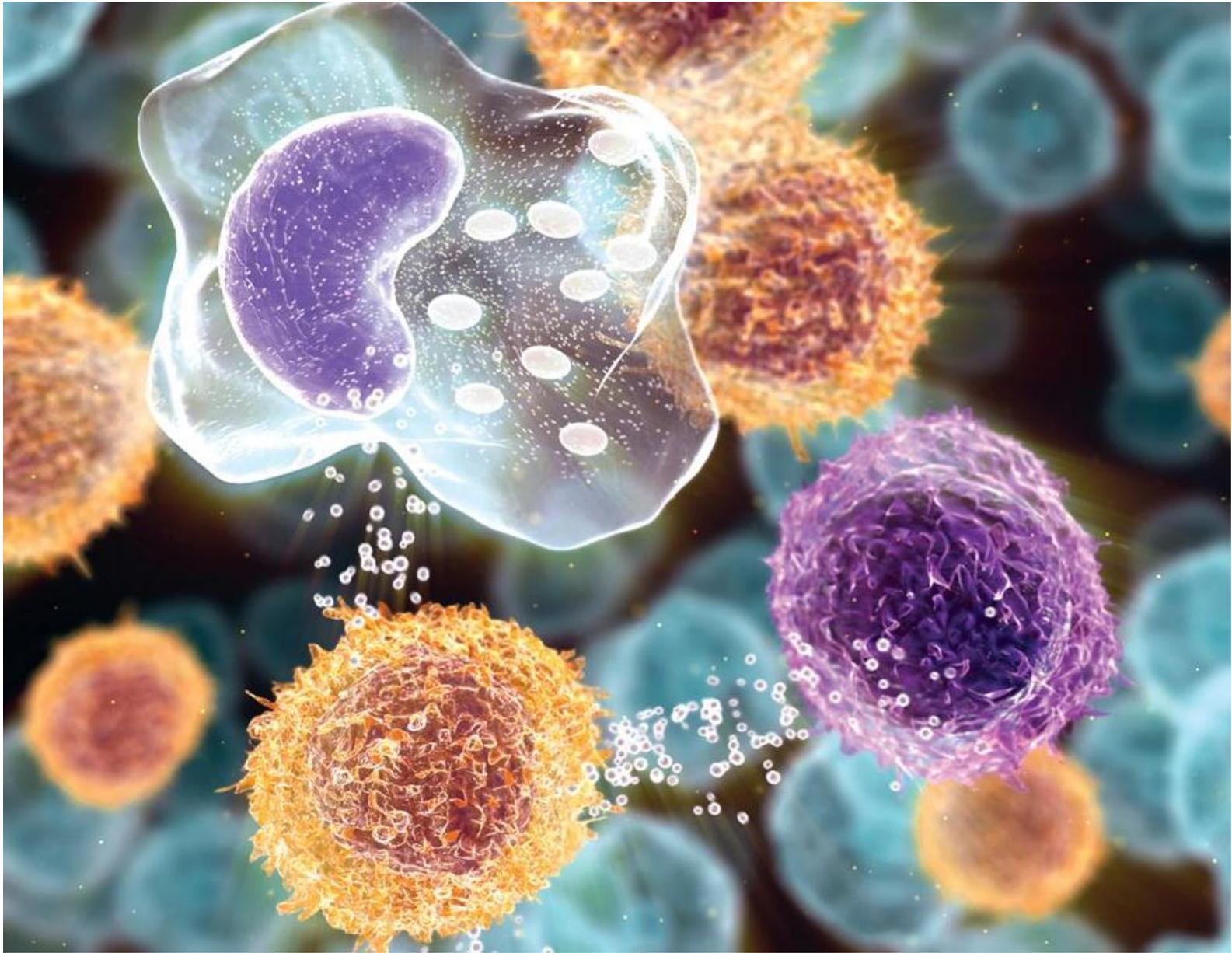


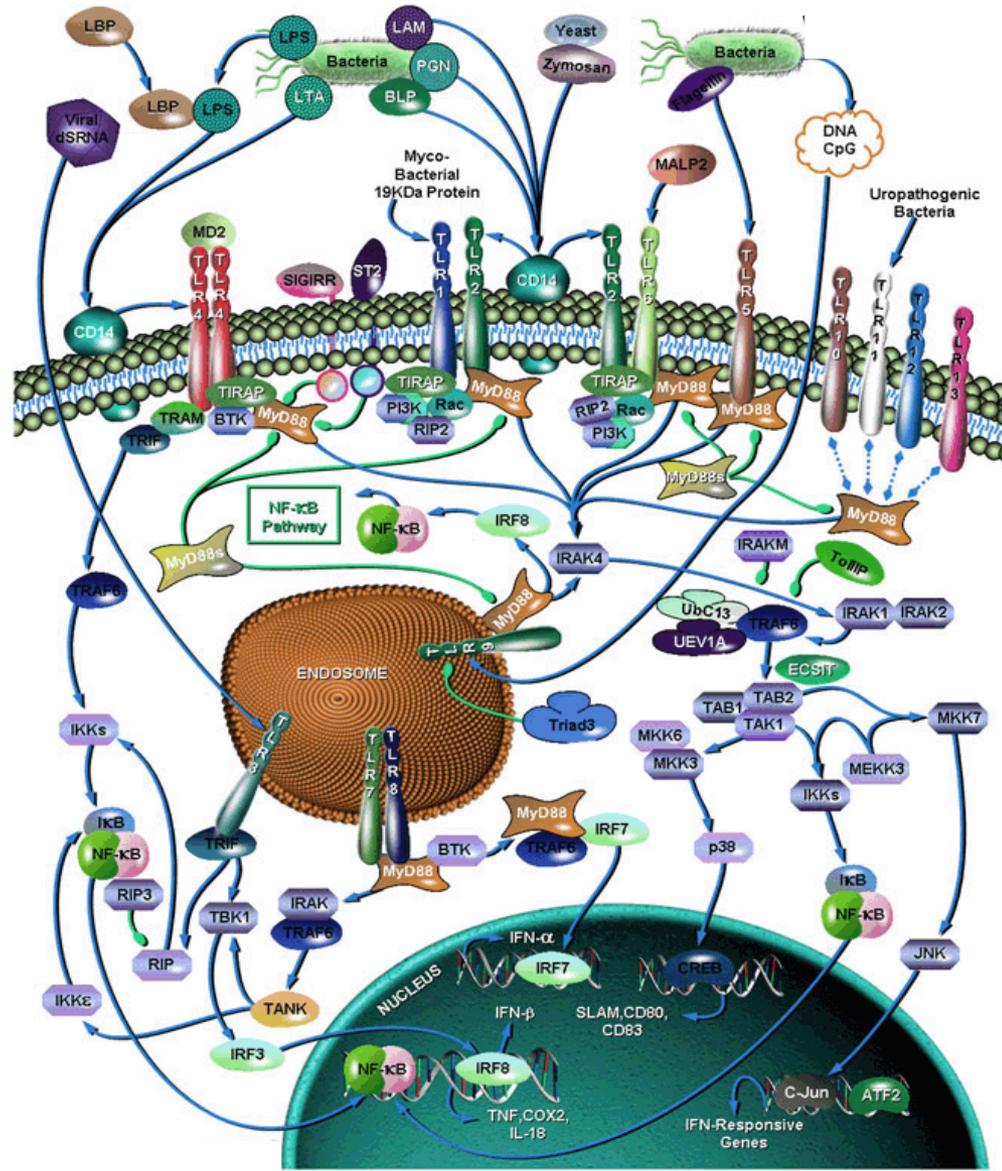
Tract Based Spatial Statistics

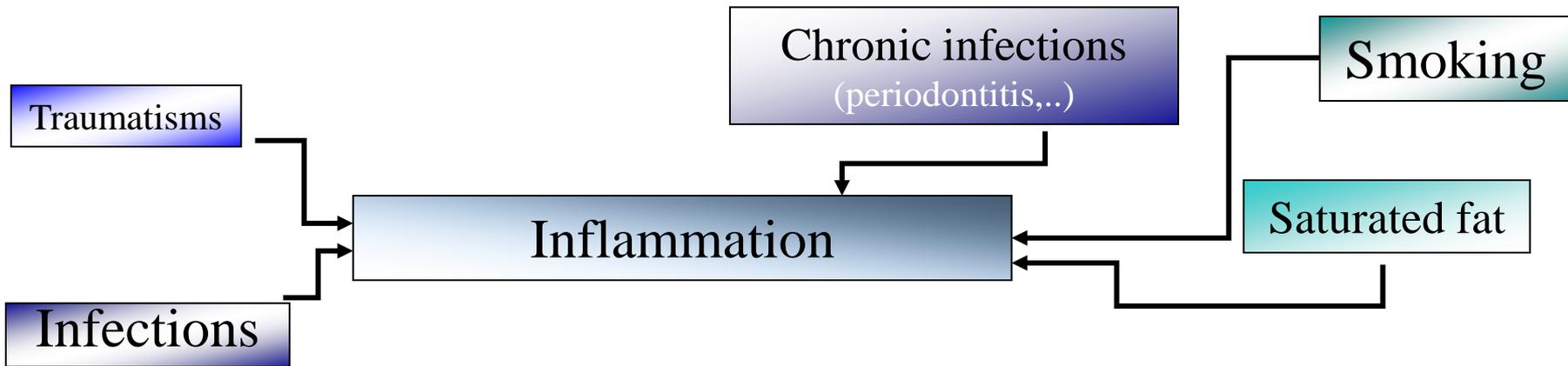


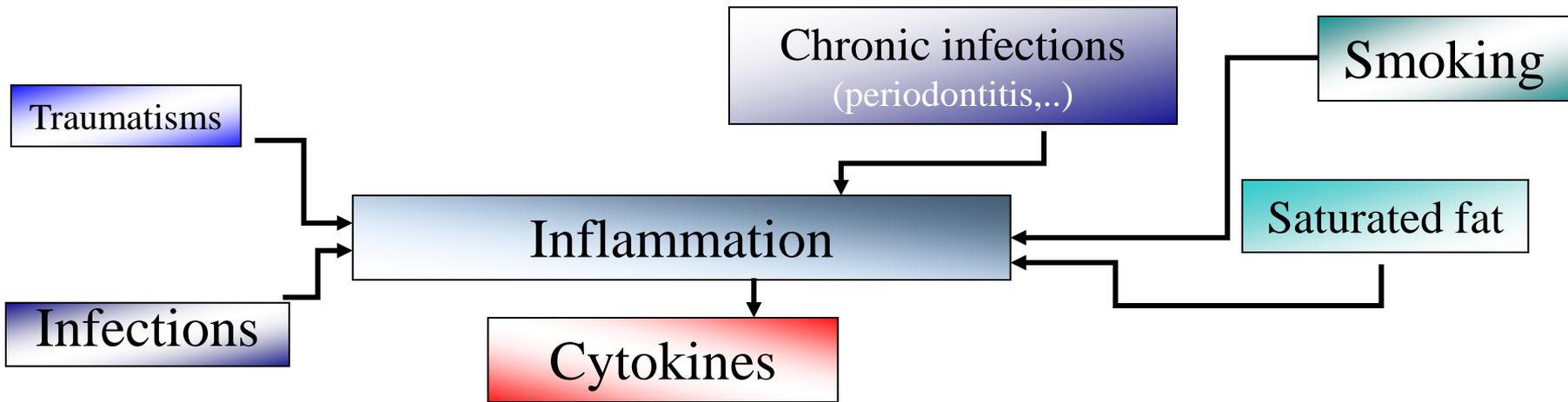
Inflamación y DM-2:

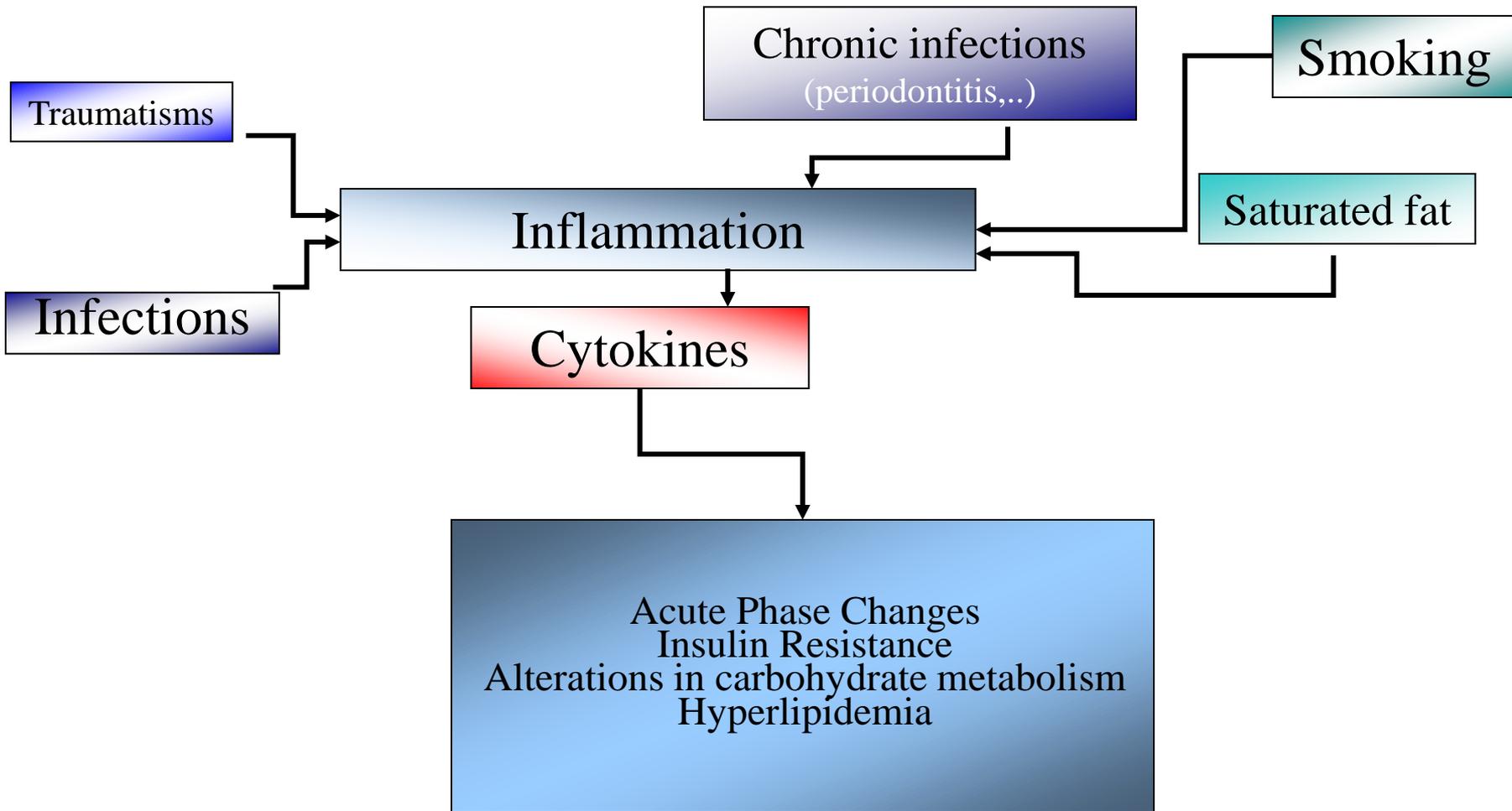
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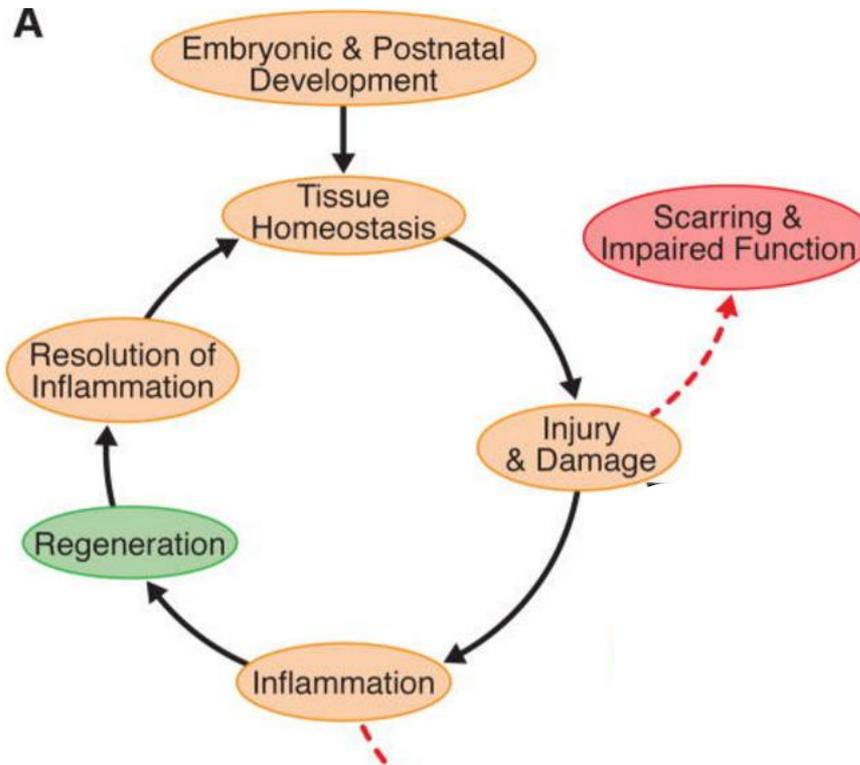


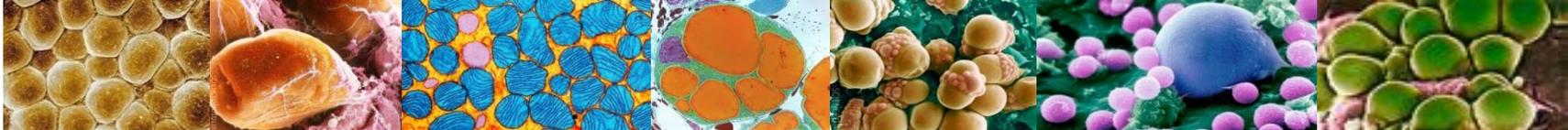




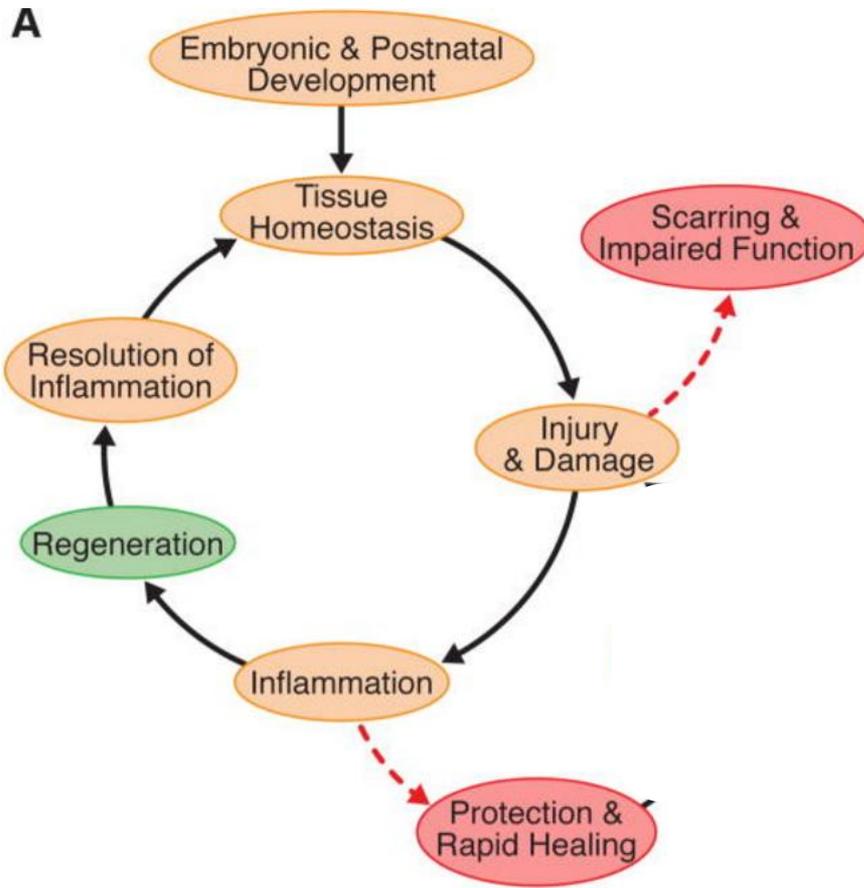


Inflamación y DM2



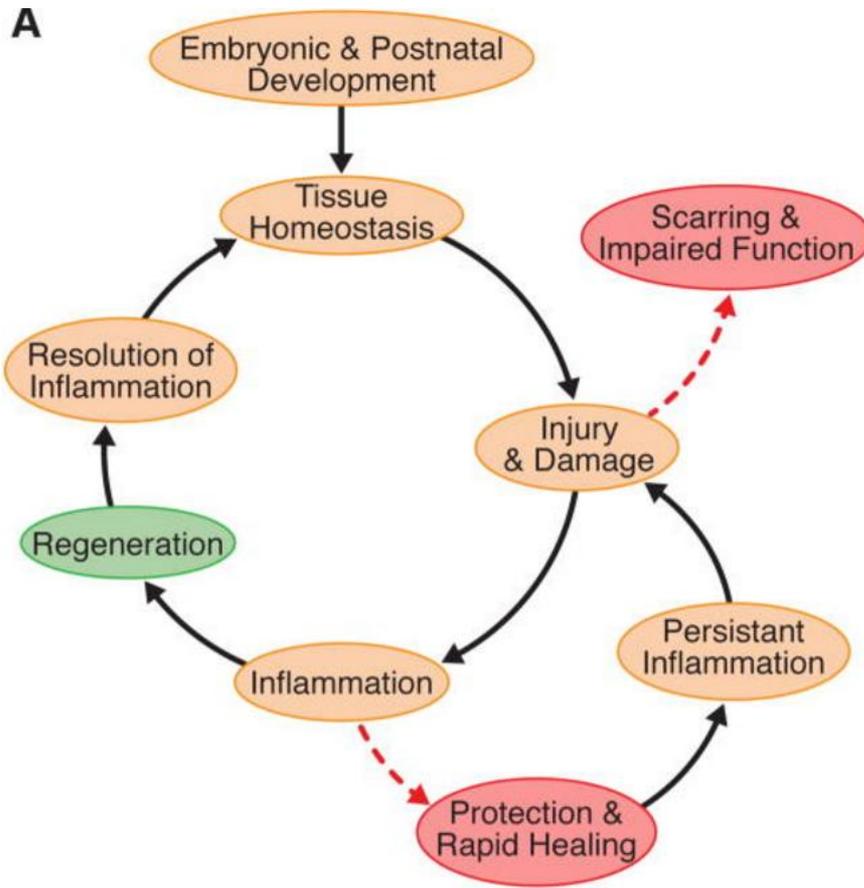


Inflamación y DM2



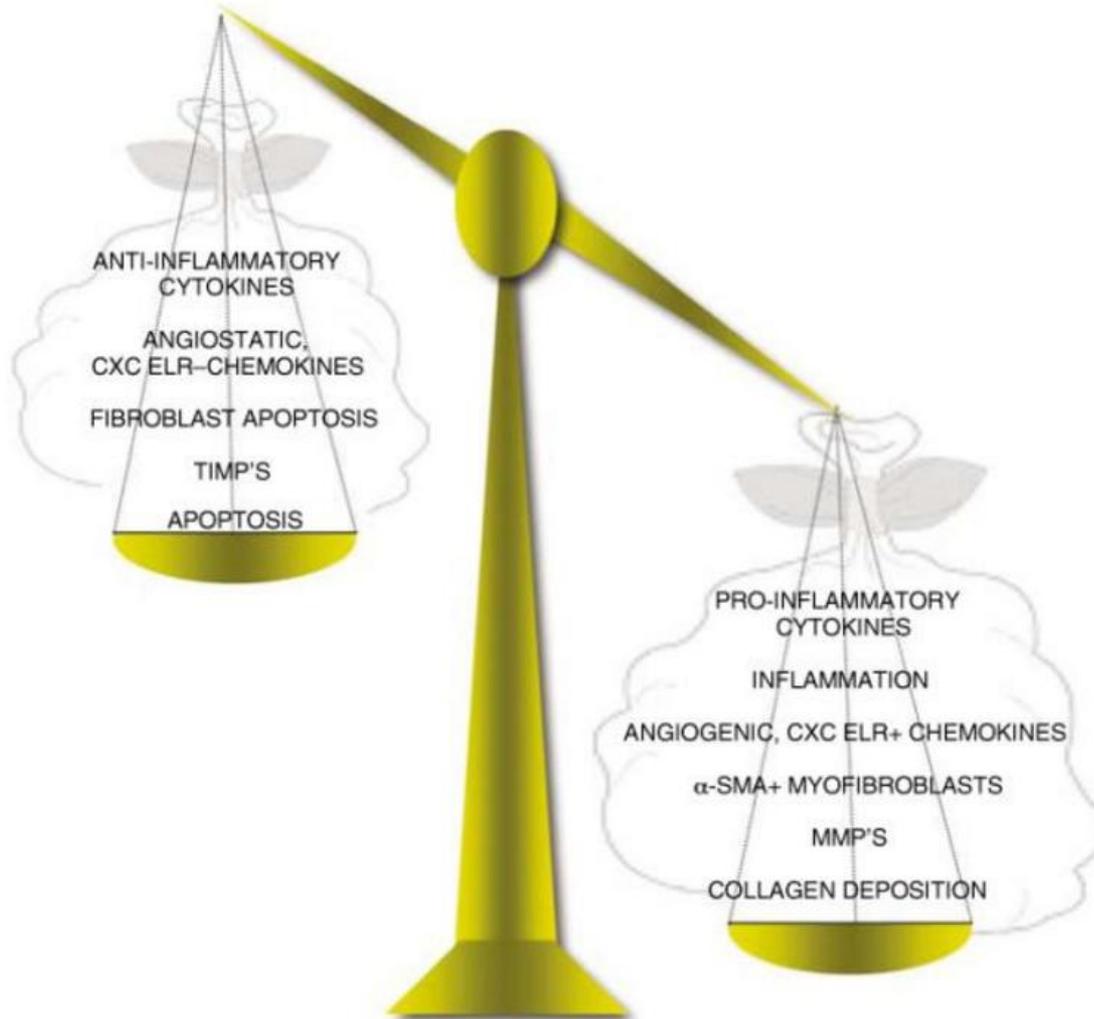


Inflamación y DM2



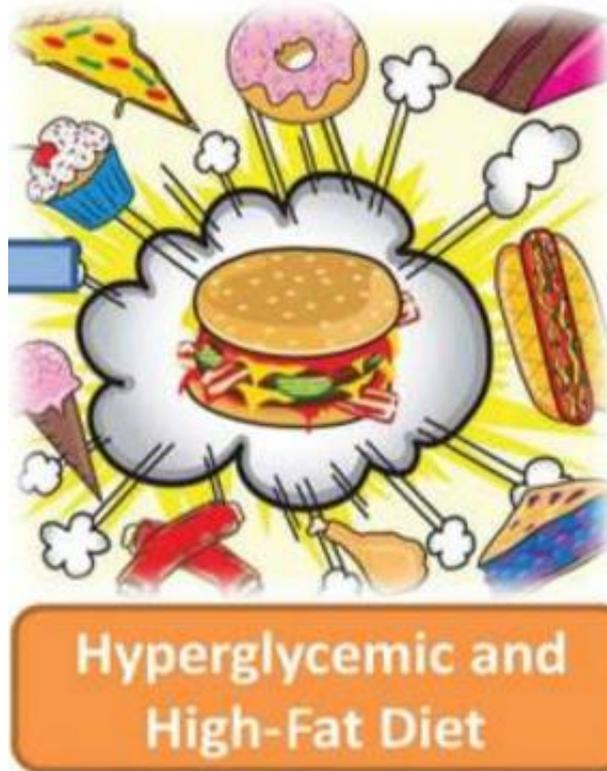


Inflamación y DM2





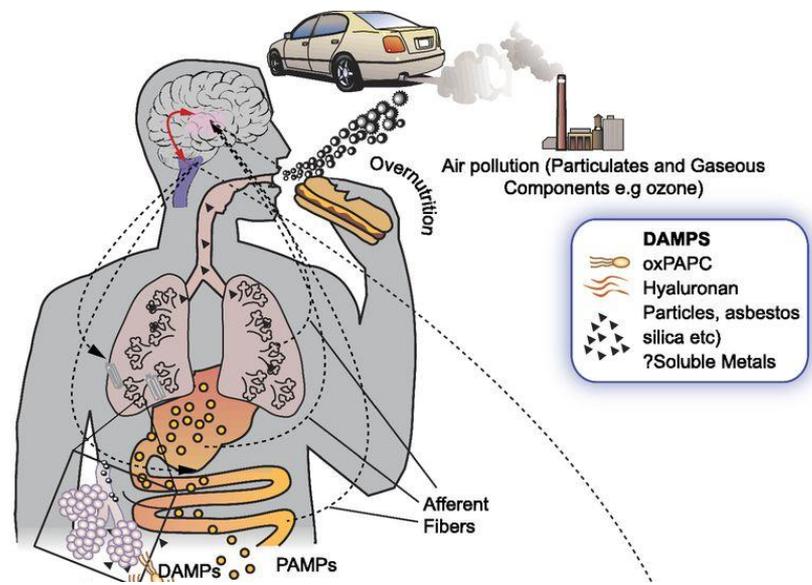
Inflamación y DM2

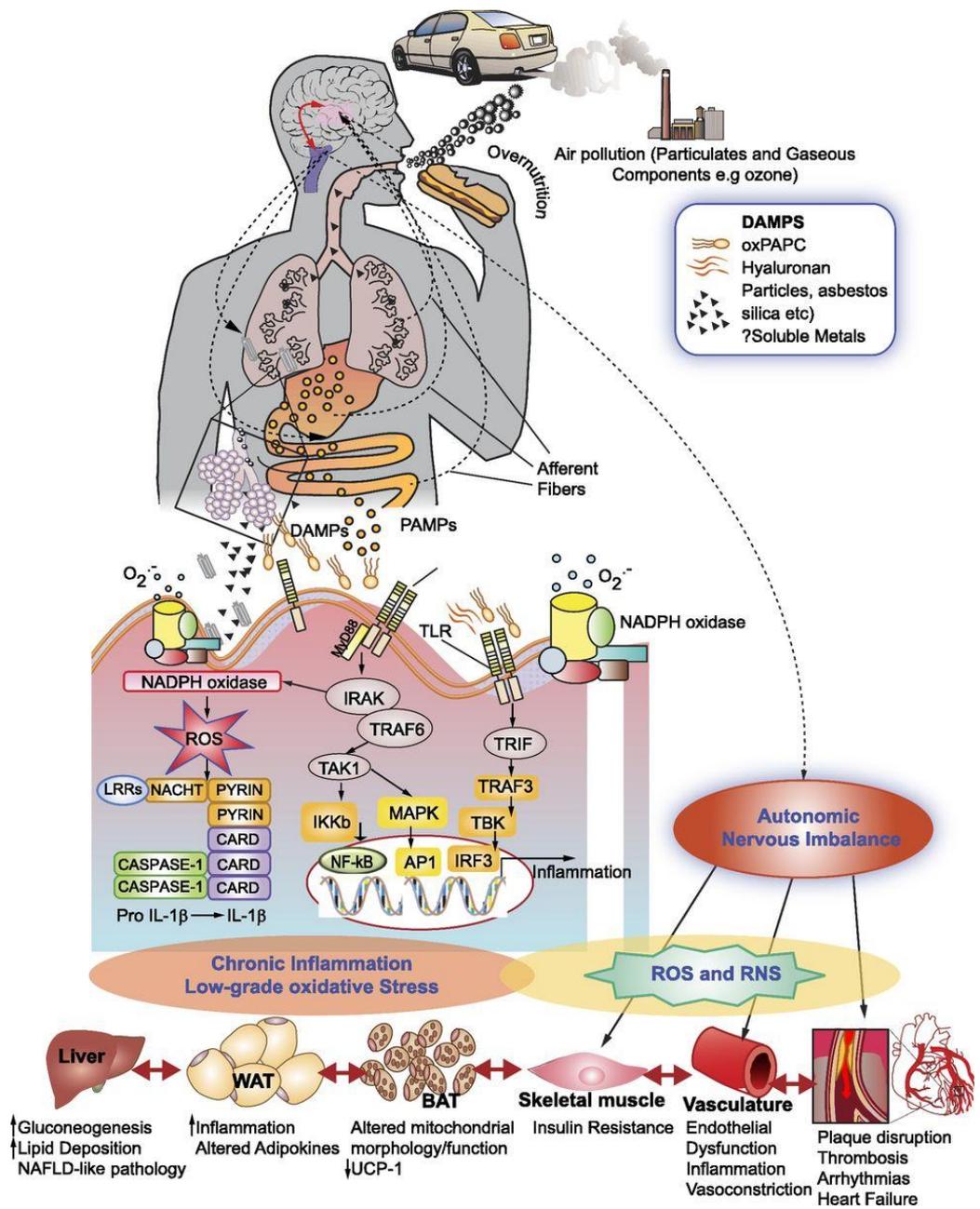




Inflamación y DM2







Relation of active, passive, and quitting smoking with incident type 2 diabetes: a systematic review and meta-analysis



An Pan*, Yeli Wang*, Mohammad Taleai, Frank B Hu, Tangchun Wu

Summary

Background Cigarette smoking remains the leading avoidable cause of disease burden worldwide, and observational studies have linked various smoking behaviours (active smoking, passive smoking, and smoking cessation) with risk of type 2 diabetes. We did a meta-analysis of prospective studies to investigate the associations between various smoking behaviours and diabetes risk.

Methods We systematically searched MEDLINE (up to May 3, 2015) and Embase (up to April 16, 2014) for reports of prospective studies, using search terms related to smoking, diabetes mellitus, and studies with a prospective design. We supplemented this strategy with manual searches of the reference lists of retrieved publications and relevant reviews. We included prospective studies that reported risk of type 2 diabetes by baseline smoking status. We calculated pooled relative risks (RRs) with 95% CIs using random-effects models, and did subgroup analyses by participant and study characteristics.

Findings We identified 88 eligible prospective studies with 5 898 795 participants and 295 446 incident cases of type 2 diabetes. The pooled RR of type 2 diabetes was 1.37 (95% CI 1.33–1.42) for comparing current smoking with non-smoking (84 studies with 5 853 952 participants), 1.14 (1.10–1.18) for comparing former smoking with never smoking (47 studies with 2 930 391 participants), and 1.22 (1.10–1.35) for comparing never smokers with and without exposure to passive smoke (seven studies with 156 439 participants). The associations persisted in all subgroups, and we identified a dose-response relation for current smoking and diabetes risk: compared with never smokers, the RRs were 1.21 (1.10–1.33) for light smokers, 1.34 (1.27–1.41) for moderate smokers, and 1.57 (1.47–1.66) for heavy smokers. Based on the assumption that the association between smoking and diabetes risk is causal, we estimated that 11.7% of cases of type 2 diabetes in men and 2.4% in women (ie, about 27.8 million cases in total worldwide) were attributable to active smoking. Compared with never smokers, the pooled RR from ten studies with 1 086 608 participants was 1.54 (95% CI 1.36–1.74) for new quitters (<5 years), 1.18 (1.07–1.29) for middle-term quitters (5–9 years), and 1.11 (1.02–1.20) for long-term quitters (≥ 10 years).

Interpretation Active and passive smoking are associated with significantly increased risks of type 2 diabetes. The risk of diabetes is increased in new quitters, but decreases substantially as the time since quitting increases. If the association between smoking and risk of type 2 diabetes is causal, public health efforts to reduce smoking could have a substantial effect on the worldwide burden of type 2 diabetes.

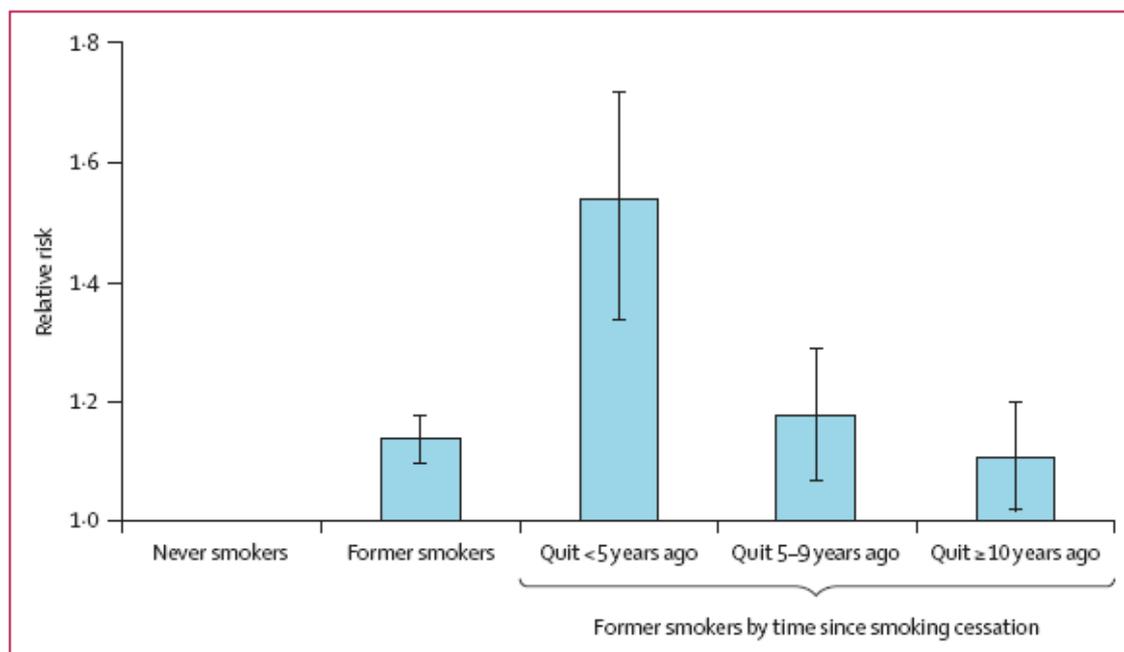
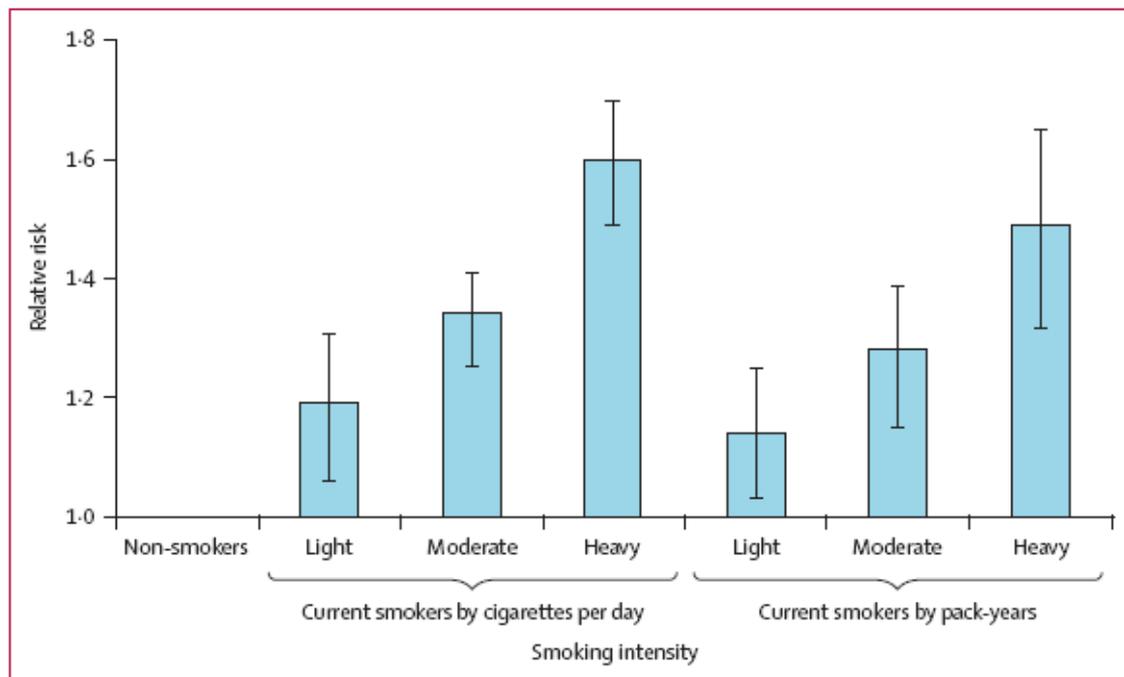
Lancet Diabetes Endocrinol 2015

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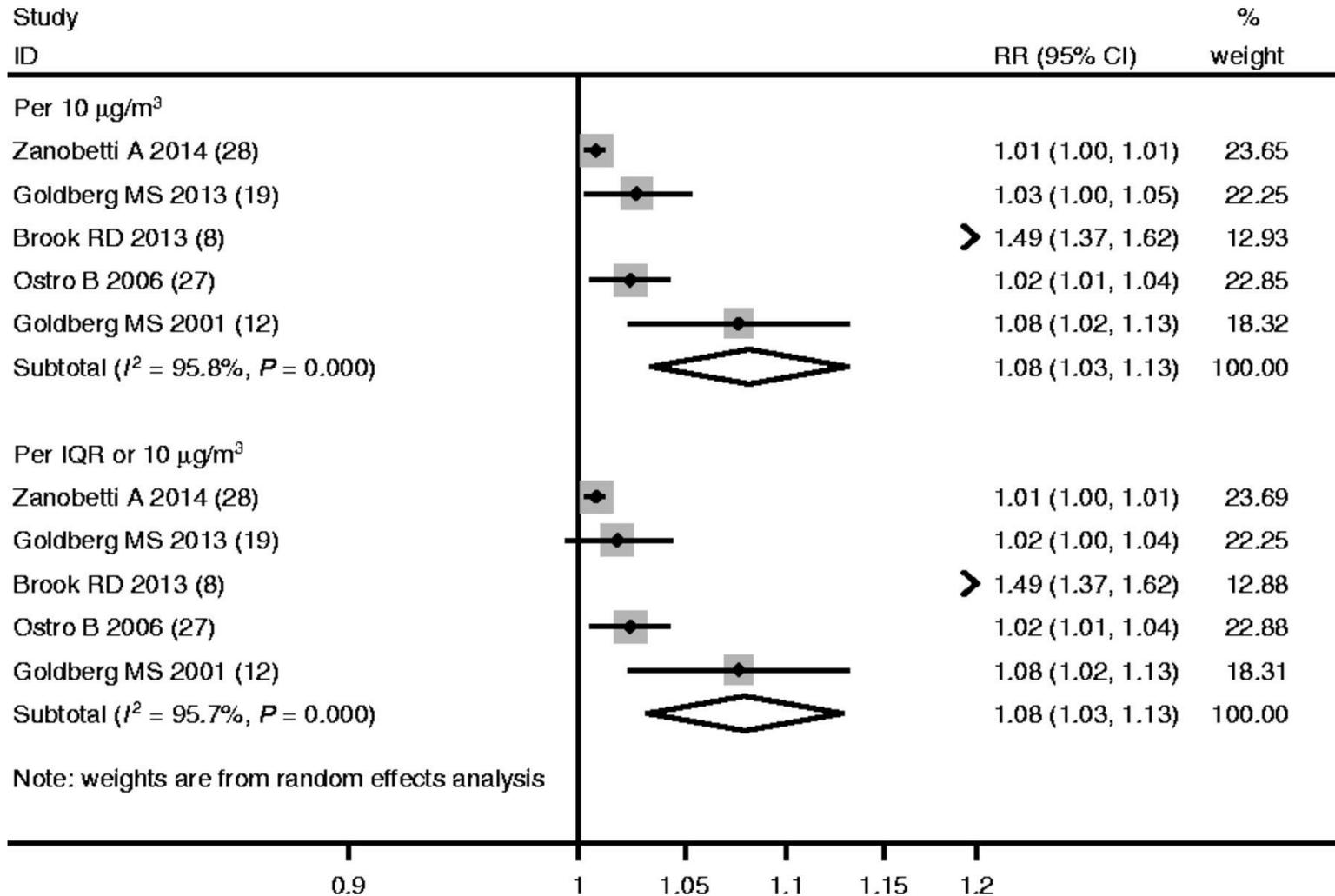
See Online/Comment
[http://dx.doi.org/10.1016/S2213-8587\(15\)00341-1](http://dx.doi.org/10.1016/S2213-8587(15)00341-1)

*Contributed equally

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Association between the high level of PM2.5 and diabetes-associated mortality.



Chengqian Li et al. Eur J Endocrinol 2014;171:R183-R190

Urmila P. Kodavanti

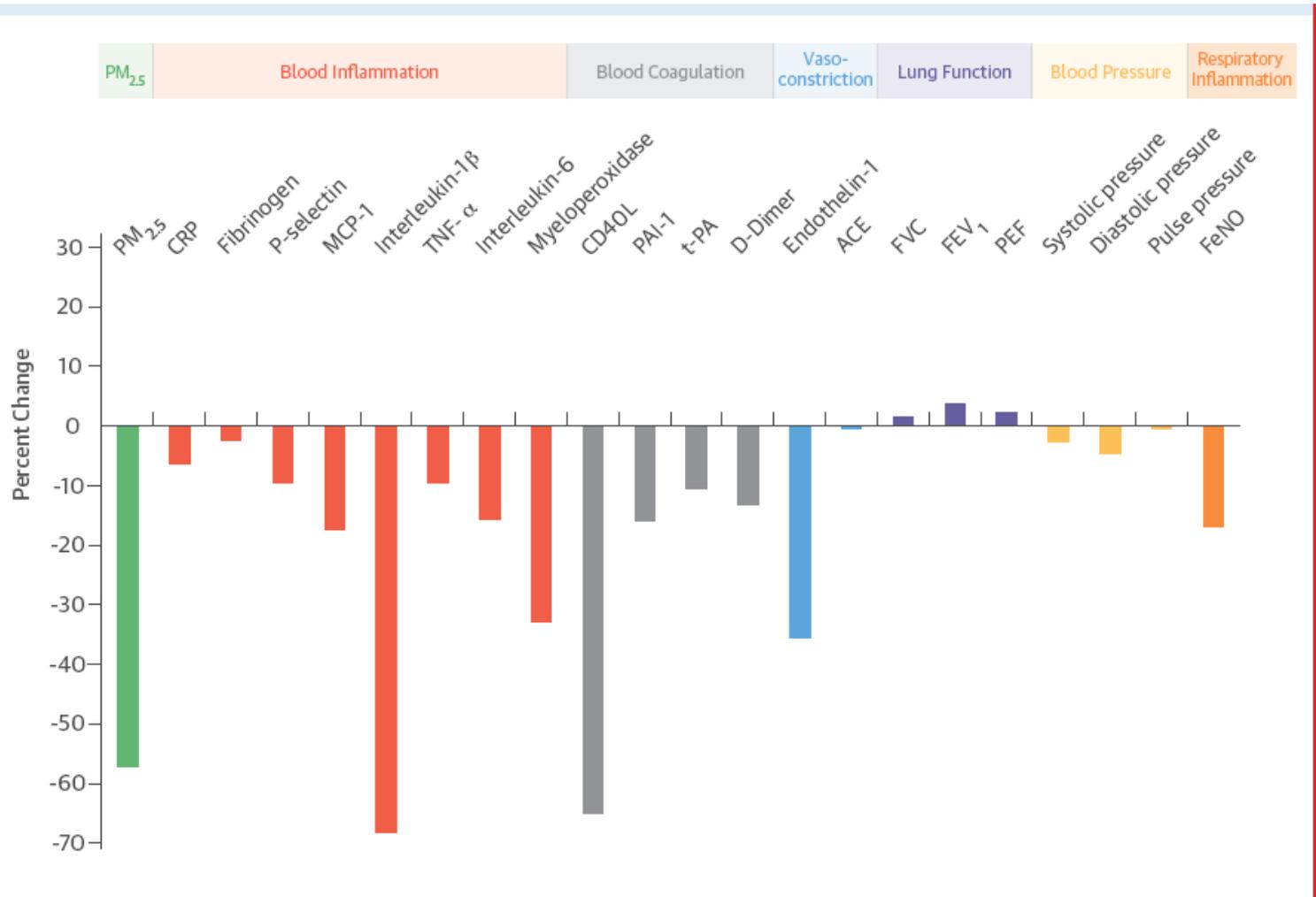


CrossMark

Air Pollution and Insulin Resistance: Do All Roads Lead to Rome?



Diabetes 2015;64:712–714 | DOI: 10.2337/db14-1682



Chen, R. et al. J Am Coll Cardiol. 2015; 65(21):2279-87.

Different colors represent different indicators of exposure and health: **green** = PM_{2.5}; **salmon** = blood inflammation; **gray** = blood coagulation; **blue** = vasoconstriction; **purple** = lung function; **yellow** = blood pressure; and **orange** = respiratory inflammation. ACE = angiotensin-converting enzyme; CD40L = CD40 ligand; CRP = C-reactive protein; FeNO = fractional exhaled nitric oxide; FEV₁ = forced expiratory volume in 1 s; FVC = forced vital capacity; MCP = monocyte chemoattractant protein; PAI = plasminogen activator inhibitor; PEF = peak expiratory flow; PM_{2.5} = particulate matter <2.5 μm in aerodynamic diameter; TNF = tumor necrosis factor; t-PA = tissue plasminogen activator.

EDITORIAL COMMENT

Personalizing Your Airspace and Your Health*



Sanjay Rajagopalan, MD,[†] Robert D. Brook, MD[‡]

Indoor and outdoor air pollution rank as the fourth and ninth leading causes of global morbidity and mortality in the most recent Global Burden of Disease (GBD) report and collectively outrank other major risk factors in terms of importance (1). The fine particulate matter <math><2.5 \mu\text{m}</math> ($\text{PM}_{2.5}$) component of inhaled particulate matter, more than other larger fractions, dominates risk through its impact on cardiovascular events (2). A substantial body of evidence supports a stereotypic activation of adverse mechanisms, including height-

and point to an increase in the proportion of patients in East Asia living above the World Health Organization (WHO) interim Target-1 of $35 \mu\text{g}/\text{m}^3$ (increased from 51% in 1998 to 2000 to 70% in 2010 to 2012) (5). In stark contrast in North America, the vast majority of the population lives below the WHO Air Quality standard of $10 \mu\text{g}/\text{m}^3$, with 20% exposed to $\text{PM}_{2.5}$ above this level (5). However, individuals living within regions meeting air quality standards can still face health risks posed by “hot spots” of exposure (e.g., near roadways, point



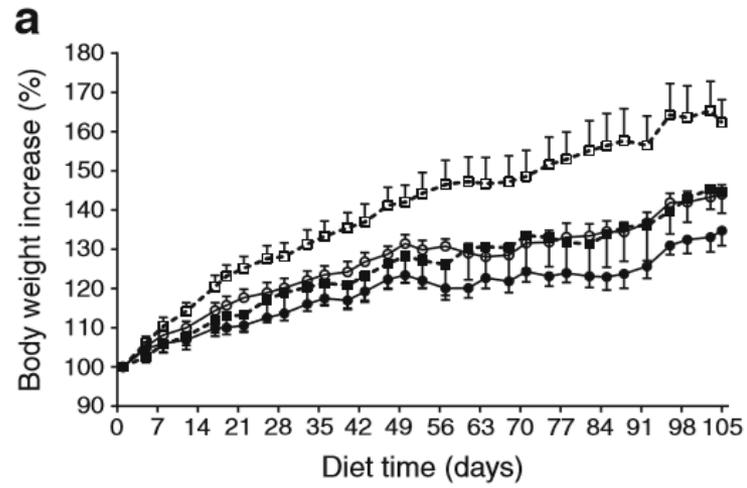
Inflamación y DM-2:

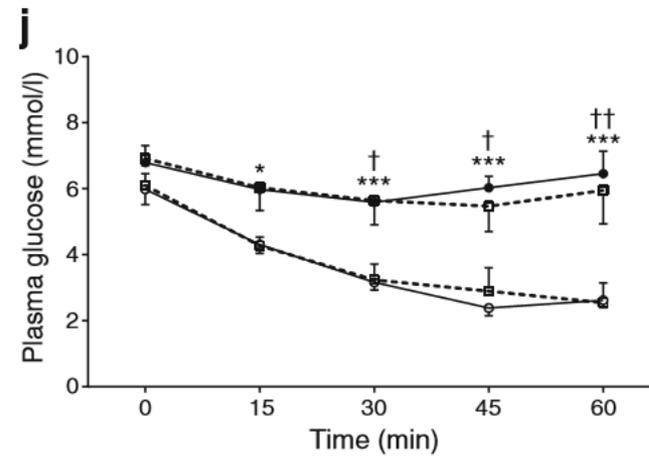
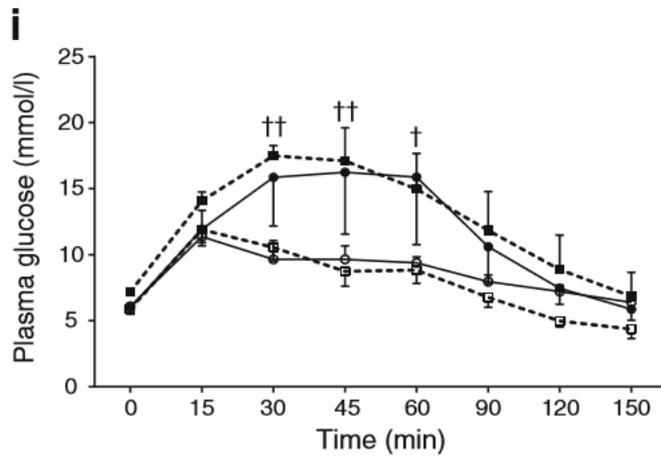
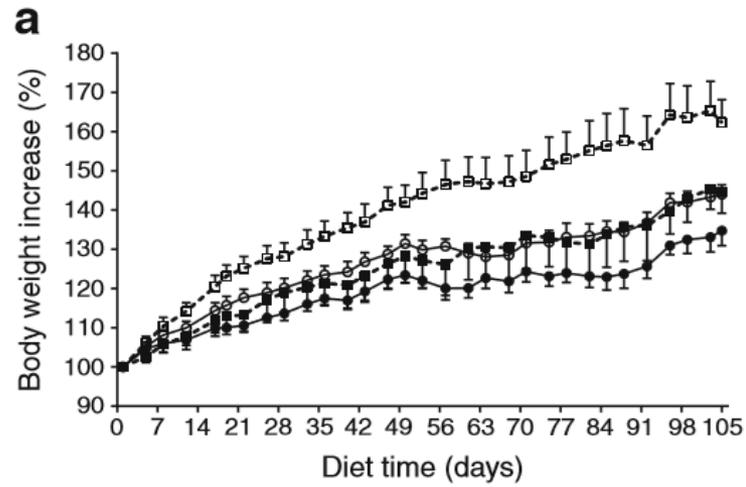
1. La inflamación siempre impacta negativamente en la fisiopatología de la DM-2.
2. La inflamación es la respuesta. Lo importante es el estímulo que la desencadena.
- 3. Algo de inflamación es bueno. Lo perjudicial es su cronificación.**
4. ¿Podemos utilizarla como base para tratar la DM-2?

Riesgo de DM-2 / Complicaciones

Inflamación





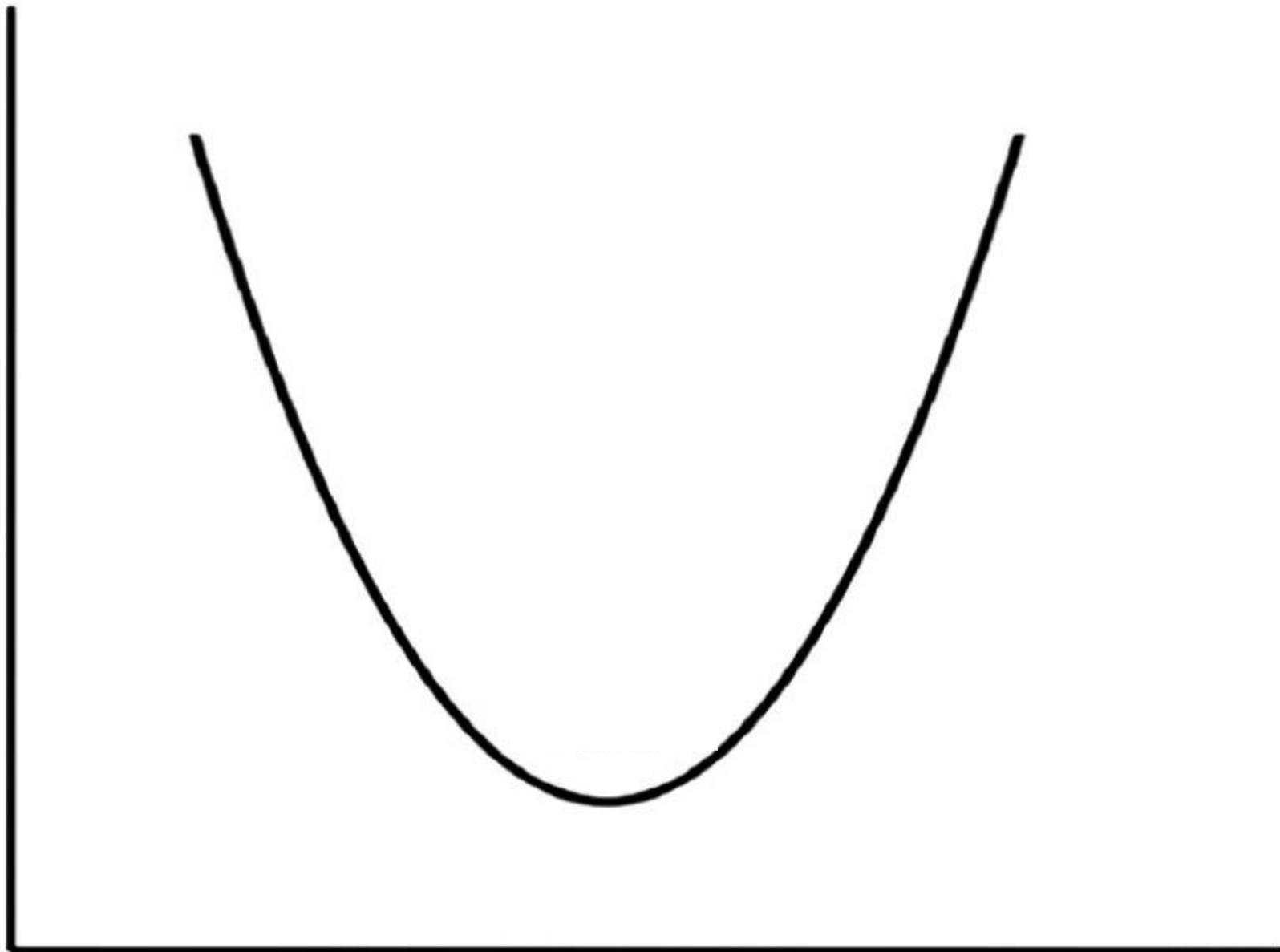


Riesgo de DM-2 / Complicaciones

Inflamación



Riesgo de DM-2 / Complicaciones

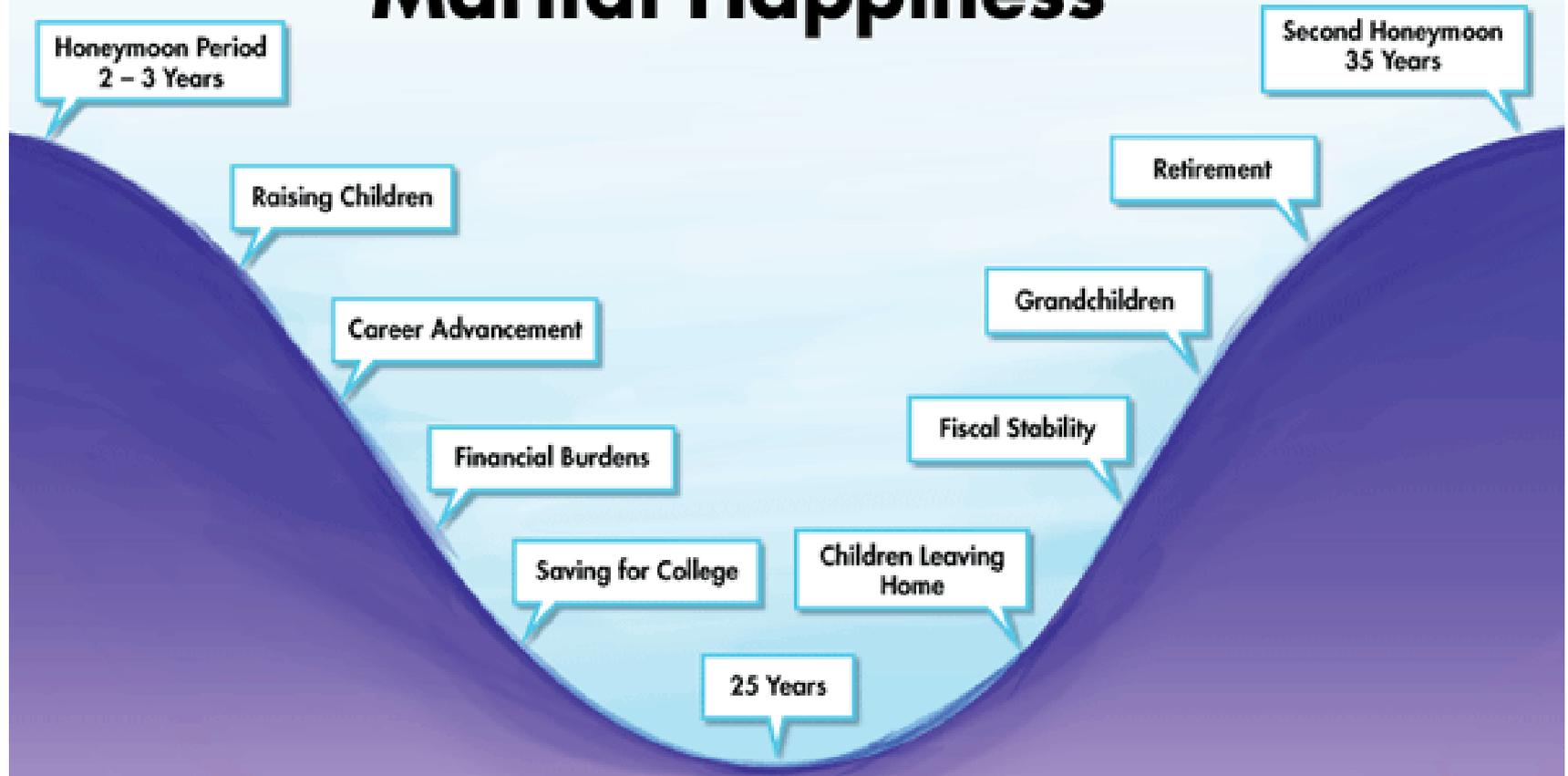


Inflamación

Mortality ↑



U-Shaped Curve of Marital Happiness



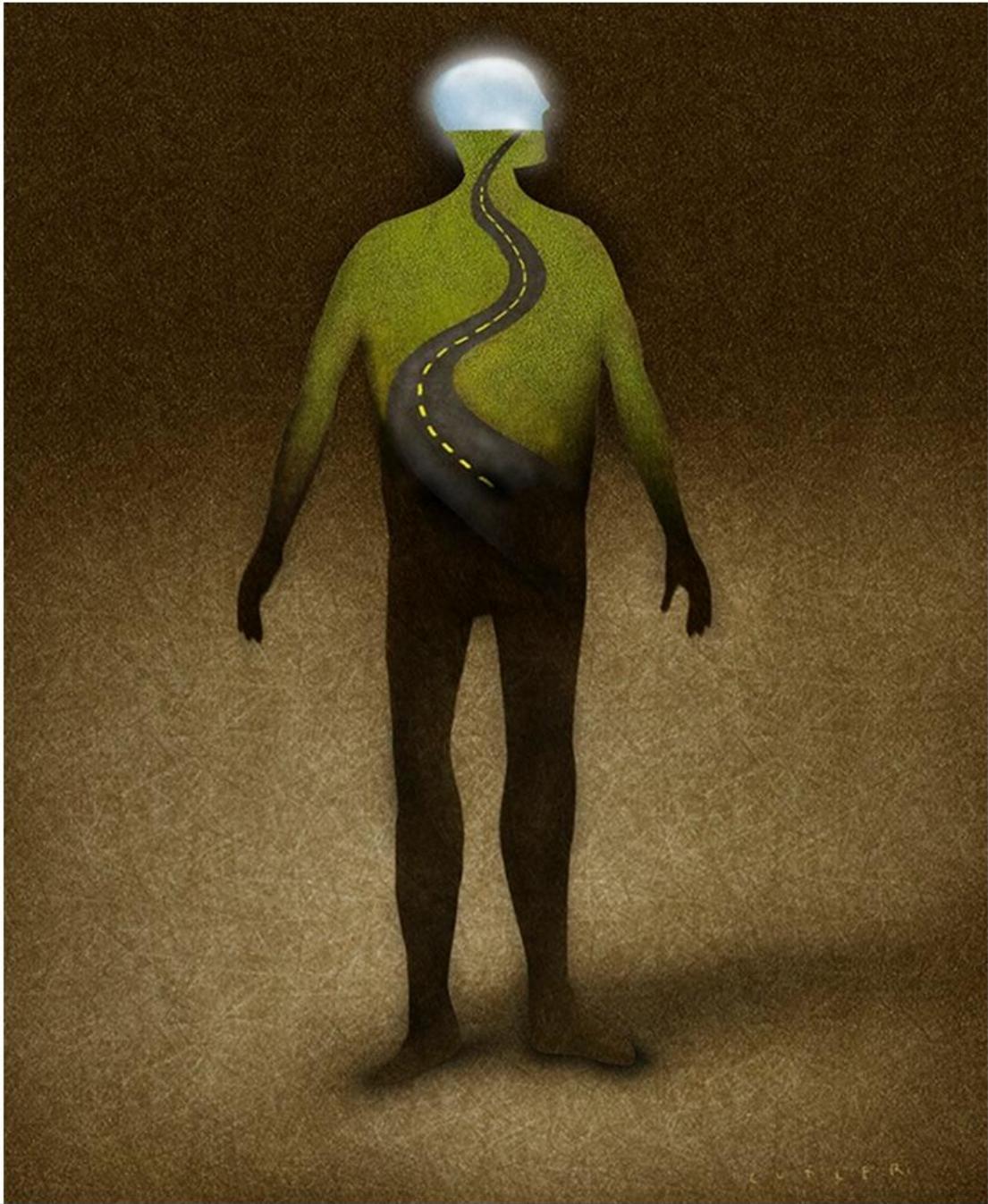


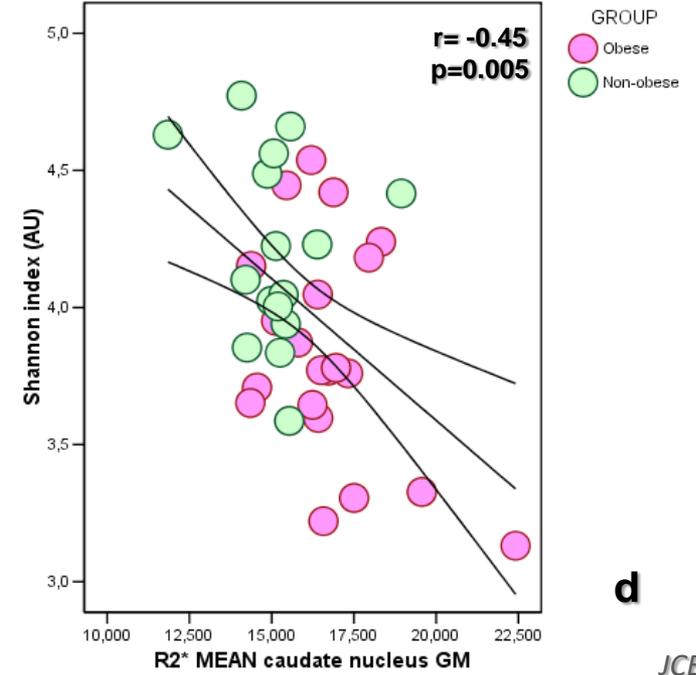
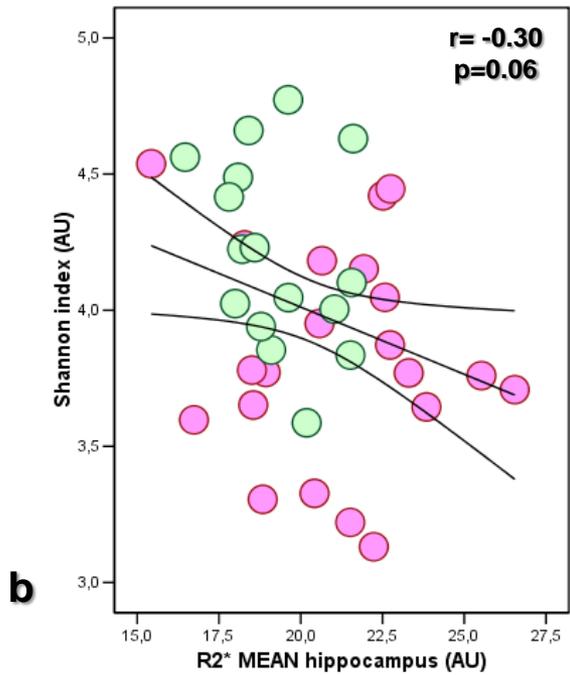
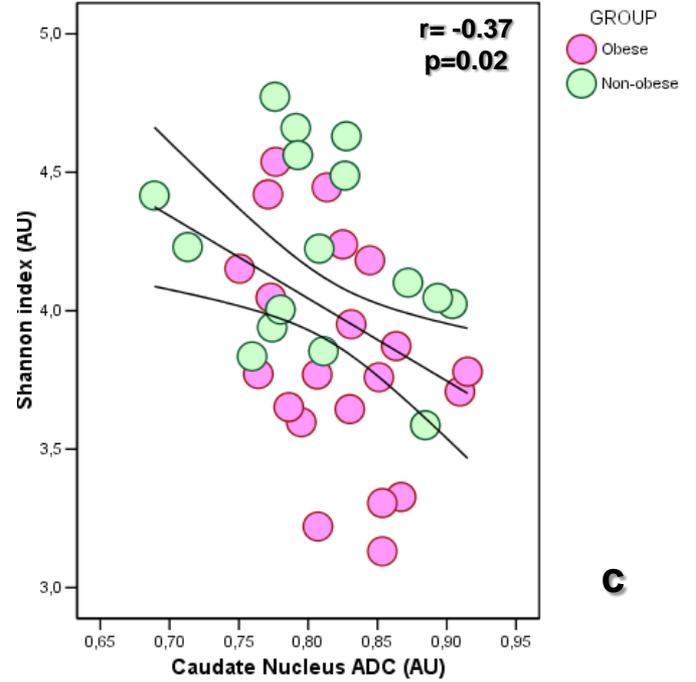
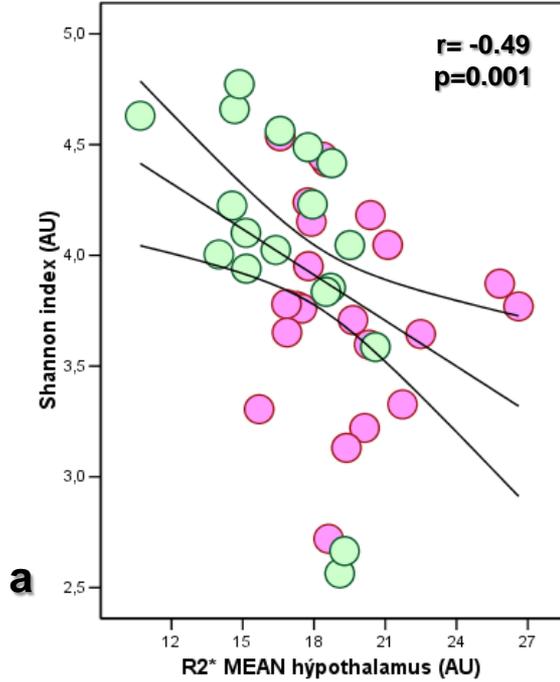


Inflamación y DM-2:

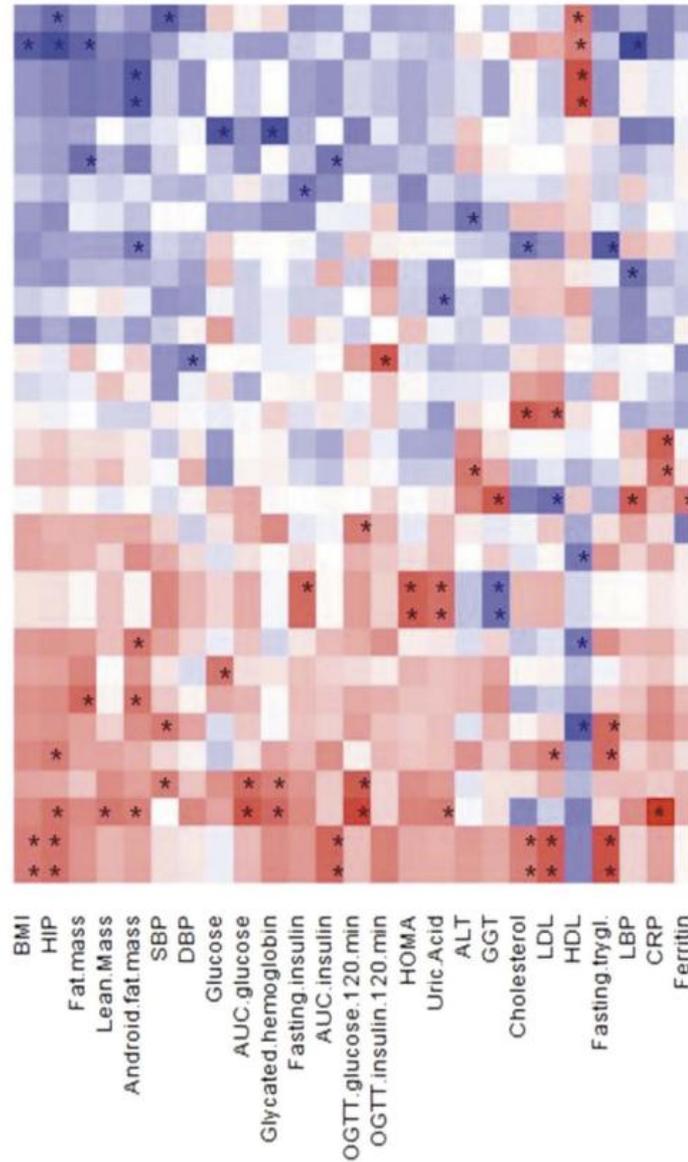
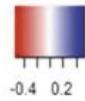
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R value

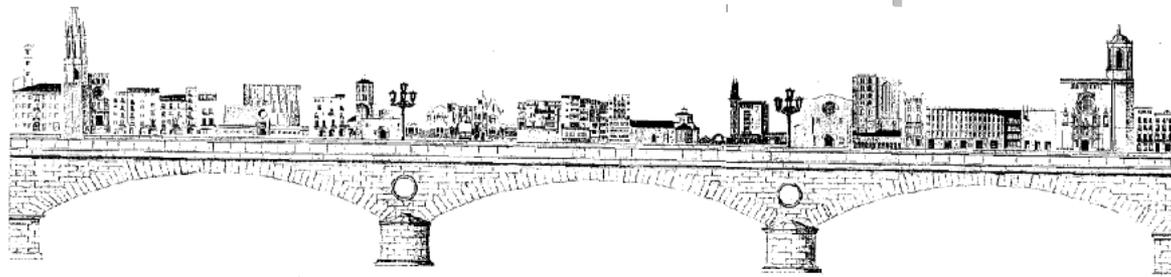


- Saccharomycetes (C)
- Tremellomycetes (C)
- Cystobasidiomycetes (C)
- Erythrobasidiaceae (F)
- Eurotium (G)
- Aspergillus (G)
- Ascomycota (P)
- Torulaspota (G)
- Dipodascaceae (F)
- Rhodotorula (G)
- Alternaria (G)
- Basidiomycota (P)
- Debaryomycetaceae (F)
- Pichiaceae (F)
- Hypocraceae (F)
- Leotiomyces (C)
- Betisia (G)
- Cladosporium (G)
- Eupenicillium (G)
- Aspergillaceae (F)
- Ceratocystidaceae (F)
- Ceratocystis (G)
- Penicillium (G)
- Moniliella (G)
- Nectriaceae (F)
- Eurotiomycetes (C)
- Zygomycota (P)
- Corticaceae (F)
- Agaricomycetes (C)
- Mucoraceae (F)
- Mucor (G)



Inflamación y DM-2: Conclusiones

1. La inflamación siempre impacta negativamente en la fisiopatología de la DM-2.
2. La inflamación es la respuesta. Lo importante es el estímulo que la desencadena.
3. Algo de inflamación es bueno. Lo perjudicial es su cronificación.
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Girona 28 d'abril del 2000

 Hospital Universitari de Girona
Doctor Josep Trueta

 ACD
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