Prevenzione e Stili di Vita:
Strumenti per una Società che cambia

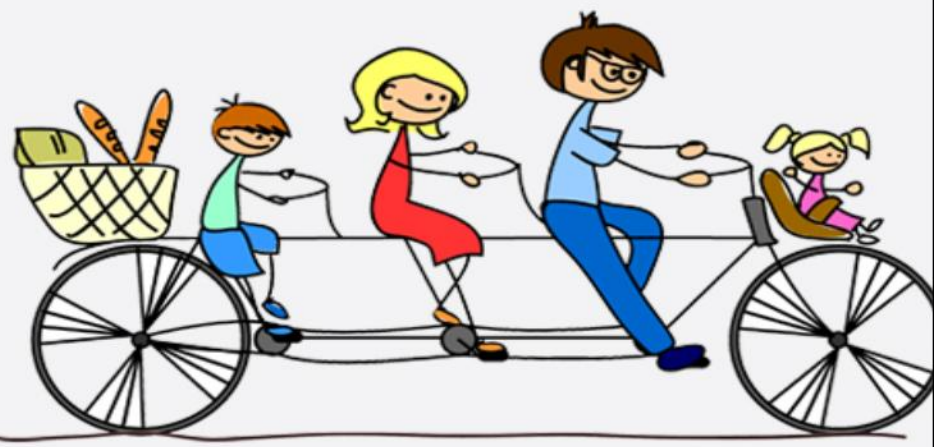
27 aprile 2012 Aula Pocchiari – ISS - Roma

SINDROME METABOLICA: PREVENIRE O CURARE?

Michele O. Carruba

Department of Pharmacology, Chemotherapy and Medical Toxicology

Center for Study and Research on Obesity



University of Milan, Italy

School of Medicine

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SINDROME METABOLICA: PREVENIRE O CURARE?

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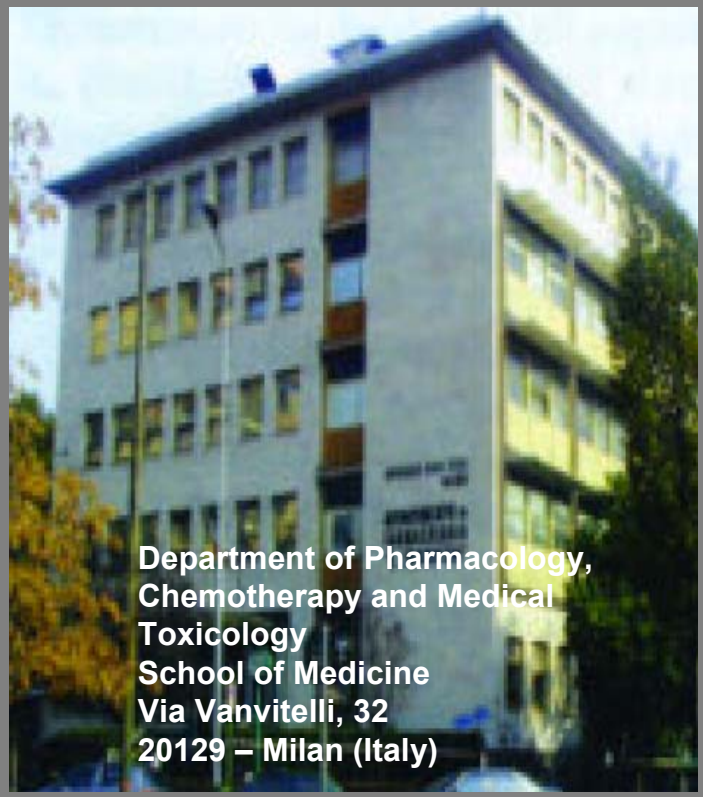
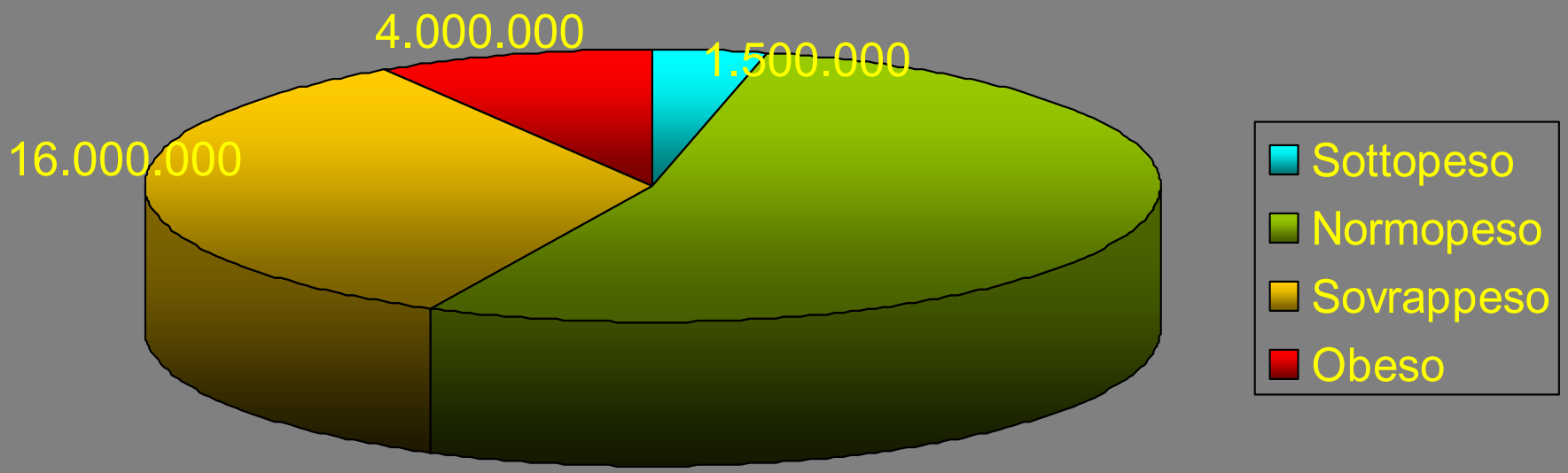
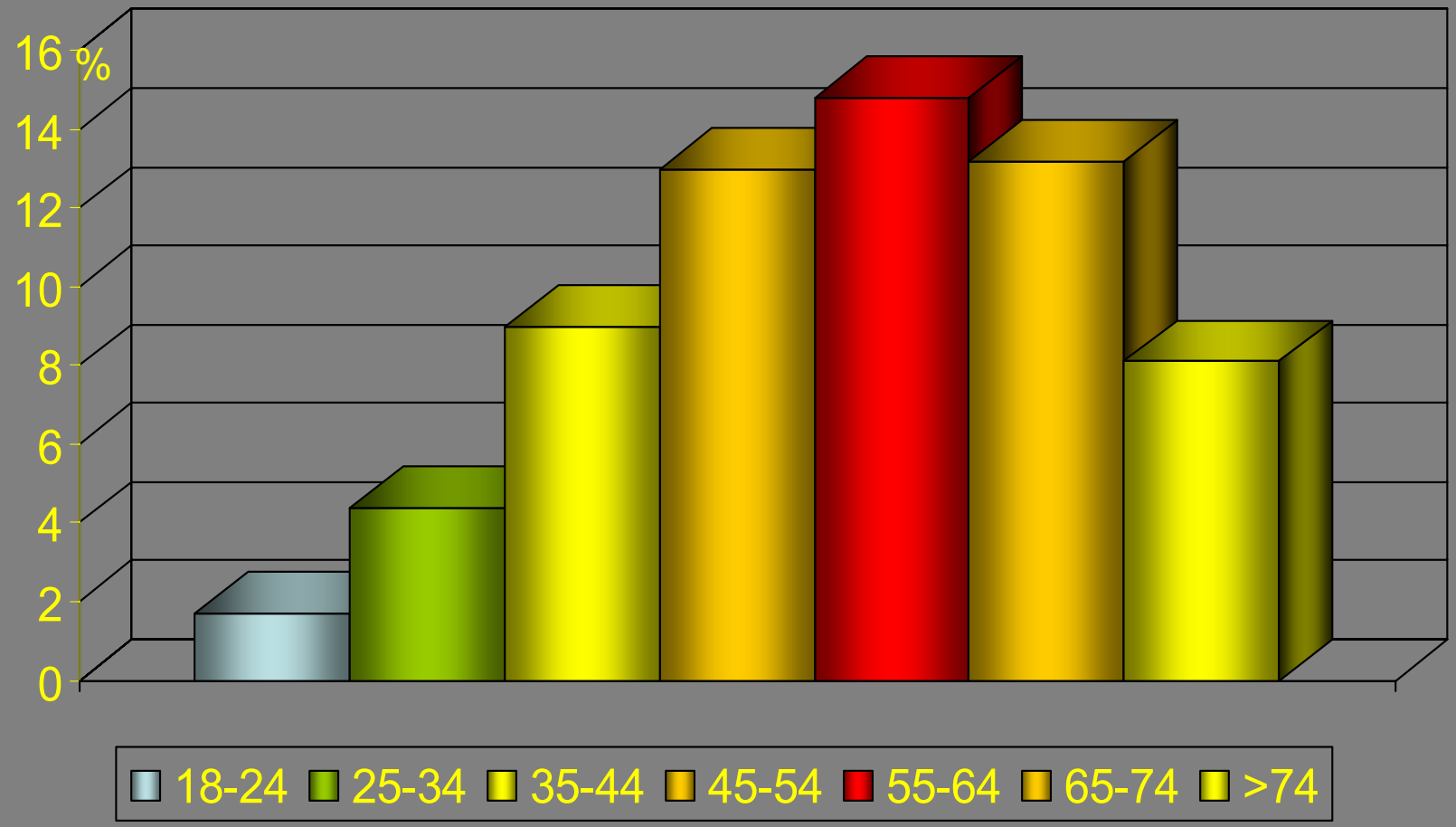


Distribuzione della popolazione italiana nelle varie condizioni di peso



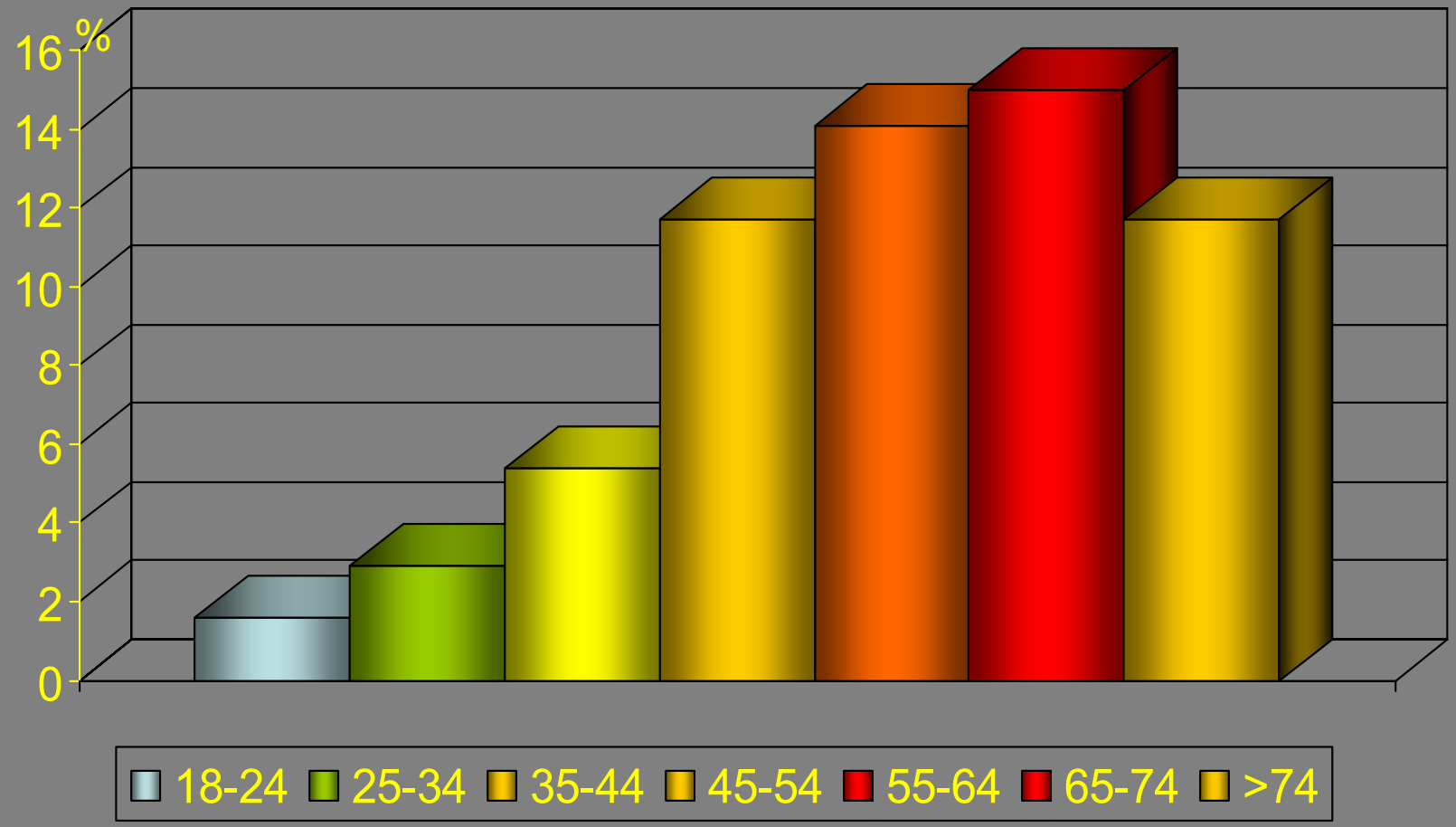
Fonte: ISTAT, 4° Rapporto sull'Obesità in Italia. Istituto Auxologico Italiano, 2002

Prevalenza dell'obesità (IMC>30) in funzione dell'età negli **UOMINI**



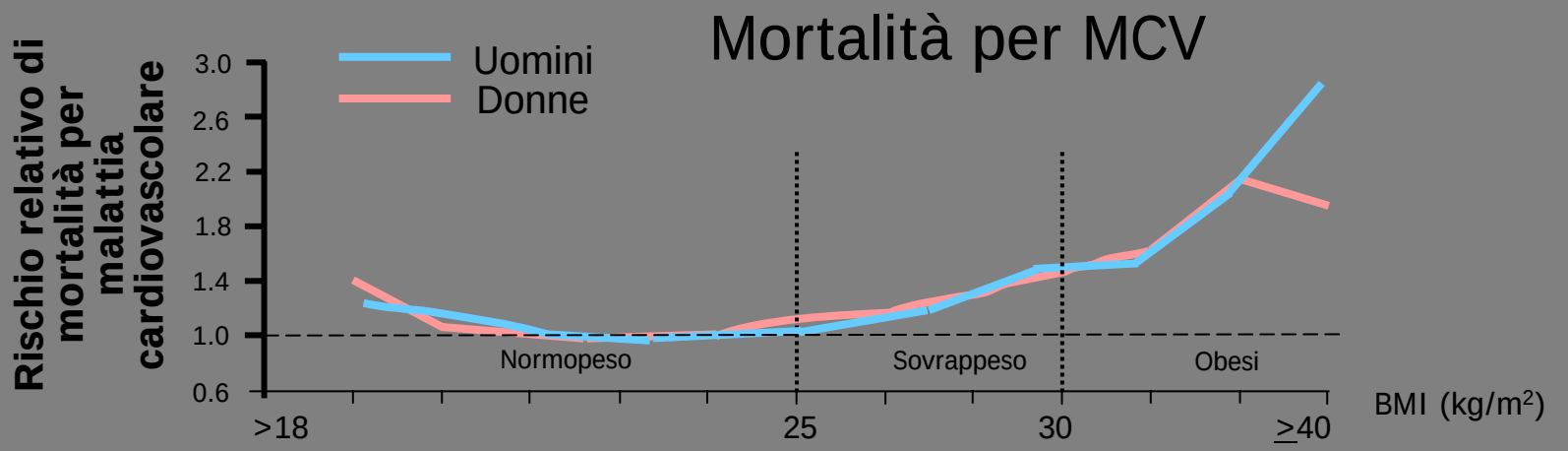
Fonte: ISTAT, 4° Rapporto sull'Obesità in Italia. Istituto Auxologico Italiano, 2002

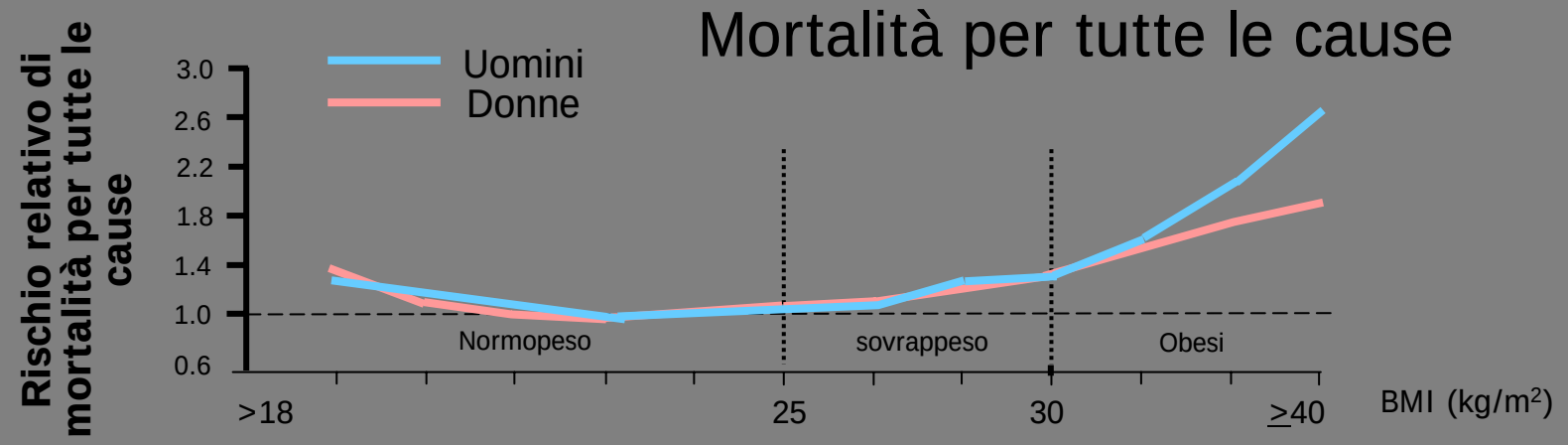
Prevalenza dell'obesità (IMC>30) in funzione dell'età nelle **DONNE**



Fonte: ISTAT, 4° Rapporto sull'Obesità in Italia. Istituto Auxologico Italiano, 2002

Sovrappeso e obesità aumentano il rischio di MCV e di mortalità per tutte le cause

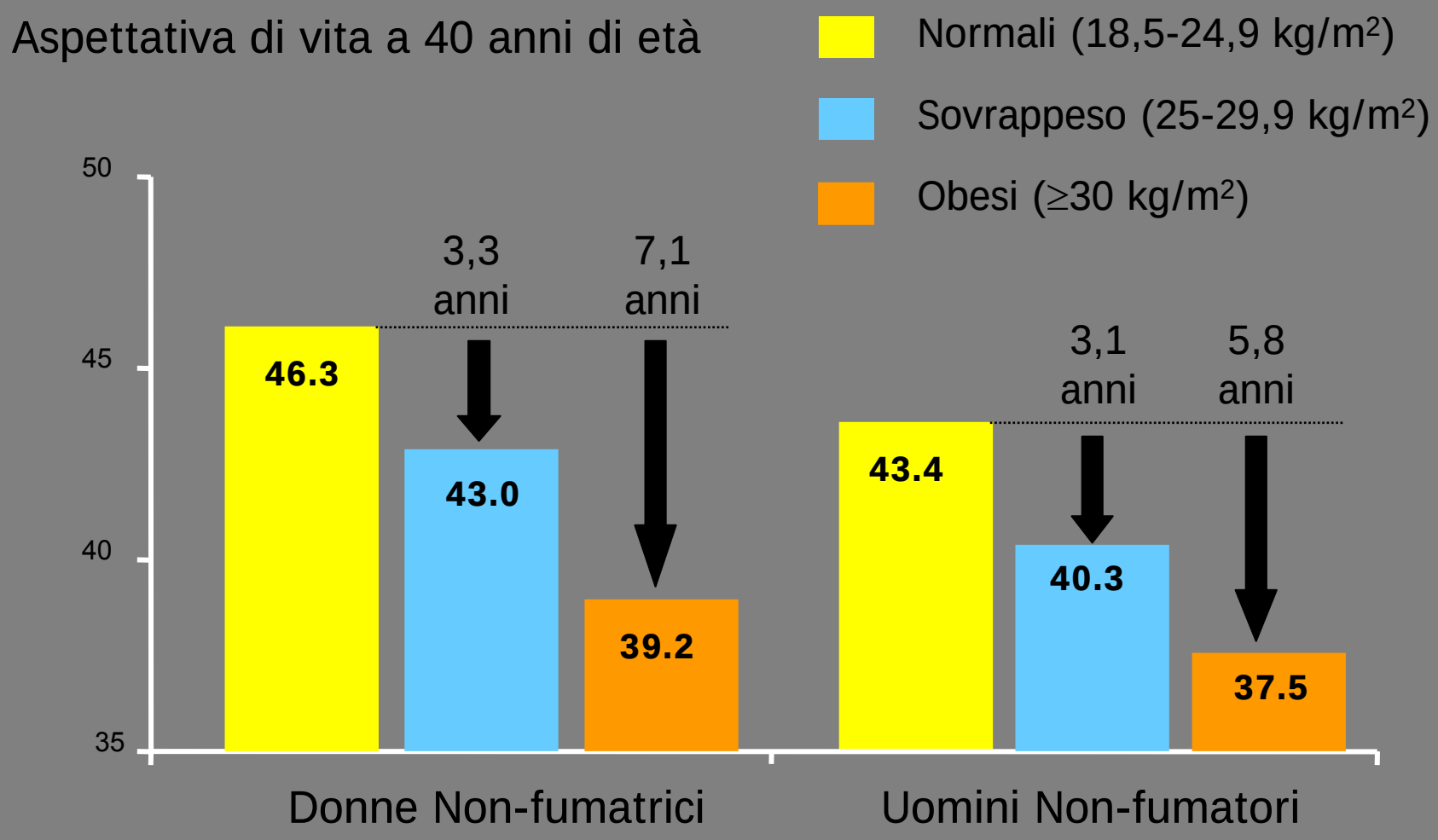




Dati relativi a 1 milione di uomini e donne seguiti per 16 anni con età media di 57 anni che non hanno mai fumato e non avevano una storia di malattia all'arruolamento.

Calle et al. *N Engl J Med.* 1999;341:1097-1105

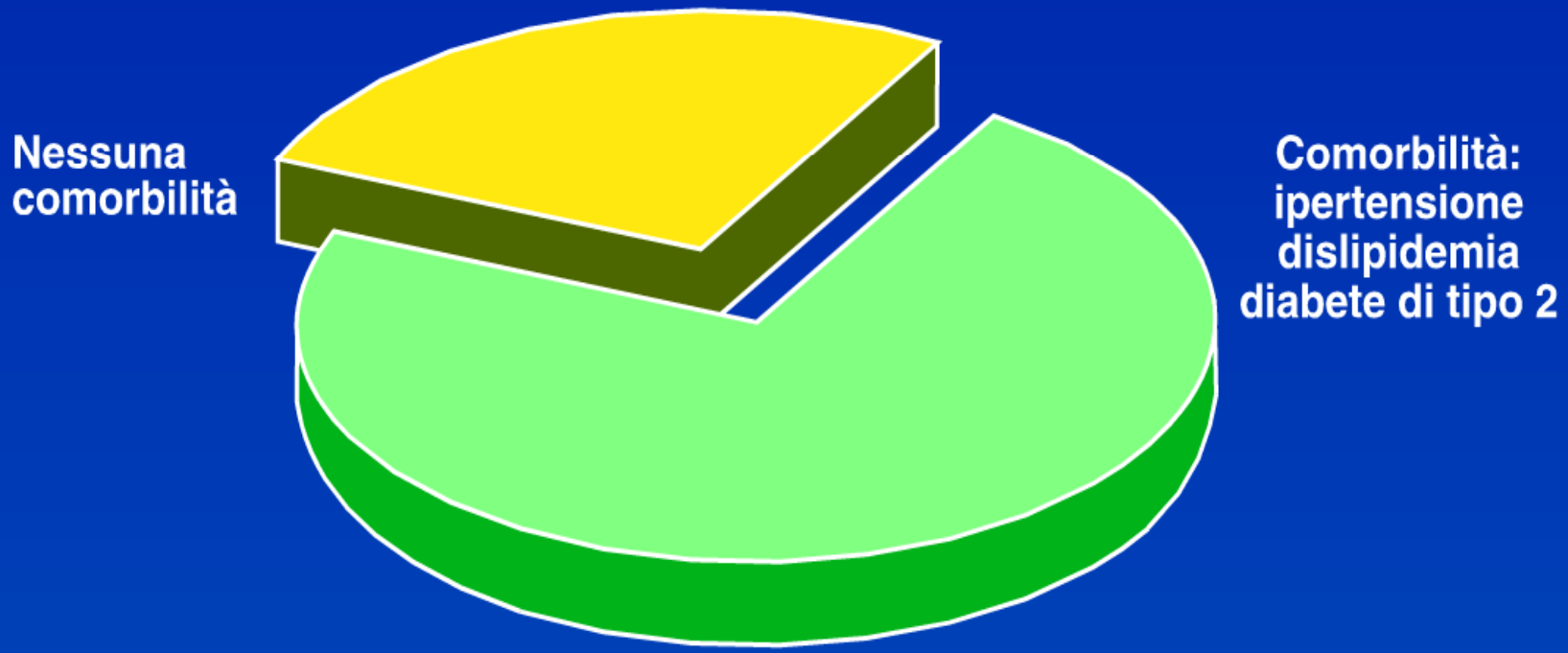
Aspettativa di vita a 40 anni: impatto dell'eccesso di peso corporeo



Peeters et al. *Ann Intern Med* 2003; 138: 24-32

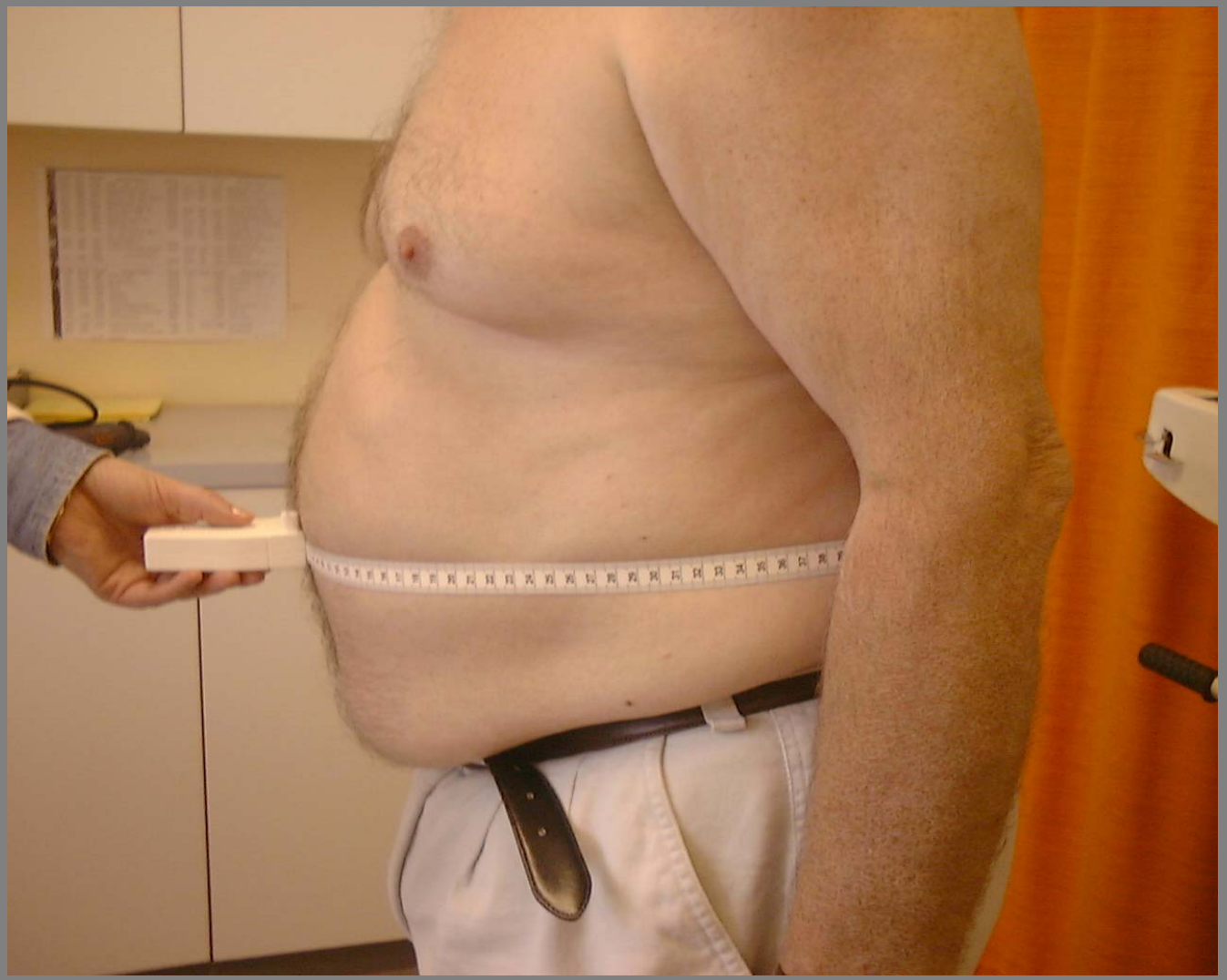
BMI e comorbilità

Pazienti con BMI ≥27 (%)

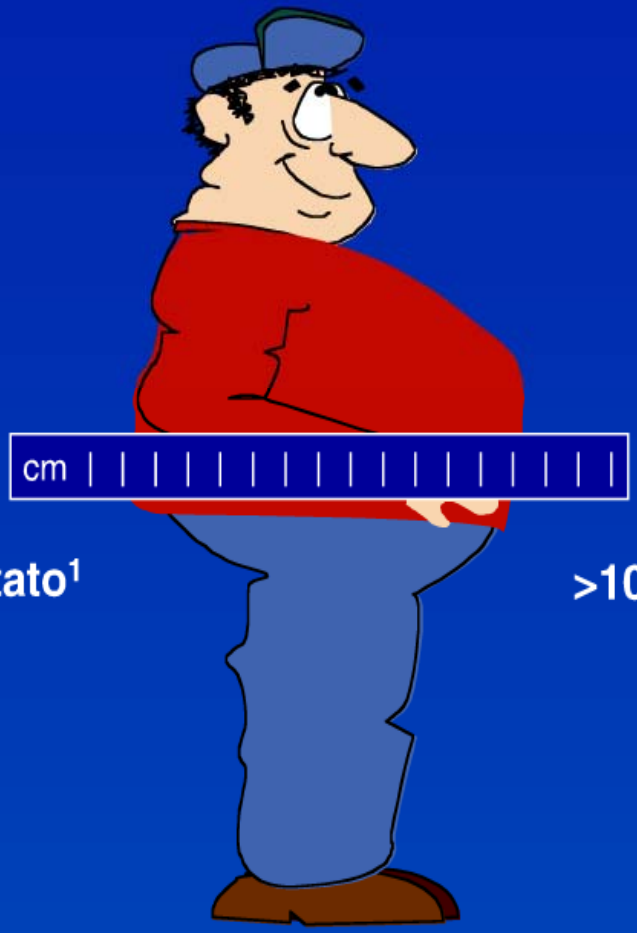


Dati non pubblicati NHANES III

Un parametro fondamentale: la circonferenza addominale



La circonferenza vita è un indicatore del tessuto adiposo viscerale



Donne

>88 cm = Rischio aumentato[1]

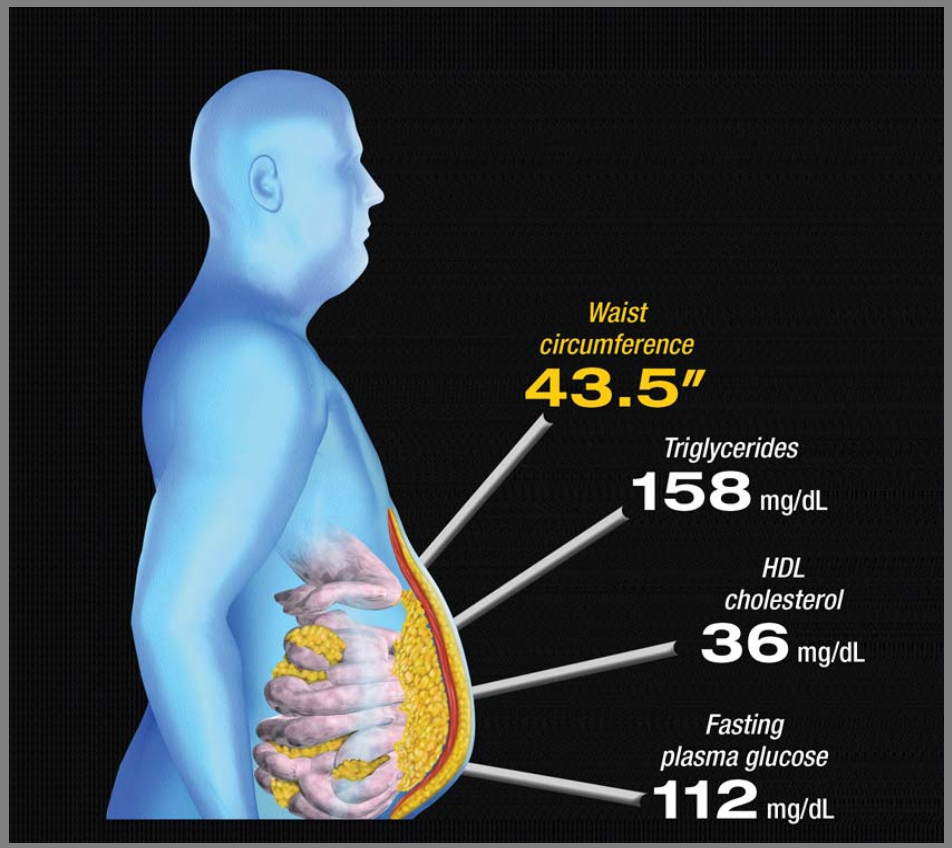
Uomini

>102 cm = Rischio aumentato[1]

[1]Lean MEJ, et al. Lancet;1998:351:853–6

Unmet clinical need associated with abdominal obesity

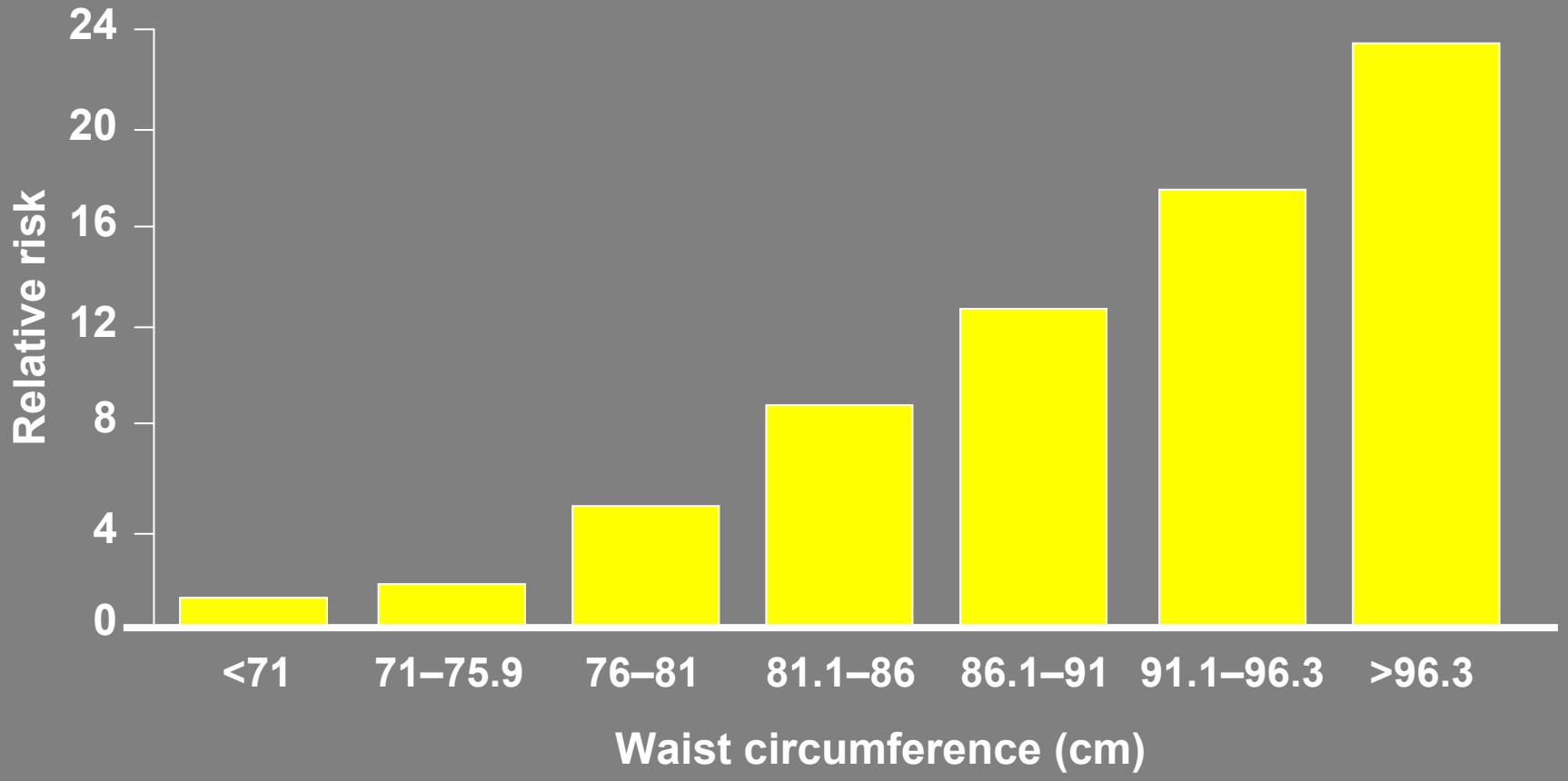
CV risk factors in a typical patient with abdominal obesity



Patients with abdominal obesity (high waist circumference) often present with one or more additional CV risk factors

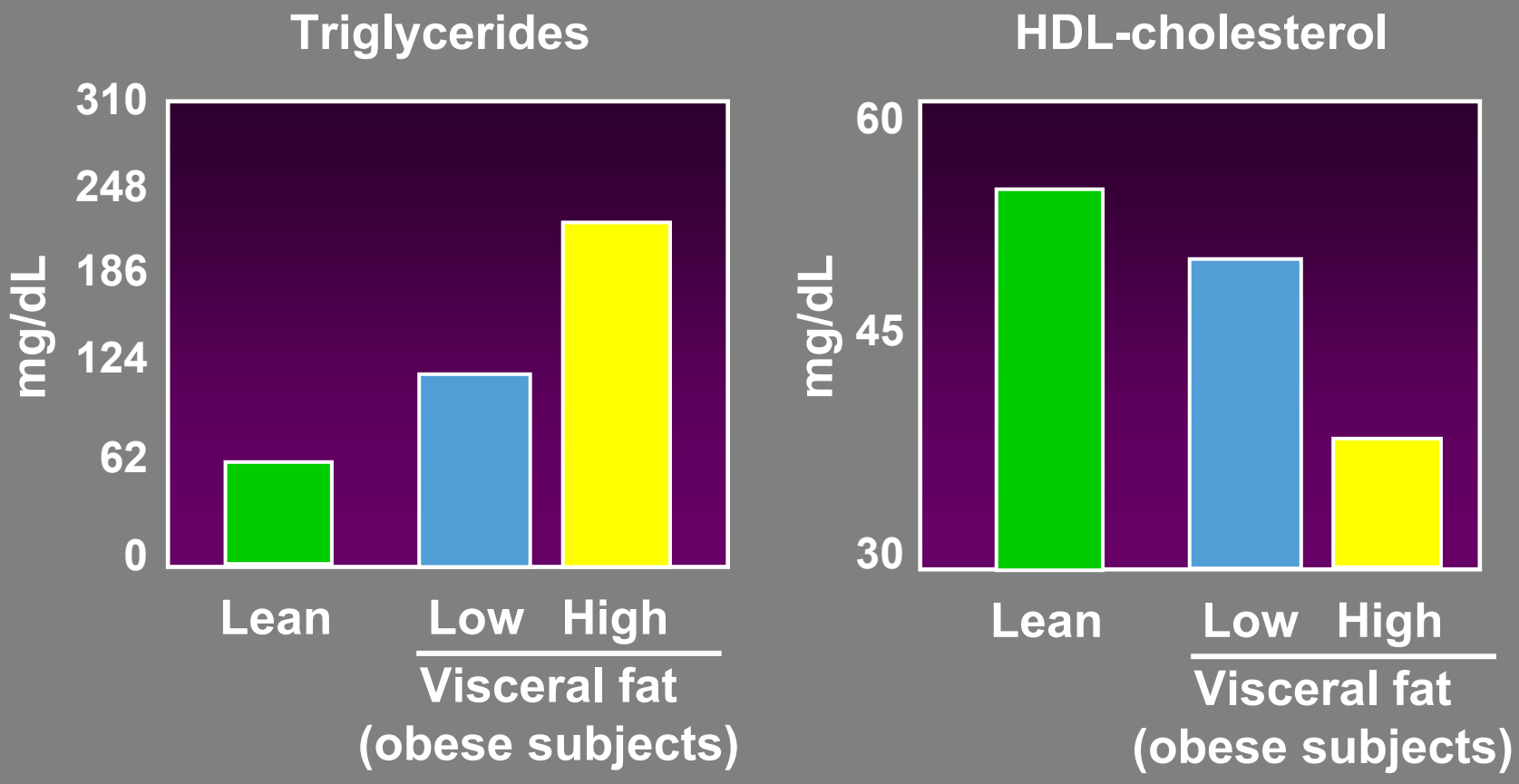
NHANES 1999–2000 cohort (data on file)

Abdominal obesity increases the risk of developing type 2 diabetes



Carey et al 1997

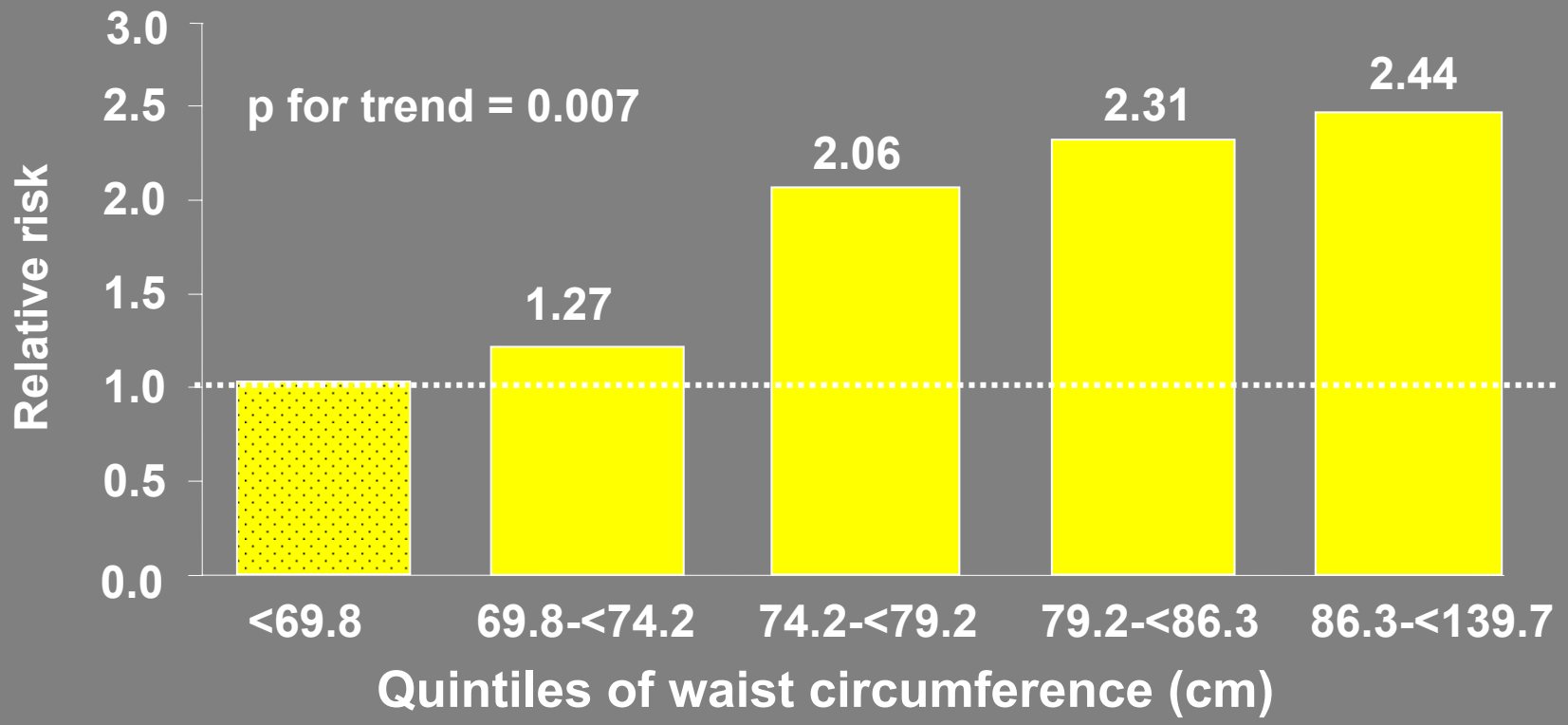
Intra-abdominal adiposity and dyslipidaemia



Pouliot et al 1992

Abdominal obesity and increased risk of CHD

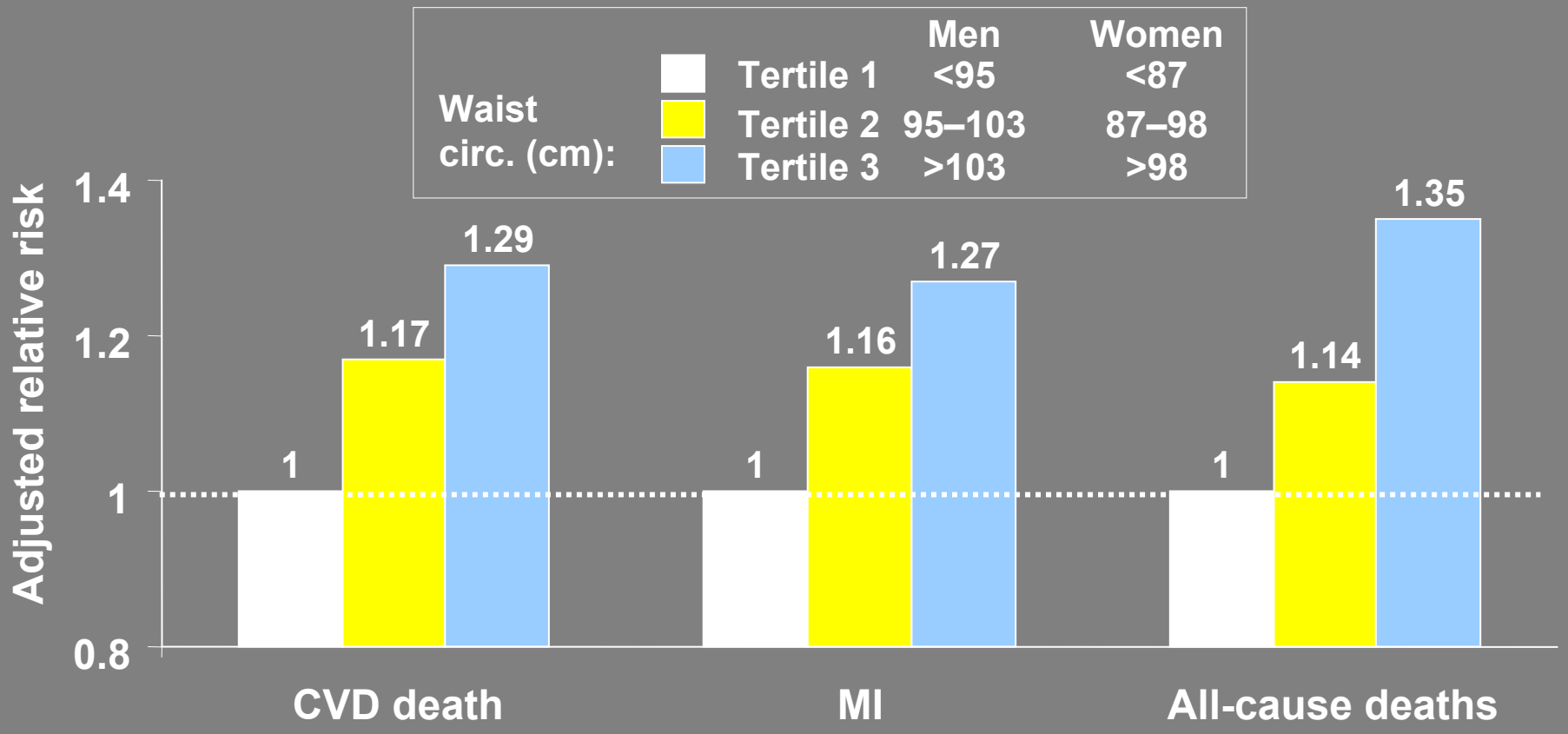
Waist circumference was independently associated with increased age-adjusted risk of CHD, even after adjusting for BMI and other CV risk factors



Rexrode et al 1998

Abdominal obesity and increased risk of cardiovascular events

The HOPE Study



Adjusted for BMI, age, smoking, sex, CVD, disease, DM, HDL-C, total-C

Dagenais et al 2005

Sindrome metabolica

Reaven GM. Annu Rev Med 1993; 44:121–31

"STUDIO SPESA" : COSTI DIRETTI ANNUI DELL'OBESITÀ IN ITALIA

BMI	Costo tot (€)	Pazienti (mlo)	%	Tot (mld)
25-29,9	984	17,5	35	17,2
30-39,9	2.136	4,5	9	9,6
> 40	2796	0,5	0,9	1,4
				28,2

Studio SPESA: composizione dei costi dell'Obesità Tot 28,2 mld Euro/anno

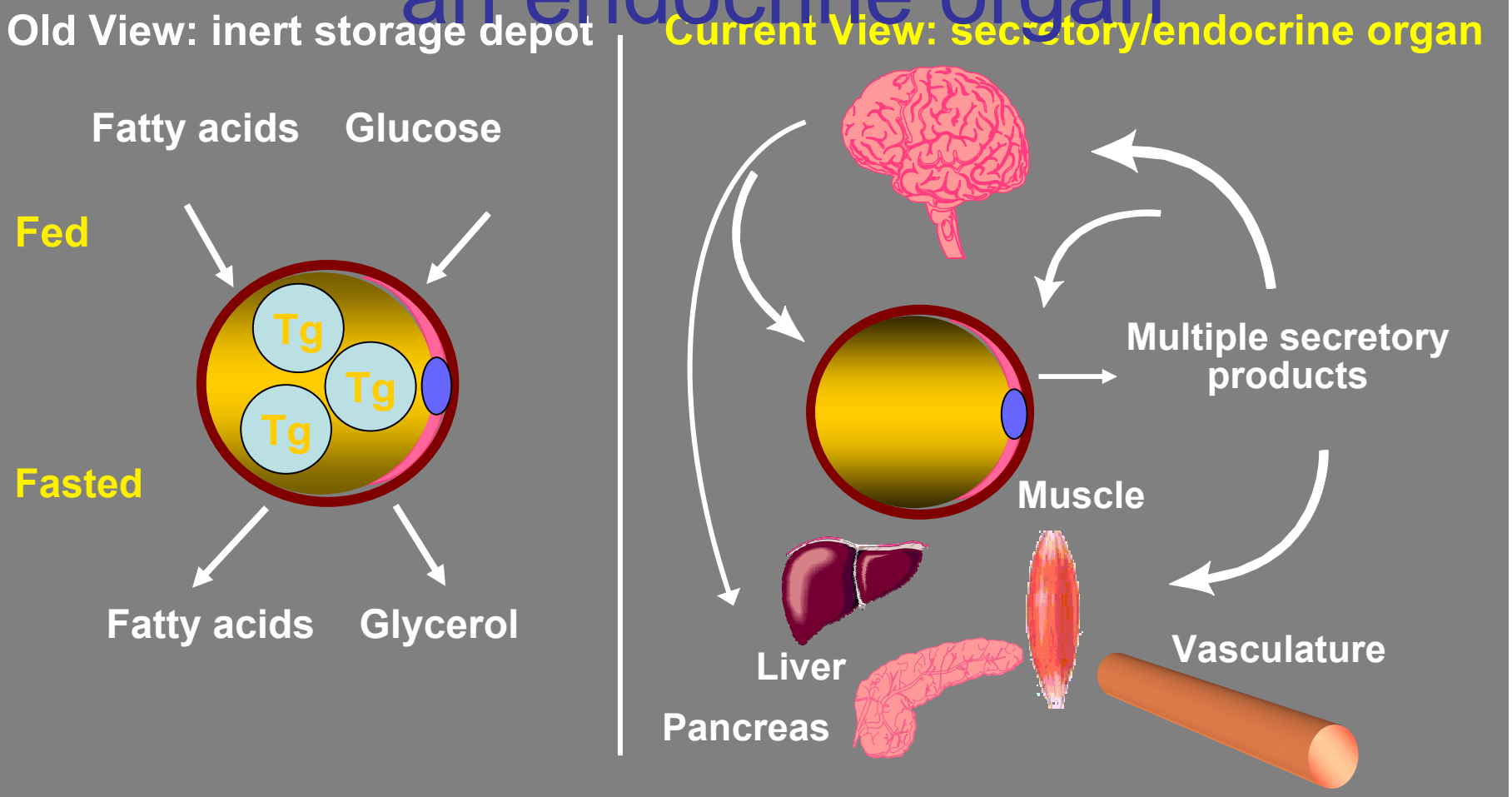
Voce di costo	Percentuale
Ospedalizzazioni	64%
Diagnostica	12%
Farmaci	7%
Visite	6%
Altro	11%

Nel 2025 costo Tot (mld) da 11 a 15,7 (+43%)
con obesità infantile + 205%

Elaborazione Centro Studi Ricerca Obesità (CSRO) e Farmacoeconomia UniMI

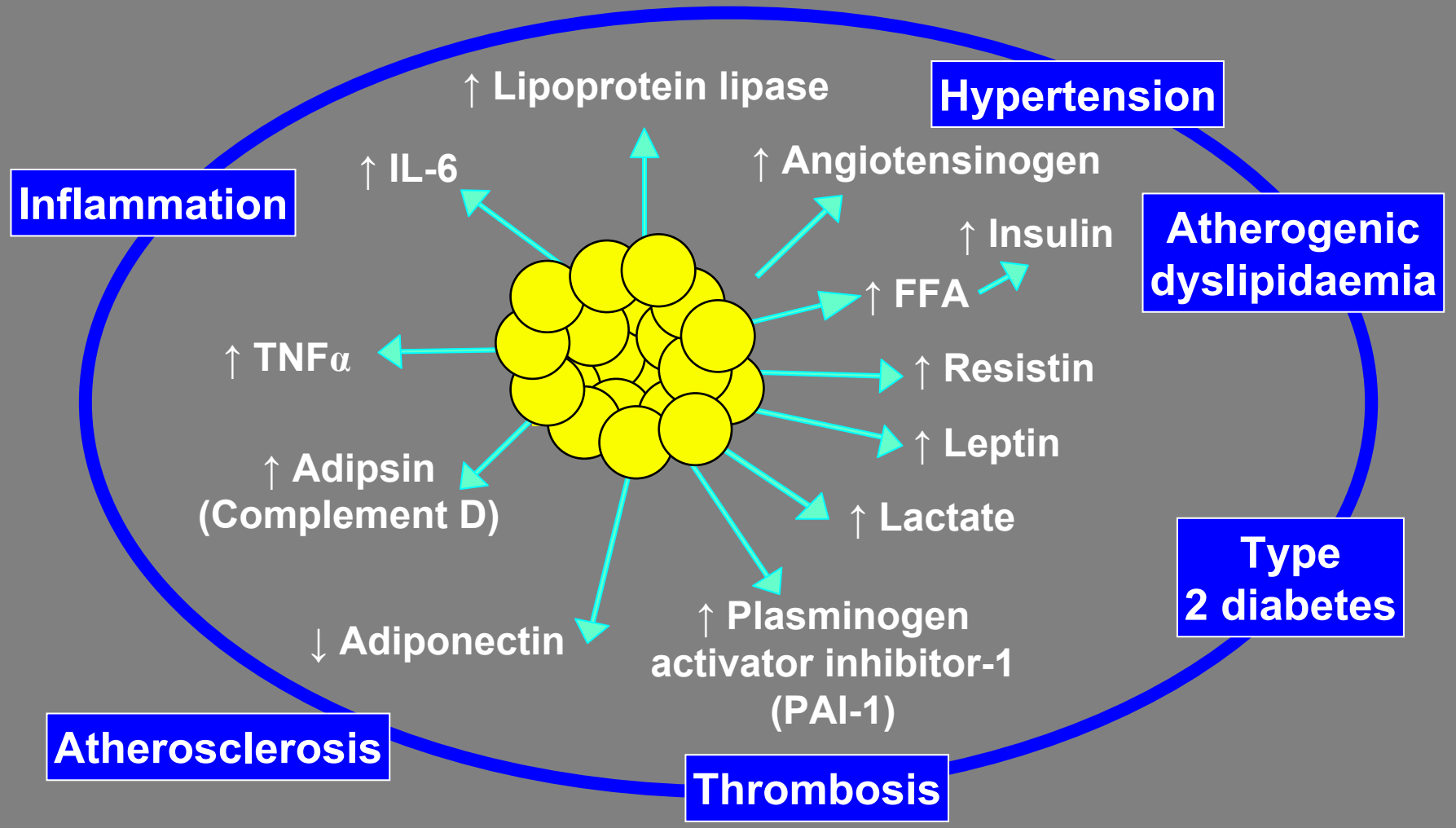
Centro Studio e Ricerca sull'Obesità (C.S.R.O.)
Dipartimento di Farmacologia, Chemioterapia e Tossicologia Medica,
Università degli Studi di Milano, Via Vanvitelli 32, Milano
Prof. **Michele Carruba**

The evolving view of adipose tissue: an endocrine organ



Lyon CJ et al 2003

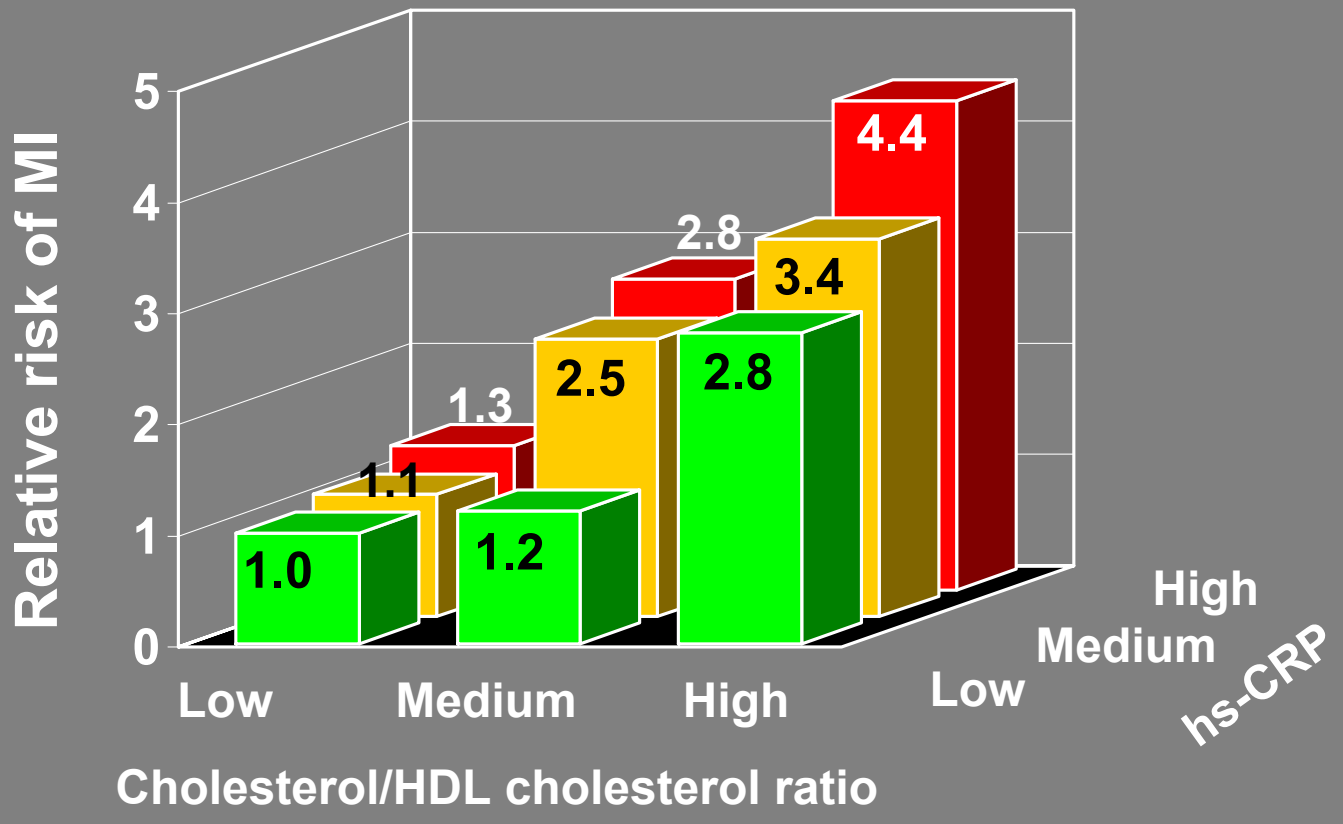
Adverse cardiometabolic effects of products of adipocytes



Lyon 2003; Trayhurn et al 2004; Eckel et al 2005

Systemic inflammation and adverse cardiovascular outcomes

Physicians' Health Study: 9-year follow-up



Ridker et al 1998

TNF-α downregulates eNOS expression and mitochondrial biogenesis in fat and muscle of obese rodents

Alessandra Valerio,[1] Annalisa Cardile,[1,2] Valeria Cozzi,[1,2] Renata Bracale,[1,2,3] Laura Tedesco,[1,2,4] Addolorata Pisconti,[2,5] Letizia Palomba,[6] Orazio Cantoni,[6] Emilio Clementi,[2,5,7] Salvador Moncada,[8] Michele O. Carruba,[1,4] and Enzo Nisoli[1,4]

[1]Integrated Laboratories Network, Center for Study and Research on Obesity, Department of Pharmacology, School of Medicine, University of Milan, Milan, Italy. [2]Department of Preclinical Sciences, University of Milan, Milan, Italy. [3]CEINGE Biotecnologie Avanzate, Naples, Italy. [4]Istituto Auxologico Italiano, Milan, Italy. [5]Stem Cell Research Institute, San Raffaele Scientific Institute, Milan, Italy. [6]Istituto di Farmacologia e Farmacognosia, University of Urbino "Carlo Bo," Urbino, Italy. [7]Eugenio Medea Scientific Institute, Lecco, Italy. [8]Wolfson Institute for Biomedical Research, University College London, London, United Kingdom.

The Journal of Clinical Investigation

Mitochondrial Biogenesis in Mammals: The Role of Endogenous Nitric Oxide

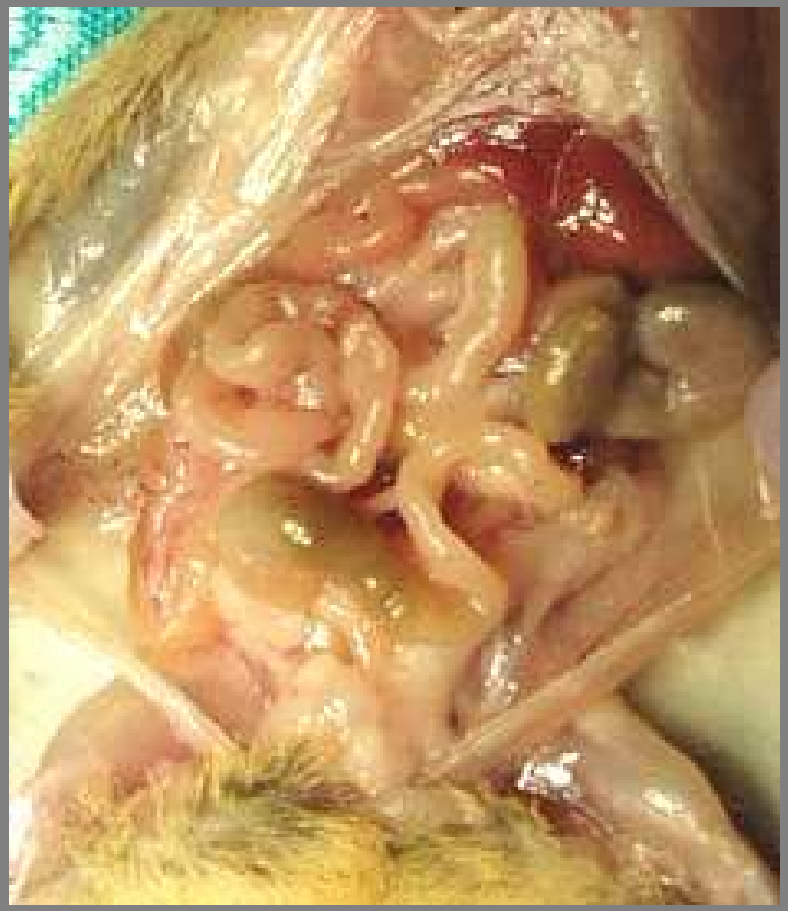
Enzo Nisoli,[1,2*†] Emilio Clementi,[3,4*] Clara Paolucci,[3] Valeria Cozzi,[1] Cristina Tonello,[1] Clara Sciorati,[3] Renata Bracale,[1] Alessandra Valerio,[5] Maura Francolini,[6] Salvador Moncada,[7] Michele O. Carruba[1,2]

Nitric oxide was found to trigger mitochondrial biogenesis in cells as diverse as brown adipocytes and 3T3-L1, U937, and HeLa cells. This effect of nitric oxide was dependent on guanosine 3′,5′-monophosphate (cGMP) and was mediated by the induction of peroxisome proliferator–activated receptor γ coactivator 1α, a master regulator of mitochondrial biogenesis. Moreover, the mitochondrial biogenesis induced by exposure to cold was markedly reduced in brown adipose tissue of endothelial nitric oxide synthase null-mutant (eNOS$^{-/-}$) mice, which had a reduced metabolic rate and accelerated weight gain as compared to wild-type mice. Thus, a nitric oxide–cGMP–dependent pathway controls mitochondrial biogenesis and body energy balance.

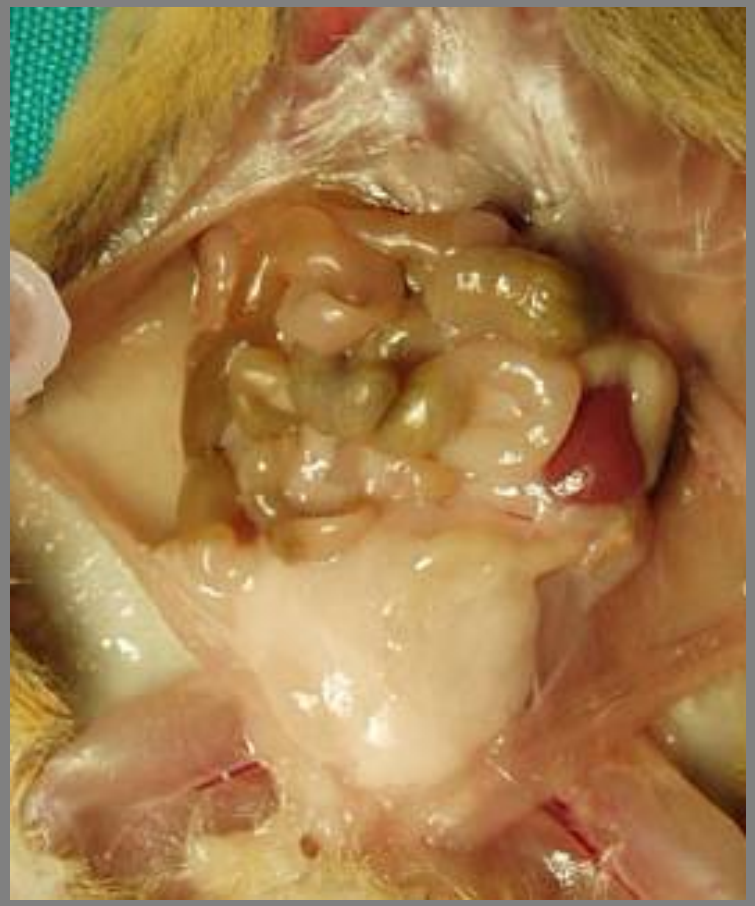
Science 299: 896-899, 2003

Visceral fat depot in eNOS$^{-/-}$ *vs.* wild-type mice

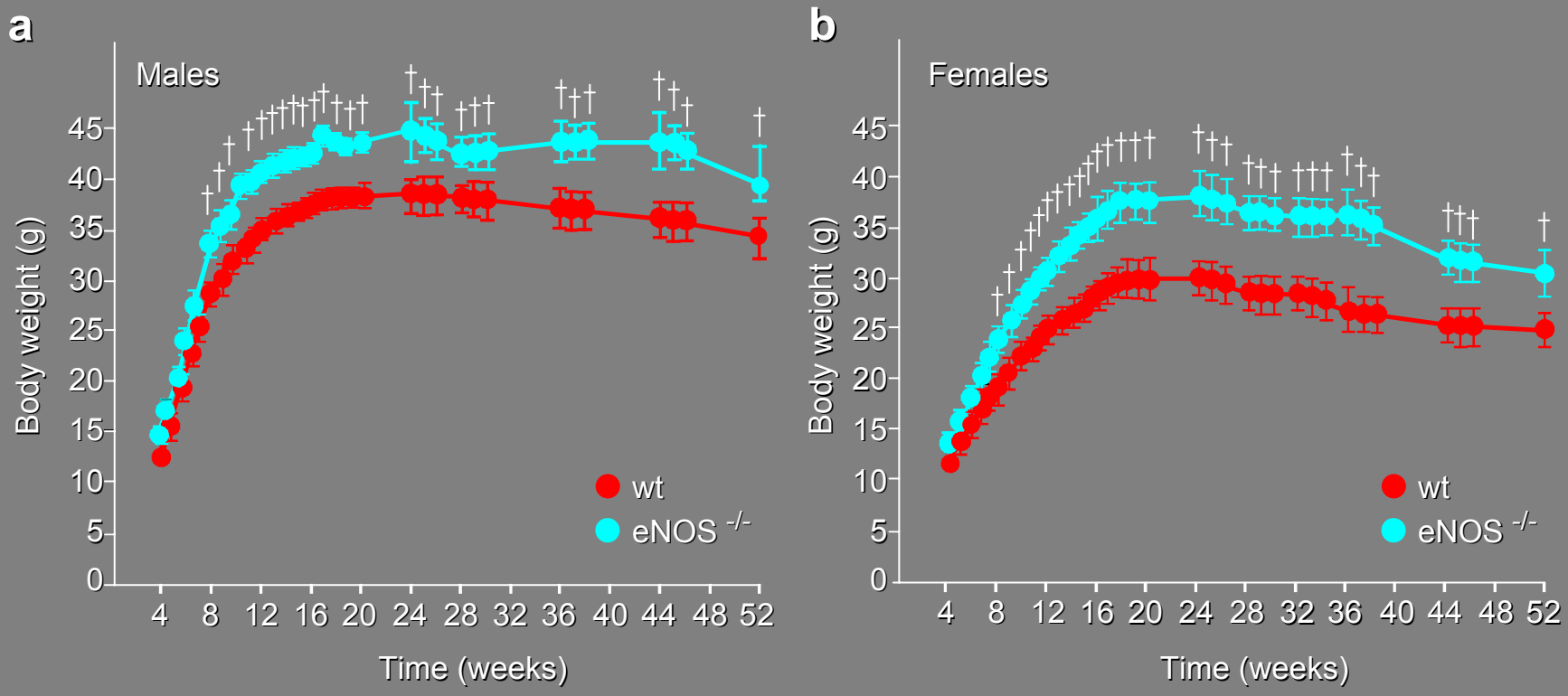
wt



eNOS$^{-/-}$



Growth curves of wild-type and eNOS$^{-/-}$ mice



Nisoli et al., Science 299, 896–899, 2003

Clustering of cardiovascular risk factors mimicking the human metabolic syndrome X in eNOS null mice

Stéphane Cook[a], *Olivier Hugli*[a], *Marc Egli*[a], *Peter Vollenweider*[a], *Rémy Burcelin*[b], *Pascal Nicod*[a], *Bernard Thorens*[b], *Urs Scherrer*[a]

[a] Department of Internal Medicine, and the Botnar Centre for Clinical Research, Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland
[b] Institute of Pharmacology and Toxicology, University of Lausanne, Lausanne, Switzerland

Summary

Aims/hypothesis: The metabolic syndrome comprises a clustering of cardiovascular risk factors but the underlying mechanism is not known. Mice with targeted disruption of endothelial nitric oxide synthase (eNOS) are hypertensive and insulin resistant. We wondered, whether eNOS deficiency in mice is associated with a phenotype mimicking the human metabolic syndrome.

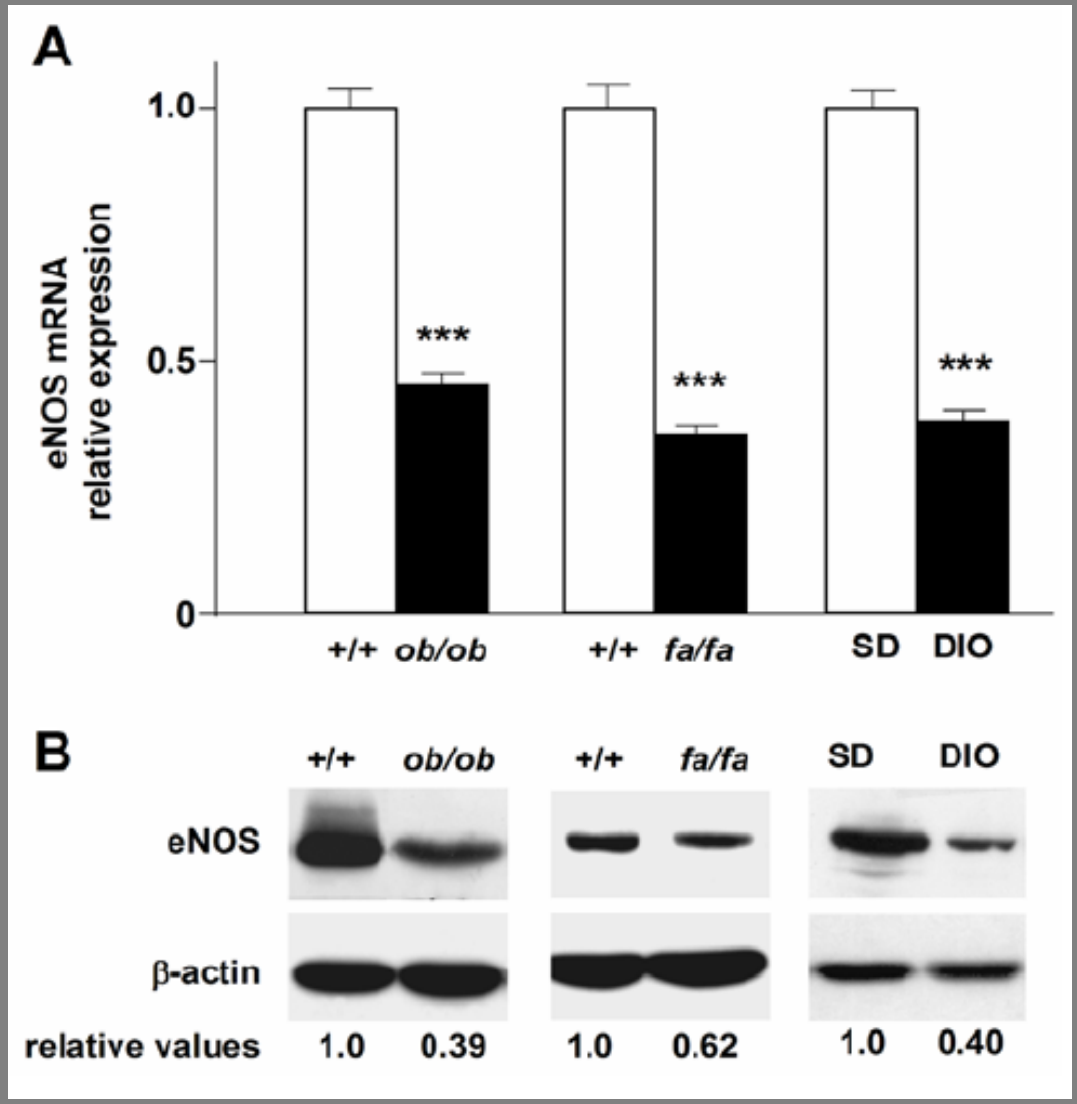
Methods and Results: In addition to arterial pressure and insulin sensitivity (euglycaemic hyperinsulinaemic clamp), we measured the plasma concentration of leptin, insulin, cholesterol, triglycerides, free fatty acids, fibrinogen and uric acid in 10 to 12 week old $eNOS^{-/-}$ and wild type mice. We also assessed glucose tolerance under basal conditions and following a metabolic stress with a high fat diet. As expected $eNOS^{-/-}$ mice were hypertensive and insulin resistant, as evidenced by fasting hyperinsulinaemia and a roughly 30 percent lower steady state glucose infusion rate during the clamp. $eNOS^{-/-}$ mice had a 1.5 to 2-fold elevation of the cholesterol, triglyceride and free fatty acid plasma concentration. Even though body weight was comparable, the leptin plasma level was 30% higher in $eNOS^{-/-}$ than in wild type mice. Finally, uric acid and fibrinogen were elevated in the $eNOS^{-/-}$ mice. Whereas under basal conditions, glucose tolerance was comparable in knock out and control mice, on a high fat diet, knock out mice became significantly more glucose intolerant than control mice.

Conclusions: A single gene defect, eNOS deficiency, causes a clustering of cardiovascular risk factors in young mice. We speculate that defective nitric oxide synthesis could trigger many of the abnormalities making up the metabolic syndrome in humans.

Key words: endothelial nitric oxide synthase; metabolic syndrome; arterial hypertension; insulin resistance; hyperlipidaemia; glucose intolerance

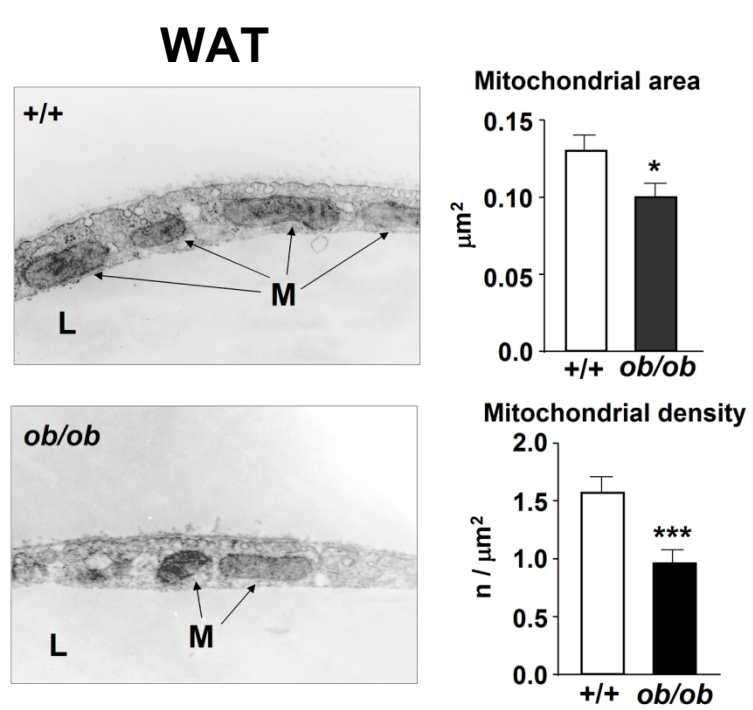
SWISS MED WKLY 133: 360–363, 2003

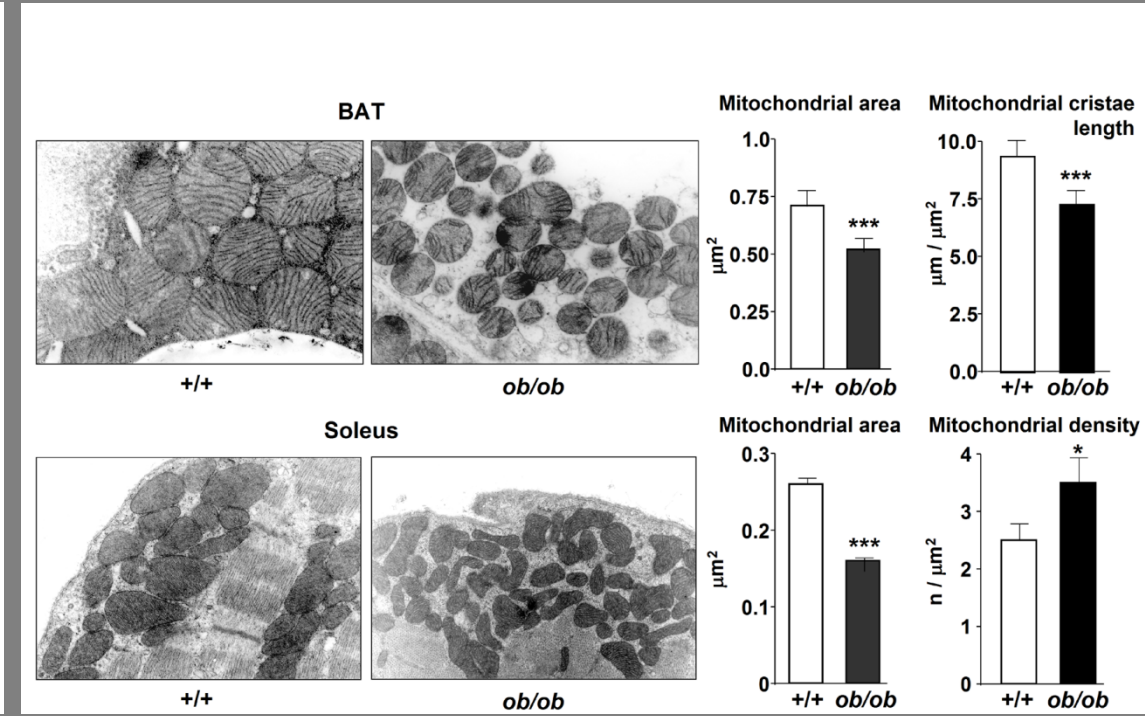
eNOS expression is reduced in WAT of obese rodents



Valerio et al., J. Clin. Invest., Oct. 2006

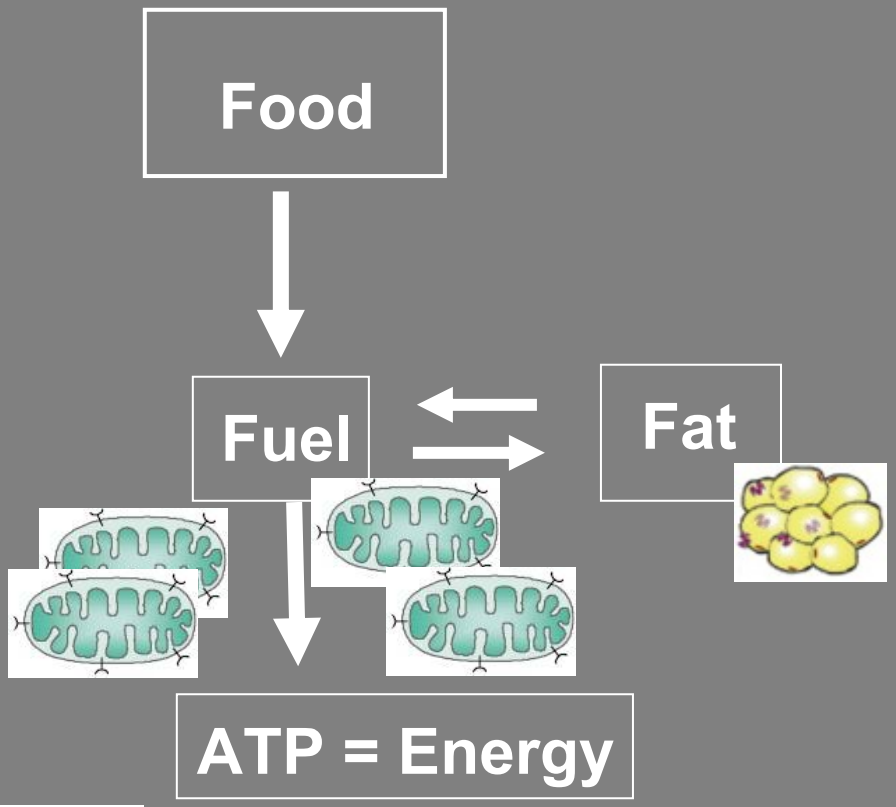
Electron microscopy analysis of WAT, BAT and muscle in *ob/ob* mice

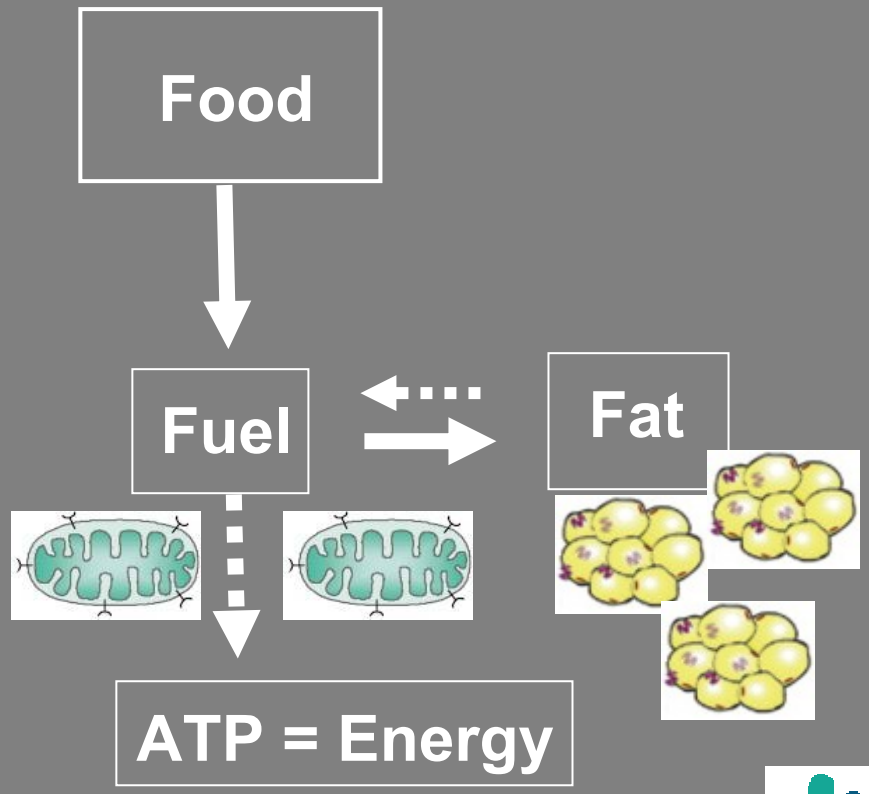




Valerio et al., J. Clin. Invest., Oct. 2006

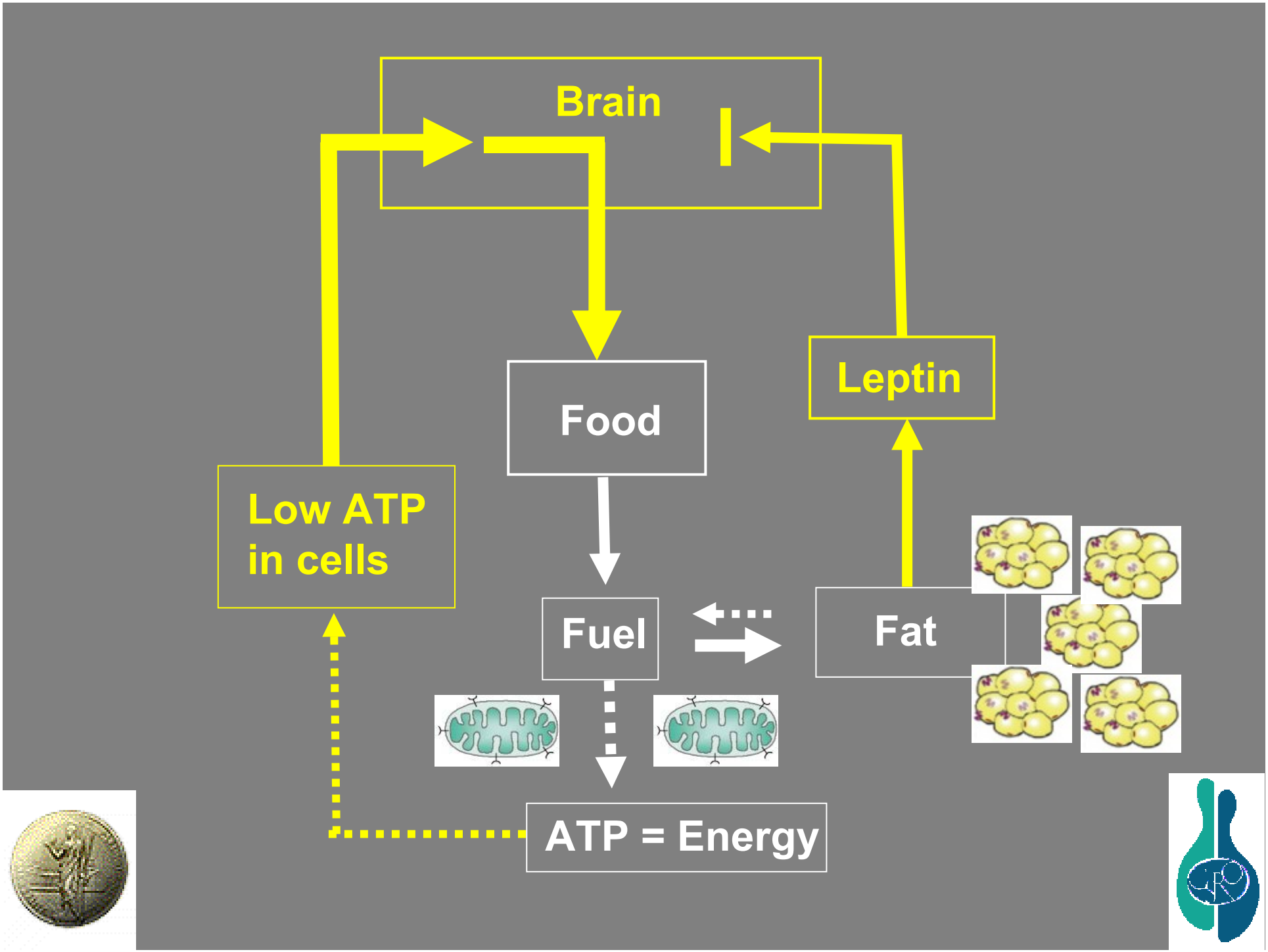
Decreased energy levels can cause and sustain obesity





Healthy subject

Obese subject


Brain
Food
Leptin
Low ATP
in cells
Fuel
Fat
ATP = Energy

Calorie Restriction Promotes Mitochondrial Biogenesis by Inducing the Expression of eNOS

Enzo Nisoli,[1,2*] Cristina Tonello,[1] Annalisa Cardile,[1] Valeria Cozzi,[1] Renata Bracale,[1] Laura Tedesco,[1] Sestina Falcone,[1,3] Alessandra Valerio,[1] Orazio Cantoni,[4] Emilio Clementi,[1,3,5] Salvador Moncada,[6] Michele O. Carruba[1,2]

Calorie restriction extends life span in organisms ranging from yeast to mammals. Here, we report that calorie restriction for either 3 or 12 months induced endothelial nitric oxide synthase (eNOS) expression and 3′,5′-cyclic guanosine monophosphate formation in various tissues of male mice. This was accompanied by mitochondrial biogenesis, with increased oxygen consumption and adenosine triphosphate production, and an enhanced expression of sirtuin 1. These effects were strongly attenuated in eNOS null-mutant mice. Thus, nitric oxide plays a fundamental role in the processes induced by calorie restriction and may be involved in the extension of life span in mammals.

Sience October 14, 2005

Quindi? Stile di vita!

Abitudini alimentari

e

Attività fisica

Adherence to a Mediterranean Diet and Survival in a Greek Population

Antonia Trichopoulou, M.D., Tina Costacou, Ph.D., Christina Bamia, Ph.D., and Dimitrios Trichopoulos, M.D.

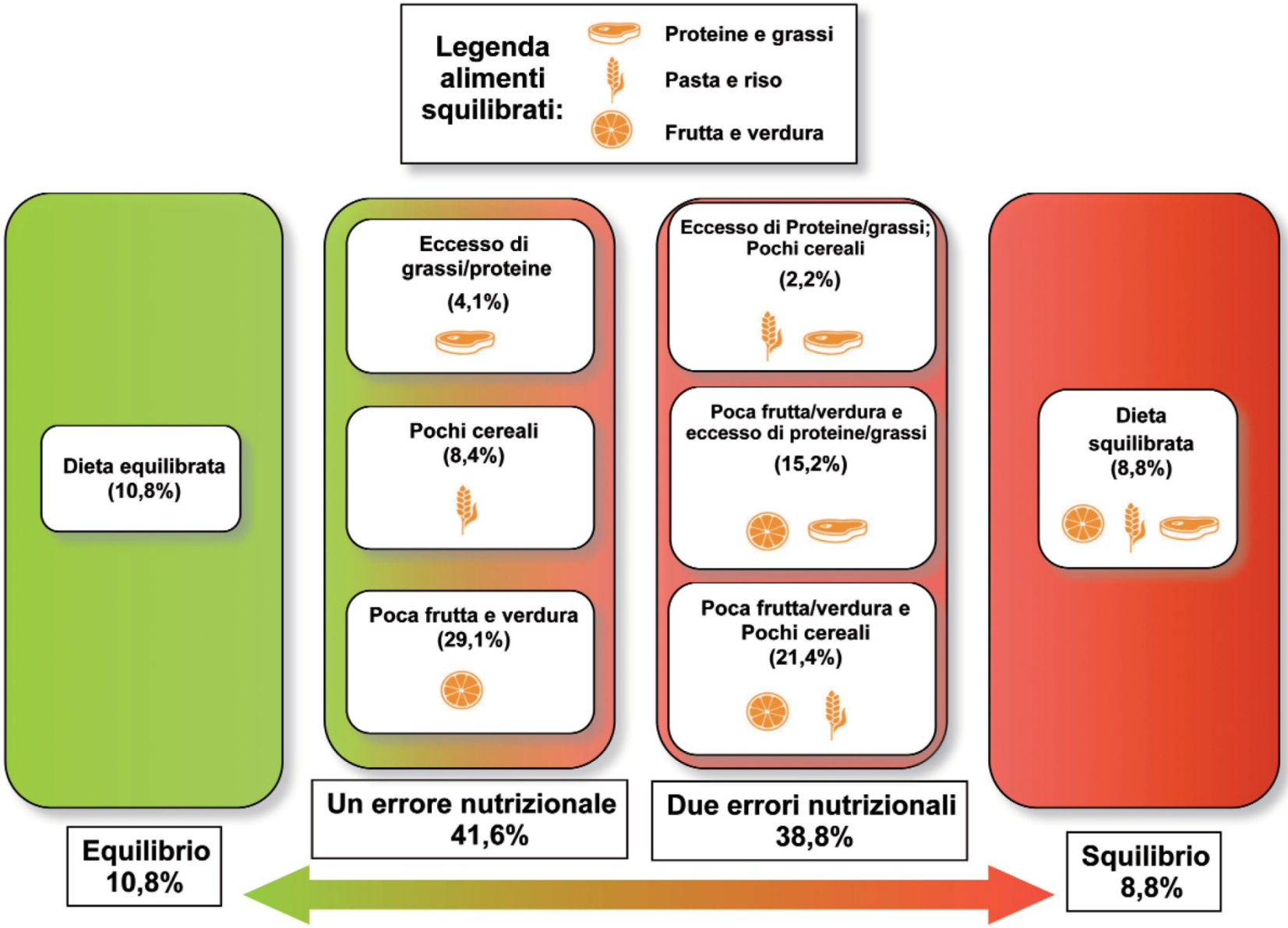
We conducted a population-based, prospective investigation involving 22,043 adults in Greece who completed an extensive, validated, food-frequency questionnaire at base line.

During a median of 44 months of follow-up, there were 275 deaths. A higher degree of adherence to the Mediterranean diet was associated with a reduction in total mortality.

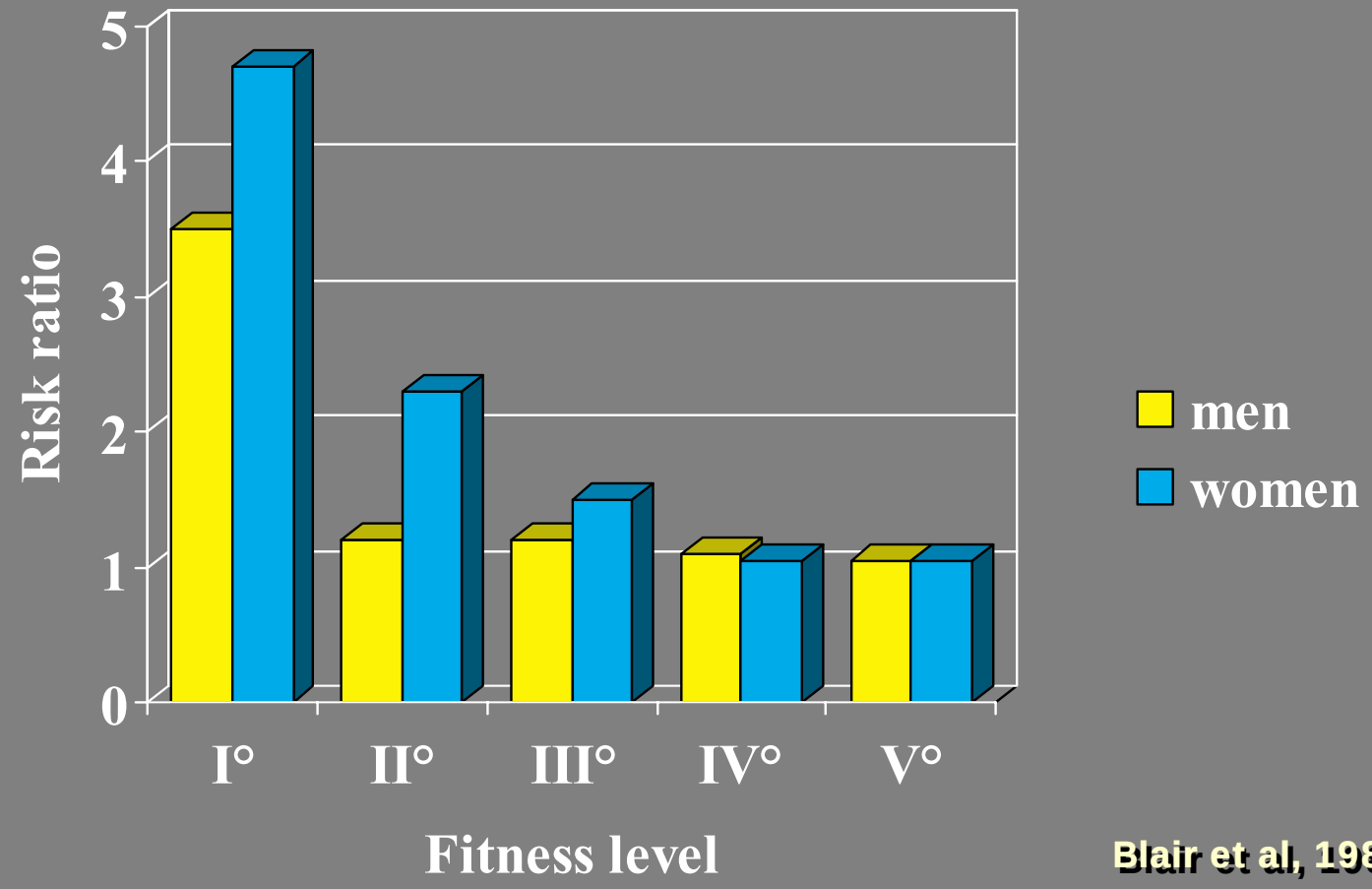
An inverse association with greater adherence to this diet was evident for both death due to coronary heart disease and death due to cancer.

Conclusions. Greater adherence to the traditional Mediterranean diet is associated with a significant reduction in total mortality.

The New England Journal of Medicine, 348: 2599-2608, 2003

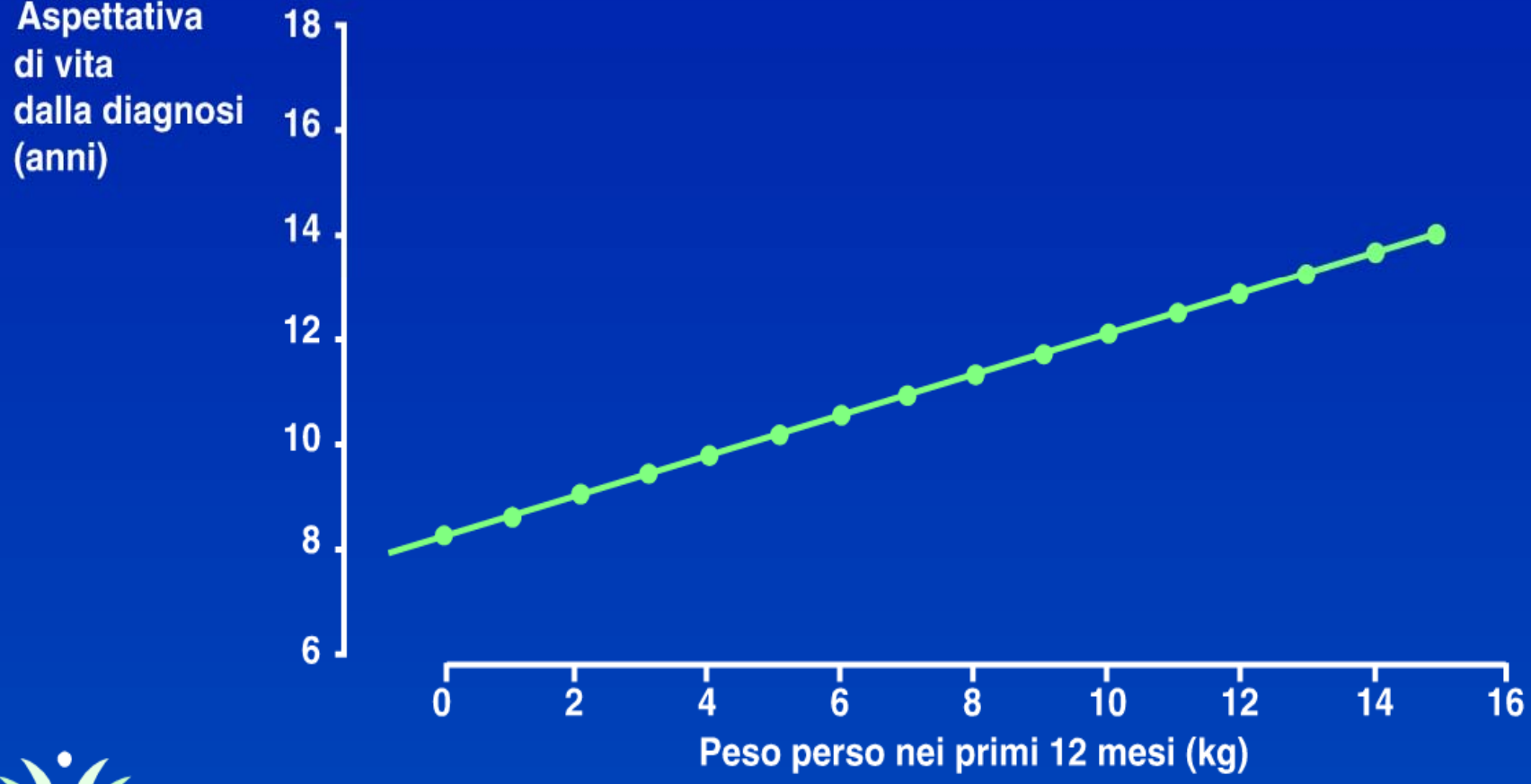

Legenda alimenti squilibrati:
Proteine e grassi
Pasta e riso
Frutta e verdura
Dieta equilibrata (10,8%)
Eccesso di grassi/proteine (4,1%)
Pochi cereali (8,4%)
Poca frutta e verdura (29,1%)
Eccesso di Proteine/grassi; Pochi cereali (2,2%)
Poca frutta/verdura e eccesso di proteine/grassi (15,2%)
Poca frutta/verdura e Pochi cereali (21,4%)
Dieta squilibrata (8,8%)
Equilibrio 10,8%
Un errore nutrizionale 41,6%
Due errori nutrizionali 38,8%
Squilibrio 8,8%

Livello di attività fisica e rischio di mortalità



Blair et al, 1989

Aspettativa di vita in pazienti obesi con diabete di tipo 2 (BMI >25 kg/m^2) in funzione della perdita di peso



Adattato con il permesso da Lean et al. Diabet Med 1990; 7: 228–33

Integrated Laboratories Network (InLaNe)

Center for Study and Research on Obesity, University of Milan

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Enzo Nisoli, M.D. Ph.D.
Cristina Tonello, Ph.D.
Valeria Cozzi, Ph.D.
Laura Tedesco, Ph.D.
Annalisa Cardile, Ph.D. student
Renata Bracale, Ph.D. Student

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Alessandra Valerio, M.D. Ph.D.
Marta Dossena, Ph.D. student

The Metabolic Syndrome: Time for a Critical Appraisal

Joint statement from the American Diabetes Association and the European Association for the Study of Diabetes

RICHARD KAHN, PHD[1]
JOHN BUSE, MD, PHD[2]
ELE FERRANNINI, MD[3]
MICHAEL STERN, MD[4]

Diabetes Care 28: 2289-2304, 2005

Table 3—*Summary of concerns regarding the metabolic syndrome*

1) Criteria are ambiguous or incomplete. Rationale for thresholds are ill defined.
2) Value of including diabetes in the definition is questionable.
3) Insulin resistance as the unifying etiology is uncertain.
4) No clear basis for including/excluding other CVD risk factors.
5) CVD risk value is variable and dependent on the specific risk factors present.
6) The CVD risk associated with the "syndrome" appears to be no greater than the sum of its parts.
7) Treatment of the syndrome is no different than the treatment for each of its components.
8) The medical value of diagnosing the syndrome is unclear.

Diabetes Care 28: 2289-2304, 2005

Properties of key adipokines

Adiponectin ↓ in IAA	**Anti-atherogenic/antidiabetic:** ↓ foam cells ↓ vascular remodelling ↑ insulin sensitivity ↓ hepatic glucose output
IL-6 ↑ in IAA	**Pro-atherogenic/pro-diabetic:** ↑ vascular inflammation ↓ insulin signalling
TNFα ↑ in IAA	**Pro-atherogenic/pro-diabetic:** ↓ insulin sensitivity in adipocytes (paracrine)
PAI-1 ↑ in IAA	**Pro-atherogenic:** ↑ atherothrombotic risk

IAA: intra-abdominal adiposity

Marette 2002

Overweight, Obesity, and Mortality from Cancer in a Prospectively Studied Cohort of U.S. Adults

Eugenia E. Calle, Ph.D., Carmen Rodriguez, M.D., M.P.H., Kimberly Walker-Thurmond, B.A., and Michael J. Thun, M.D.

The heaviest members of this cohort (body-mass index of at least 40) had death rates from all cancers combined that were 52 percent higher (for men) and 62 percent higher (for women) than the rates in men and women of normal weight.

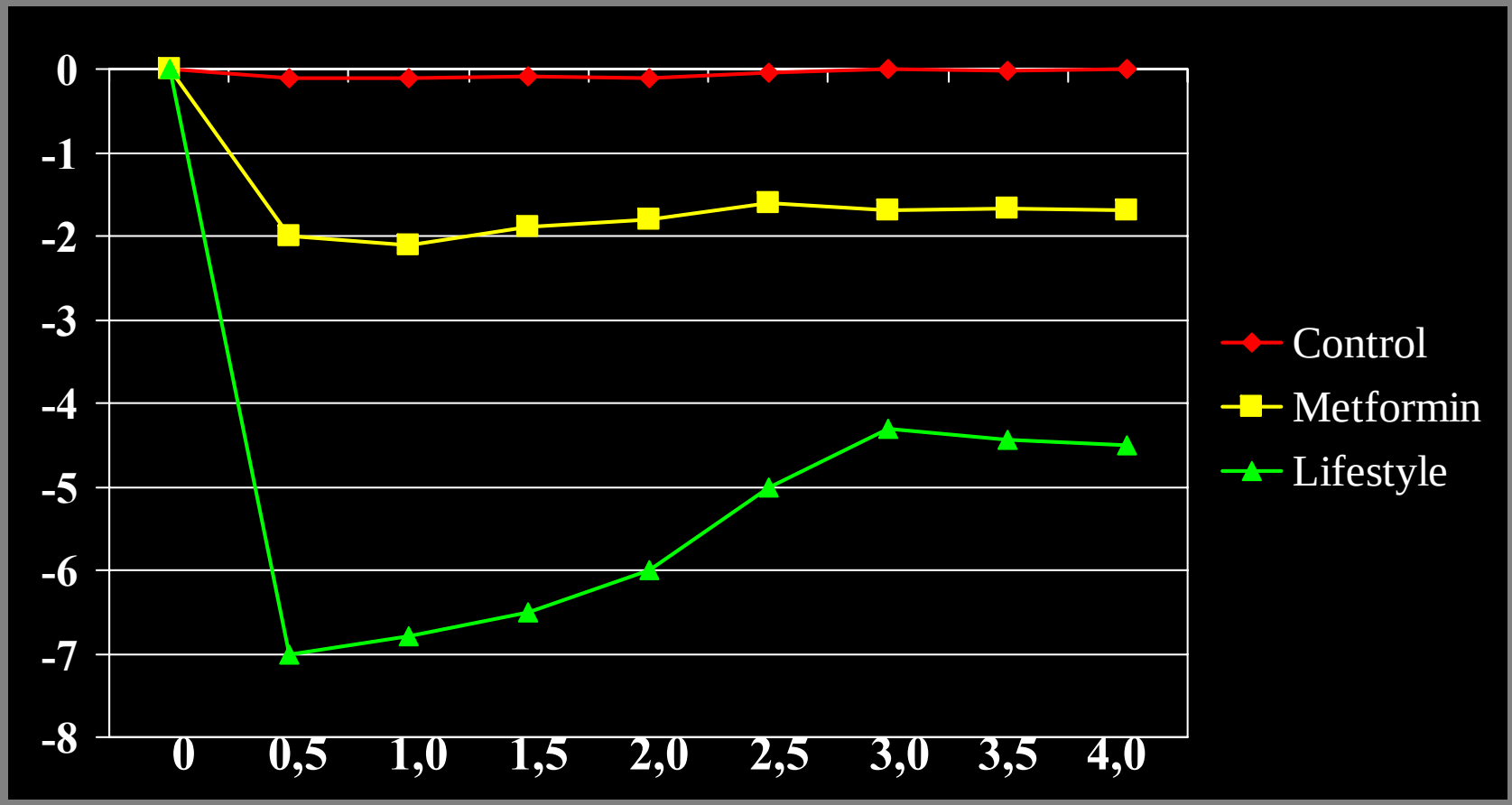
In both men and women, body-mass index was also significantly associated with higher rates of death due to cancer of the esophagus, colon and rectum, liver, gallbladder, pancreas, and kidney; the same was true for death due to non-Hodgkin's lymphoma and multiple myeloma. Significant trends of increasing risk with higher body-mass-index values were observed for death from cancers of the stomach and prostate in men and for death from cancers of the breast, uterus, cervix, and ovary in women.

Conclusions. Increased body weight was associated with increased death rates for all cancers combined and for cancers at multiple specific sites.

The New England Journal of Medicine, 348: 1625-1638, 2003

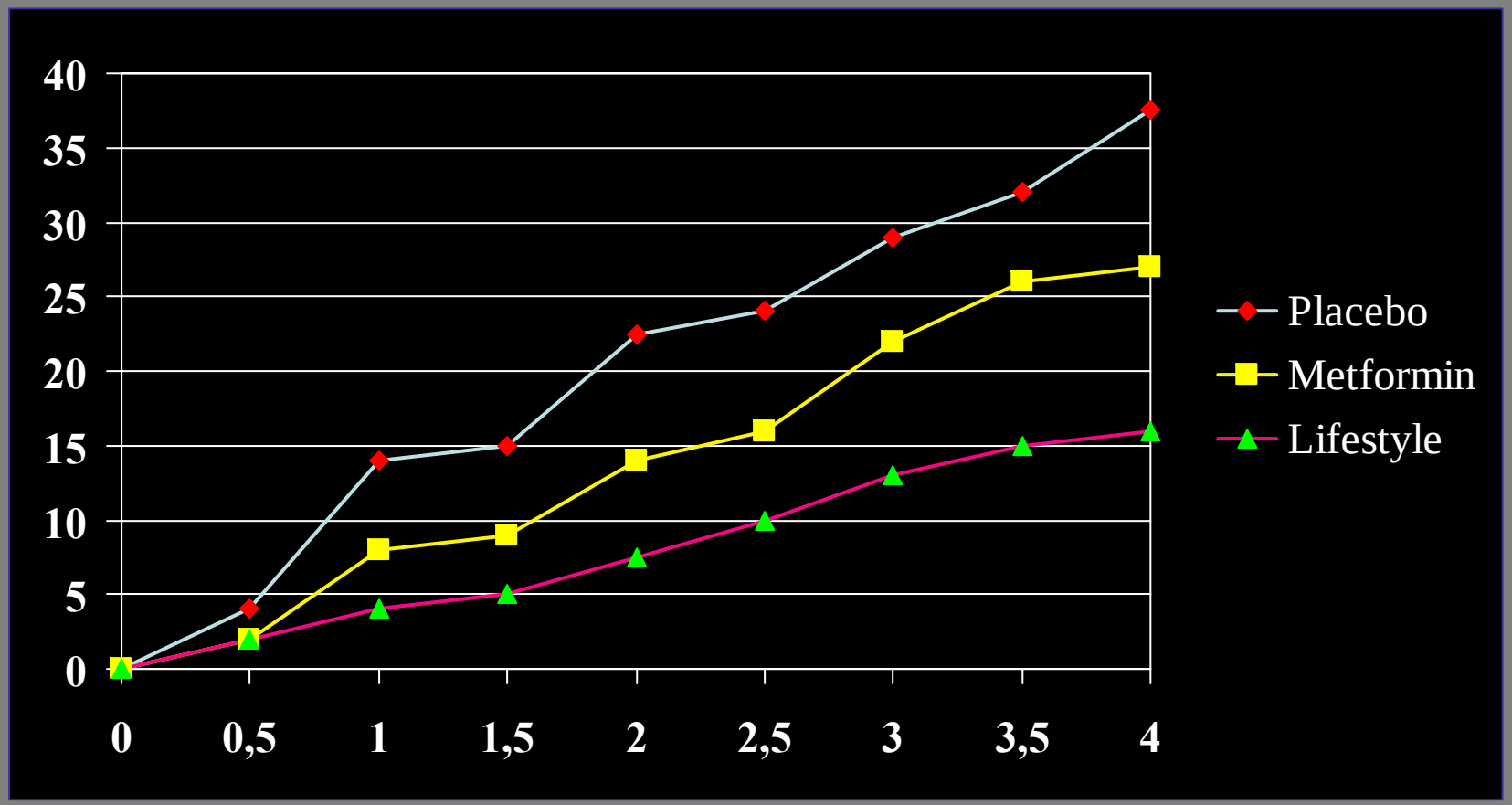
Changes in Body Weight (Kg)

(from, DPPRG, 2002)



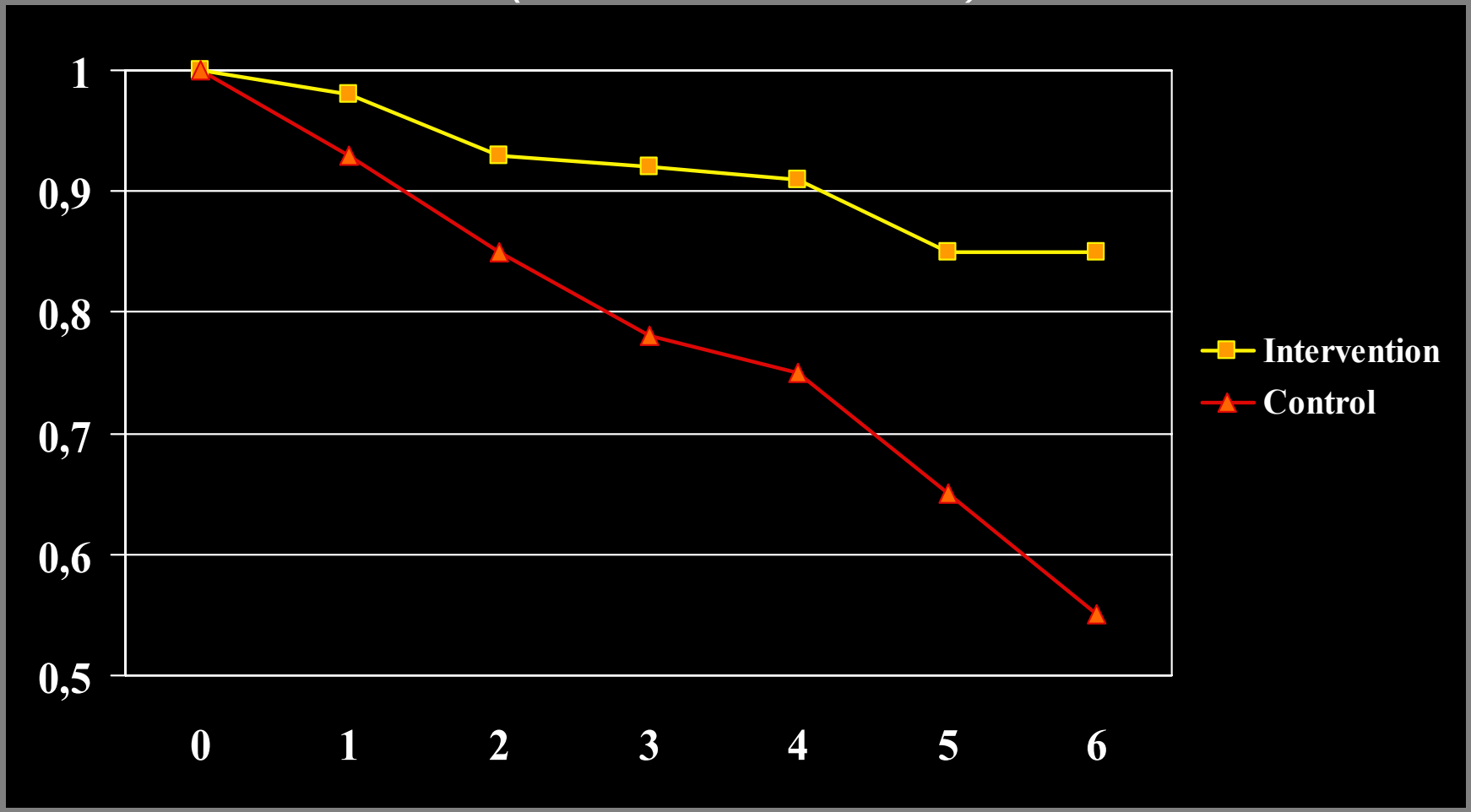
Cumulative incidence of diabetes

(from, DPPRG, 2002)



Probabilty of remaining free of diabetes

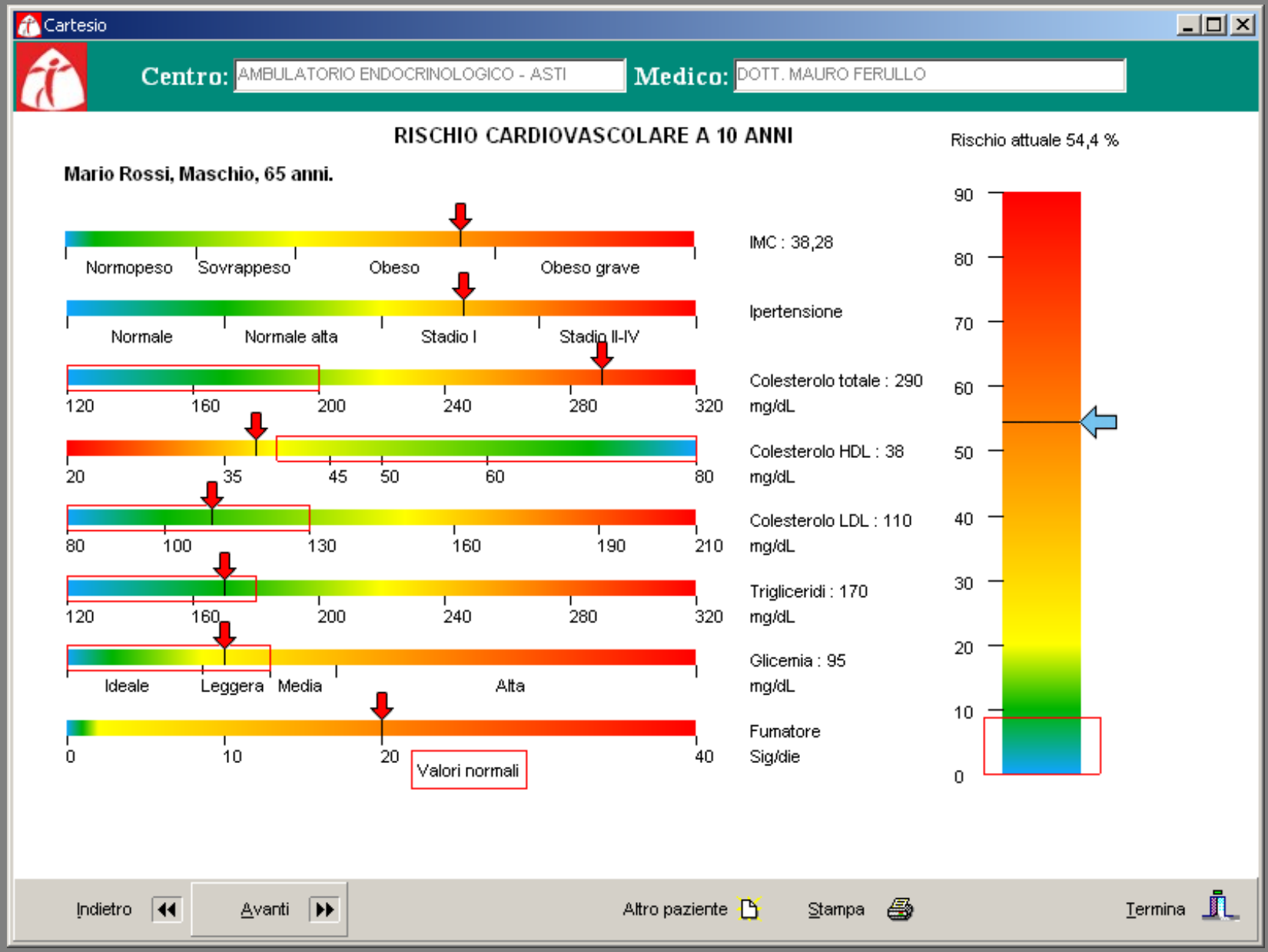
(Tuomilehto et al, 2001)



DALLA RICERCA AL TERRITORIO

Il Rischio

- **Ogni anno 270.000 soggetti vengono colpiti da un attacco cardiaco che è fatale nel 50% dei casi e non arriva neanche al ricovero nel 30% dei casi**
- **Nell'analisi Framingham Study si è rivoluzionato il concetto di rischio: non più singoli fattori (fumo, ipertensione, ipercolesterolemia), ma una valutazione del Rischio Assoluto derivante da un'analisi multifattoriale**


Cartesio
Centro: AMBULATORIO ENDOCRINOLOGICO - ASTI
Medico: DOTT. MAURO FERULLO
RISCHIO CARDIOVASCOLARE A 10 ANNI
Rischio attuale 54,4 %
Mario Rossi, Maschio, 65 anni.
Normopeso
Sovrappeso
Obeso
Obeso grave
IMC : 38,28
Normale
Normale alta
Stadio I
Stadio II-IV
Ipertensione
120
160
200
240
280
320
Colesterolo totale : 290 mg/dL
20
35
45
50
60
80
Colesterolo HDL : 38 mg/dL
80
100
130
160
190
210
Colesterolo LDL : 110 mg/dL
Trigliceridi : 170 mg/dL
Ideale
Leggera
Media
Alta
Glicemia : 95 mg/dL
0
10
20
40
Valori normali
Fumatore Sig/die
90
80
70
60
50
40
30
20
10
0
Indietro
Avanti
Altro paziente
Stampa
Termina

Cartesio

Centro: AMBULATORIO ENDOCRINOLOGICO - ASTI **Medico:** DOTT. MAURO FERULLO

MODIFICA E RICALCOLA IL RISCHIO CARDIOVASCOLARE A 10 ANNI

Mario Rossi, Maschio, 65 anni.

Peso : 88

IMC : 34,38

Normopeso Sovrappeso Obeso Obeso grave

PAS : 123

PAD : 95

Ipertensione

Normale Normale alta Stadio I Stadio II-IV

Colesterolo totale : 226

120 160 200 240 280 320 mg/dL

Fumo: Sì No

Diabete: Sì No

Legenda: fattori che determinano la riduzione del rischio

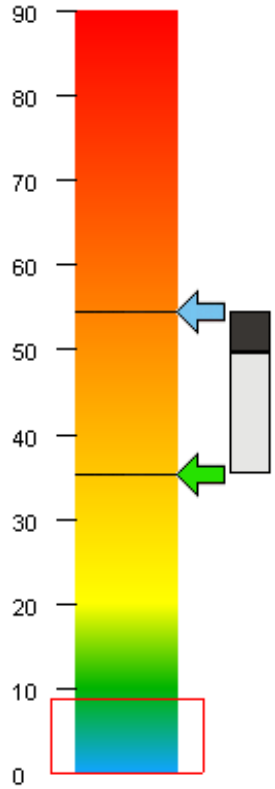
Obesità Fumo Diabete

Ipertensione Colesterolo

Valori normali

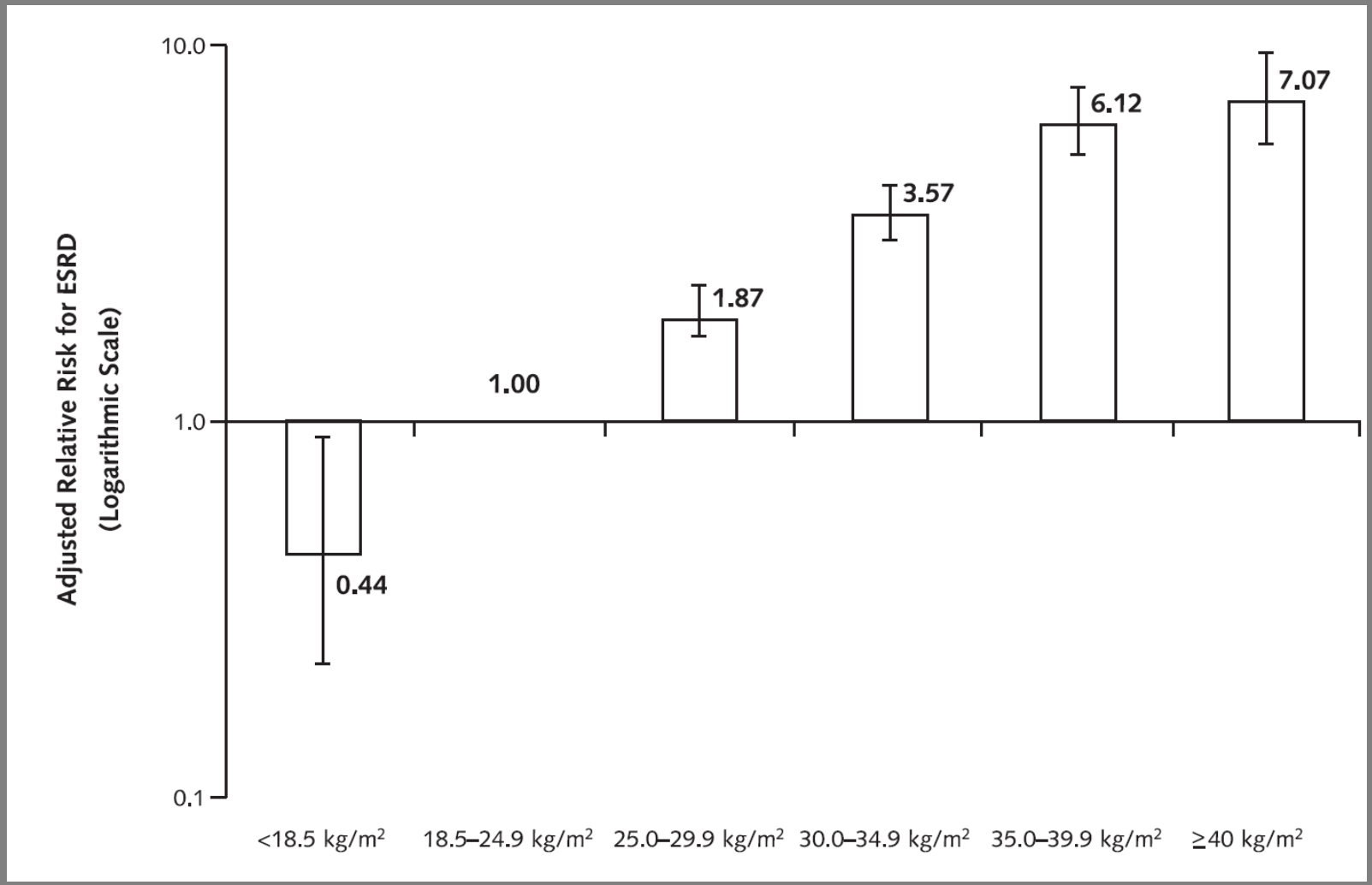
Rischio attuale 54,4 %

Rischio calcolato 35,3 %



Indietro Avanti Altro paziente Stampa Ripristina Termina

Adjusted relative risk for end-stage renal disease (ESRD) by body mass index (BMI)



Ann Intern Med. 144: 21-28, 2006

NCEP ATP III: identificazione della Sindrome Metabolica

Diagnosi posta in base alla presenza di 3 o più dei seguenti

Fattori di rischio	Valore
Obesità viscerale (circonferenza vita †)	
Uomini	>102 cm (>40 pollici)
Donne	>88 cm (>35 pollici)
Trigliceridi	≥150 mg/dL
Colesterolo HDL	
Uomini	<40 mg/dL
Donne	<50 mg/dL
Pressione arteriosa	≥130 / ≥85 mmHg
Glicemia a digiuno	≥110 mg/dL

† Alcuni pazienti maschi possono sviluppare fattori di rischio metabolici multipli anche se la circonferenza-vita è aumentata solo marginalmente (es., 94–102 cm [37–40 pollici]).

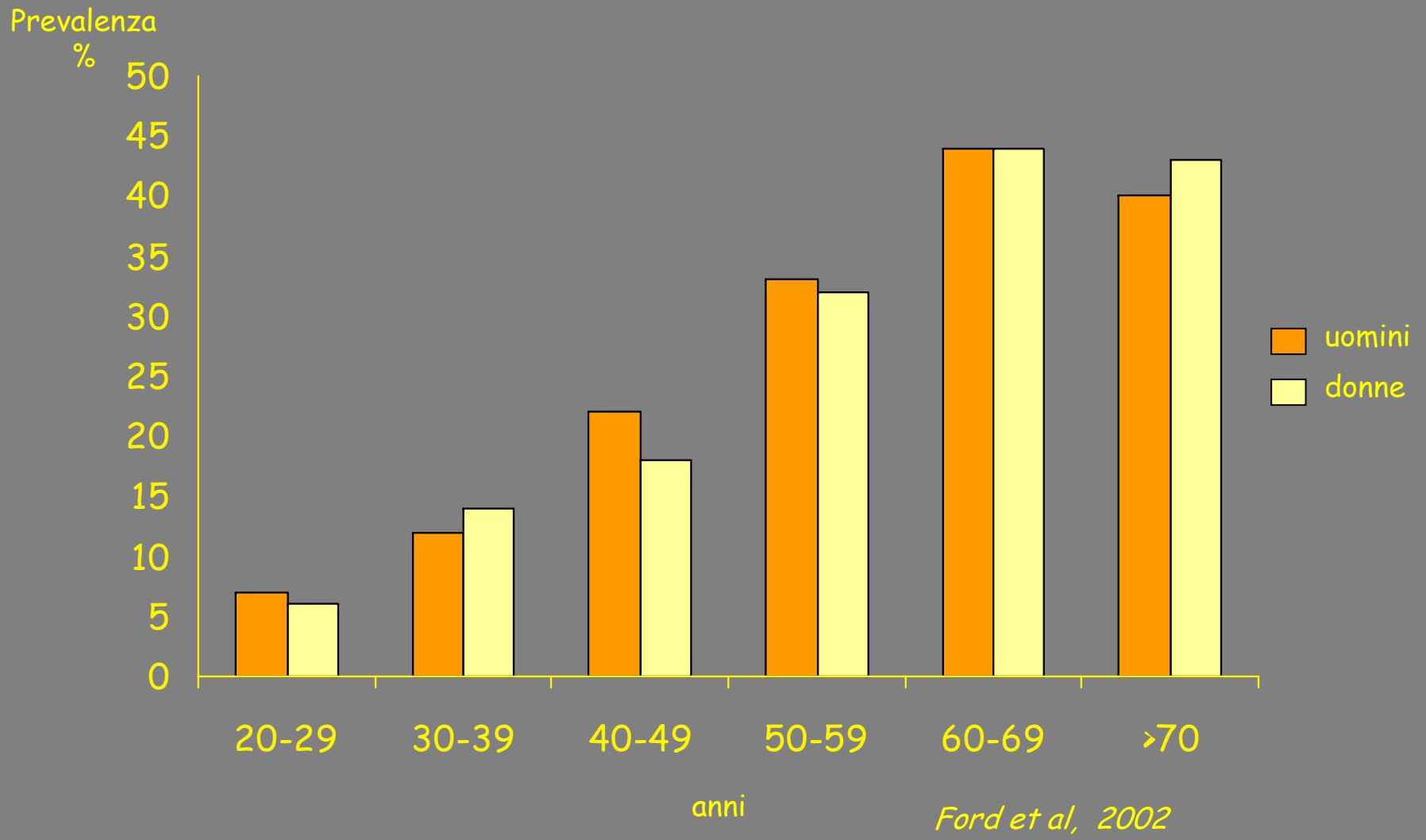
NCEP ATP III. JAMA. 2001;285(19):2486-2497

Sindrome metabolica – nuova Consensus IDF (International Diabetes federation)

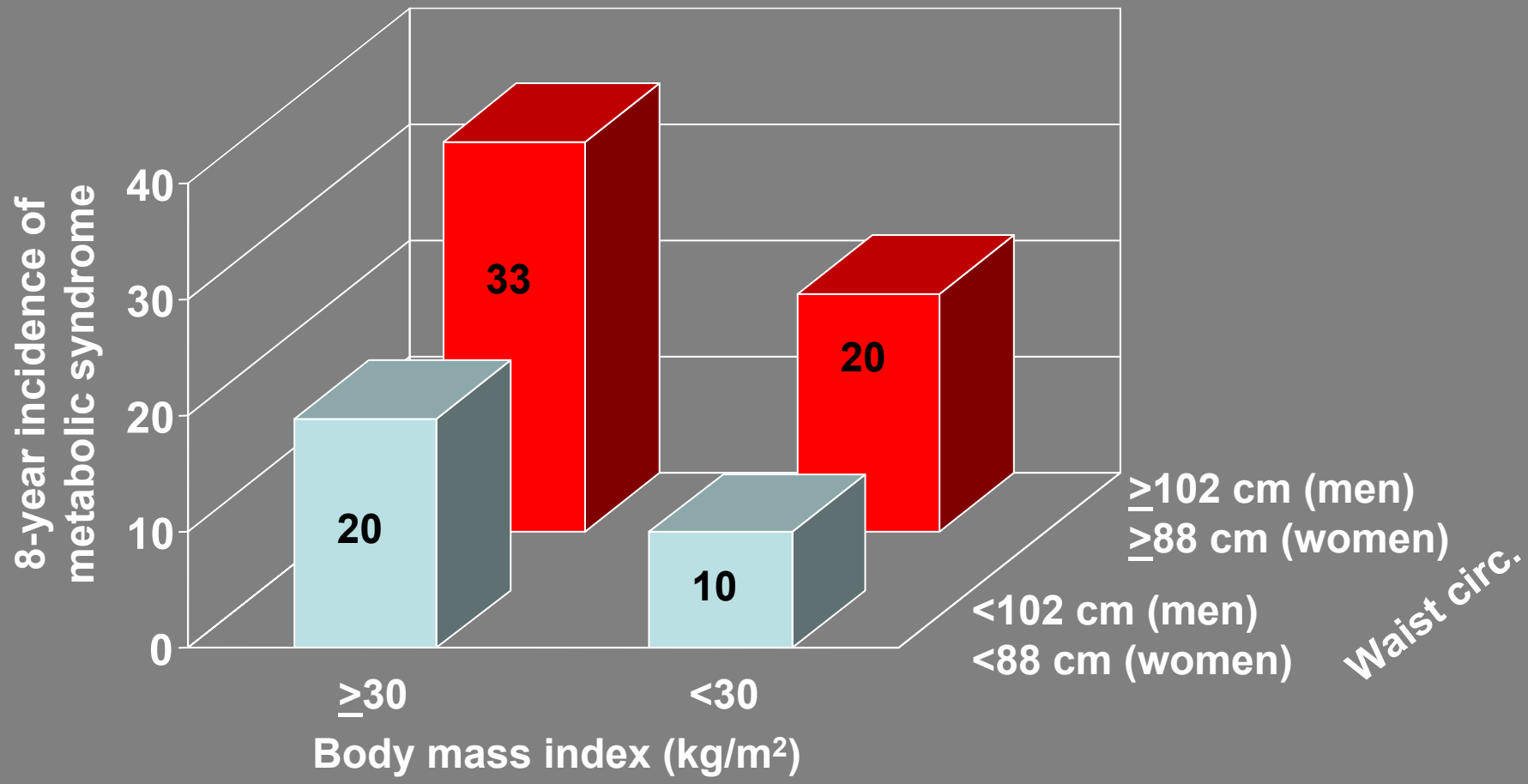
Obesità centrale (cm) Uomini Donne	 ≥94 ≥ 80
Più la presenza di altri due tra i seguenti fattori di rischio:	
Trigliceridi (mg/dL)	>150 *(o trattamento specifico)*
Colesterolo HDL (mg/dL) Uomini Donne	 <40 <50 *(o trattamento specifico)*
Pressione sistolica/diastolica (mm Hg)	≥ 130/ ≥ 85 *(o trattamento per ipertensione precedentemente diagnosticata)*
Glicemia a digiuno (mg/dL)	≥ 100 *(o precedente diagnosi di diabete di tipo 2)*

The IDF consensus worldwide definition of the metabolic syndrome, *Berlin, PMS, www.idf.org*

PREVALENZA DELLA SINDROME METABOLICA IN ADULTI AMERICANI (8814 soggetti, 1988-1994)

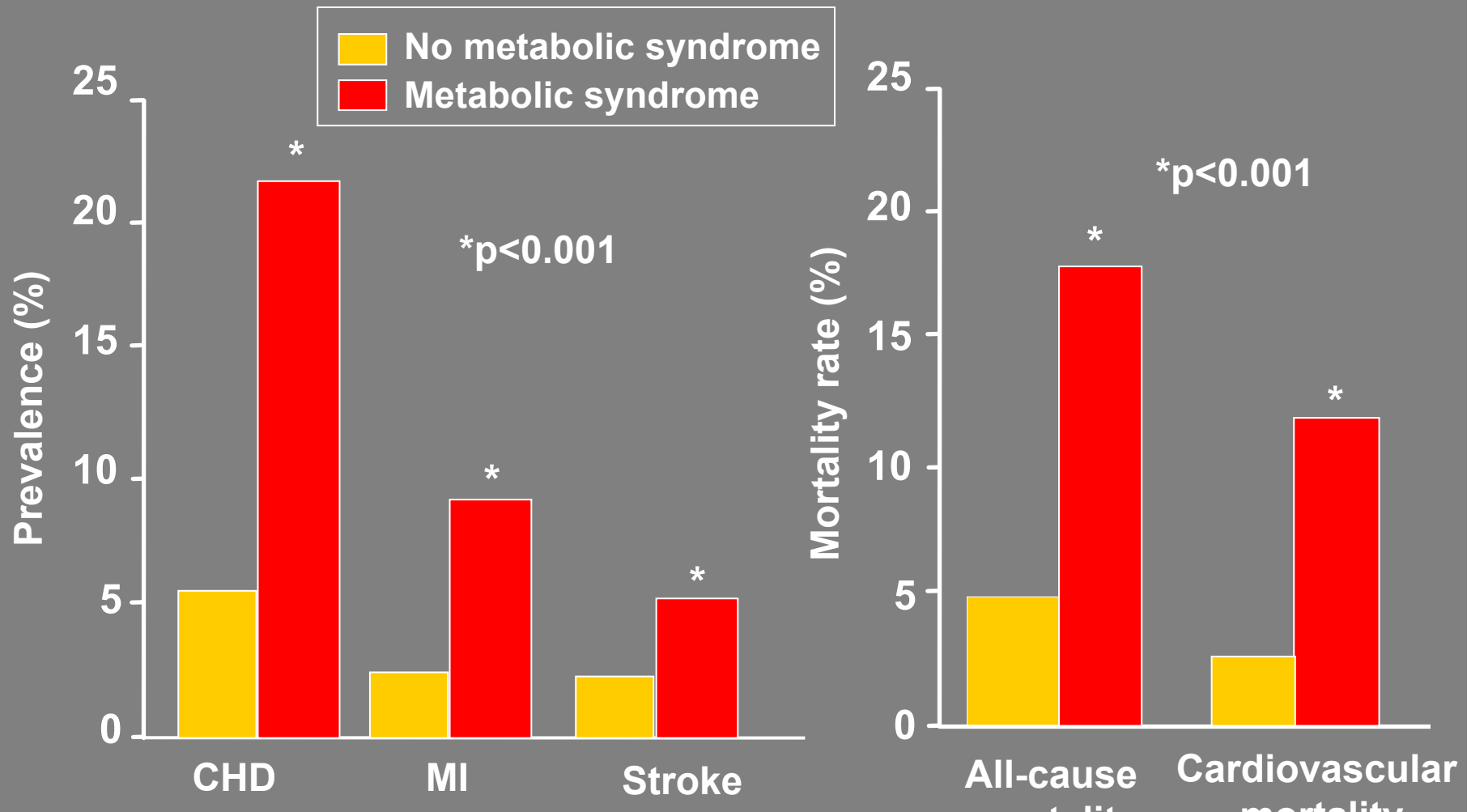


Abdominal obesity predicts the metabolic syndrome



Han et al 2002

Metabolic syndrome has a negative impact on CV health and mortality

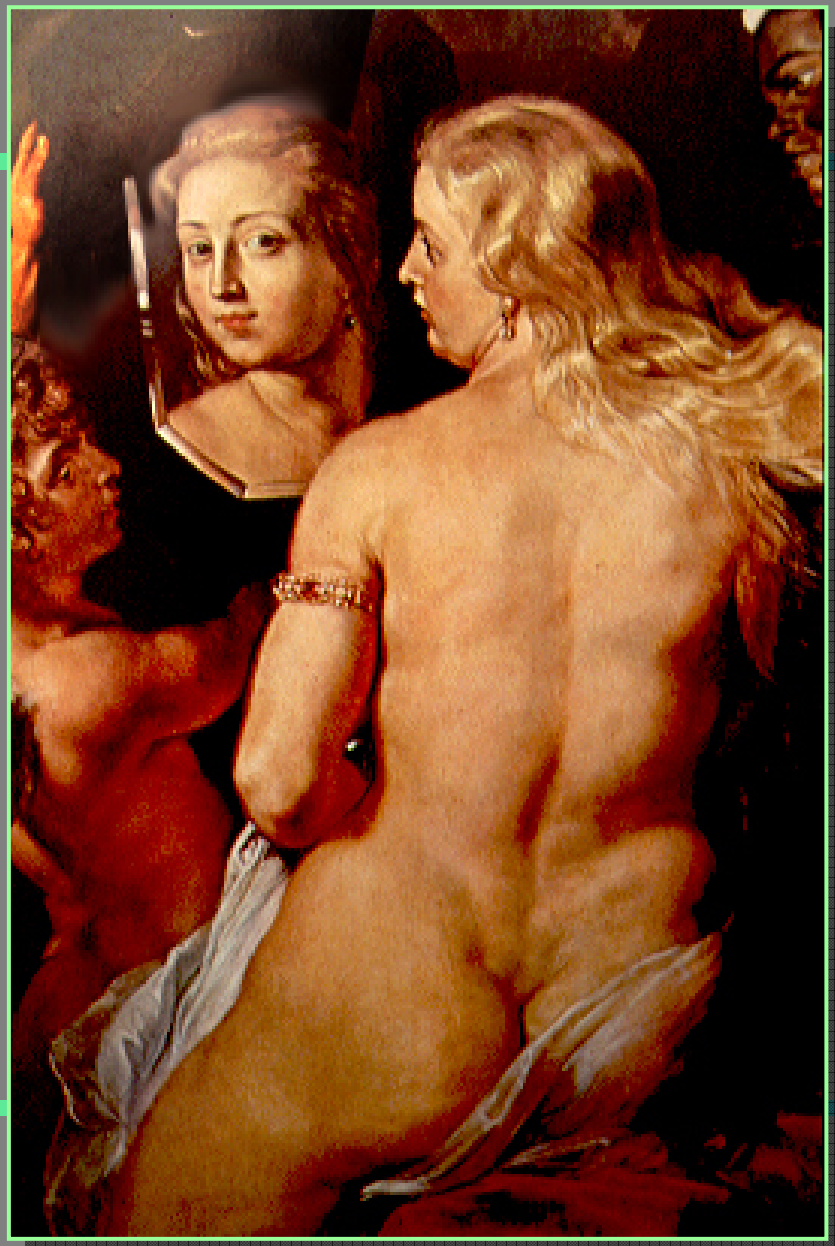


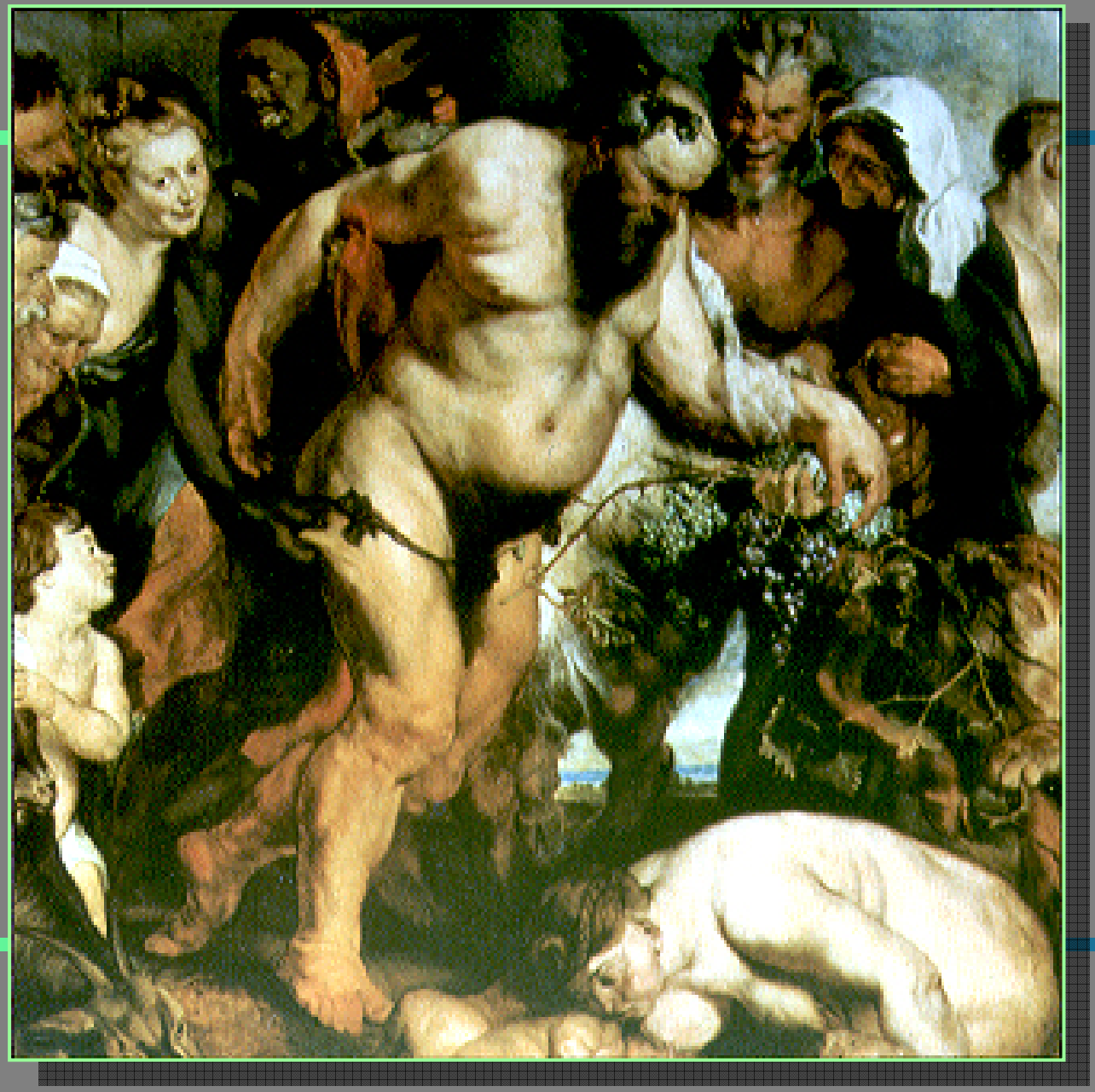
Isomaa et al 2001

Why is abdominal obesity harmful?

- **Abdominal obesity**
  - is often associated with other CV risk factors
  - is an independent CV risk factor
- **Adipocytes are metabolically active endocrine organs, not simply inert fat storage**

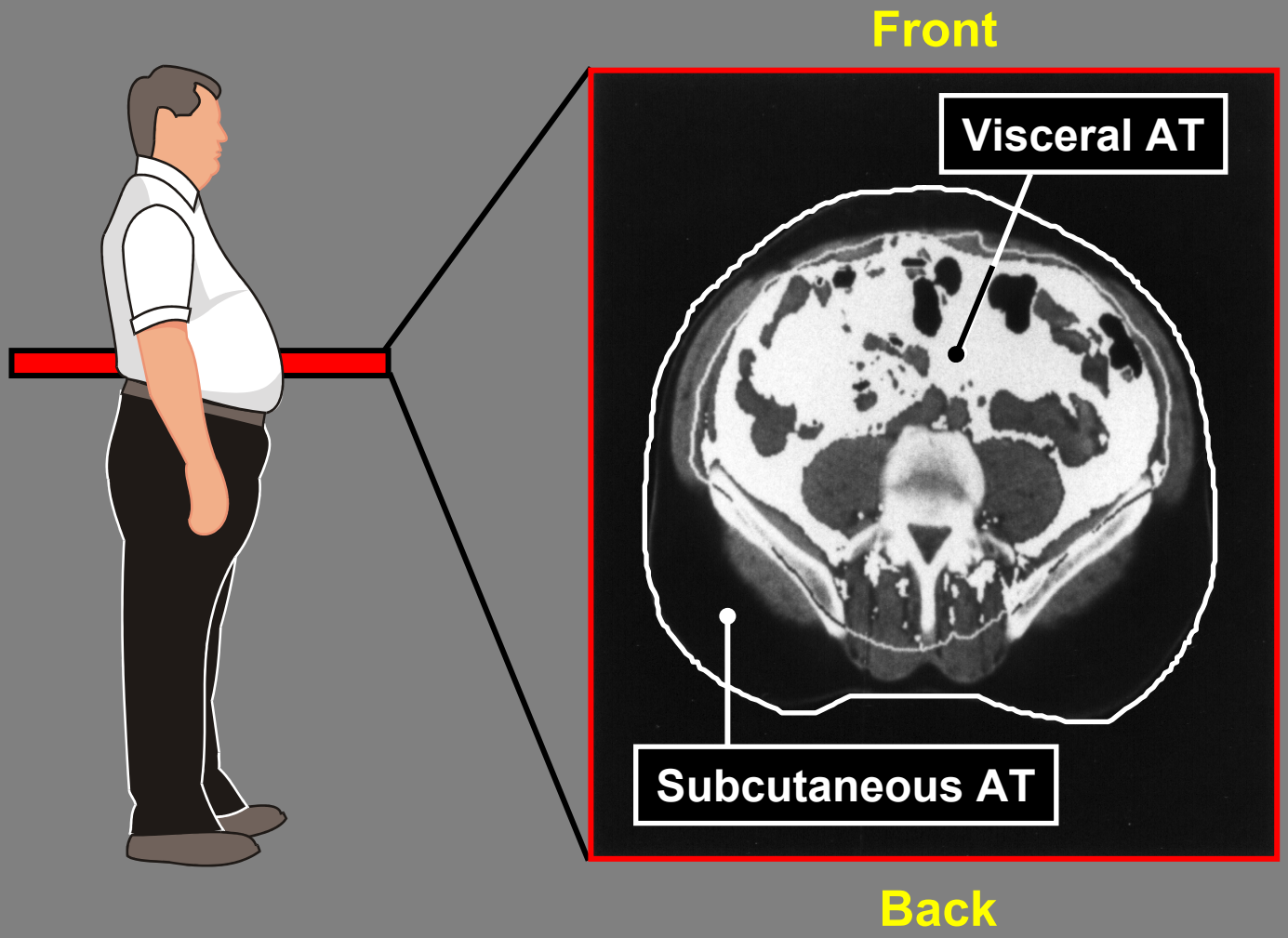
Wajchenberg 2000



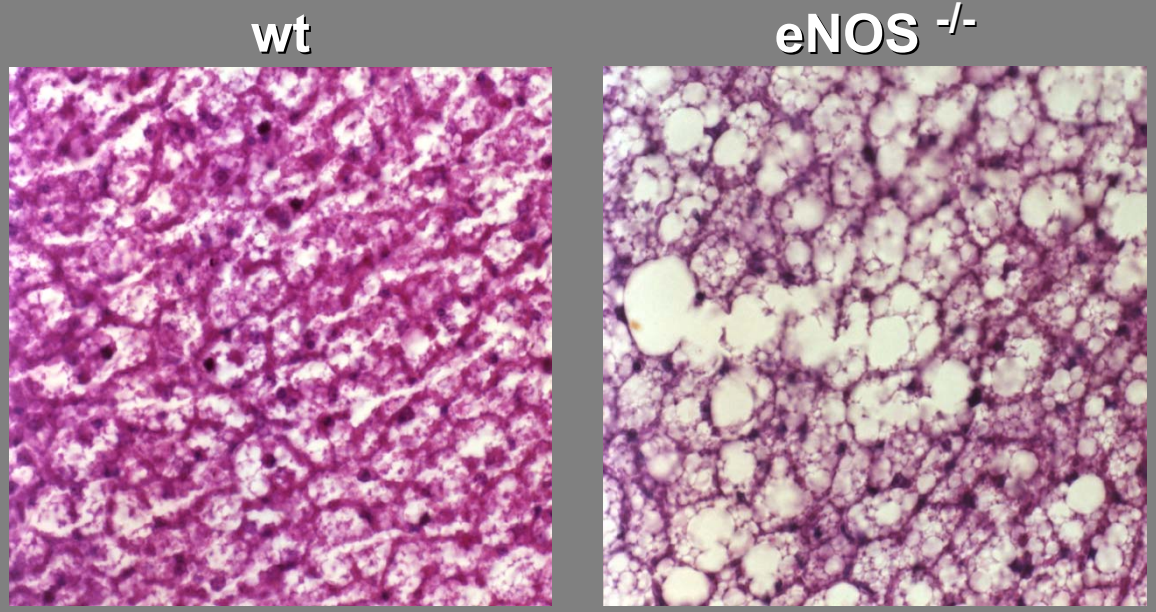


Intra-Abdominal (Visceral) Fat

The dangerous inner fat!

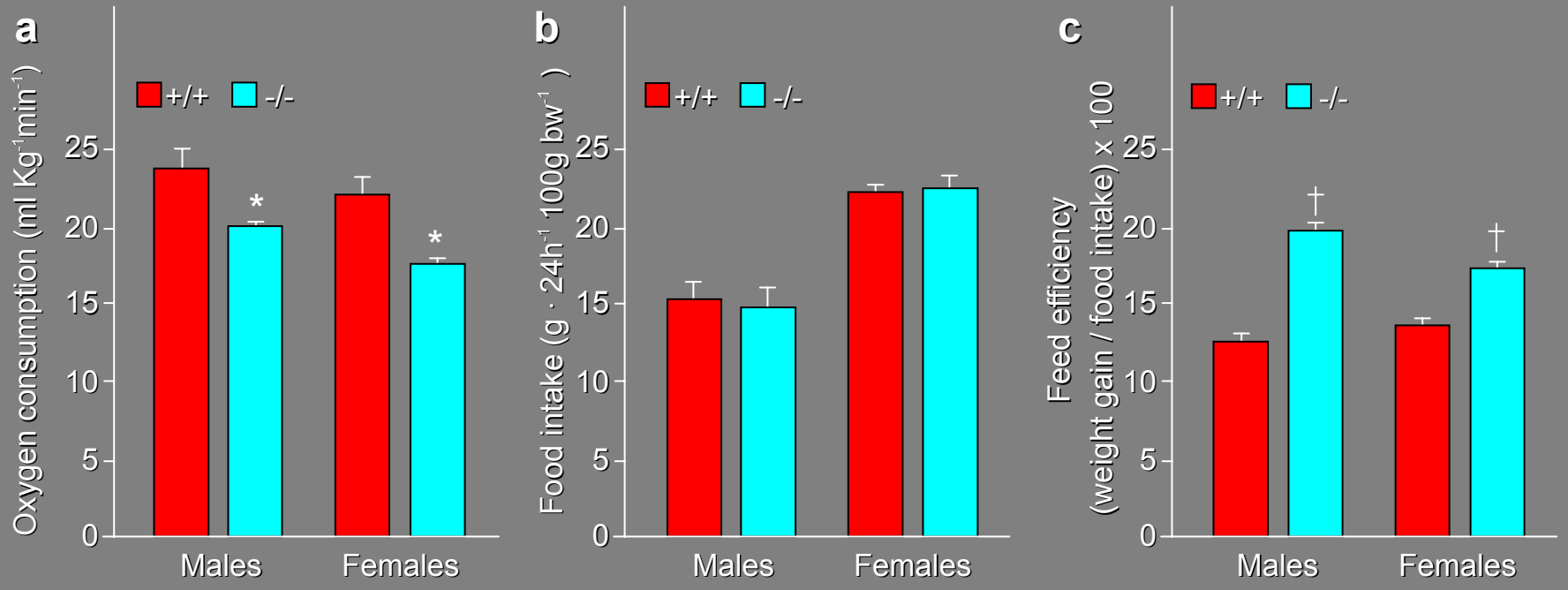


Lack of eNOS expression reduces mitochondrial biogenesis in BAT of eNOS$^{-/-}$ mice



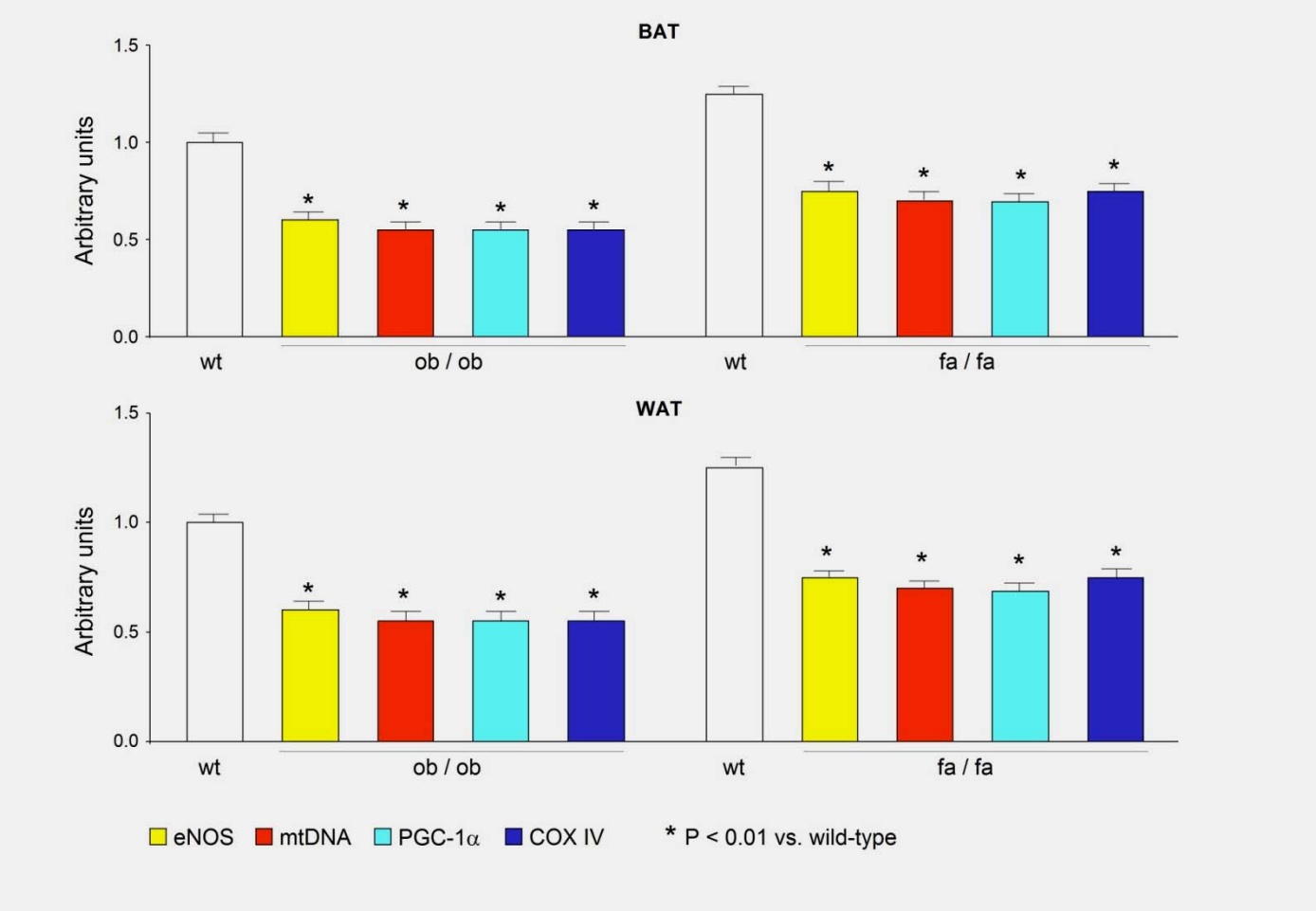
Nisoli et al., Science 299, 896–899, 2003

Oxygen consumption, food intake, and food efficiency of wild-type and eNOS$^{-/-}$ mice



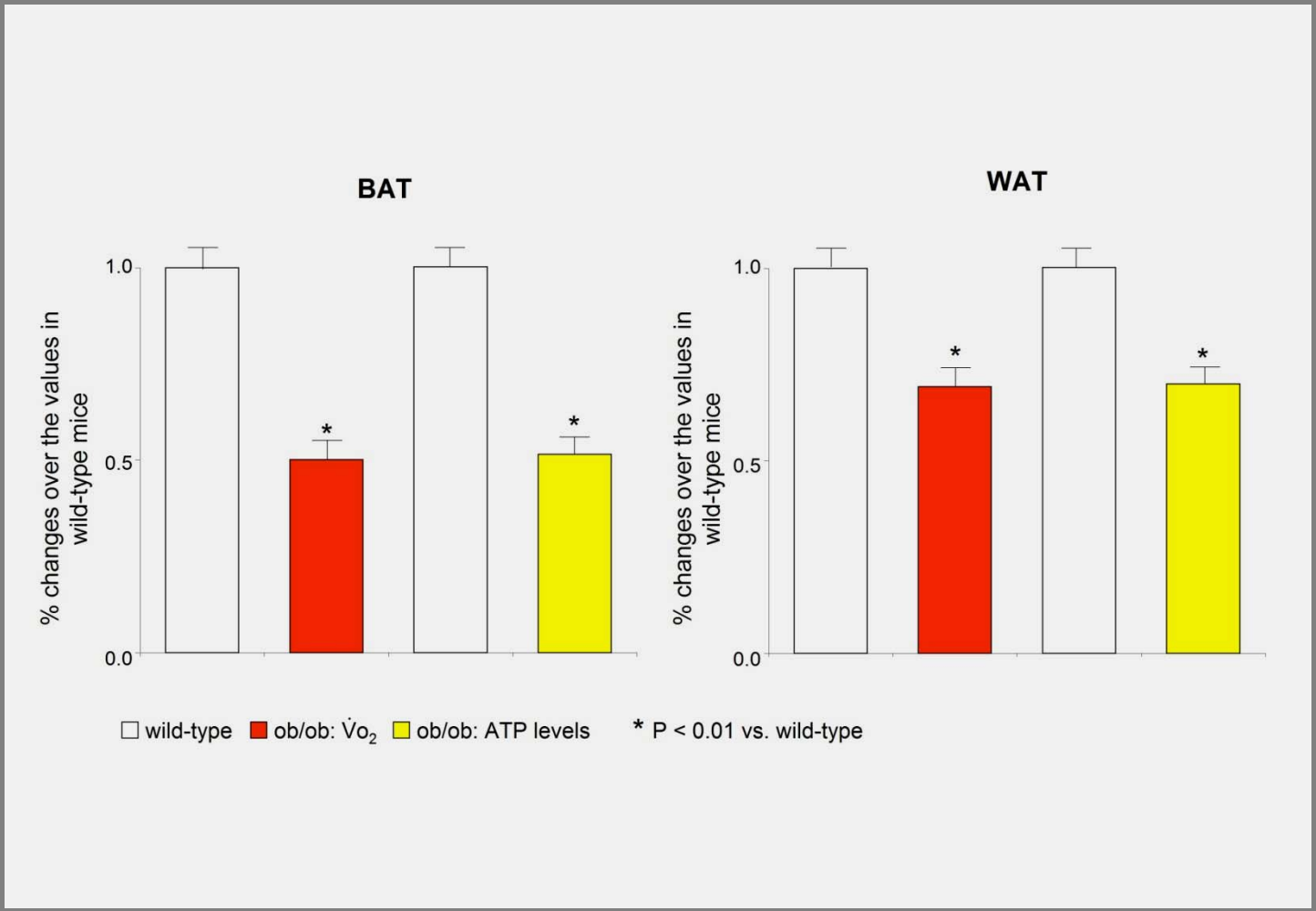
Nisoli et al., Science 299, 896–899, 2003

eNOS, PGC-1α, COX IV gene expression, and mtDNA levels are reduced in both WAT and BAT of obese rodents

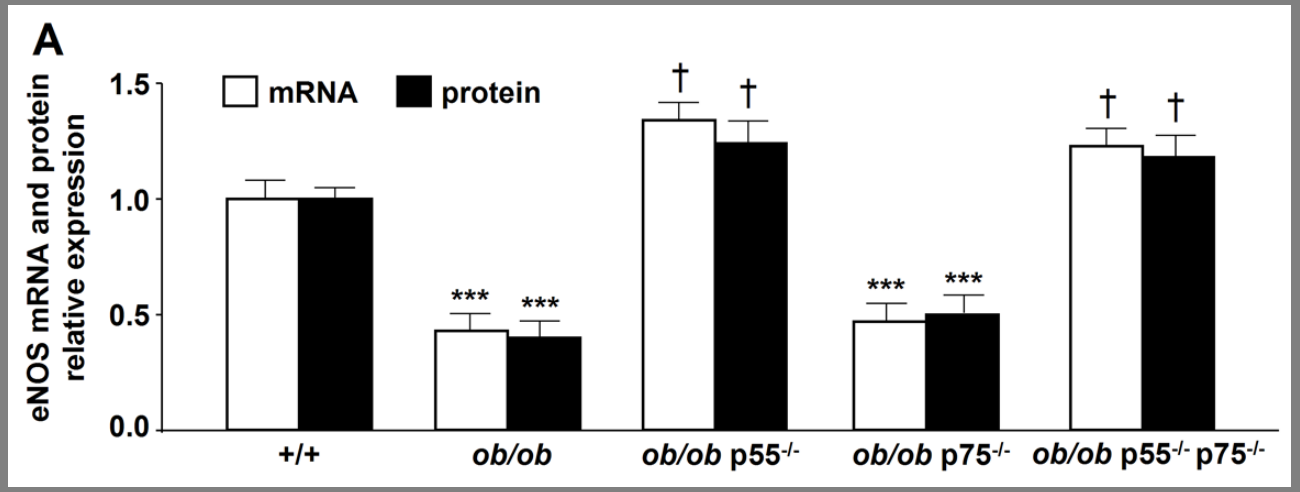


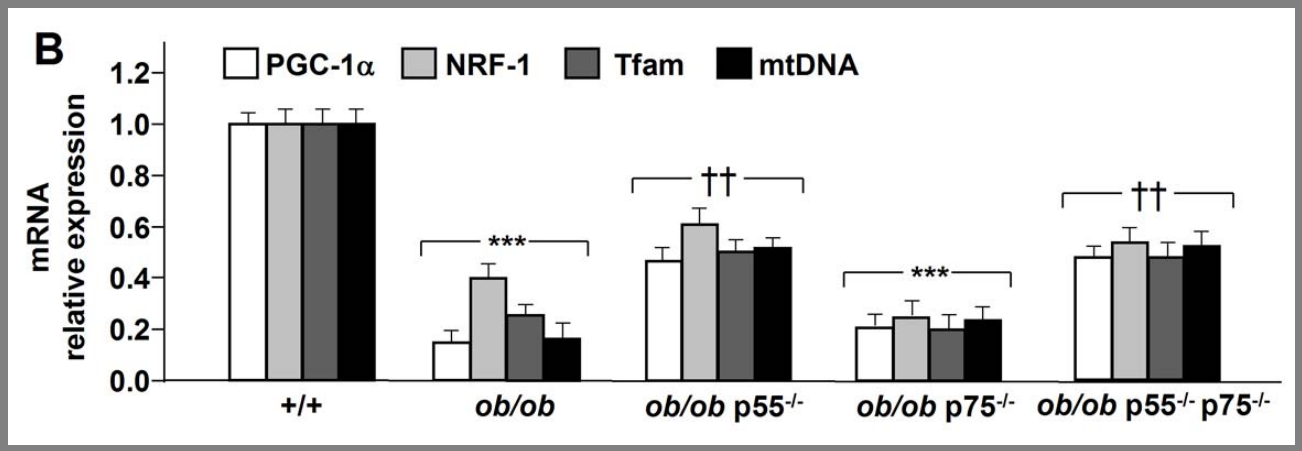
Valerio et al., J. Clin. Invest., Oct. 2006

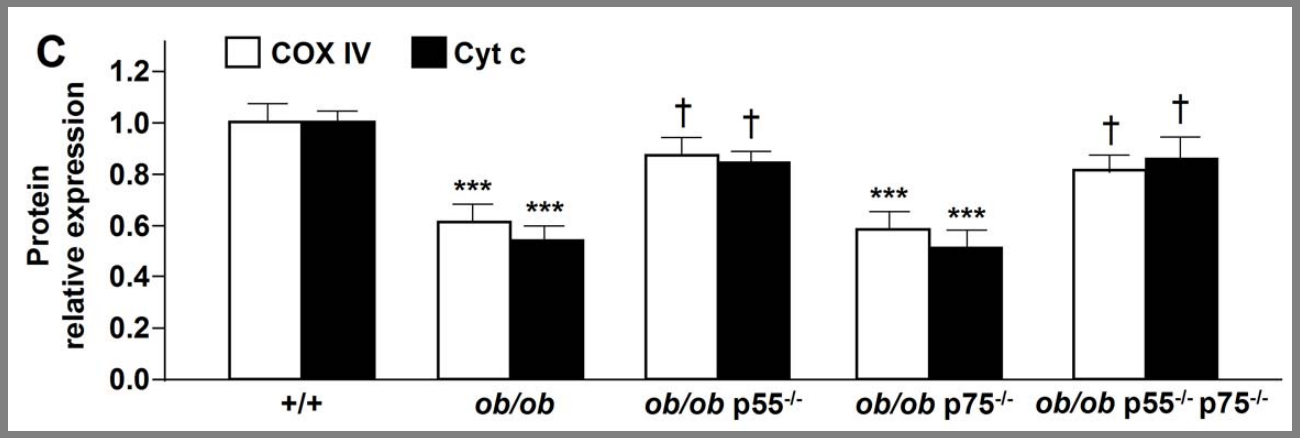
Oxygen consumption and ATP levels are decreased in WAT and BAT of *ob/ob* mice



Valerio et al., J. Clin. Invest., Oct. 2006



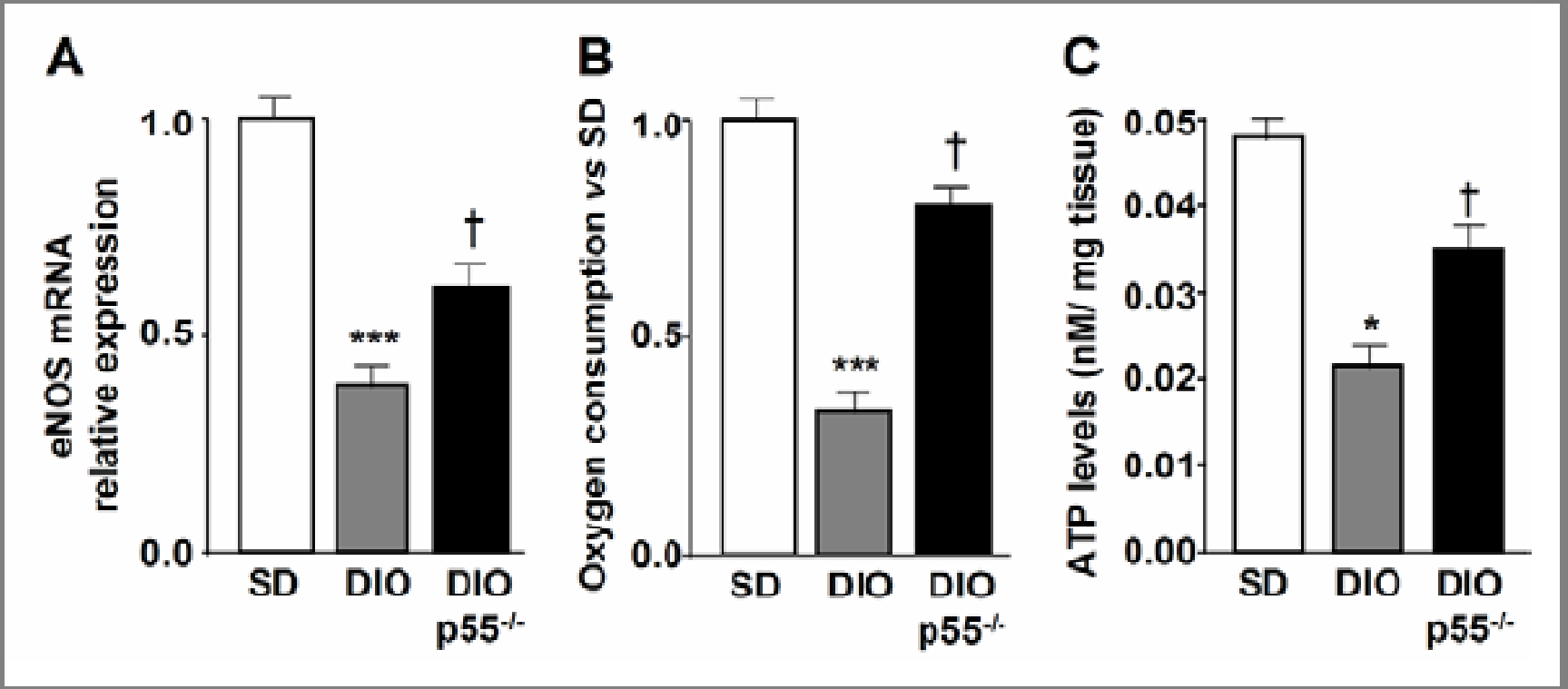




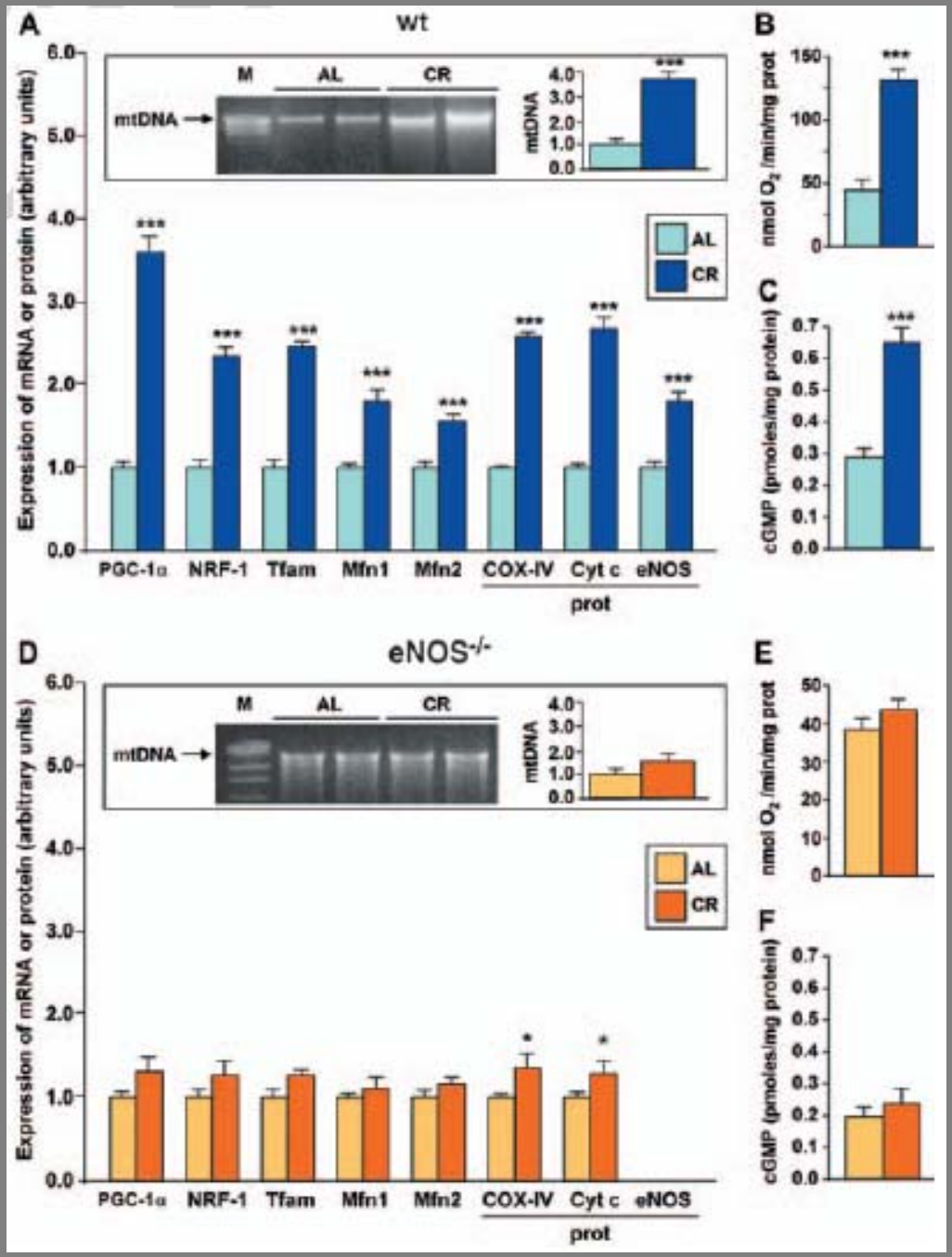
Mitochondrial biogenesis is partially normalized in WAT of *ob/ob* mice in which TNF-α signaling has been genetically deleted

Valerio et al., J. Clin. Invest., Oct. 2006

Mitochondrial biogenesis is partially normalized in WAT of DIO mice in which TNF-α receptor 1 (p55) has been genetically deleted



Valerio et al., J. Clin. Invest., Oct. 2006



CR induces mitochondrial biogenesis in WAT of wild-type (wt) but not eNOS$^{-/-}$ mice through eNOS expression and cGMP formation.

Nisoli et al., Science 310: 314-317, 2005

DIETA MEDITERRANEA

- Calorie
- Bilanciamento
- Carboidrati (IG)
- Lipidi (saturi/insaturi)
- Fibra
- Vitamine e antiossidanti
- Numero pasti
- Piramide (QB)

I nutrienti della dieta mediterranea

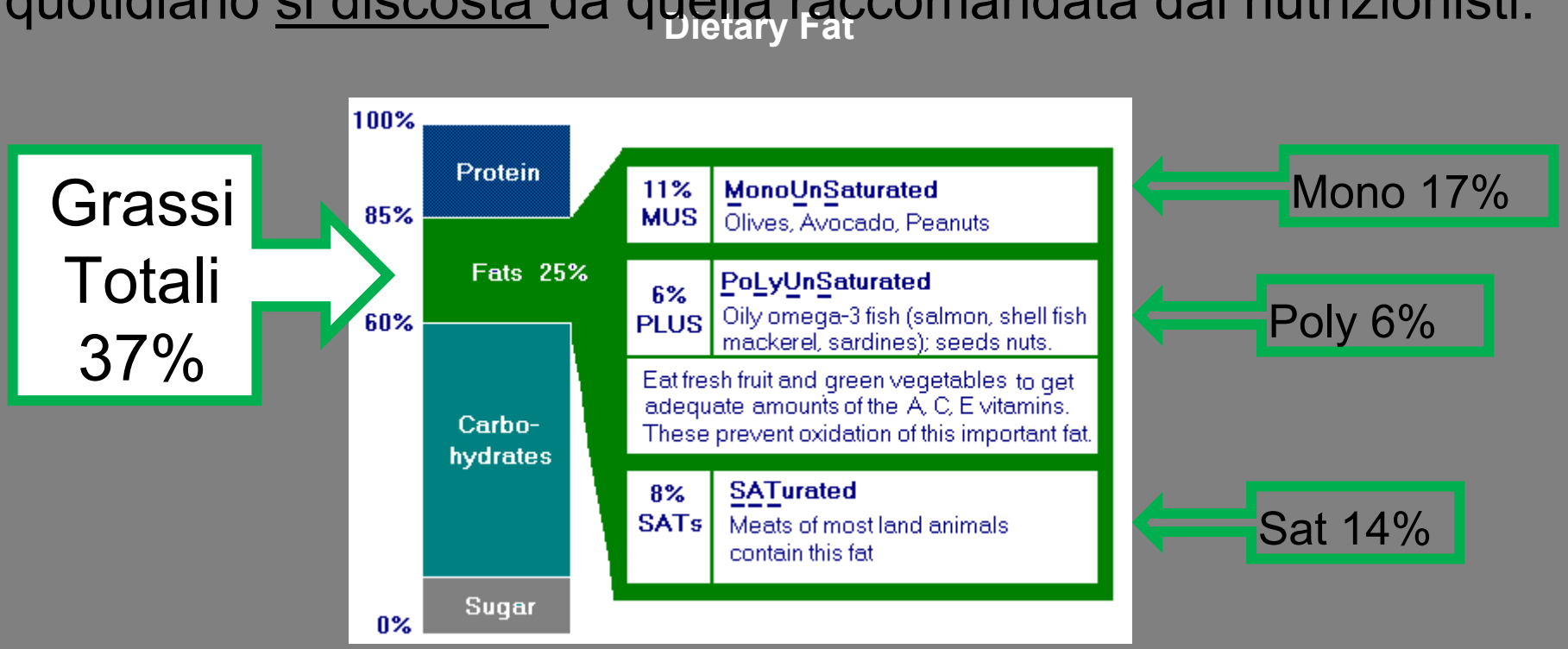
Fabbisogno giornaliero per un apporto bilanciato di nutrienti

Nutriente	Percentuale sul totale dell'apporto calorico
Carboidrati	Dal 50 al 55 %
di cui saccarosio	Meno del 10 %
Lipidi	Dal 25 al 30 %
di cui acidi grassi saturi	Meno del 10 %
Proteine	Dal 10 al 15 %

(Fonte : FAO/OMS)

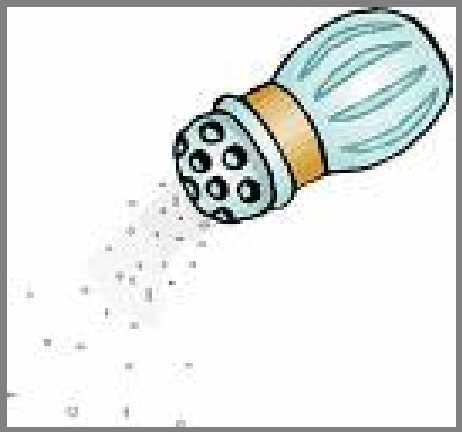
Il consumo di grassi alimentari

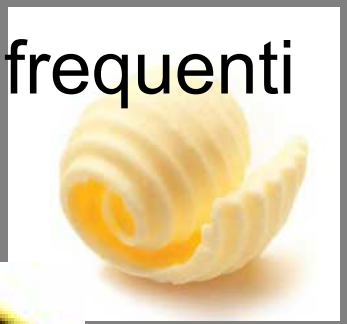
La percentuale di grassi sul totale dell'apporto energetico quotidiano si discosta da quella raccomandata dai nutrizionisti:

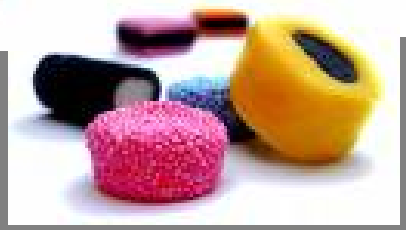


Eccessi

- Zuccheri semplici
- Grassi saturi
- Sale
- Proteine di origine animale
- Porzioni troppo abbondanti
- Spuntini troppo frequenti

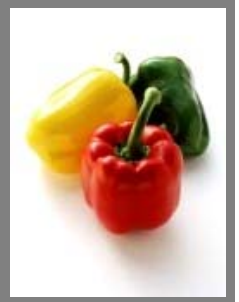


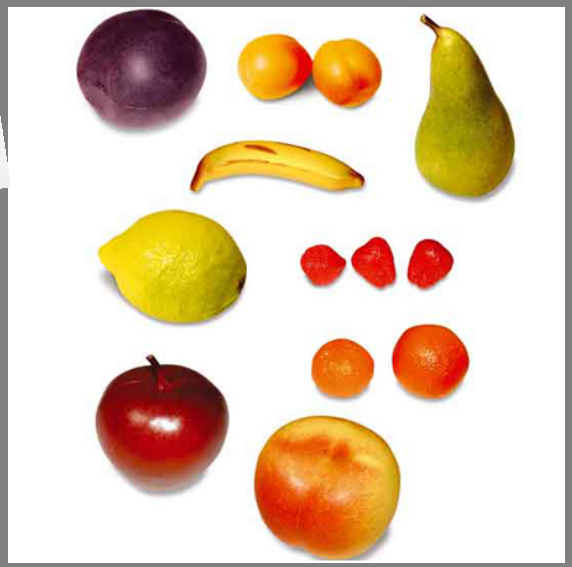




Carenze

- Frutta e verdura
- Fibre
- Principi nutritivi fondamentali
- Carboidrati complessi





La piramide alimentare

Per un corretto approccio nutrizionale è uso comune classificare gli alimenti in **7 gruppi principali** in accordo alle specifiche proprietà nutritive di ogni gruppo.