Dipartimento di Scienze Agrarie Alimentari e Agro-Ambientali
Università di Pisa

2016
ANNO INTERNAZIONALE DEI LEGUMI

NUTRA FOOD
Università di Pisa

GIORNATA DI STUDIO

I LEGUMI: SEMI NUTRIENTI PER UN FUTURO SOSTENIBILE

2 dicembre 2016 - ore 9:00-17:30

Sala Convegni del Dipartimento di Scienze Agrarie, Alimentari e Agro-ambientali
Via del Borghetto 80, Pisa

Annibale Carracci 'Il Mangiafagioli', 1584-1585

Castelluccio di Norcia, Umbria, nel 2015, patria delle lenticchie e della roveja

Più legumi, meno diabete

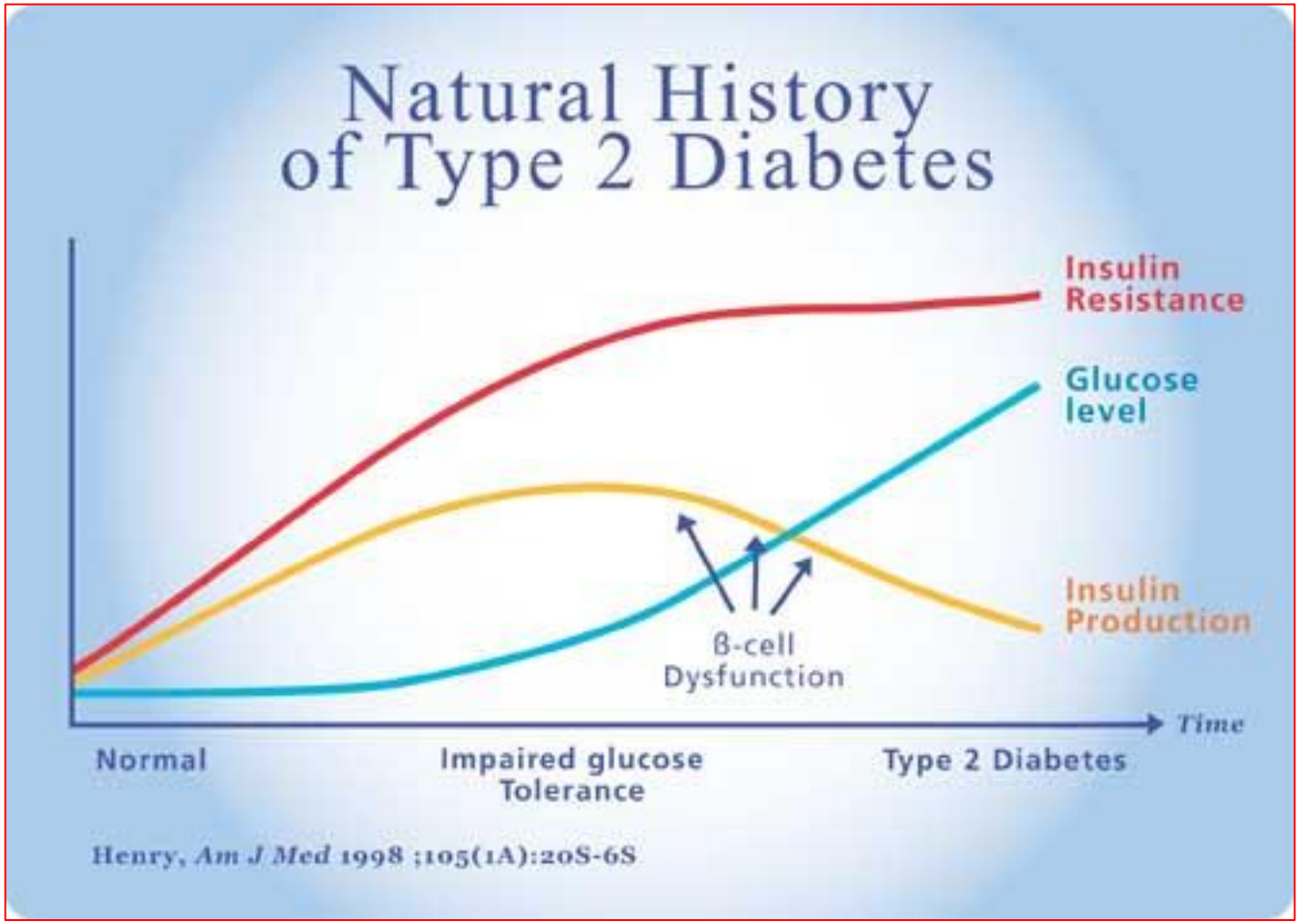
Piero Marchetti – Dipartimento di Medicina Clinica e Sperimentale (Università di Pisa)

Il Diabete Mellito

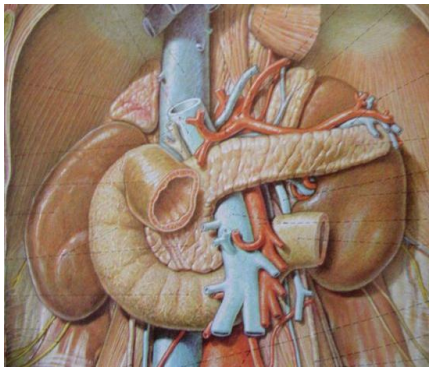
Il diabete mellito è una sindrome cronica dovuta a carenza assoluta o relativa di insulina, associata a gradi variabili di resistenza all'azione dell'insulina stessa da parte dei tessuti periferici, e caratterizzata da alterazioni del metabolismo glucidico, lipidico e proteico

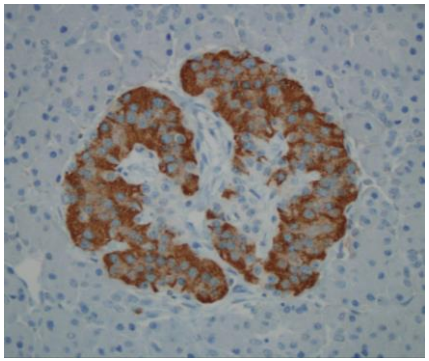
Classificazione del Diabete Mellito

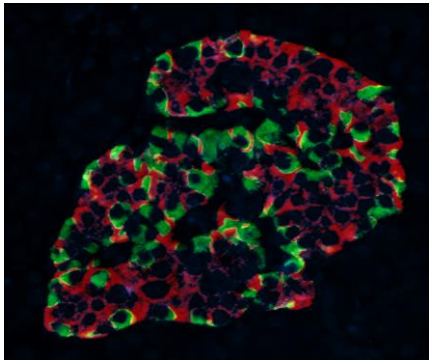
- Tipo 1
- Tipo 2
- Altri tipi specifici
- Diabete gestazionale


Natural History
of Type 2 Diabetes
Insulin
Resistance
Glucose
level
Insulin
Production
ß-cell
Dysfunction
Time
Normal
Impaired glucose
Tolerance
Type 2 Diabetes
Henry, Am J Med 1998 ;105(1A):20S-6S

Il pancreas endocrino







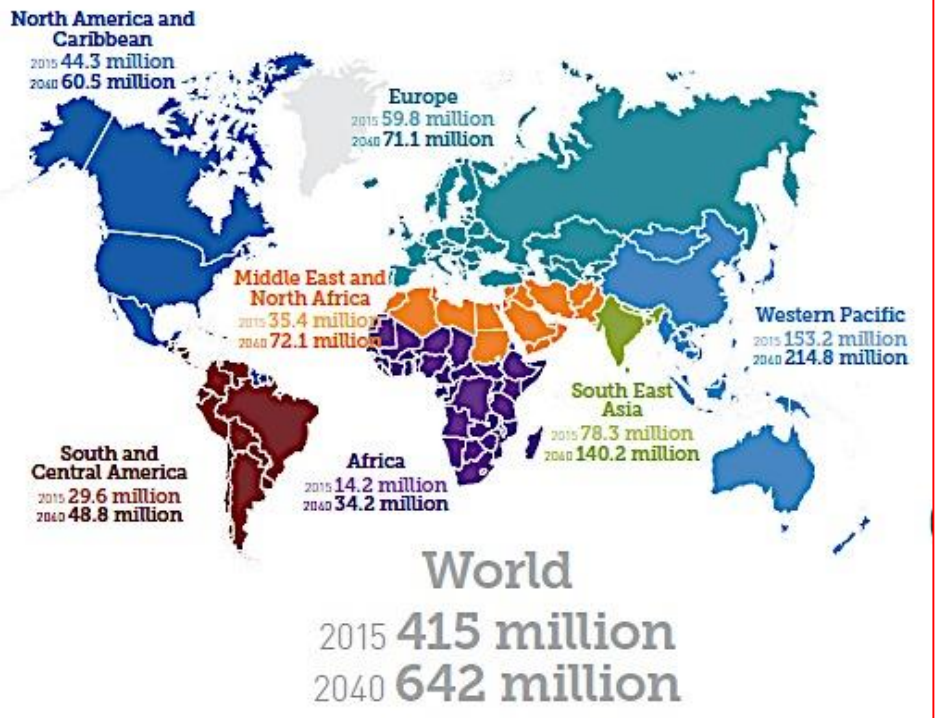
1,000,000 islets/pancreas

Approximately 1,000 cells/islet

50-80% of islet cells are beta cells

500-800,000,000 beta cells in a pancreas

Estimated number of people with diabetes worldwide and per region in 2015 and 2040 (20-79 years)



IDF 2015

Il diabete in Italia

- 4 M di persone che sanno di avere il diabete
- 1 M di persone che hanno il diabete senza saperlo
- 200.000 nuovi casi all'anno
- 500.000 diabetici con un problema serio ai reni
- 350.000 diabetici che hanno avuto un infarto
- 200.000 diabetici con un problema serio agli occhi
- 175.000 diabetici che hanno avuto un ictus
- 150.000 diabetici che hanno avuto un problema importante ai piedi
- 4.000 € per paziente per anno

SID – Diabete Ricerca

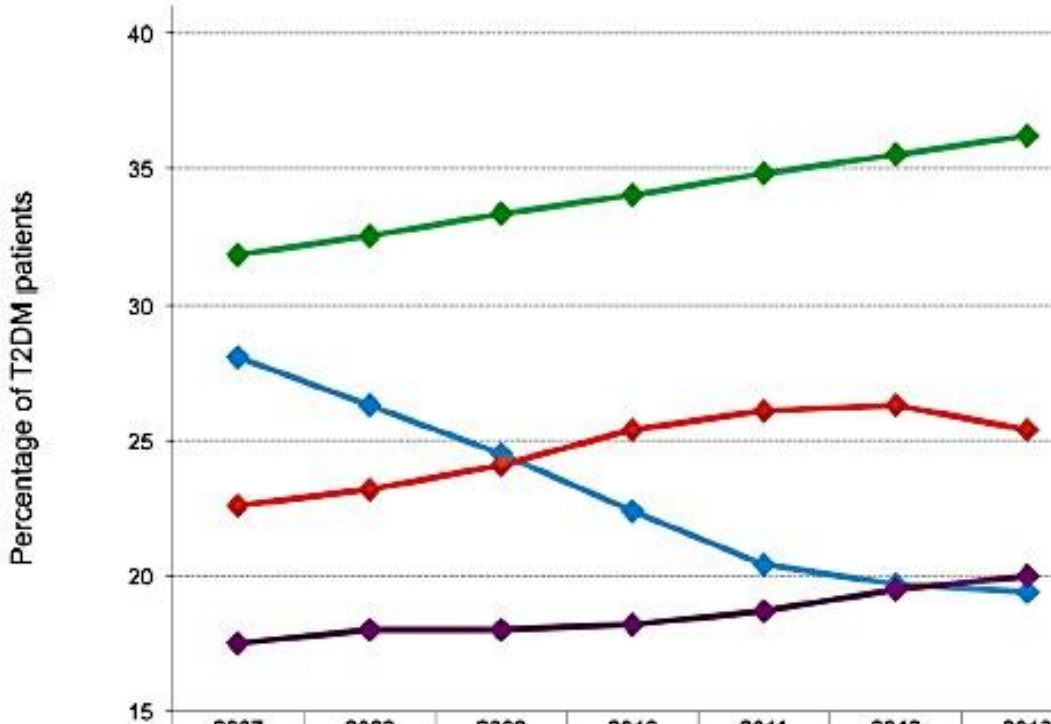
ACTOS 28CPR 15MG
ACTOS 28CPR 30MG
ACTOS 28CPR 45MG
ACTRAPHANE 30 NOV 5CART 300UI
ACTRAPHANE 30 SC 10ML 100UI/ML
ACTRAPHANE 50 NOV 5CART 3ML100
ACTRAPID NOVOL 5CART 3ML100UI/
ACTRAPID SC EV 1FL 10ML100UI/M
AMARYL 30CPR 2MG
AMARYL 30CPR 3MG
AMARYL 30CPR 4MG
APIDRA 5CART OPTIC.3ML 100U/ML
APIDRA SC 1FL 10ML 100U/ML
APIDRA SC 5 OPTISET 3ML100U/ML
APIDRA SC 5CART 3ML 100U/ML
APIDRA SOLOST.SC 5PEN 100UI/ML
AVAGLIM 28CPR RIV 4MG+4MG
AVANDAMET 112CPR RIV 2MG+500MG
AVANDAMET 56CPR RIV 2MG+1000MG
AVANDAMET 56CPR RIV 4MG+1000MG
AVANDIA 28CPR RIV 4MG
AVANDIA 28CPR RIV 8MG
BIDIABE 20CPR 125MG+30MG
BIEUGLUCON M 40CPR RIV 400+2,5
BYETTA SC 1PEN 1,2ML 5MCG
BYETTA SC 1PEN 2,4ML 10MCG
COMPETACT 56CPR RIV 15MG+850MG
DAONIL 30CPR 5MG
DIABEMIDE 20CPR 250MG
DIABORALE 20CPR 500MG
DIABREZIDE 40CPR 80MG
DIAGLIMET 36CPR RIV 5MG+500MG
DIALINAX 60CPR 500MG+2,5MG
DIAMEL 30CPR 2MG
DIAMICRON 40CPR 80MG
DIAMICRON 60CPR 30MG R.M.
DRAMION 60CPR 30MG R.M.
EFFICIB 56CPR RIV 50MG+1000MG
EFFICIB 56CPR RIV 50MG+850MG
EUCREAS 60CPR 50MG+1000MG
EUCREAS 60CPR 50MG+850MG
EUGLUCON 30CPR 5MG
GALTES 40CPR 80MG
GALVUS 56CPR 50MG
GLIBEN 30CPR 5MG
GLIBEN F 30CPR RIV 5MG+25MG
GLIBOMET 40CPR RIV 400MG+2,5MG
GLIBOMET 60CPR RIV 400MG+5MG
GLIBORAL 30CPR 5MG
GLICLAZIDE ALMUS 40CPR DIV80MG
GLICLAZIDE ALTER 40CPR DIV 80M
GLICLAZIDE BIG 40CPR 80MG

GLICLAZIDE DOC 40CPR 80MG
GLICLAZIDE EG 40CPR DIV 80MG
GLICLAZIDE MG 60CPR 30MG R.M.
GLICLAZIDE MOLTENI 40CPR 80MG
GLICLAZIDE M.G. 40CPR 80MG
GLICLAZIDE PENSA 40CPR 80MG
GLICLAZIDE TEVA 60CPR 30MG R.M
GLICLAZIDE WINTH 40CPR 80MG
GLICOBASE 40CPR 100MG
GLICONORM 36CPR RIV 5MG+500MG
GLICOREST 36CPR RIV 5MG+500MG
GLIMEPIRIDE ACT 30CPR 2MG
GLIMEPIRIDE ACT 30CPR 3MG
GLIMEPIRIDE ACT 30CPR 4MG
GLIMEPIRIDE ANG 30CPR 2MG BLIS
GLIMEPIRIDE DOC 30CPR 2MG
GLIMEPIRIDE EG 30CPR 2MG BLIST
GLIMEPIRIDE GERMED 30CPR 2MG
GLIMEPIRIDE HEX 30CPR 2MG BLIS
GLIMEPIRIDE M.G. 30CPR 2MG BLI
GLIMEPIRIDE SAN 30CPR 2MG BLIS
GLIMEPIRIDE TAD 30CPR 2MG
GLIMEPIRIDE TEVA 30CPR 2MG
GLUCOBAY 40CPR 100MG
GLUCOBAY 40CPR 100MG
GLUCOBAY 40CPR 50MG
GLUCOBLOC 40CPR 80MG DIV
GLUCOMIDE 40CPS 500MG+2,5MG
GLUCOPHAGE 30CPR RIV 500MG
GLUCOPHAGE 40CPR RIV 850MG
GLUCOPHAGE 60CPR RIV 1000MG
GLURENOR 40CPR 30MG
HUMALOG BASAL 5KWIKPEN 3ML 100
HUMALOG KWIKPEN 5PEN 3ML 100U/
HUMALOG MIX25 5KWIKPEN 3ML100U
HUMALOG MIX50 5KWIKPEN 3ML100U
HUMALOG NPL=>HUMALOG BASAL 5CA
HUMALOG SC 1FL 10ML 100U/ML
HUMALOG SC 5CART 3ML 100U/ML
HUMALOG SC BASAL 5CART 3ML100U
HUMALOG SC MIX 25 5CART 3ML
HUMALOG SC MIX 50 5CART 3ML
HUMALOG SC MIX25 F 100U/ML10ML

HUMULIN 30/70 1F 10ML 100U/ML
HUMULIN 30/70 5CART 3ML100U/ML
HUMULIN I 1F 10ML 100U/ML
HUMULIN I 5CART 3ML 100UI/ML
HUMULIN R 1F 10ML 100U/ML
HUMULIN R 5CART 3ML 100U/ML
INSUMAN RAP EV SC 5F 5ML100UI/
INSUMAN RAP EV SC 5ML 100UI/ML
INSUMAN RAP OPT EV SC5CART 3ML
JANUMET 56CPR RIV 50MG+1000MG
JANUMET 56CPR RIV 50MG+850MG
JANUVIA 28CPR RIV 100MG
LANTUS OPTIS.SC 5PEN 100UI/ML
LANTUS OPT.CK SC 5CART 100UI/M
LANTUS SC 1FL 100UI/ML 10ML
LANTUS SC 5CART 3ML 100UI/ML
LANTUS SOLOST.SC 5PEN 100UI/ML
LEVEMIR FLEX 5PEN 3ML 100UI/ML
METBAY 30CPR 500MG
METFONORM 30CPR RIV 500MG
METFONORM 40CPR RIV 850MG
METFONORM 60CPR RIV 1000MG
METFORAL 30CPR RIV 850MG
METFORAL 50CPR RIV 500MG
METFORALMILLE 60CPR RIV 1000MG
METFORMINA EG 60CPR RIV 1000MG
METFORMINA HEXAL 60CPR RIV 1G

METFORMINA HEX.AG 30CPR RIV850
METFORMINA HEX.AG 50CPR RIV500
METFORMINA M.S. 60CPR 1000MG
METFORMINA TEVA 30CPR 500MG OP
METFORMINA TEVA 40CPR 850MG OP
METFORMINA TEVA 60CPR RIV 1G
MINIDIAB 30CPR 5MG
NOVOMIX 30 FLEX 5PEN 3ML 100U/
NOVOMIX 50 FLEX 5PEN 3ML 100U/
NOVOMIX 70 FLEX 5PEN 3ML 100U/
NOVONORM 90CPR 0,5MG
NOVONORM 90CPR 1MG
NOVONORM 90CPR 2MG
NOVORAPID FLEX 5PEN 3ML 100U/M
NOVORAPID PENFILL SC 5CART 3ML
PLEIAMIDE 40CPR RIV 125+400MG
PROTAPHANE NOVOL5CART 3ML300UI
PROTAPHANE SC 1FL 10ML100UI/ML
SOLOSA 30CPR 2MG
SOLOSA 30CPR 3MG
SOLOSA 30CPR 4MG
SUGUAN M 40CPR RIV 400+2,5MG
TANDEMACT 28CPR 30MG+4MG
TESAVEL 28CPR RIV 100MG
VELMETIA 56CPR RIV 50MG+1000MG
VELMETIA 56CPR RIV 50MG+850MG
XELEVIA 28CPR RIV 100MG
ZUGLIMET 30CPR RIV 500MG
ZUGLIMET 40CPR RIV 850MG
ZUGLIMET 60CPR RIV 1000MG

Increasing use of antidiabetic drugs



	2007	2008	2009	2010	2011	2012	2013	Change 2007-2013 (95% CI)
NIAD monotherapy	31.8	32.5	33.3	34.0	34.8	35.5	36.2	4.40 (4.16 to 4.64)
No drugs	28.1	26.3	24.5	22.4	20.4	19.7	19.4	-9.70 (-9.48 to -9-92)
NIAD in combination	22.6	23.2	24.1	25.4	26.1	26.3	25.4	2.80 (2.58 to 3.02)
Insulin ± NIAD	17.5	18.0	18.0	18.2	18.7	19.5	20.0	2.5 (2.2 to 2.8)

Mata-Cases M et al, BMI Open 2016

Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy Dovepress
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Open Access Full Text Article ORIGINAL RESEARCH

Trends in medication use in patients with type 2 diabetes mellitus: a long-term view of real-world treatment between 2000 and 2015

This article was published in the following Dove Press journal:
Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy
1 November 2016
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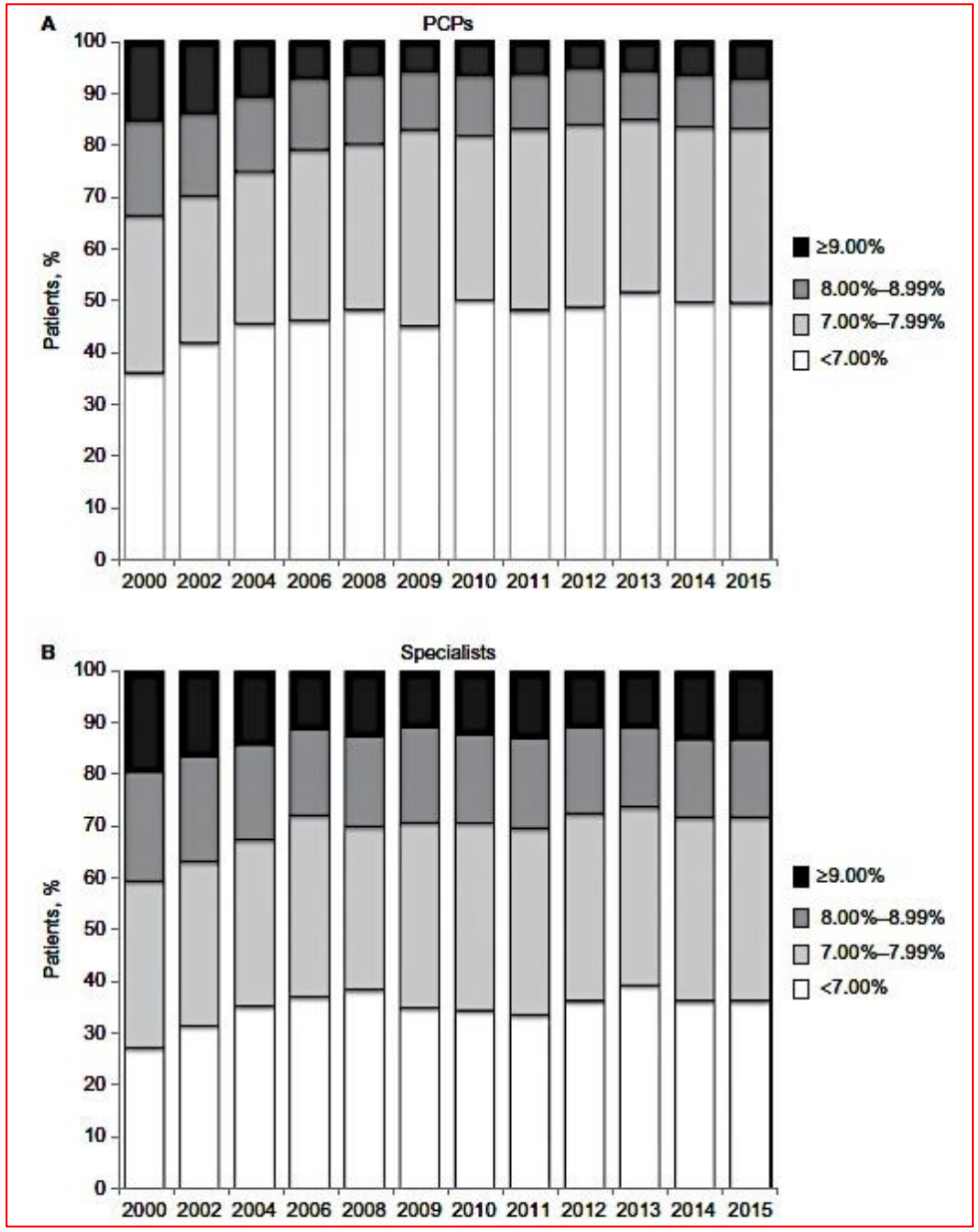
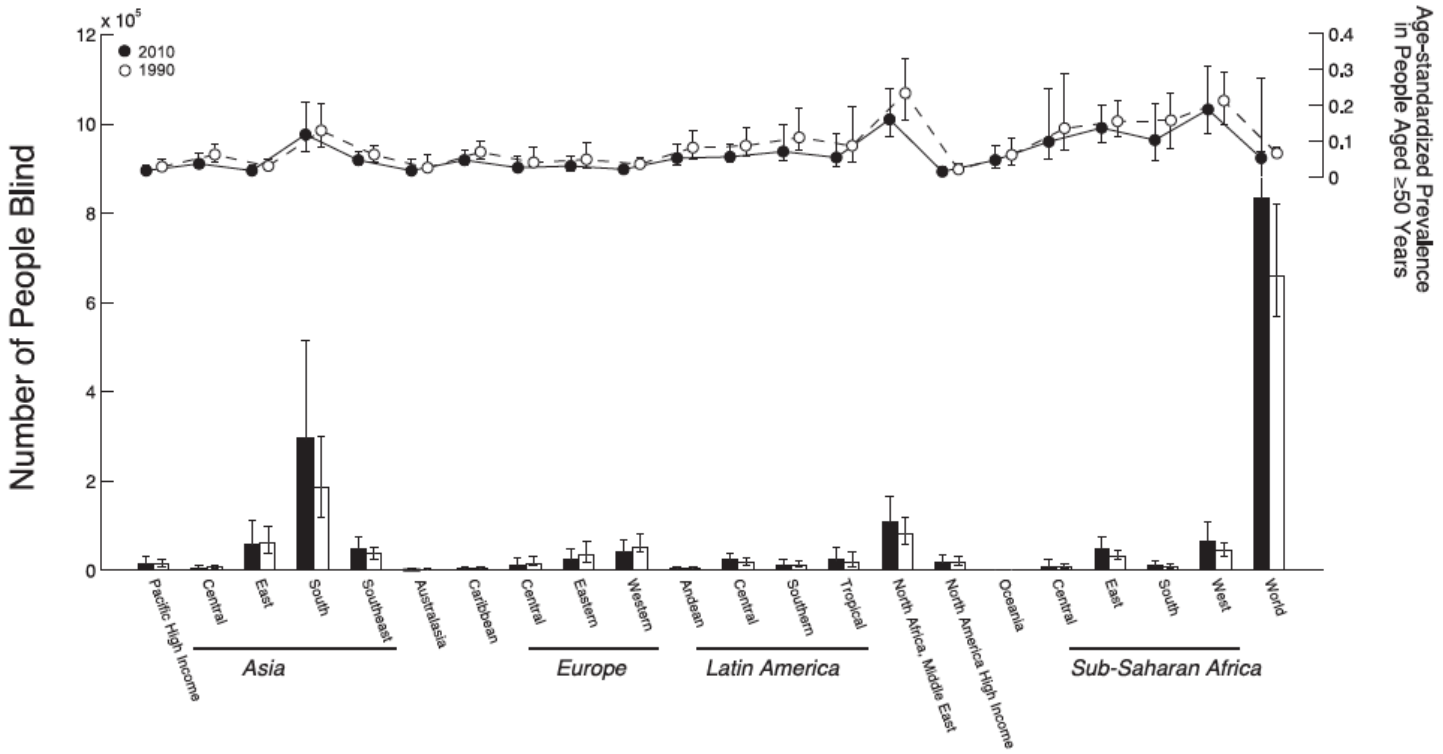
Victoria Higgins
James Piercy
Adam Roughley
Gary Milligan
Andrea Leith
James Siddall
Mike Benford



Global Estimates on the Number of People Blind or Visually Impaired by Diabetic Retinopathy: A Meta-analysis From 1990 to 2010

*Janet L. Leasher,[1] Rupert R.A. Bourne,[2] Seth R. Flaxman,[3] Jost B. Jonas,[4] Jill Keeffe,[5] Kovin Naidoo,[6,7] Konrad Pesudovs,[8] Holly Price,[2] Richard A. White,[9] Tien Y. Wong,[10] Serge Resnikoff,[7] and Hugh R. Taylor,[11] on behalf of the Vision Loss Expert Group of the Global Burden of Disease Study**

Diabetes Care 2016;39:1643–1649 | DOI: 10.2337/dc15-2171



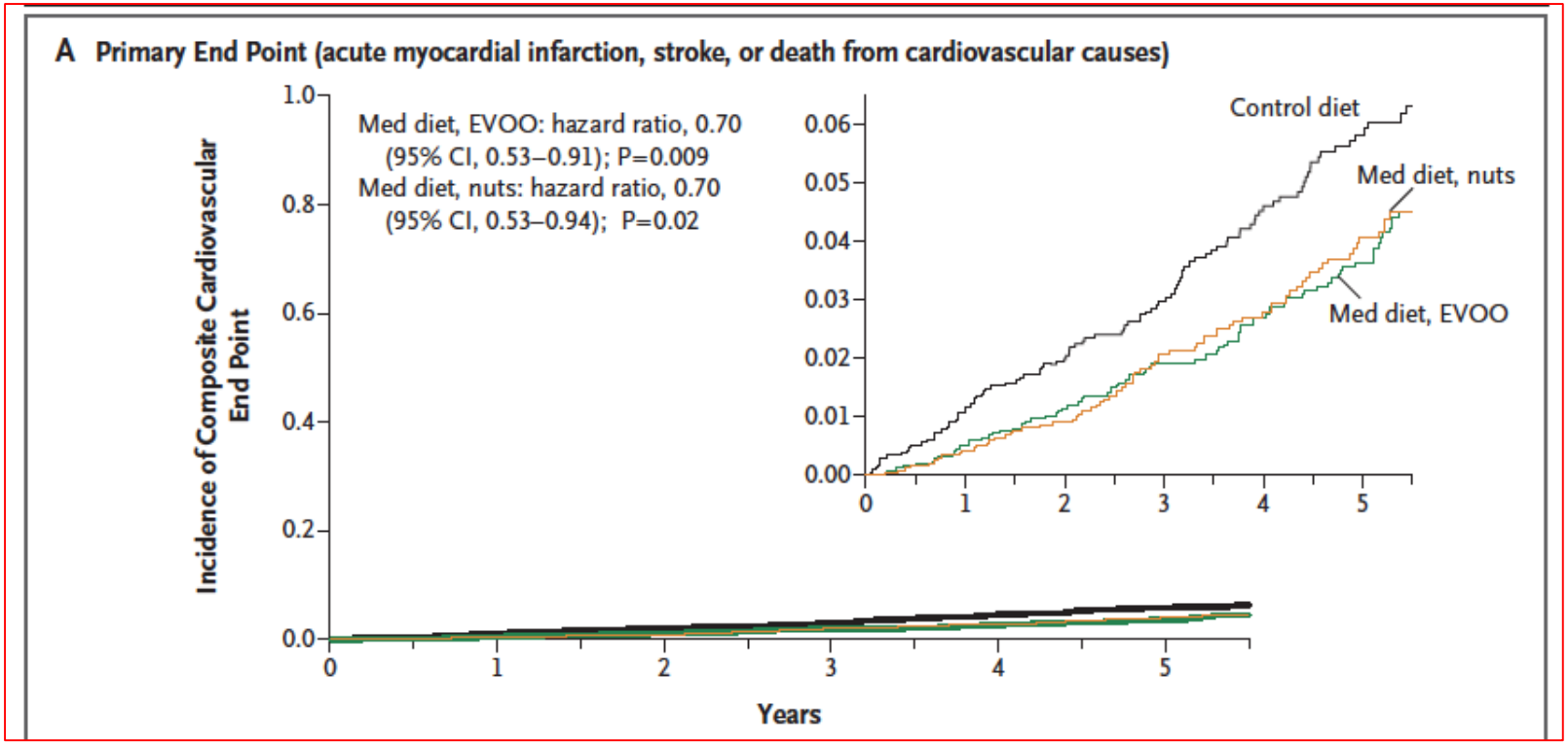

C'è qualcosa di nuovo oggi nel sole,
anzi d'antico:

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 APRIL 4, 2013 VOL. 368 NO. 14

Primary Prevention of Cardiovascular Disease with a Mediterranean Diet

Ramón Estruch, M.D., Ph.D., Emilio Ros, M.D., Ph.D., Jordi Salas-Salvadó, M.D., Ph.D., Maria-Isabel Covas, D.Pharm., Ph.D., Dolores Corella, D.Pharm., Ph.D., Fernando Arós, M.D., Ph.D., Enrique Gómez-Gracia, M.D., Ph.D., Valentina Ruiz-Gutiérrez, Ph.D., Miquel Fiol, M.D., Ph.D., José Lapetra, M.D., Ph.D., Rosa Maria Lamuela-Raventos, D.Pharm., Ph.D., Lluís Serra-Majem, M.D., Ph.D., Xavier Pintó, M.D., Ph.D., Josep Basora, M.D., Ph.D., Miguel Angel Muñoz, M.D., Ph.D., José V. Sorlí, M.D., Ph.D., José Alfredo Martínez, D.Pharm, M.D., Ph.D., and Miguel Angel Martínez-González, M.D., Ph.D., for the PREDIMED Study Investigators*



Bo et al. J Transl Med (2016) 14:91
DOI 10.1186/s12967-016-0851-7

Journal of
Translational Medicine

RESEARCH Open Access

Predictive role of the Mediterranean diet on mortality in individuals at low cardiovascular risk: a 12-year follow-up population-based cohort study

Simona Bo[1*], Valentina Ponzo[1], Ilaria Goitre[1], Maurizio Fadda[2], Andrea Pezzana[3], Guglielmo Beccuti[1], Roberto Gambino[1], Maurizio Cassader[1], Laura Soldati[4] and Fabio Broglio[1]

Abstract

Background: Adherence to the Mediterranean diet reduces the risk of all-cause and cardiovascular (CV) mortality and the incidence of CV events. However, most previous studies were performed in high-risk individuals. Our objective was to assess whether the adherence to the Mediterranean diet, evaluated by the MED score, was associated with all-cause and CV mortality and incidence of CV events in individuals at low CV risk from a population-based cohort, after a 12-year mean follow-up.

Methods: A cohort of 1658 individuals completed a validated food-frequency questionnaire in 2001–2003. The MED score was calculated by a 0–9 scale. Anthropometric, laboratory measurements, and the vital status were collected at baseline and during 2014. The baseline CV risk was estimated by the Framingham risk score. Participants were divided into two groups: individuals at low risk (CV < 10) and individuals with CV risk ≥10.

Results: During a 12-year mean follow-up, 220 deaths, 84 due to CV diseases, and 125 incident CV events occurred. The adherence to the Mediterranean diet was low in 768 (score 0–2), medium in 685 (score 4–5) and high in 205 (score >6) individuals. Values of BMI, waist circumference, fasting glucose and insulin significantly decreased from low to high diet adherence only in participants with CV risk ≥10. In a Cox-regression model, the hazard ratios (HRs) in low-risk individuals per unit of MED score were: HR = 0.83 (95 % CI 0.72–0.96) for all-cause mortality, HR = 0.75 (95 % CI 0.58–0.96) for CV mortality, and HR = 0.79 (95 % CI 0.65–0.97) for CV events, after multiple adjustments. In individuals with CV risk ≥10, the MED score predicted incident CV events (HR = 0.85; 95 % CI 0.72–0.99), while the associations with all-cause (HR = 1.02; 95 % CI 0.90–1.15) and CV mortality (0.94; 95 % CI 0.76–1.15) were not significant.

Conclusions: Greater adherence to the Mediterranean diet was associated with reduced fatal and non fatal CV events, especially in individuals at low CV risk, thus suggesting the usefulness of promoting this nutritional pattern in particular in healthier individuals.

Keywords: All-cause mortality, Cardiovascular risk, Cardiovascular mortality, Mediterranean diet

DIABETES RESEARCH AND CLINICAL PRACTICE 89 (2010) 97–102

Contents lists available at ScienceDirect

Diabetes Research and Clinical Practice

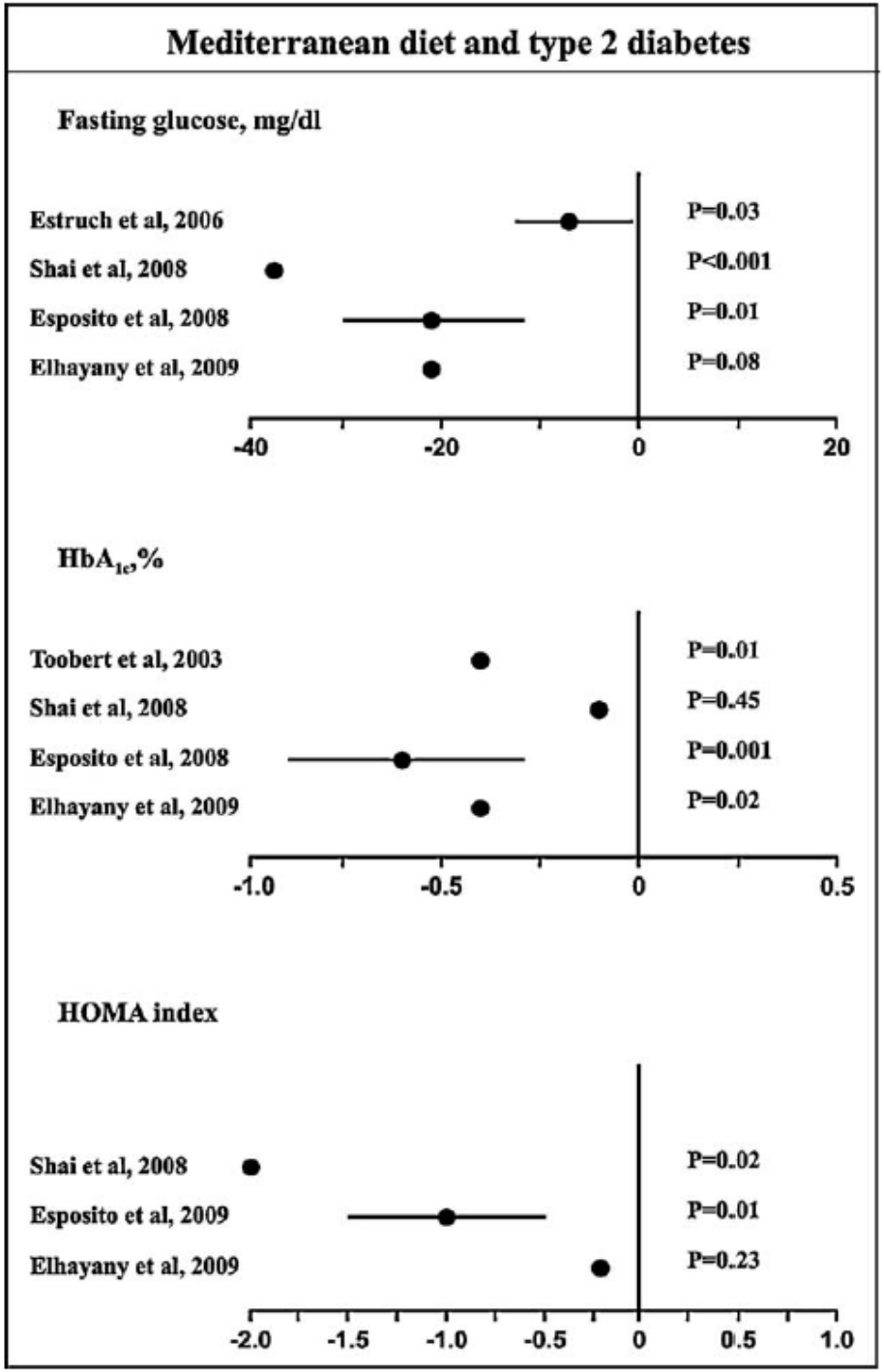
journal homepage: www.elsevier.com/locate/diabres

International Diabetes Federation

Review

Prevention and control of type 2 diabetes by Mediterranean diet: A systematic review

Katherine Esposito[a], *Maria Ida Maiorino*[a], *Antonio Ceriello*[b], *Dario Giugliano*[a,*]



Can J Diabetes 40 (2016) 355–363

Contents lists available at ScienceDirect

Canadian Journal of Diabetes

journal homepage:
www.canadianjournalofdiabetes.com

Review

The Role of Pulses in the Dietary Management of Diabetes

Dan Ramdath PhD [a,*], Simone Renwick [a,b], Alison M. Duncan PhD, RD [c]

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obesity reviews

doi: 10.1111/obr.12144

Etiology and Pathophysiology

A review of the nutritional value of legumes and their effects on obesity and its related co-morbidities

C. J. Rebello[1,2], F. L. Greenway[2] and J. W. Finley[1]

[1]School of Nutrition and Food Sciences, Louisiana State University, Baton Rouge, LA, USA; [2]Pennington Biomedical Research Center, Louisiana State University, Baton Rouge, LA, USA

Legume nutrition benefits

Type	Fibre (g)	Pyridoxine (mg)	Magnesium (mg)	Zinc (mg)	Phenolic content (ug/100g)
Lentils	15.6	0.35	71	2.51	25.8
Beans	10-15	0.2-0.4	70-120	1.5-2.2	15-18
Chickpeas	12.5	0.23	79	2.51	20.5
Lupins	4.6	0.015	90	2.59	19.4
Soy beans	10.3	0.402	148	1.98	/

Legume bioactive substances

- Alpha-amylase inhibitors (isoform 1)
- Alpha-glucosidase inhibitors (vitexin, isovitexin – flavonoids – polyphenols)

- Daidzein and Genistein (flavonoids – polyphenols)
  - Daidzein and Genistein are converted to equol and 5-hydroxy-equol by human intestinal *Slackia isoflavoniconvertens*

Microbiota intestinale

- Insieme dei microorganismi che si trovano nel tubo digerente dell'uomo
  - 600-1.000 specie differenti di microorganismi
  - Trilioni di microorganismi (10 volte il num. di cellule umane)
  - 10 milioni di geni (30.000 geni nel genoma umano)

Review

Journal of INTERNAL MEDICINE

doi: 10.1111/joim.12508

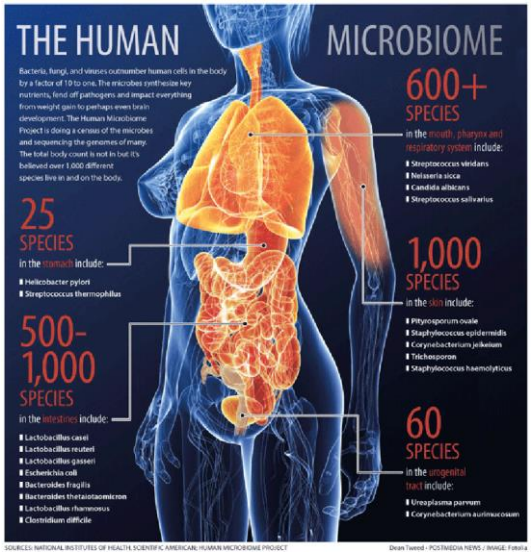
The gut microbiota and metabolic disease: current understanding and future perspectives

T. Arora[1] & F. Bäckhed[1,2]

From the [1]Wallenberg Laboratory and Sahlgrenska Center for Cardiovascular and Metabolic Research, Department of Molecular and Clinical Medicine, Institute of Medicine, University of Gothenburg, Gothenburg, Sweden; and [2]Novo Nordisk Foundation Center for Basic Metabolic Research, Section for Metabolic Receptology and Enteroendocrinology, Faculty of Health Sciences, University of Copenhagen, Copenhagen, Denmark

Abstract. Arora T and Bäckhed F (University of Gothenburg, Gothenburg, Sweden; University of Copenhagen, Copenhagen, Denmark). The gut microbiota and metabolic disease: current understanding and future perspectives. (Review). *J Intern Med* 2016; **280**: 339–349.

of butyrate-producing bacteria are reduced in patients with type 2 diabetes, whereas levels of *Lactobacillus* sp. are increased. Recent data suggest that the reduced levels of butyrate-producing bacteria might be causally linked to type 2 diabetes. Bariatric surgery, which promotes long-term weight loss and diabetes remission, alters the gut



REVIEW

doi:10.1038/nature18846

Diet–microbiota interactions as moderators of human metabolism

Justin L. Sonnenburg[1] & Fredrik Bäckhed[2,3]

It is widely accepted that obesity and associated metabolic diseases, including type 2 diabetes, are intimately linked to diet. However, the gut microbiota has also become a focus for research at the intersection of diet and metabolic health. Mechanisms that link the gut microbiota with obesity are coming to light through a powerful combination of translation-focused animal models and studies in humans. A body of knowledge is accumulating that points to the gut microbiota as a mediator of dietary impact on the host metabolic status. Efforts are focusing on the establishment of causal relationships in people and the prospect of therapeutic interventions such as personalized nutrition.

Worldwide, obesity has more than doubled since 1980 according to the World Health Organization. In 2014, more than 1.9 billion adults were overweight, and over 600 million of those people were obese. Obesity results from a positive energy balance, which occurs when the amount of energy ingested exceeds the amount expended, and it is a strong risk factor for other metabolic complications such as type 2 diabetes. Type 2 diabetes is increasing in prevalence in low-income countries, and in 2014, approximately 422 million adults worldwide had diabetes. The condition is characterized by high blood sugar, resistance to insulin and a relative lack of insulin. Insulin resistance is also associated with an increased flux of free fatty acids that contribute to diabetic dyslipidaemia, which is characterized by a high concentration of triglycerides in blood plasma, a low concentration of high-density lipoprotein (HDL) cholesterol and an increased concentration of small, dense low-density lipoprotein cholesterol particles[1]. Dyslipidaemia is one of the major risk factors for cardiovascular disease in people with diabetes. Accordingly, abnormal metabolism of glucose and lipids is the hallmark of metabolic syndrome, which is defined by central (abdominal) obesity and the presence of two or more of four factors — elevated triglycerides, reduced HDL cholesterol, high blood pressure, and increased fasting blood glucose. As governments and health organizations struggle to find solutions to these largely preventable health issues, a rapidly expanding area of research that is focused on the microbes that live within our digestive tract is offering fresh and interesting insights and potential avenues for intervention.

The human gut is a bioreactor with a microbiota that typically encompasses hundreds or thousands of bacterial taxa, which predominantly belong to two phyla: Firmicutes and Bacteroidetes[2–5]. Tremendous strides have been taken over the past decade towards mapping the composition and basic functional attributes of the gut microbiota of people from industrialized countries[3,5]. This ensemble of organisms has coevolved with the human host and complements the coding potential of our own genome with 500-fold more genes[6]. However, the annotation, and consequently the biological function, of many of these remain poorly defined.

The observation that germ-free mice, which lack a microbiota, have reduced adiposity and improved tolerance to glucose and insulin when compared with conventional (colonized) counterparts[7] jump-started a decade of research that focused on the clarification of underlying mechanisms. Germ-free mice are protected from diet-induced obesity when fed a Western-style diet[8–10], which further supports a link between the gut microbiota and the host metabolism. The altered microbiota that is observed in genetically obese mice[11,12] is sufficient to promote increased adiposity in lean mice that receive a microbiota transplant[12], demonstrating that the microbiota contributes to the regulation of adiposity. The importance and generalizability of these initial findings are strengthened by reports of alterations in the gut microbiota of obese people[4,13–15], which confer the obese or adiposity phenotypes when transferred to mice[14,16].

Here, we review the large body of data that is shaping our understanding of how the gut microbiota can alter the absorption, metabolism and storage of calories. Despite broad agreement that gut microbes modify how the human body responds to components of diet to influence metabolism, the mechanisms that underlie this process are exceptionally complex and the data can be difficult to reconcile. The picture that is emerging suggests that obesity is associated with reduced diversity of the gut microbiota[13,17]. Systemic inflammation and microbial metabolites, such as bile acids and short-chain fatty acids, are also commonly implicated. The ability to easily access and reprogramme the composition and function of the microbiota make it an attractive target for intervention.

Diet as an important modulator of the gut microbiota

Extensive research on the gut microbiota has shown that diet modulates the composition and function of this community of microbes in humans and other mammals[18–25], with the earliest literature[26] published almost 100 years ago. Human intervention studies from the past decade have revealed the extent to which different aspects of the microbiota can be influenced through dietary change; this can be summarized by three main themes.

The first theme is that the microbiota of the human gut responds rapidly to large changes in diet. The existence of these fast, diet-induced dynamics is supported by evidence from people who switch between plant- and meat-based diets, who add more than 30 grams per day of specific dietary fibres to their diet or who follow either a high-fibre–low-fat diet or a low-fibre–high-fat diet for 10 days; in all cases, the composition and function of the microbiota shifted over 1–2 days[18,20,23]. Such marked shifts in response to nutrient availability are perhaps unsurprising given that populations of microbes can double within an hour and the gut extensively purges the community every 24–48 hours. This responsiveness might represent an advantageous feature of enlisting

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High-level adherence to a Mediterranean diet beneficially impacts the gut microbiota and associated metabolome.

De Filippis F[1], Pellegrini N[2], Vannini L[3], Jeffery IB[4], La Storia A[1], Laghi L[3], Serrazanetti DI[5], Di Cagno R[6], Ferrocino I[7], Lazzi C[2], Turroni S[8], Cocolin L[7], Brigidi P[8], Neviani E[2], Gobbetti M[6], O'Toole PW[4], Ercolini D[1].

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Abstract

OBJECTIVES: Habitual diet plays a major role in shaping the composition of the gut microbiota, and also determines the repertoire of microbial metabolites that can influence the host. The typical Western diet corresponds to that of an omnivore; however, the Mediterranean diet (MD), common in the Western Mediterranean culture, is to date a nutritionally recommended dietary pattern that includes high-level consumption of cereals, fruit, vegetables and legumes. To investigate the potential benefits of the MD in this cross-sectional survey, we assessed the gut microbiota and metabolome in a cohort of Italian individuals in relation to their habitual diets.

DESIGN AND RESULTS: We retrieved daily dietary information and assessed gut microbiota and metabolome in 153 individuals habitually following omnivore, vegetarian or vegan diets. The majority of vegan and vegetarian subjects and 30% of omnivore subjects had a high adherence to the MD. We were able to stratify individuals according to both diet type and adherence to the MD on the basis of their dietary patterns and associated microbiota. We detected significant associations between consumption of vegetable-based diets and increased levels of faecal short-chain fatty acids, Prevotella and some fibre-degrading Firmicutes, whose role in human gut warrants further research. Conversely, we detected higher urinary trimethylamine oxide levels in individuals with lower adherence to the MD.

CONCLUSIONS: High-level consumption of plant foodstuffs consistent with an MD is associated with beneficial microbiome-related metabolomic profiles in subjects ostensibly consuming a Western diet.

Conclusioni

- Il consumo di legumi, in particolare nell'ambito della dieta mediterranea, è associato a multipli benefici di tipo metabolico, incluso un ruolo nella prevenzione e nella cura del diabete mellito
- E' auspicabile un sempre maggiore coinvolgimento di varie figure professionali per diffondere e implementare le conoscenze circa i vantaggi derivanti dall'uso alimentare dei legumi

Dipartimento di Scienze Agrarie Alimentari e Agro-Ambientali
Università di Pisa

2016
ANNO INTERNAZIONALE DEI LEGUMI

NUTRA FOOD
Università di Pisa

GIORNATA DI STUDIO

I LEGUMI: SEMI NUTRIENTI PER UN FUTURO SOSTENIBILE

2 dicembre 2016 - ore 9:00-17:30

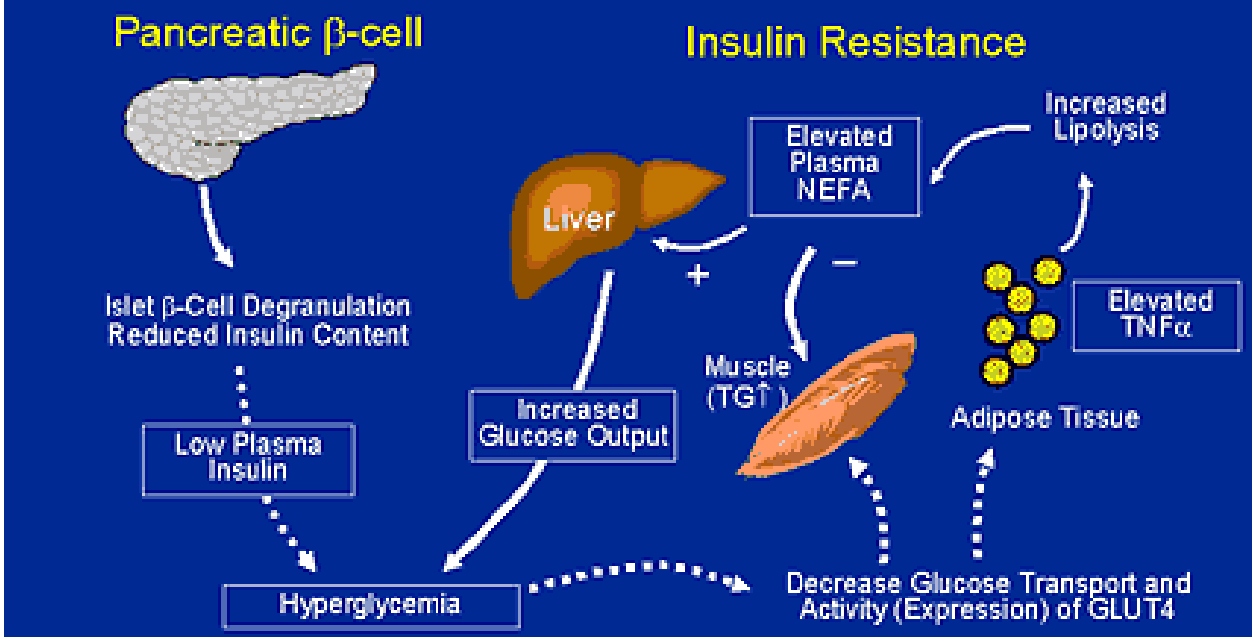
Sala Convegni del Dipartimento di Scienze Agrarie, Alimentari e Agro-ambientali
Via del Borghetto 80, Pisa

Annibale Carracci 'Il Mangiafagioli', 1584-1585

Castelluccio di Norcia, Umbria, nel 2015, patria delle lenticchie e della roveja

Grazie per la vostra attenzione!!!

Insulin Resistance and β-Cell Dysfunction Produce Hyperglycemia in Type 2 Diabetes Mellitus



NEFA = nonesterified fatty acid.
TG = triglyceride.

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Primary Prevention of Cardiovascular Disease with a Mediterranean Diet

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Table 1. Summary of Dietary Recommendations to Participants in the Mediterranean-Diet Groups and the Control-Diet Group.

Food	Goal
Mediterranean diet	
Recommended	
Olive oil*	≥4 tbsp/day
Tree nuts and peanuts†	≥3 servings/wk
Fresh fruits	≥3 servings/day
Vegetables	≥2 servings/day
Fish (especially fatty fish), seafood	≥3 servings/wk
Legumes	≥3 servings/wk
Sofrito‡	≥2 servings/wk
White meat	Instead of red meat
Wine with meals (optionally, only for habitual drinkers)	≥7 glasses/wk
Discouraged	
Soda drinks	<1 drink/day
Commercial bakery goods, sweets, and pastries§	<3 servings/wk
Spread fats	<1 serving/day
Red and processed meats	<1 serving/day
Low-fat diet (control)	
Recommended	
Low-fat dairy products	≥3 servings/day
Bread, potatoes, pasta, rice	≥3 servings/day
Fresh fruits	≥3 servings/day
Vegetables	≥2 servings/day
Lean fish and seafood	≥3 servings/wk
Discouraged	
Vegetable oils (including olive oil)	≤2 tbsp/day
Commercial bakery goods, sweets, and pastries§	≤1 serving/wk
Nuts and fried snacks	≤1 serving /wk
Red and processed fatty meats	≤1 serving/wk
Visible fat in meats and soups¶	Always remove
Fatty fish, seafood canned in oil	≤1 serving/wk
Spread fats	≤1 serving/wk
Sofrito‡	≤2 servings/wk