INQUINAMENTO DA MATERIALE PARTICOLATO AMBIENTALE: SCELTE INDIVIDUALI PER IMPLEMENTARE LA SALUTE CARDIO-METABOLICA

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L'Organizzazione Mondiale della Sanità (OMS) riconosce l'inquinamento ambientale come un importante fattore di rischio per patologie. L'esposizione al particolato ambientale $PM_{2.5}$ (particelle con diametro uguale a 2.5 micron,) è divenuta globalmente la quinta causa di morte, responsabile di 4.2 milioni di morti nel mondo (Tabella 1). Il particolato ambientale consiste di una miscela di particelle (solide e liquide, inorganiche e organiche) sospese nell'aria ambientale, in larga parte derivate dalle combustioni del carburante dei veicoli a motore. Ne sono principali componenti solfati, ammoniaca, nitrati, carbone nero, cloruro di sodio, polveri minerali, e acqua.

L'esposizione al materiale particolato è associata alla malattia cardiovascolare e al diabete tipo 2, due patologie strettamente connesse. Il legame fra diabete e inquinamento ambientale è di grande rilevanza per due ordini di fattori. In primo luogo, l'iperglicemia rappresenta il terzo fattore di rischio per morte nel mondo, con 5.3 milioni di morti nel 2015 (Tabella 1). In secondo luogo, il numero globale di diabetici nel mondo continua ad aumentare, con previsioni allarmanti di 642 milioni nel 2040. Una recente meta-analisi ha confermato l'associazione fra esposizione a PM e incidenza del diabete: il rischio di diabete aumenta di un valore compreso tra il 10 e il 27% per un incremento dell'esposizione a 10 $\mu g/m^3$ di $PM_{2.5}$. I meccanismi che potrebbero sottendere a questa associazione includono la disfunzione endoteliale, l'infiammazione vascolare, l'insulino-resistenza epatica, gli elevati livelli di HbA1c e di pressione arteriosa, e la compromissione delle funzioni autonomiche. Interessante notare come questi meccanismi fiusiopatologici siano già stati riconosciuti da differenti organizzazioni internazionali (American Heart Association e European Society of Cardiology) come gli intermediari dell'aumentato rischio cardiovascolare associato all'esposizione al PM.

Benché le principali fonti di inquinamento ambientale siano al di fuori del controllo umano, esiste ancora un margine per un'azione personale. Guardare ai principali fattori di rischio per mortalità risulta di sicuro importante nella prospettiva di innalzare l'aspettativa di vita di individui ad alto rischio di patologie cardio-metaboliche o affette da diabete o malattie cardiovascolari. Modulare i fattori di rischio legati all'alimentazione, riducendo il consumo di sodio ed implementando quello di cereali integrali, frutta, noci, semi e verdure, potrebbe contribuire ad equilibrare gli effetti nocivi di altri fattori di rischio non dietetici. Numerose evidenze scientifiche da trials randomizzati controllati dimostrano che pattern alimentari salutari, come ad esempio la dieta di tipo Mediterraneo, sono associati al miglioramento del profilo cardio-metabolico di individui ad alto rischio prevenendo la comparsa di diabete tipo 2, migliorando il controllo glico-metabolico e riducendo il rischio di eventi cardiovascolari del 30%. Uno stile di vita salutare (assenza dell'abitudine al fumo, assenza di obesità, attività fisica svolta almeno una volta a settimana, e dieta salutare) riduce del 50% circa il rischio di malattia coronaria in individui ad alto rischio genetico, dando forza al concetto che non è mai troppo tardi e non è mai inutile aderire ad un sano stile di vita.

Tabella 1. Fattori di rischio per morte nel mondo

Fattore di rischio	Ranking	Morti (milioni)
Pressione arteriosa elevata	1	10.7
Fumo	2	6.4
Iperglicemia	3	5.3
Ipercolesterolemia	4	4.4
$PM_{2.5}$ ambientale	5	4.2
Elevata assunzione di sodio	6	4.1
Body mass index elevato	7	4.0
Basso consumo di cereali integrali	8	3.2
Basso consumo di frutta	9	2.9
Inquinamento domestico	10	2.8
Ridotta filtrazione glomerulare	11	2.45
Uso di alcohol	12	2.4
Basso consumo di noci e semi	13	2.1
Basso consumo di vegetali	14	2.0
Ridotti livelli di attività fisica	15	1.6

Adattata da State of Global Air 2017.

I° meeting
Club SIE Endocrinologia Ambientale
Roma, 19 giugno 2017
Sala Polifunzionale
della
Presidenza del Consiglio dei Ministri
Via Santa Maria in via 37b

Inquinamento da materiale particolato ambientale: scelte individuali per implementare la salute cardio-metabolica

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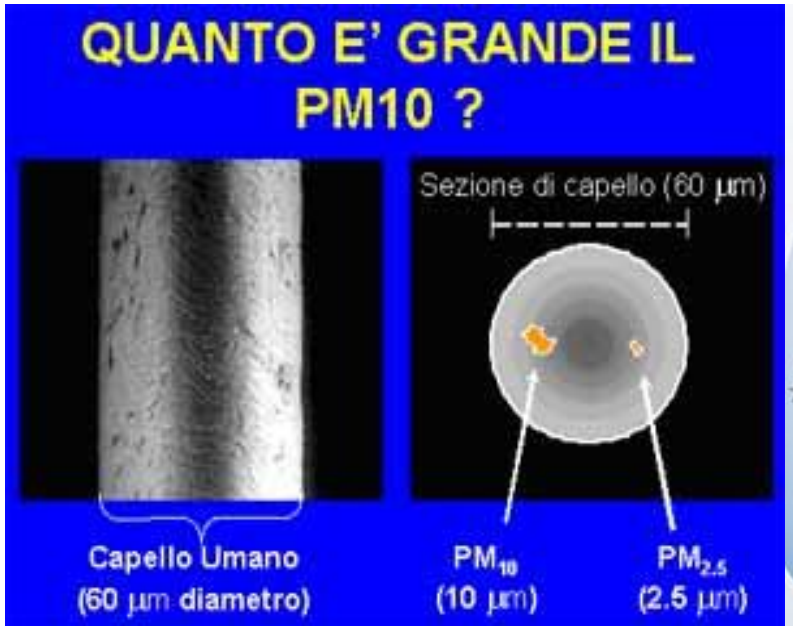
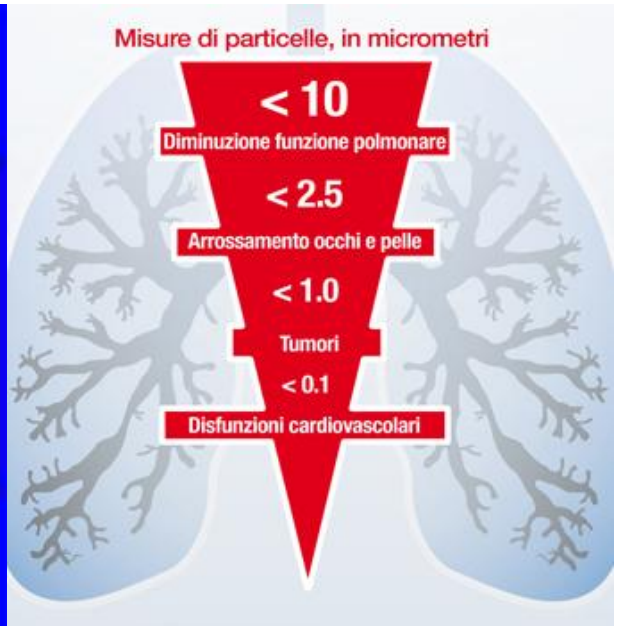
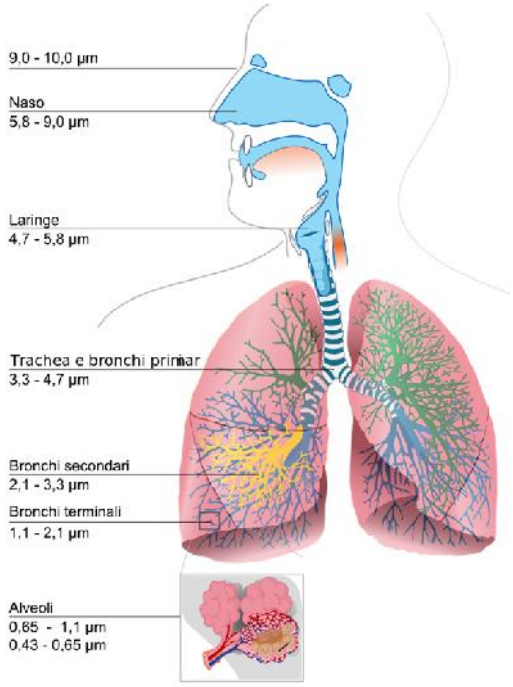
Particolato

- Particolato, particolato sospeso, pulviscolo atmosferico, polveri sottili, polveri totali sospese (PTS), sono termini che identificano comunemente l'insieme delle sostanze sospese in aria (fibre, particelle carboniose, metalli, silice, inquinanti liquidi o solidi).

- Il particolato è l'inquinante che oggi è considerato di maggiore impatto nelle aree urbane, ed è composto da tutte quelle particelle solide e liquide disperse nell'atmosfera, con un diametro che va da pochi nanometri fino ai 500 µm e oltre.







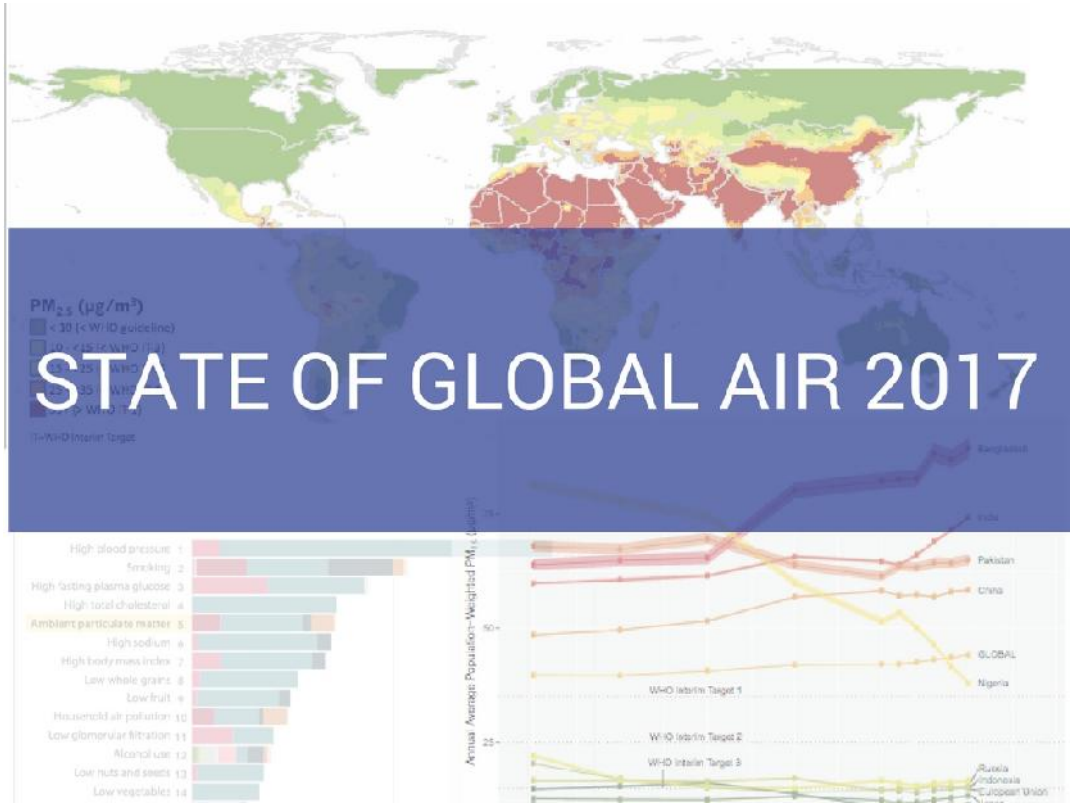
Outdoor air pollution-caused deaths – breakdown by disease:

- 40% – ischaemic heart disease;
- 40% – stroke;
- 11% – chronic obstructive pulmonary disease (COPD);
- 6% - lung cancer; and
- 3% – acute lower respiratory infections in children.

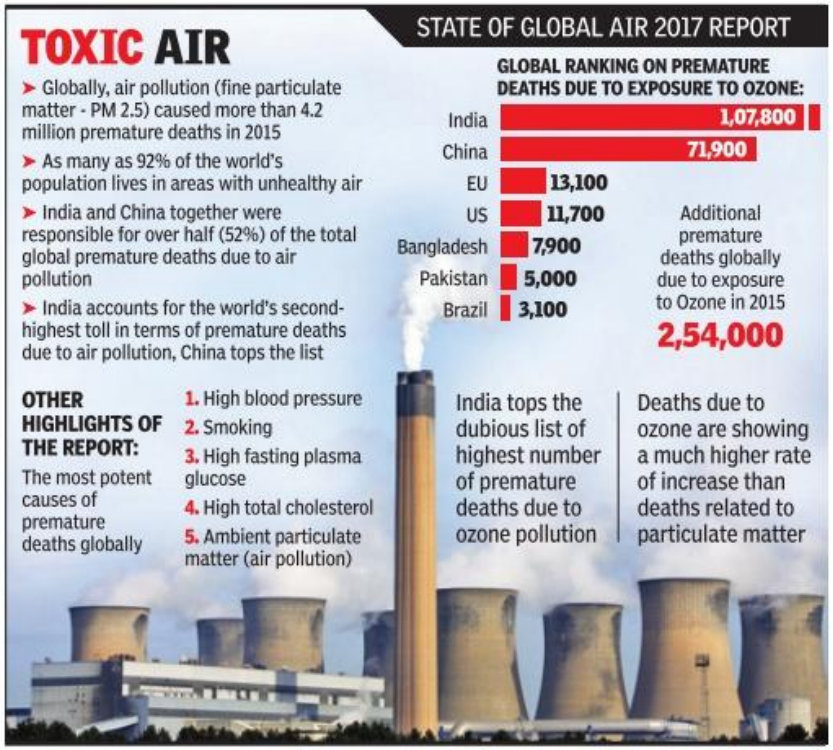
Indoor air pollution-caused deaths – breakdown by disease:

- 34% - stroke;
- 26% - ischaemic heart disease;
- 22% - COPD;
- 12% - acute lower respiratory infections in children; and
- 6% - lung cancer.

Air pollution is contamination of the indoor or outdoor environment by any chemical, physical or biological agent that modifies the natural characteristics of the atmosphere. Household combustion devices, motor vehicles, industrial facilities and forest fires are common sources of air pollution. Pollutants of major public health concern include particulate matter, carbon monoxide, ozone, nitrogen dioxide and sulfur dioxide. Outdoor and indoor air pollution cause respiratory and other diseases, which can be fatal.

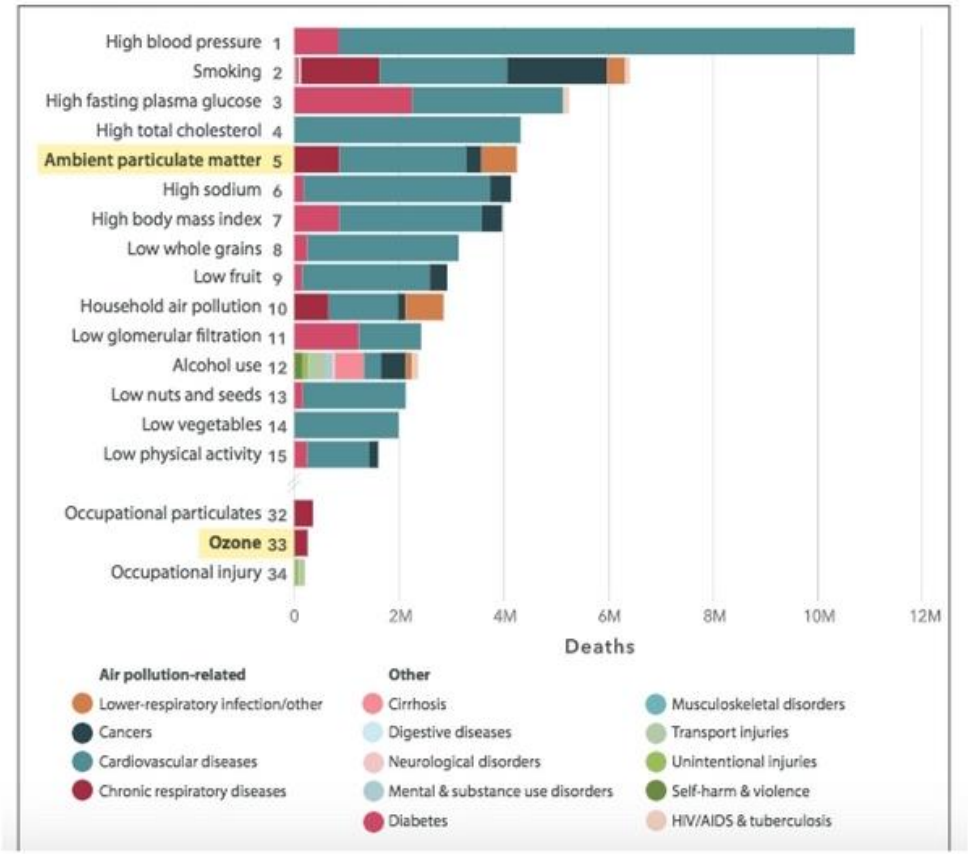


STATE OF GLOBAL AIR 2017



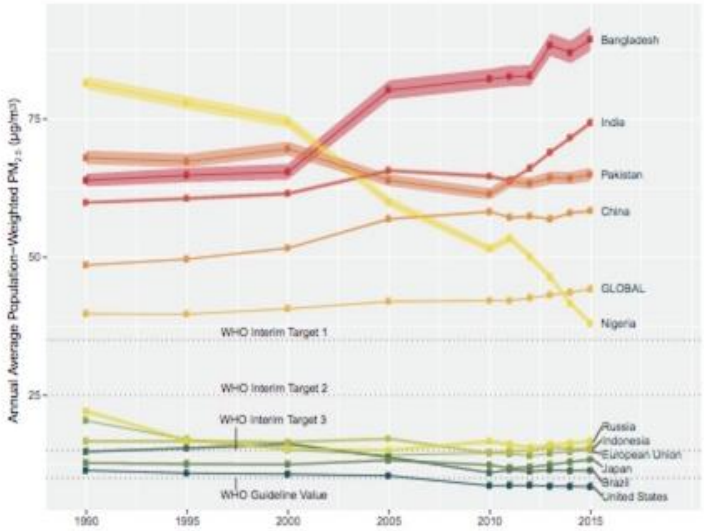
HEI IHME UBC

The State of Global Air website is a collaboration between the Health Effects Institute and the Institute for Health Metrics and Evaluation, with expert input from the University of British Columbia.



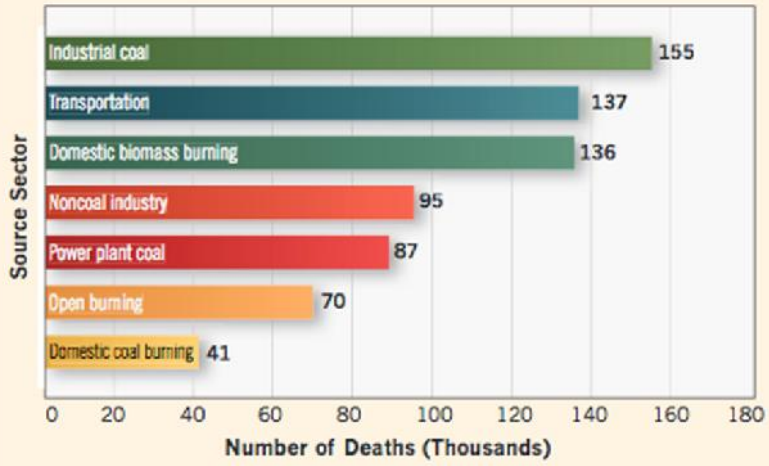
The latest publication by the GBD in 2015 reported that exposure to PM2.5 is the **5th biggest contributor to mortality and disease globally** and accounts for 4.2 million deaths from heart disease, stroke, lung cancer, chronic lung disease and respiratory infections. Their recommendation is to continue tracking and analyzing pollution and PM2.5 in order to better understand how to reduce this burden on the health system and society as a whole.

Trends in annual average population-weighted $PM_{2.5}$ concentrations in the 10 most populous countries plus European Union.



Explore the data on the State of Global Air interactive site.

Source sector contributions to deaths attributable to $PM_{2.5}$ in China in 2013.



Padova è la città più inquinata d'Europa (Agenzia Europea Ambiente)

- *La Pianura Padana è tra le aree maggiormente colpite in Europa, ove i valori dell'ozono sono tre volte superiori alla soglia limite*.
- Il traffico è il primo elemento responsabile inquinante, seguito dalle industrie, agricoltura e riscaldamento abitativo

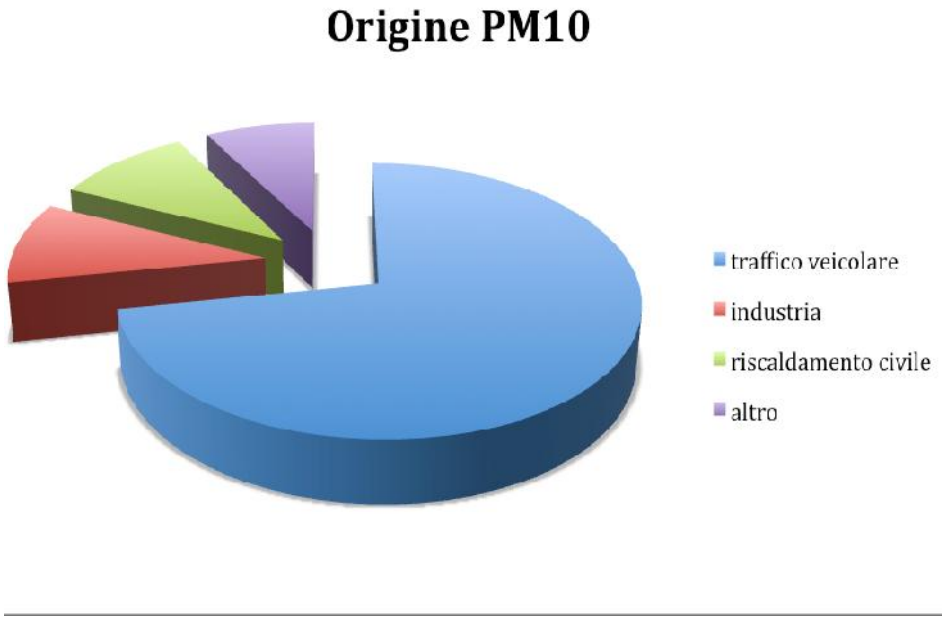
"La maglia nera spetta a Padova": per i primi novanta giorni dell'anno (2015) ha respirato mediamente 53 µg di polveri sottili per ogni m^3 d'aria. Milano e Reggio Emilia entrambe a quota 51,2 µg (il limite di legge annuale è di 40 µg/m^3).

I fattori naturali

- polvere, terra, sale marino ("aerosol marino«,) incendi, microrganismi, pollini e spore, erosione di rocce, eruzioni vulcaniche, polvere cosmica.

I fattori antropici

- **emissioni della combustione dei motori (autocarri, automobili, aerei, navi)**
- emissioni del riscaldamento domestico (in particolare gasolio, carbone e legna)
- residui dell'usura del manto stradale, dei freni e delle gomme delle vetture
- emissioni di lavorazioni meccaniche, dei cementifici, dei cantieri
- lavorazioni agricole
- inceneritori e centrali elettriche
- fumo di tabacco



Particolato: quanto ne possiamo respirare?

D. Lgs 155/2010

Valore Limite per la media annuale	40 µg/m³
Valore limite giornaliero (24-ore)	50 µg/m³
Numero massimo di superamenti consentiti in un anno civile	35 gg/anno

Dal 2011 è scattato l'obbligo per il monitoraggio del $PM_{2,5}$, con l'obiettivo di raggiungere al 2015 un valore limite medio annuo fissato a 25 µg/m³

WHO and air pollution

Fact sheet N°292
Updated February 2016
Fact sheet N°313
Updated March 2014

Key facts

- Air pollution is a major environmental risk to health. By reducing air pollution levels, countries can reduce the burden of disease from stroke, heart disease, lung cancer, and both chronic and acute respiratory diseases, including asthma.

- Ambient (outdoor air pollution) in both cities and rural areas was estimated to cause **3.7 million premature deaths worldwide per year in 2012**; this mortality is due to exposure to small particulate matter of 10 microns or less in diameter (PM10), which cause cardiovascular and respiratory disease, and cancers.

Guideline values

$PM_{2.5}$

10 µg/m^3 annual mean

25 µg/m^3 24-hour mean

PM_{10}

20 µg/m^3 annual mean

50 µg/m^3 24-hour mean

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Commenti 55

Smog, l'europarlamento raddoppia i limiti di emissioni per le auto

Con pochi voti di scarto, approvata la modifica del regolamento sugli ossidi di azoto, i precursori delle polveri sottili. Socialisti e verdi si sono opposti ma hanno perso

di ANTONIO CIANCIULLO

Lo leggo dopo | 03 febbraio 2016

Articoli Correlati | **Dall'archivio** Il caso Volkswagen

55 Commenti

ROMA – Largo alle polveri sottili, quelle che corrodono i nostri polmoni, quelle che fanno scattare la febbre da smog

le polveri sono schizzate rapidamente a 100 e poi addirittura a 125 microgrammi per metro cubo, a fronte di un limite di 50.

Endocrine (2016) 51:32–37
DOI 10.1007/s12020-015-0638-2

MINI REVIEW

Particulate matter pollutants and risk of type 2 diabetes: a time for concern?

Katherine Esposito[1] · Michela Petrizzo[3] · Maria Ida Maiorino[2] · Giuseppe Bellastella[2] · Dario Giugliano[2]

The risk of future diabetes associated with exposure to 10 µg/m^3 increase of PM2.5 has been quantified in the range of 10 to 27 %

Table 1 Summary of meta-analyses exploring the association between particulate matter pollutants and diabetes ri

Author, year (reference)	Design	Exposure	Variable	Effect size (95 % CI)
Balti [20]	Cohort ($n = 5$)	$PM_{2.5}$	10 µg/m^3 increase	HR: 1.11 (1.03–1.2)
Wang [21]	Cohort ($n = 5$)	$PM_{2.5}$	10 µg/m^3 increase	RR: 1.27 (0.97–1.66)
	Cohort (3)	PM_{10}	10 µg/m^3 increase	RR: 1.15 (1.02–1.30)
Janghobani [22]	Cohort ($n = 2$)	$PM_{2.5}$	10 µg/m^3 increase	RR: 1.05 (0.99–1.1)
	Time-series (2)		or IQR increase	
	Cohort ($n = 1$)	PM_{10}	10 µg/m^3 increase	RR: 1.008 (1.003–1.013)
	Time-series ($n = 1$)		or IQR increase	
	Case-cross ($n = 2$)			
Li [23]	Time-series ($n = 3$)	$PM_{2.5}$	10 µg/m^3 increase	RR: 1.08 (1.03–1.13)
	Cohort ($n = 1$)			
	Case-Cross ($n = 1$)			
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Eze [24]	Cohort ($n = 3$)	$PM_{2.5}$	10 µg/m^3 increase	RR: 1.10 (1.02–1.18)

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The risk of diabetes mortality associated with PM2.5 is around 1 % for each increment exposure of 10 µg/m3 of both PM2.5 and PM10.

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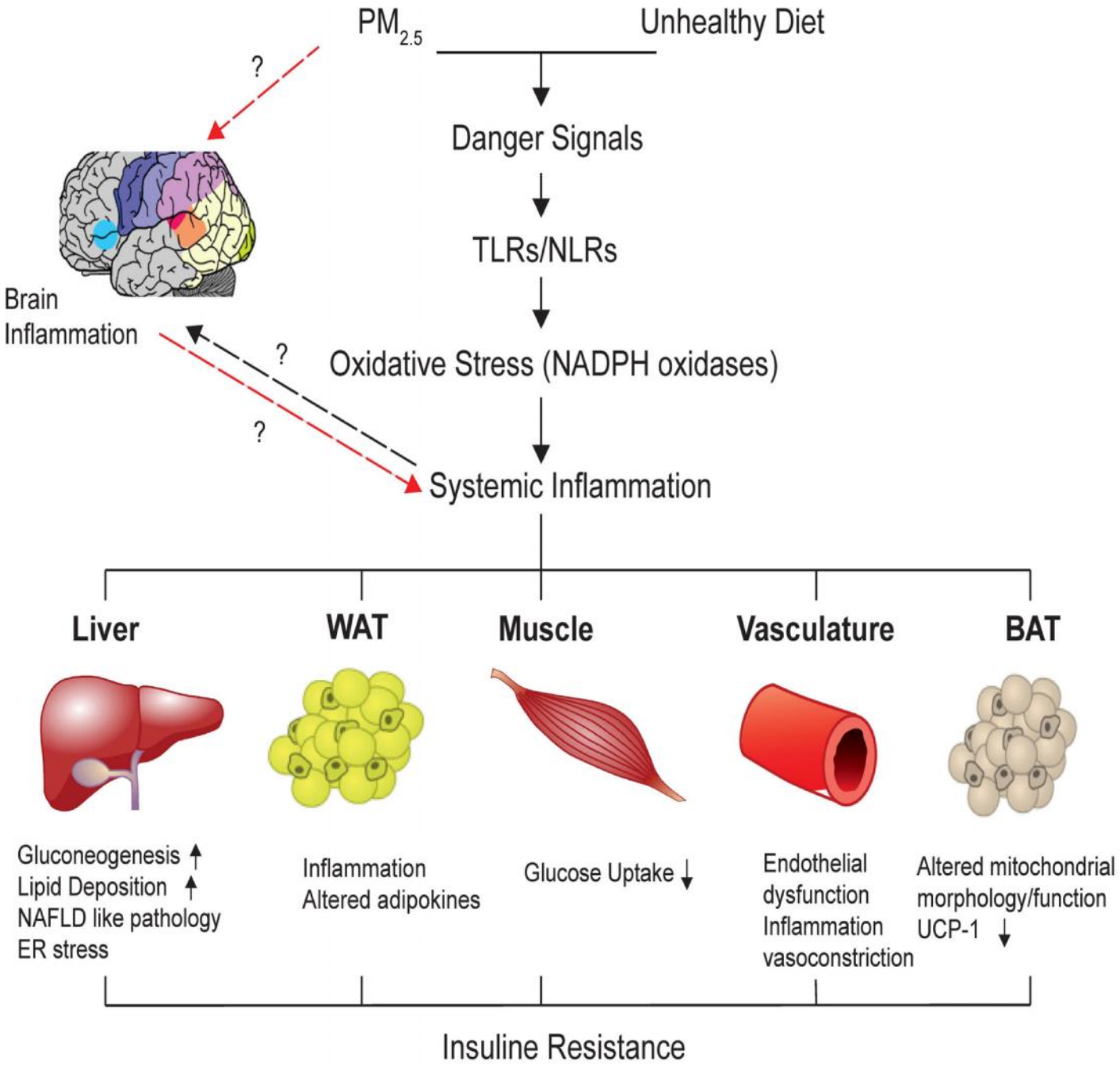
PM2.5
Unhealthy Diet
?
Danger Signals
TLRs/NLRs
Brain
Inflammation
?
Oxidative Stress (NADPH oxidases)
?
Systemic Inflammation
Liver
WAT
Muscle
Vasculature
BAT
Gluconeogenesis ↑
Lipid Deposition ↑
NAFLD like pathology
ER stress
Inflammation
Altered adipokines
Glucose Uptake ↓
Endothelial
dysfunction
Inflammation
vasoconstriction
Altered mitochondrial
morphology/function
UCP-1 ↓
Insuline Resistance

Circulation

American Heart Association®
Learn and Live

AHA Scientific Statement

Particulate Matter Air Pollution and Cardiovascular Disease

An Update to the Scientific Statement From the American Heart Association

Robert D. Brook, MD, Chair; Sanjay Rajagopalan, MD; C. Arden Pope III, PhD; Jeffrey R. Brook, PhD; Aruni Bhatnagar, PhD, FAHA; Ana V. Diez-Roux, MD, PhD, MPH; Fernando Holguin, MD; Yuling Hong, MD, PhD, FAHA; Russell V. Luepker, MD, MS, FAHA; Murray A. Mittleman, MD, DrPH, FAHA; Annette Peters, PhD; David Siscovick, MD, MPH, FAHA; Sidney C. Smith, Jr, MD, FAHA; Laurie Whitsel, PhD; Joel D. Kaufman, MD, MPH; on behalf of the American Heart Association Council on Epidemiology and Prevention, Council on the Kidney in Cardiovascular Disease, and Council on Nutrition, Physical Activity and Metabolism

AHA Scientific Statement

Particulate Matter Air Pollution and Cardiovascular Disease

An Update to the Scientific Statement From the American Heart Association

There have been well over 100 published daily time-series studies reporting small but statistically significant PM-mortality associations that have been the subject of quantitative reviews or meta-analyses. Time-series and case-crossover studies explore associations between short-term changes in air pollution and daily changes in death counts.

There appeared to be no lower-limit threshold below which PM10 was not associated with excess mortality across all regions.

Evidence summary from time-series analyses

There is a small, yet consistent association between increased mortality and short-term elevations in PM10 and PM2.5:

- 0.4% to 1.0% increase in daily mortality (and cardiovascular death specifically) due to a 10-μg/m^3 elevation in PM2.5 during the preceding 1 to 5 days.

AHA Scientific Statement

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Evidence Summary from cohort studies

- The overall evidence from the cohort studies demonstrates on average an approximate 10% increase in all-cause mortality per 10 μg/m^3 elevation in long-term average PM2.5 exposure. The mortality risk specifically related to CVD appears to be elevated to a similar (or perhaps even greater) extent, ranging from 3% to 76% (Table 3).

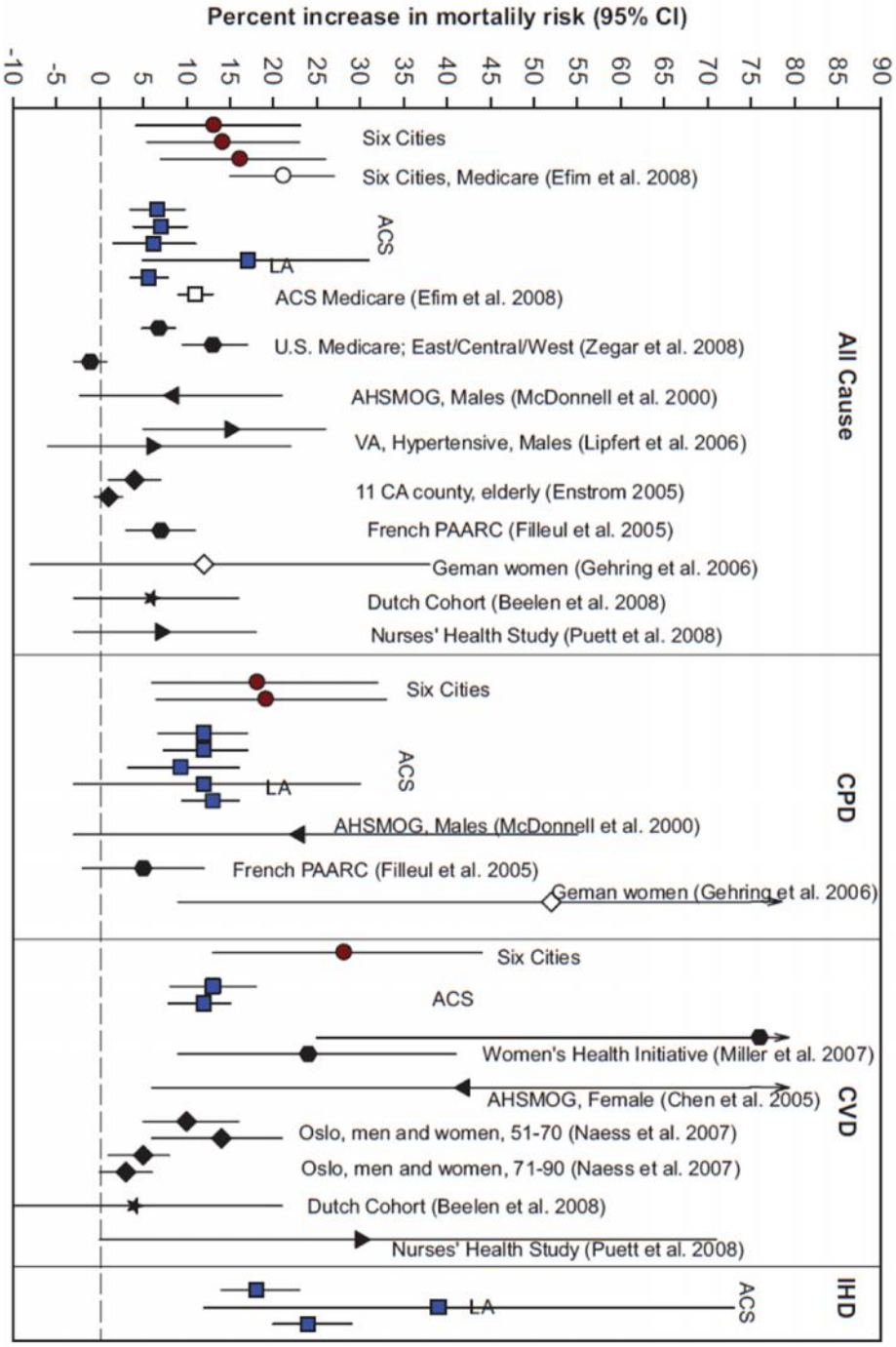

Figure. Risk estimates provided by several cohort studies per increment of 10 µg/m^3 in PM2.5 or PM10.

Pollution exposure on a local scale (across US counties) over a 2-decade period: a decrease of 10 µg/m^3 in the long-term PM2.5 concentration was related to an increase in mean life expectancy of 0.61 $\pm$ 0.2 years .

AHA Scientific Statement

Particulate Matter Air Pollution and Cardiovascular Disease

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Evidence Summary for Hospitalization Rates

Excess cardiovascular mortality and increased rates of hospitalizations are similarly associated with day-to-day changes in PM air pollution, with significant differences between geographic regions

Evidence Summary for specific Cardiovascular Events

The existing level of overall evidence is

- strong for an effect of PM on ischemic heart disease
- moderate (yet growing) for heart failure and ischemic stroke
- modest or mixed for peripheral vascular and cardiac arrhythmia/arrest

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Table 6. Overall Summary of Epidemiological Evidence of the Cardiovascular Effects of $PM_{2.5}$, Traffic-Related, or Combustion-Related Air Pollution Exposure at Ambient Levels

Health Outcomes	Short-Term Exposure (Days)	Longer-Term Exposure (Months to Years)
Subclinical cardiovascular end points and/or surrogate measures in human studies		
Surrogate markers of atherosclerosis	N/A	↑
Systemic inflammation	↑ ↑	↑
Systemic oxidative stress	↑	
Endothelial cell activation/ blood coagulation	↑ ↑	↑
Vascular/endothelial dysfunction	↑ ↑	
BP	↑ ↑	
Altered HRV	↑ ↑ ↑	↑
Cardiac ischemia	↑	
Arrhythmias	↑	

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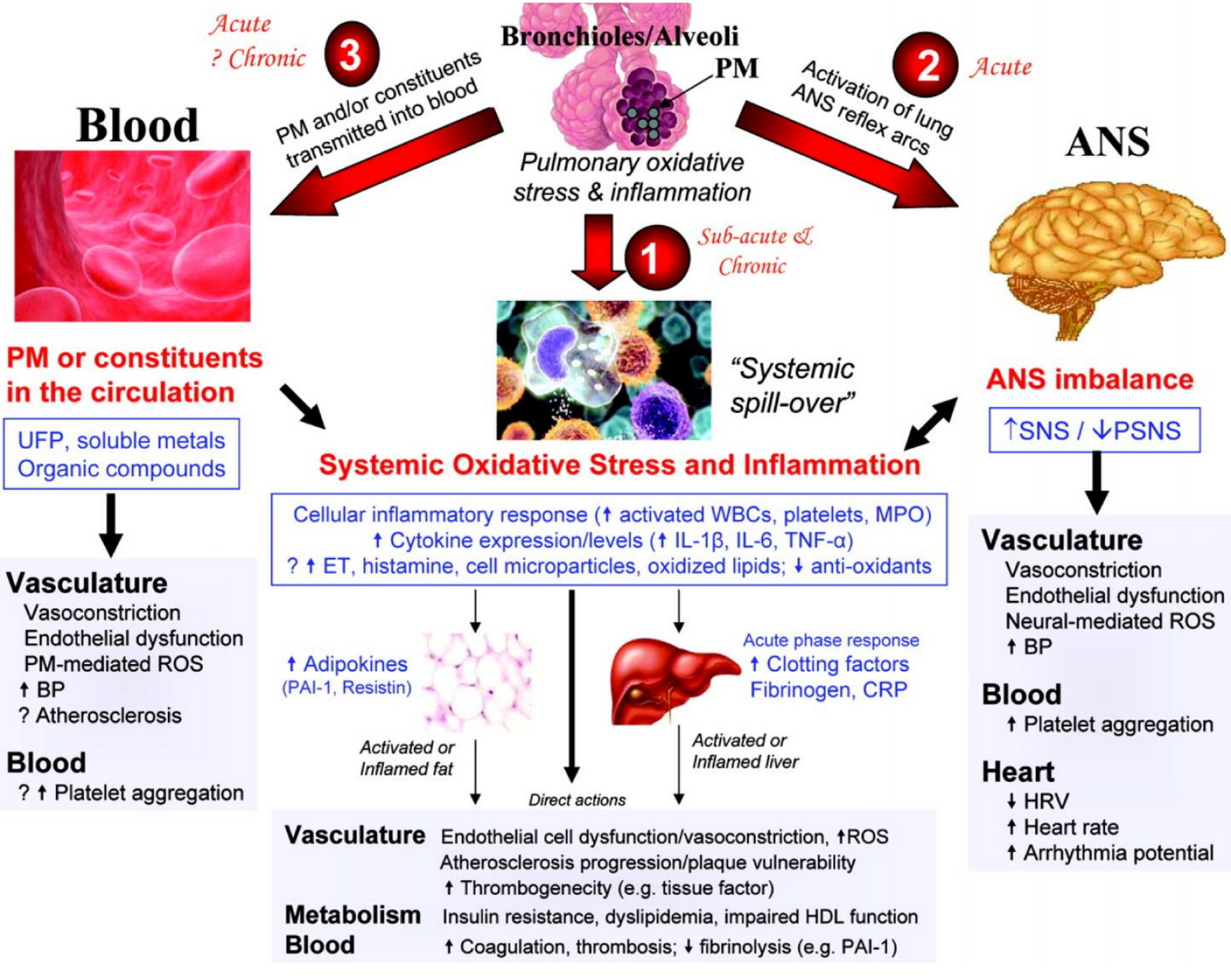
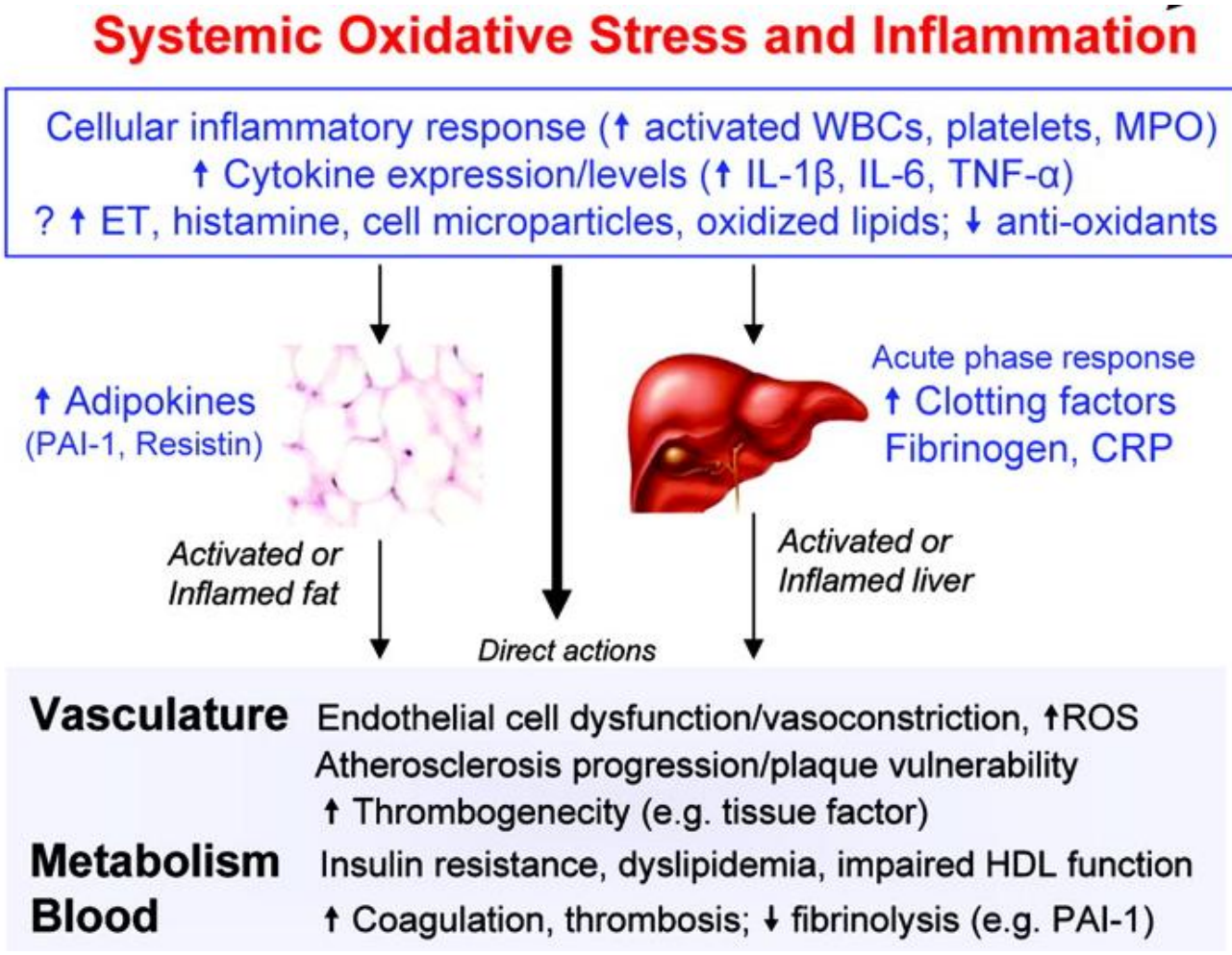
Health Outcomes	Short-Term Exposure (Days)	Longer-Term Exposure (Months to Years)
Clinical cardiovascular end points from epidemiological studies at ambient pollution concentrations		
Cardiovascular mortality	↑ ↑ ↑	↑ ↑ ↑
Cardiovascular hospitalizations	↑ ↑ ↑	↑
Ischemic heart disease*	↑ ↑ ↑	↑ ↑ ↑
Heart failure*	↑ ↑	↑
Ischemic stroke*	↑ ↑	↑
Vascular diseases	↑	↑ †
Cardiac arrhythmia/cardiac arrest	↑	↑


Acute
? Chronic
3
PM and/or constituents transmitted into blood
Bronchioles/Alveoli
PM
2
Acute
Activation of lung ANS reflex arcs
Blood
ANS
Pulmonary oxidative stress & inflammation
1
Sub-acute & Chronic
"Systemic spill-over"
PM or constituents in the circulation
UFP, soluble metals
Organic compounds
Systemic Oxidative Stress and Inflammation
Cellular inflammatory response (↑ activated WBCs, platelets, MPO)
↑ Cytokine expression/levels (↑ IL-1β, IL-6, TNF-α)
? ↑ ET, histamine, cell microparticles, oxidized lipids; ↓ anti-oxidants
ANS imbalance
↑SNS / ↓PSNS
Vasculature
Vasoconstriction
Endothelial dysfunction
PM-mediated ROS
↑ BP
? Atherosclerosis
Blood
? ↑ Platelet aggregation
↑ Adipokines
(PAI-1, Resistin)
Activated or Inflamed fat
Direct actions
Acute phase response
↑ Clotting factors
Fibrinogen, CRP
Activated or Inflamed liver
Vasculature
Vasoconstriction
Endothelial dysfunction
Neural-mediated ROS
↑ BP
Blood
↑ Platelet aggregation
Heart
↓ HRV
↑ Heart rate
↑ Arrhythmia potential
Vasculature
Endothelial cell dysfunction/vasoconstriction, ↑ROS
Atherosclerosis progression/plaque vulnerability
↑ Thrombogenecity (e.g. tissue factor)
Metabolism
Insulin resistance, dyslipidemia, impaired HDL function
Blood
↑ Coagulation, thrombosis; ↓ fibrinolysis (e.g. PAI-1)



There are a number of possible biological pathways linking air pollutants to diabetes, including:

- Endothelial dysfunction
- Dysregulation of the visceral adipose tissue through inflammation
- Hepatic insulin resistance
- Elevated hemoglobin A1c level
- Elevated blood pressure
- Alterations in autonomic tone,

All may increase insulin resistance.

Environment International 80 (2015) 26–32

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

Chronic disease prevalence in women and air pollution — A 30-year longitudinal cohort study

Women from Ontario who enrolled in the Canadian National Breast Screening Study (CNBSS) from 1980 to 1985 (n = 29,549)

PR = 1.28 (95% CI, 1.16-1.41)
PR = 3.13 (95% CI, 3.13-3.48) BMI ≥ 30

Conclusions
This study estimated significant elevated prevalent rate ratios per unit increase in PM2.5 in nine of the ten chronic diseases studied.

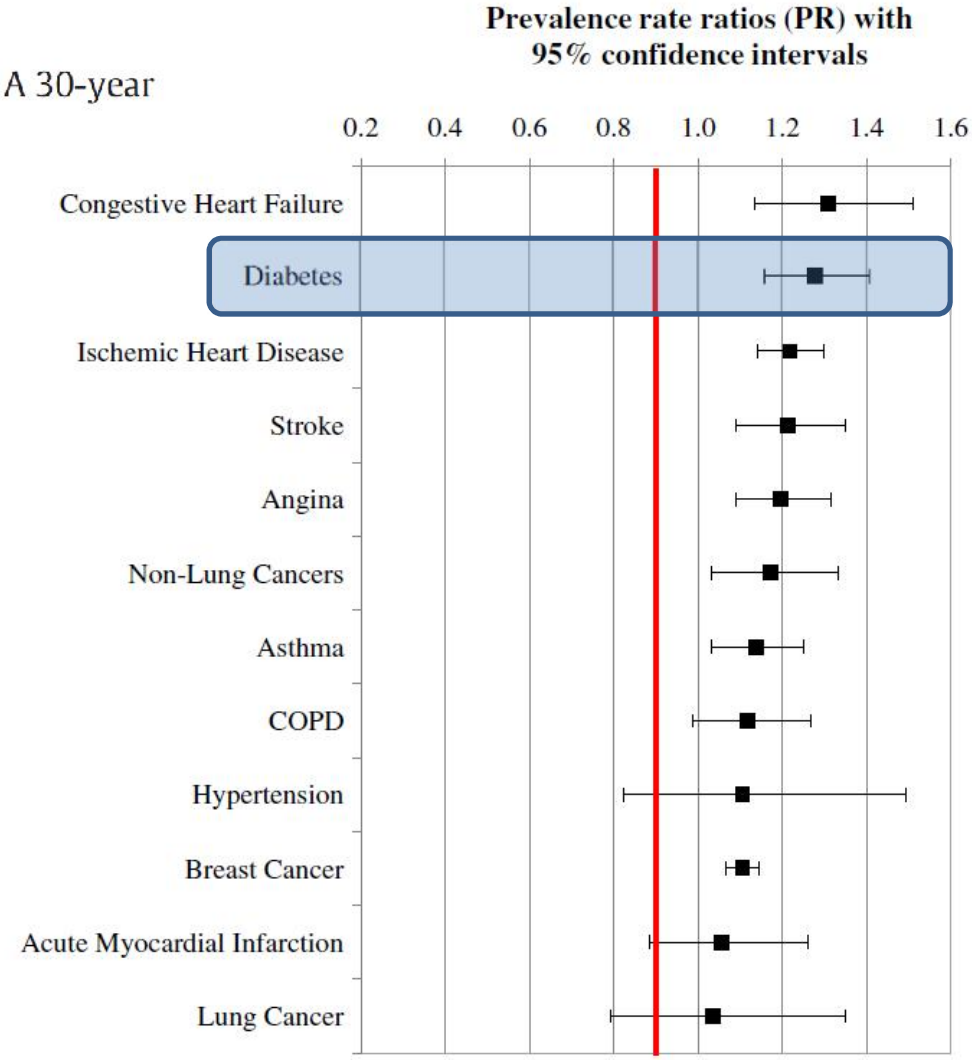


Fig. 1. Adjusted prevalence rate ratios (PRs) of chronic diseases per unit (10 μg/m^3) increase in $PM_{2.5}$.

PM and diabetes risk: The next step

- Non-traditional factors, such as air pollution that are pervasive in the urban environment, may provide low-level synergism with other dominant factors in accelerating propensity for type 2 diabetes.

- Although most sources of outdoor air pollution are well beyond the control of people, individuals should be informed that there are means to reduce the burden of air pollutants on diabetes risk.

In the National Health and Nutrition Examination Survey 2003–2004, increased fruit and vegetable intake, reflected by serum carotenoid concentrations, was associated with the reduced probability of developing type 2 diabetes in US adults with elevated serum concentrations of polychlorinated biphenyls, persistent organic pollutants.

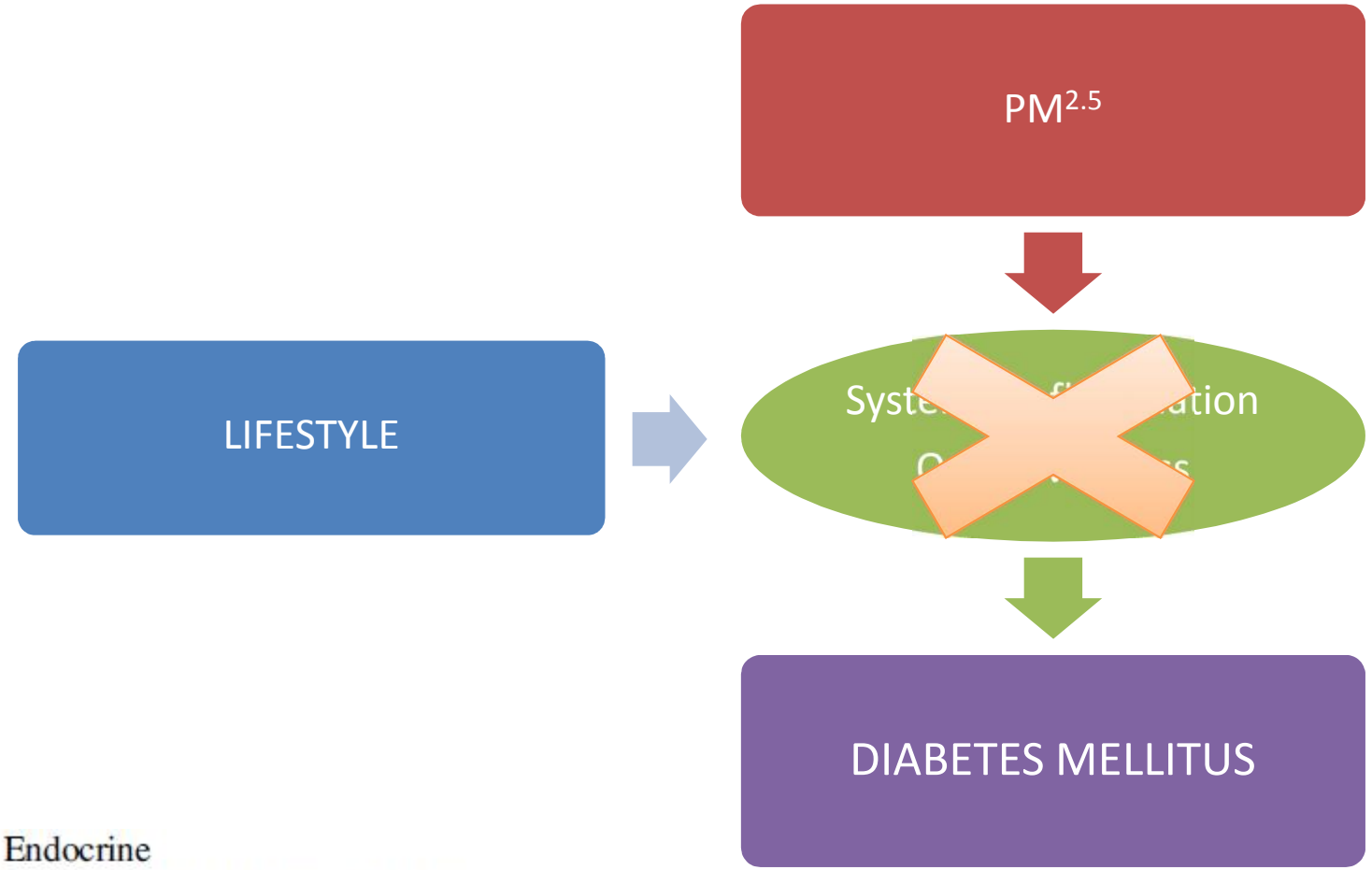
- Non-traditional factors, such as air pollution that are pervasive in the urban environment, may provide low-level synergism with other dominant factors in accelerating propensity for type 2 diabetes.

- Although there are no reported studies about the possible synergism between poor dietary habits and PM exposure on the risk of type 2 diabetes, the larger risk in obese women may suggest a possible role for poor diets.

VIEWPOINT

Particulate matter air pollution: individual choices for improving cardiometabolic well-being

Katherine Esposito[1] · Giuseppe Bellastella[1] · Maria Ida Maiorino[1] · Dario Giugliano[1]



Endocrine
DOI 10.1007/s12020-017-1326-1

Endocrine. 2017 Jun 3. doi: 10.1007/s12020-017-1326-1. [Epub ahead of print]

Particulate matter air pollution: individual choices for improving cardiometabolic well-being.

Esposito K[1], Bellastella G[1], Maiorino MI[1], Giugliano D[2].

Author information

Abstract

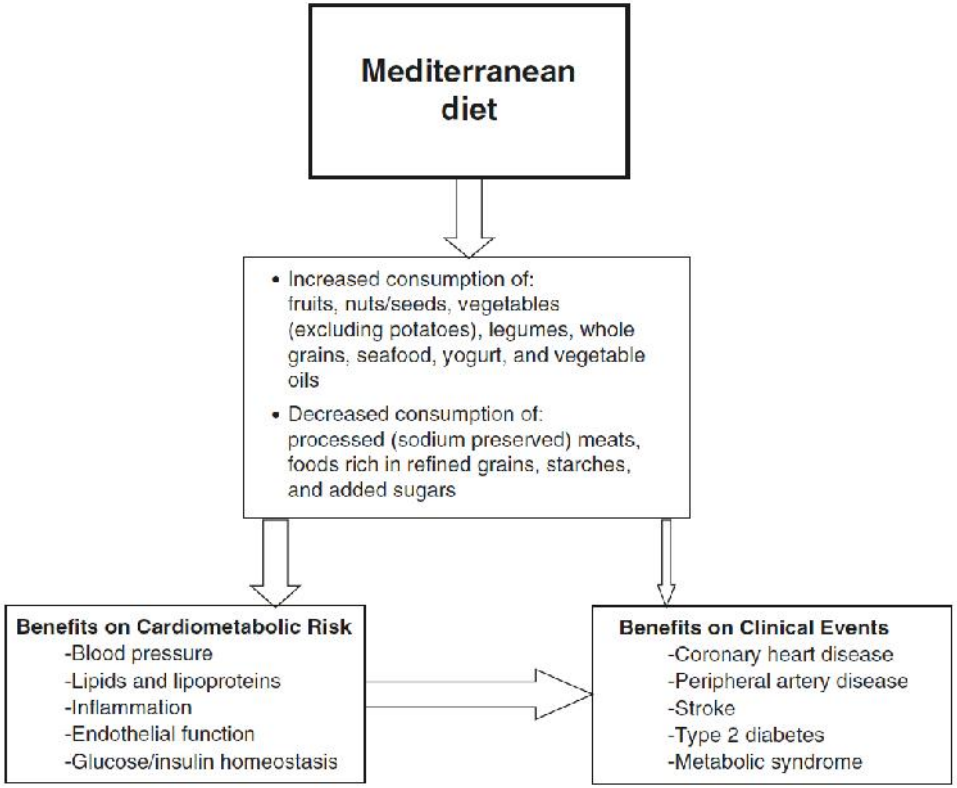
Exposure to small particulate matter ($PM_{2.5}$) has become the 5th highest ranking risk factor for death, responsible for 4.2 million deaths worldwide. PM pollution is also associated with cardiovascular disease and type 2 diabetes, and may contribute to deteriorate the already poor cardiometabolic outlook of the diabetic patient. Although most sources of outdoor air pollution are well beyond the control of individuals, there is still room for personal action. Health behaviors (smoking cessation, avoiding obesity, and increasing physical activity) may increase the poor life expectancy of individuals in the lowest income quartile of the Western population; moreover, a favorable lifestyle, (no current smoking, no obesity, physical activity at least once weekly, and a healthy diet pattern), may cut by nearly 50% the risk of coronary heart disease among people at high genetic risk. Things seem not immutable, as individual healthy choices do matter.

Endocrine (2017) 56:27–32
DOI 10.1007/s12020-016-1018-2

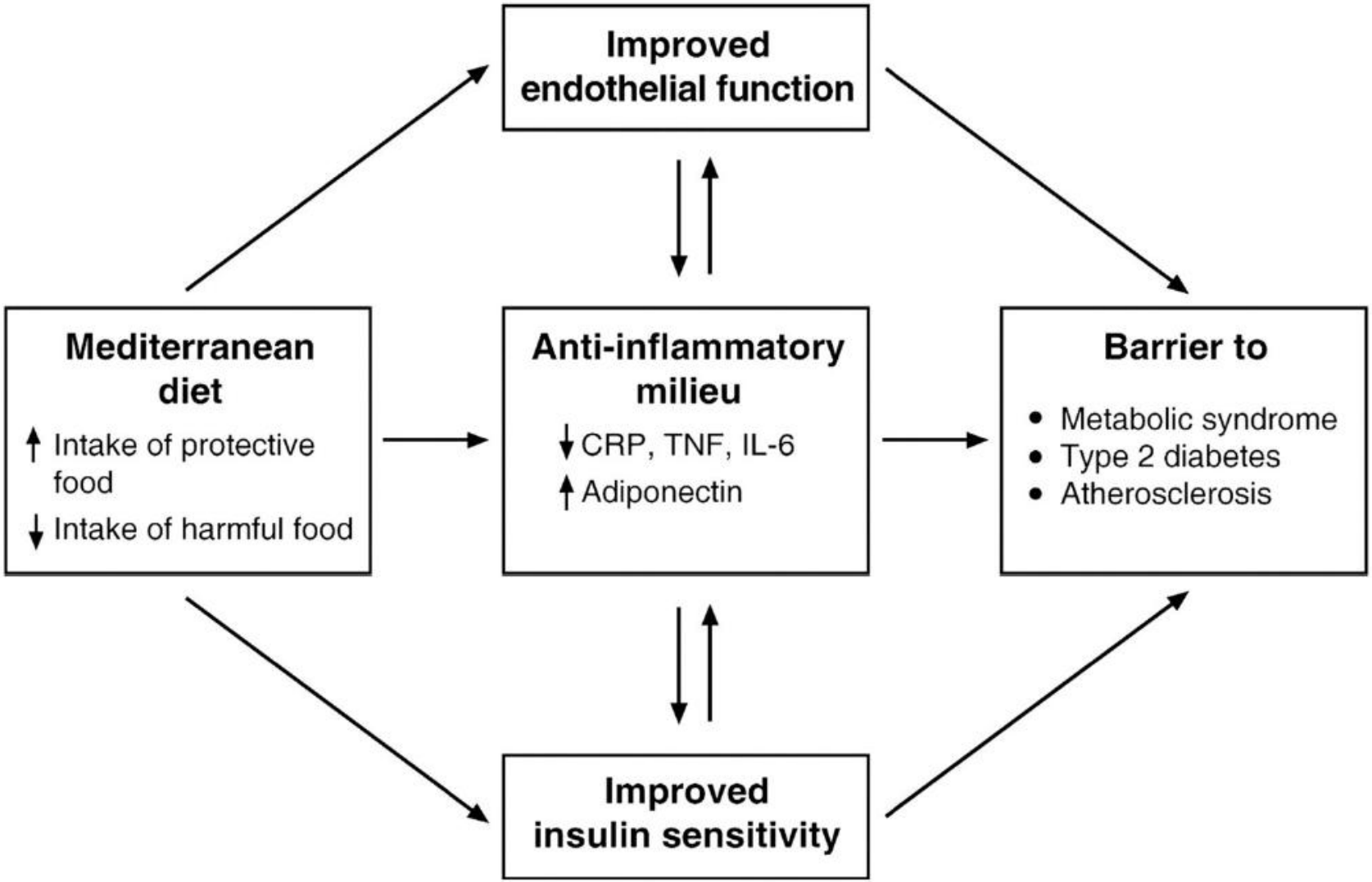
VIEWPOINT

Mediterranean diet for type 2 diabetes: cardiometabolic benefits

Katherine Esposito[1] · Maria Ida Maiorino[2] · Giuseppe Bellastella[2] · Demosthenes B. Panagiotakos[3] · Dario Giugliano[2]



Prospective studies show that higher adherence to the Mediterranean diet is associated with a 20–23% reduced risk of developing type 2 diabetes, while the results of randomized controlled trials show that Mediterranean diet reduces glycosylated hemoglobin levels by 0.30–0.47 %, and is also associated with a 28–30% reduced risk for cardiovascular events.


Improved endothelial function
Mediterranean diet
↑ Intake of protective food
↓ Intake of harmful food
Anti-inflammatory milieu
↓ CRP, TNF, IL-6
↑ Adiponectin
Barrier to
• Metabolic syndrome
• Type 2 diabetes
• Atherosclerosis
Improved insulin sensitivity

The NEW ENGLAND JOURNAL of MEDICINE

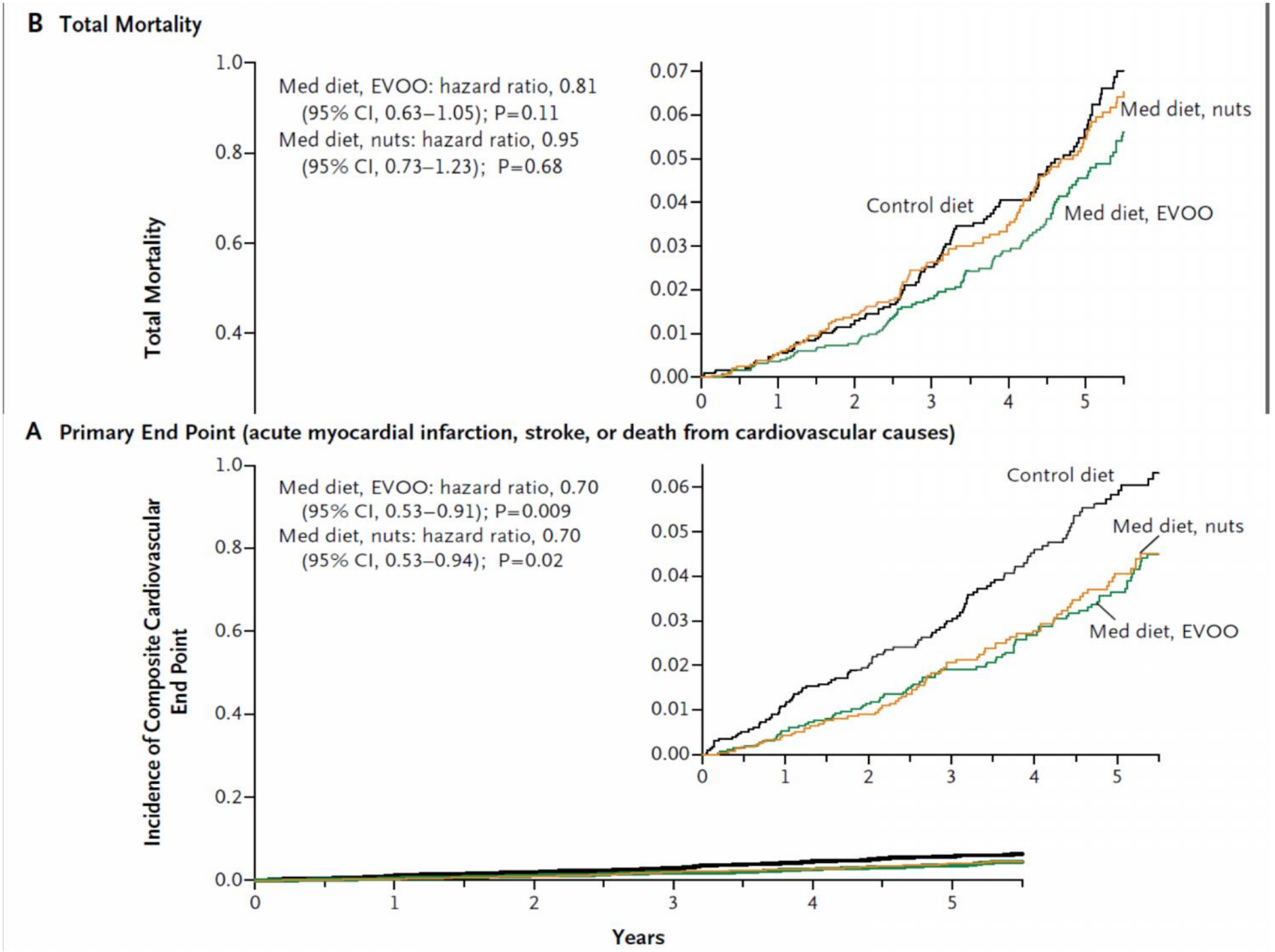
ORIGINAL ARTICLE

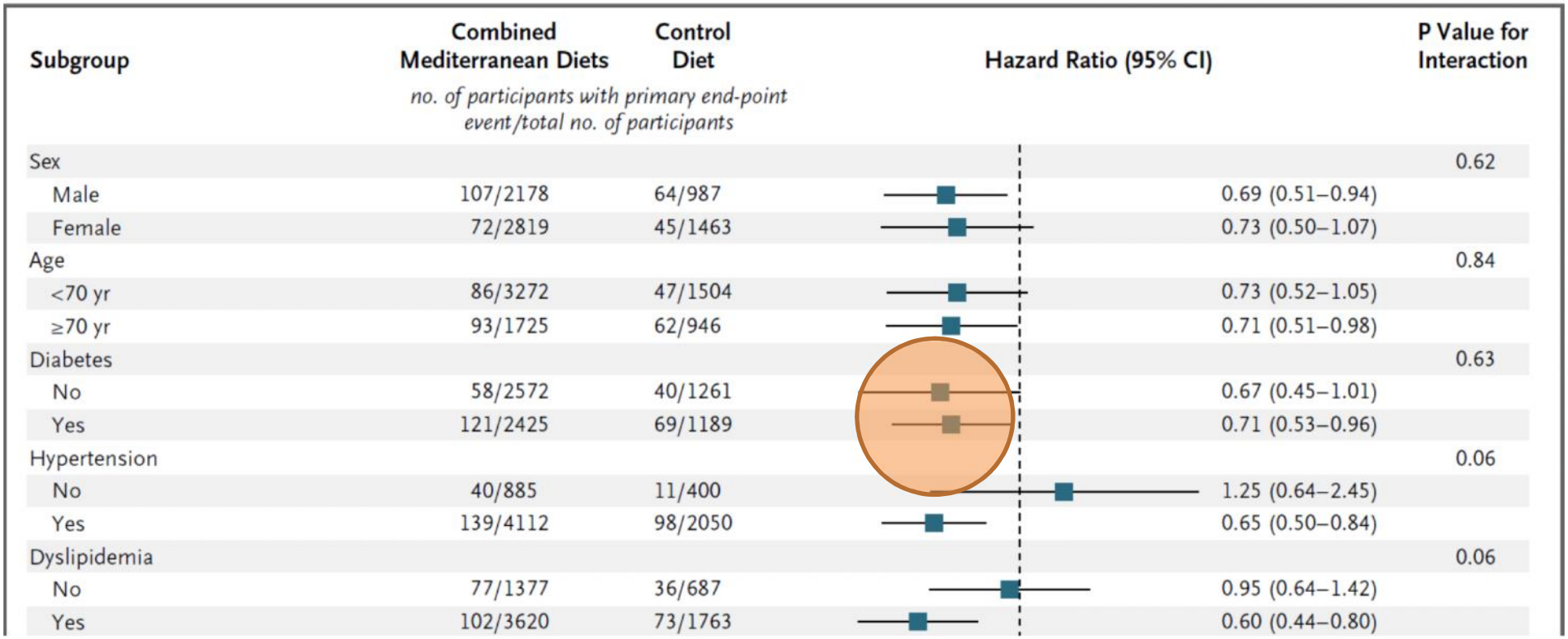
N Engl J Med 2013;368:1279-90.

Primary Prevention of Cardiovascular Disease with a Mediterranean Diet

METHODS

In a multicenter trial in Spain, we randomly assigned participants who were at high cardiovascular risk, but with no cardiovascular disease at enrollment, to one of three diets: a Mediterranean diet supplemented with extra-virgin olive oil, a Mediterranean diet supplemented with mixed nuts, or a control diet (advice to reduce dietary fat). Participants received quarterly individual and group educational sessions and, depending on group assignment, free provision of extra-virgin olive oil, mixed nuts, or small nonfood gifts. The primary end point was the rate of major cardiovascular events (myocardial infarction, stroke, or death from cardiovascular causes). On the basis of the results of an interim analysis, the trial was stopped after a median follow-up of 4.8 years.


B Total Mortality
Total Mortality
1.0
0.8
0.6
0.4
Med diet, EVOO: hazard ratio, 0.81
(95% CI, 0.63–1.05); P=0.11
Med diet, nuts: hazard ratio, 0.95
(95% CI, 0.73–1.23); P=0.68
0.07
0.06
0.05
0.04
0.03
0.02
0.01
0.00
Control diet
Med diet, nuts
Med diet, EVOO
0
1
2
3
4
5
A Primary End Point (acute myocardial infarction, stroke, or death from cardiovascular causes)
Incidence of Composite Cardiovascular End Point
1.0
0.8
0.6
0.4
0.2
0.0
Med diet, EVOO: hazard ratio, 0.70
(95% CI, 0.53–0.91); P=0.009
Med diet, nuts: hazard ratio, 0.70
(95% CI, 0.53–0.94); P=0.02
0.06
0.05
0.04
0.03
0.02
0.01
0.00
Control diet
Med diet, nuts
Med diet, EVOO
0
1
2
3
4
5
Years



Subgroup	Combined Mediterranean Diets	Control Diet	Hazard Ratio (95% CI)	P Value for Interaction
	no. of participants with primary end-point event/total no. of participants			
Sex				0.62
Male	107/2178	64/987	0.69 (0.51–0.94)	
Female	72/2819	45/1463	0.73 (0.50–1.07)	
Age				0.84
<70 yr	86/3272	47/1504	0.73 (0.52–1.05)	
≥70 yr	93/1725	62/946	0.71 (0.51–0.98)	
Diabetes				0.63
No	58/2572	40/1261	0.67 (0.45–1.01)	
Yes	121/2425	69/1189	0.71 (0.53–0.96)	
Hypertension				0.06
No	40/885	11/400	1.25 (0.64–2.45)	
Yes	139/4112	98/2050	0.65 (0.50–0.84)	
Dyslipidemia				0.06
No	77/1377	36/687	0.95 (0.64–1.42)	
Yes	102/3620	73/1763	0.60 (0.44–0.80)	

The NEW ENGLAND JOURNAL of MEDICINE

CORRESPONDENCE

Katherine Esposito, M.D., Ph.D.
Dario Giugliano, M.D., Ph.D.

n engl j med 369;674-675, august 15, 2013

Mediterranean Diet for Primary Prevention of Cardiovascular Disease

In addition to reducing the need for a first diabetes drug, a Mediterranean diet improves long-term cardiovascular outlook and insulin sensitivity in type 2 diabetes.

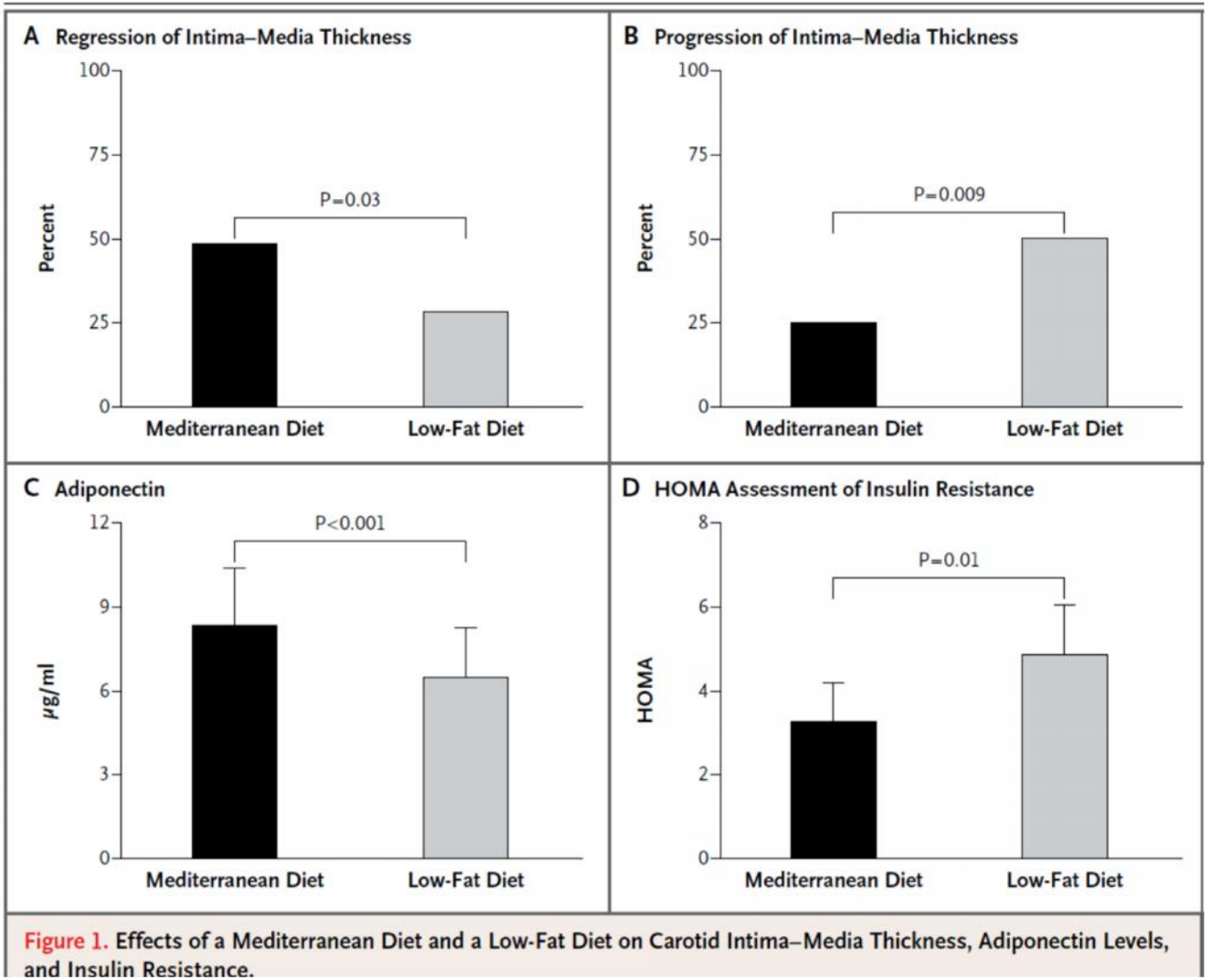


Figure 1. Effects of a Mediterranean Diet and a Low-Fat Diet on Carotid Intima–Media Thickness, Adiponectin Levels, and Insulin Resistance.

Table 5—Current recommendations for CVD risk factor management in type 2 diabetes mellitus

Risk factor	Relevant statement or guideline	Specific recommendation and Level of Evidence
Nutrition	American Heart Association / American Diabetes Association	Reduction of energy intake for overweight or obese patients (*ADA Level of Evidence A*).
		Individualized medical nutrition therapy for all patients with diabetes mellitus (*ADA Level of Evidence A*).
		Carbohydrate monitoring as an important strategy for glycemic control (*ADA Level of Evidence B*).
		Consumption of fruits, legumes, vegetables, whole grains, and dairy products in place of other carbohydrate sources (*ADA Level of Evidence B*).
		Mediterranean-style dietary pattern may improve glycemic control and CVD risk factors (*ADA Level of Evidence B*).
		Limit of sodium to <2,300 mg/day, similar to recommendations for the general population (*ADA Level of Evidence B*; note that the AHA differs and recommends sodium <1,500 mg/day).

Evert AB, et al. Nutrition therapy recommendations for the management of adults with diabetes. Diabetes Care 2014;37(Suppl. 1):S120–S143

Annals of Internal Medicine

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1 September 2009 Volume 151 Issue 5

VIEW CHECKED ABSTRACTS | CLEAR

Previous Issue

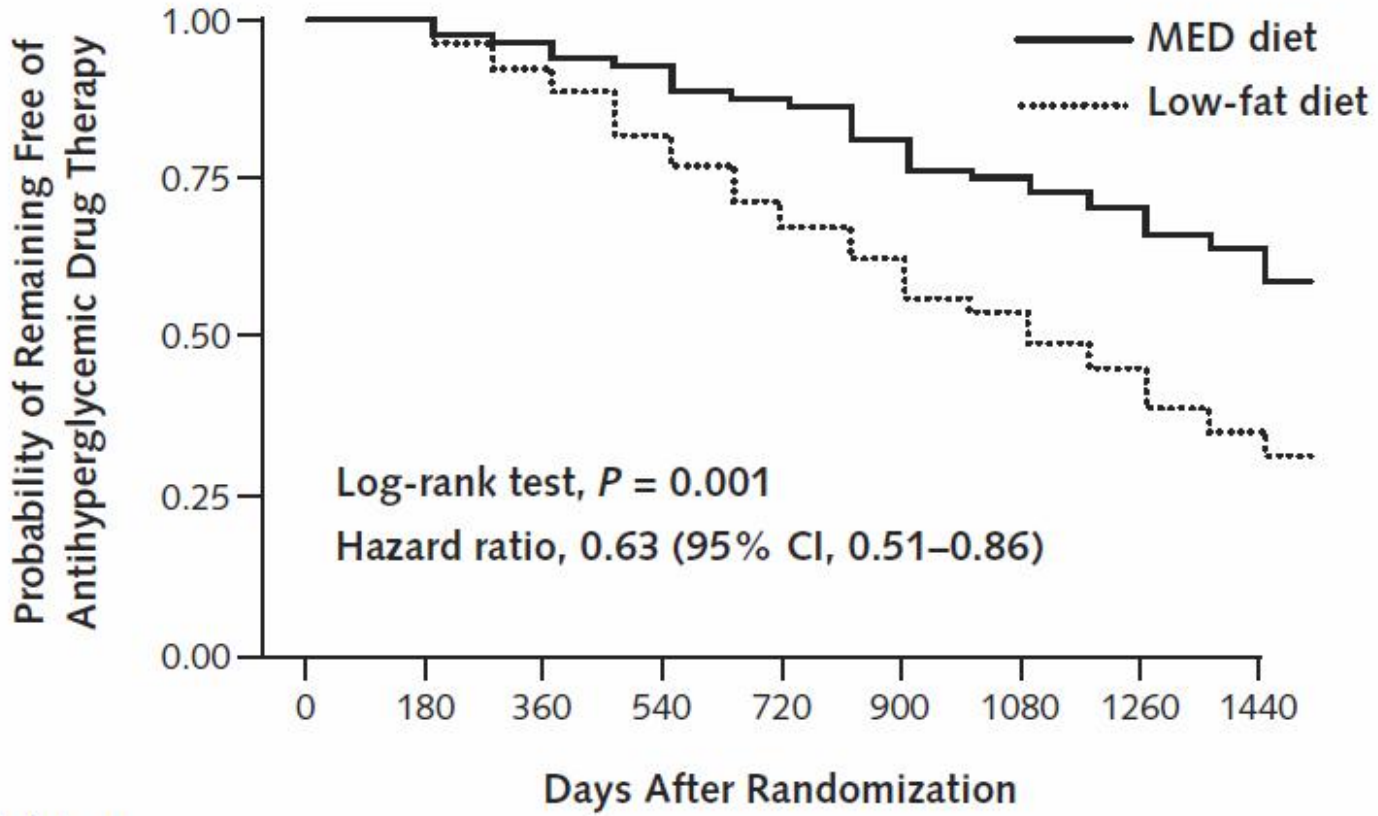
Effects of a Mediterranean-Style Diet on the Need for Antihyperglycemic Drug Therapy in Patients With Newly Diagnosed Type 2 Diabetes: A Randomized Trial

Katherine Esposito, Maria Ida Maiorino, Miryam Ciotola, Carmen Di Palo, Paola Scognamiglio, Maurizio Gicchino, Michela Petrizzo, Franco Saccomanno, Flora Beneduce, Antonio Ceriello, and Dario Giugliano

Esposito and associates compared the effects of low-carbohydrate, Mediterranean-style and low-fat, calorie-restricted diets on the need for antihyperglycemic drug therapy in 215 overweight patients with newly diagnosed type 2 diabetes. After 4 years, 44% of the Mediterranean-style diet group and 70% of the low-fat diet group required treatment of diabetes, and the Mediterranean-style diet group lost more weight and experienced greater improvements in some glycemic control and coronary risk measures.

Abstract | Full Text | PDF | Summary for Patients | Video News Release

Figure 2. **Probability of remaining free of antihyperglycemic drug therapy.**

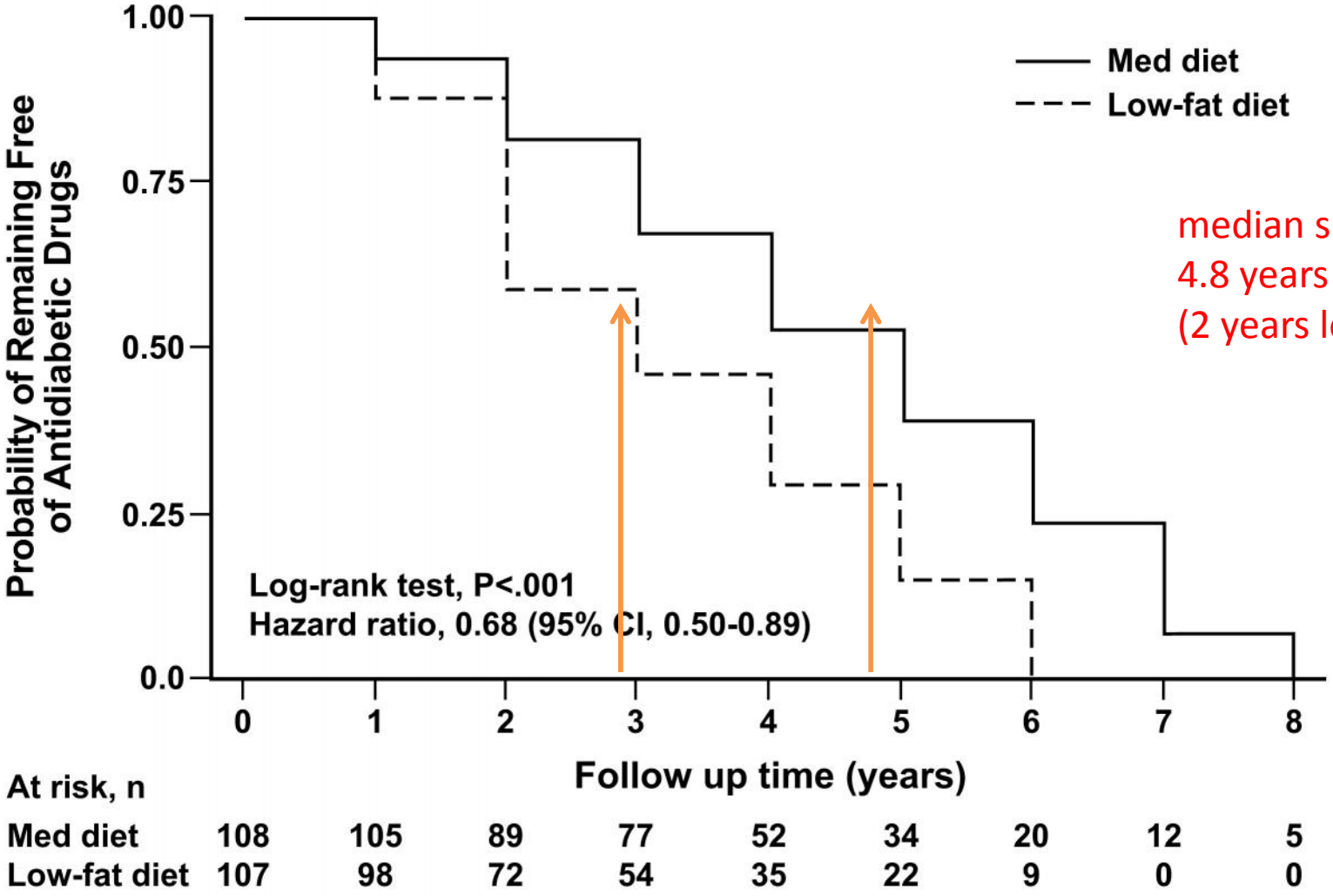


At risk, *n*										
MED diet	108	108	105	101	89	80	77	66	52	49
Low-fat diet	107	107	98	87	72	64	54	45	35	29

The Effects of a Mediterranean Diet on the Need for Diabetes Drugs and Remission of Newly Diagnosed Type 2 Diabetes: Follow-up of a Randomized Trial

Katherine Esposito,[1] Maria Ida Maiorino,[1] Michela Petrizzo,[1] Giuseppe Bellastella,[2] and Dario Giugliano[2]

Diabetes Care 2014;37:1824–1830 | DOI: 10.2337/dc13-2899



median survival time
4.8 years vs 2.8 years
(2 years longer)

The Effects of a Mediterranean Diet on the Need for Diabetes Drugs and Remission of Newly Diagnosed Type 2 Diabetes: Follow-up of a Randomized Trial

Katherine Esposito,[1] Maria Ida Maiorino,[1] Michela Petrizzo,[1] Giuseppe Bellastella,[2] and Dario Giugliano[2]

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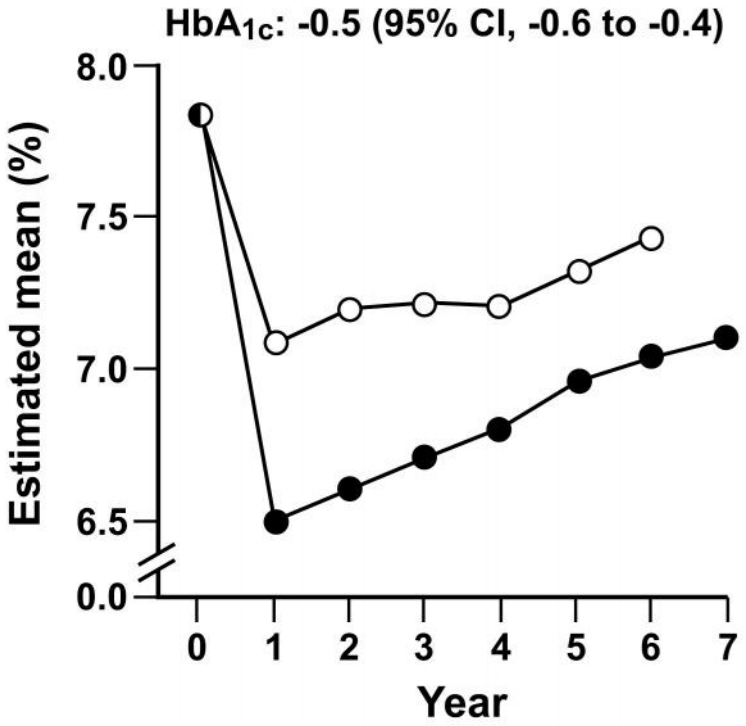

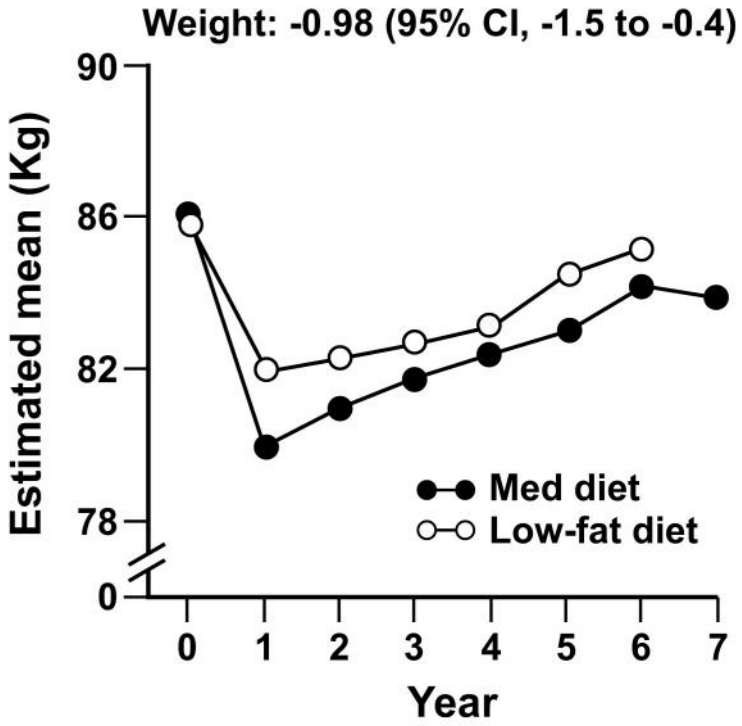

CONCLUSIONS

In patients with newly diagnosed type 2 diabetes, a LCMD resulted in a greater reduction of HbA1c levels, higher rate of diabetes remission, and delayed need for diabetes medication compared with a low-fat diet.

The Effects of a Mediterranean Diet on the Need for Diabetes Drugs and Remission of Newly Diagnosed Type 2 Diabetes: Follow-up of a Randomized Trial

Katherine Esposito,[1] Maria Ida Maiorino,[1] Michela Petrizzo,[1] Giuseppe Bellastella,[2] and Dario Giugliano[2]

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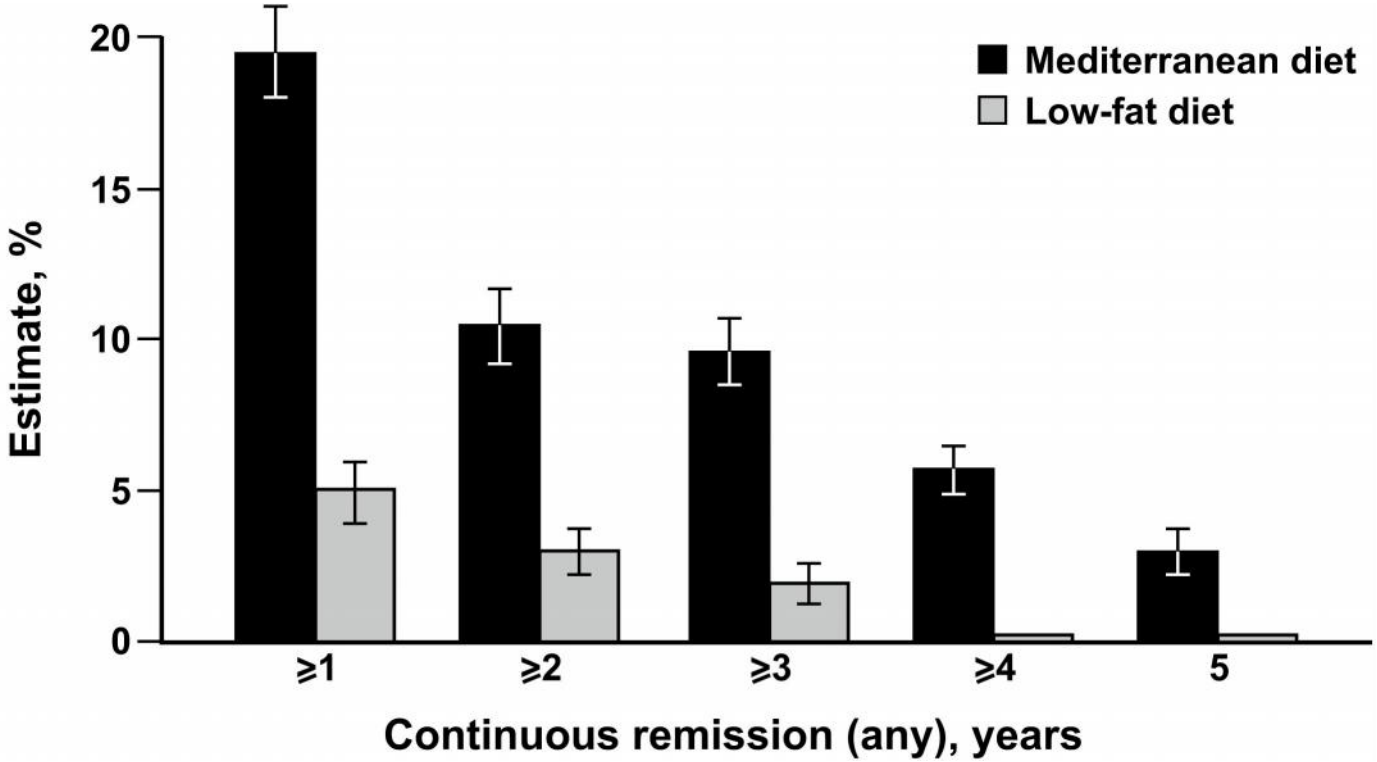
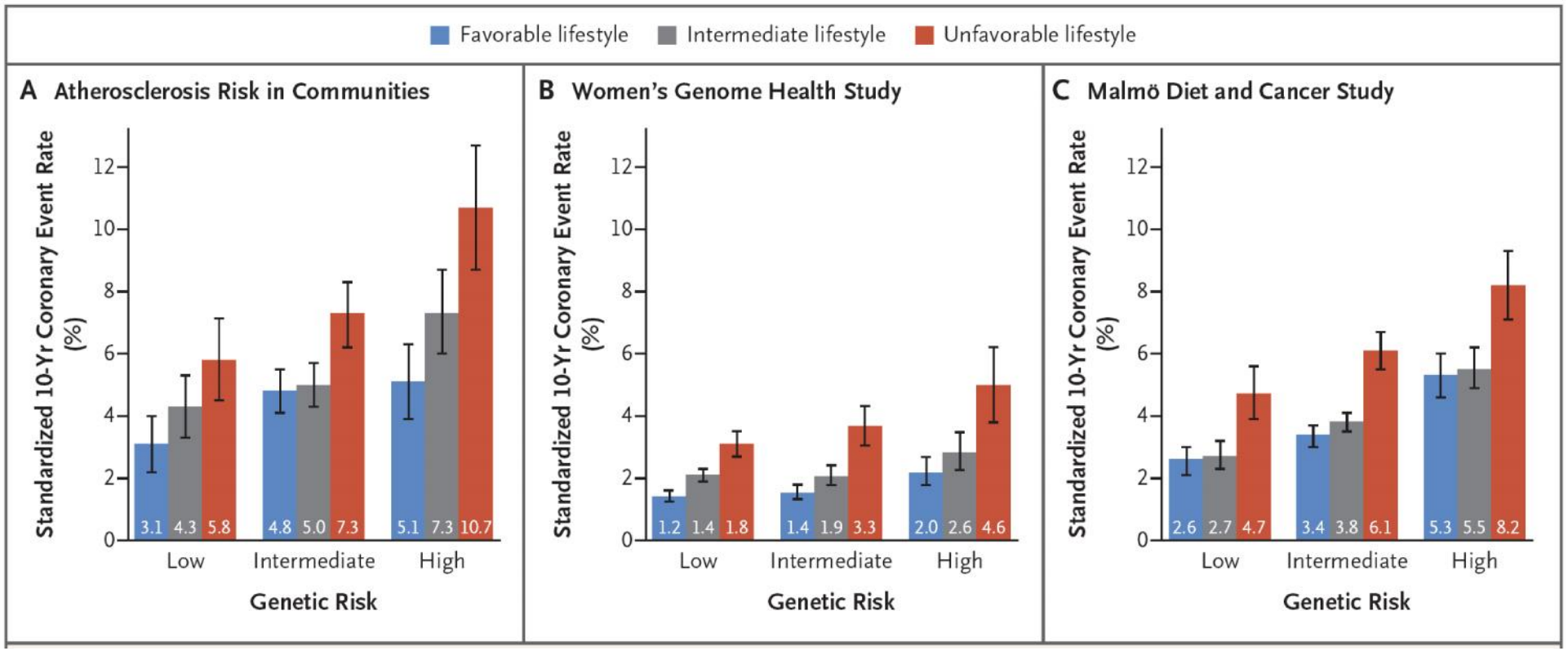


- Partial remission of diabetes was defined as a transition from meeting diabetes criteria to a prediabetes level of glycemia (FPG: 100-126 mg/dL and HbA1c of 5.7%-6.5%)
- Complete remission was defined as transition from diabetes criteria to full normalization of glucose (FPG <100 mg/dL and HbA1c <5.7%).

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Genetic Risk, Adherence to a Healthy Lifestyle, and Coronary Disease



N Engl J Med. 2016 Dec 15;375(24):2349-2358

A favorable lifestyle, as defined as at least three of the four healthy lifestyle factors

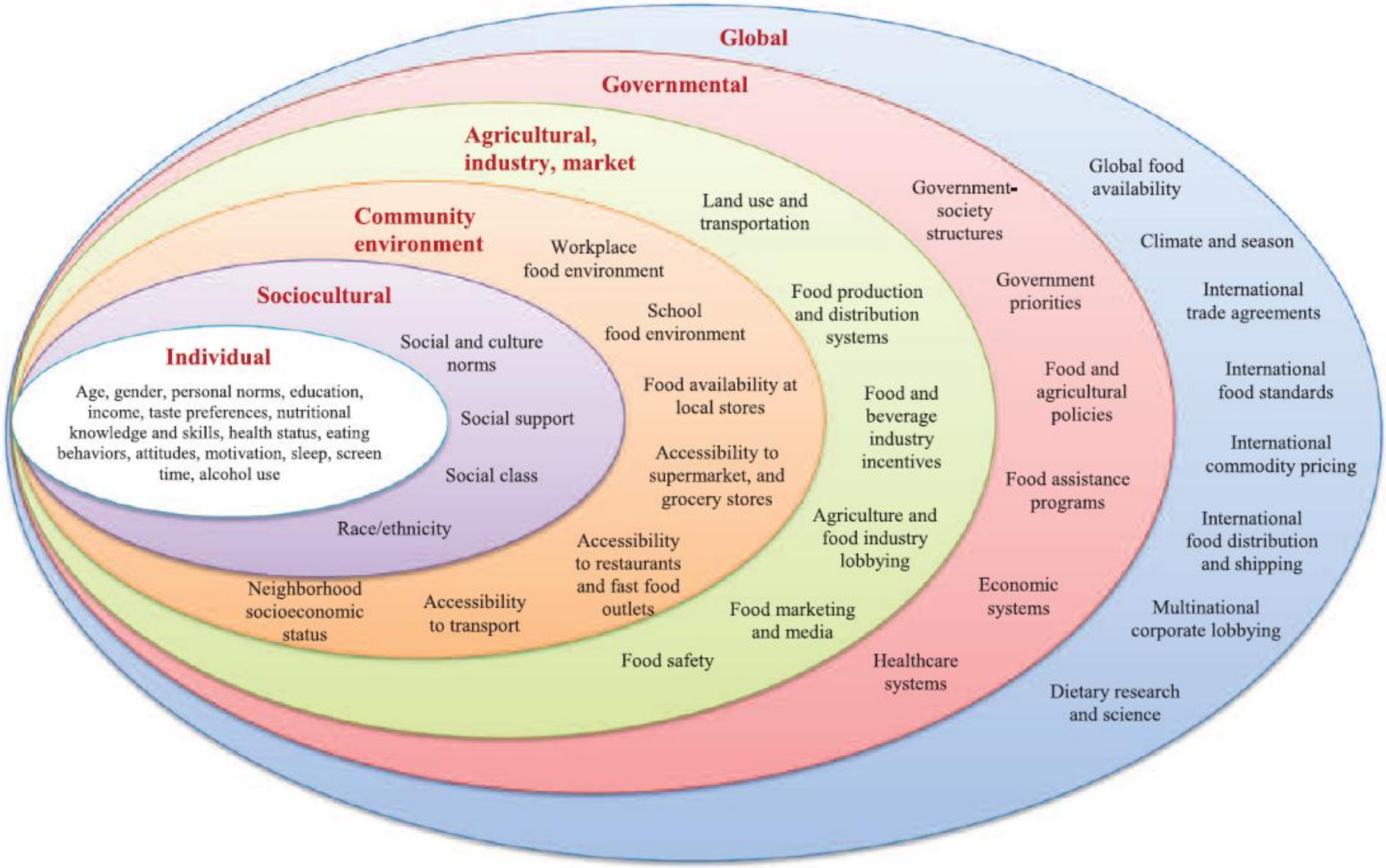
- **No current smoking**
- **No obesity**
- **Physical activity at least once weekly**
- **A healthy diet pattern**

may cut by nearly 50% the risk of coronary heart disease among people at high genetic risk

CONCLUSIONS

Across four studies involving 55,685 participants, genetic and lifestyle factors were independently associated with susceptibility to coronary artery disease. Among participants at high genetic risk, a favorable lifestyle was associated with a nearly 50% lower relative risk of coronary artery disease than was an unfavorable lifestyle. (Funded by the National Institutes of Health and others.)

Barriere ed opportunità del mangiar sano



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