Regulación e introducción de Nuevos Alimentos y Suplementos Alimenticios en México

Javier Luna Carrasco

Tendencias en México

LA SALUD ESCALA 1 LUGAR EN EL RANKING DE PREOCUPACIONES PARA EL 2011, Y SE ANTEPONE AL CALENTAMIENTO GLOBAL

Más preocupados por inseguridad, inflación y salud que en 2010

CRISIS ECONÓMICA GLOBAL
Es mas preocupante en México que en LATAM (+13.8)

Fuente: Kantar Worldpanel Latam - ConsumerWatch 2011. % de hogares que consideran estos temas entre sus 3 principales preocupaciones

GORDITOS Y

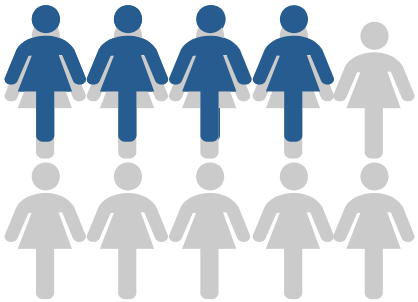
SIN CULPA

71%

De los mexicanos sufren sobrepeso y obesidad

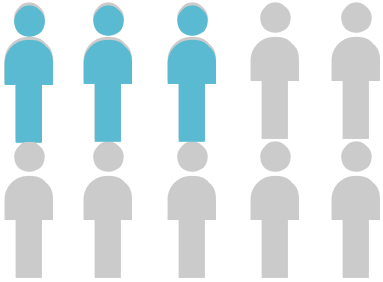
31% de los mexicanos sufren obesidad y NO hacen mucho al respecto para tratarlo

EL SOBREPESO: UNA PREOCUPACIÓN MEXICANA
¿QUIÉN LO SUFRE MÁS? ¿ELLA O ÉL?



44%

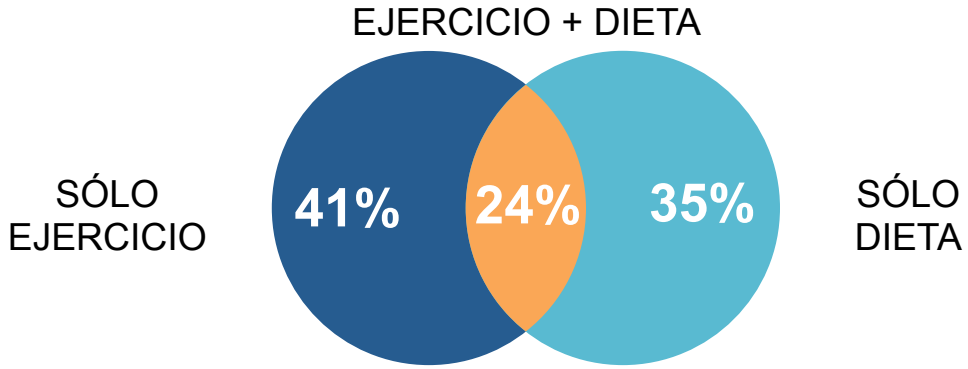
31%



¿Y CUÁNTOS HACEN ALGO PARA COMBATIRLO?

3 x 1

CON EJERCICIO, DIETA, O AMBAS:



ES EL EJERCICIO FÍSICO DONDE SE CONCENTRA LA APUESTA

EN MÉXICO…

GANA CON VENTAJA EL COMER MEJOR

¿CUÁL ES LA TENDENCIA?

¿COMER MENOS?	¿COMER MEJOR?
grasas	
20%	45%

El promedio de personas que hacen Dieta en LATAM es del 30%!

¿QUÉ IMPLICA COMER MEJOR PARA EL MEXICANO?

MÁS SANO Y EQUILIBRADO

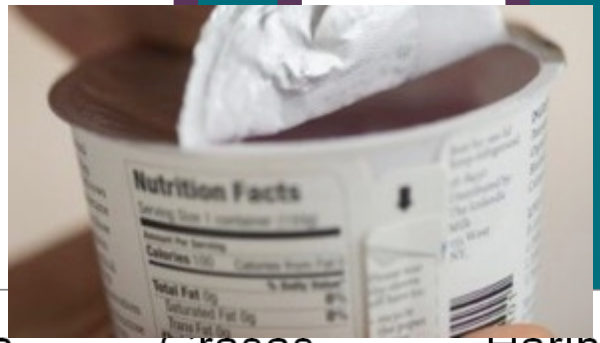
He cambiado mi alimentación hacia comidas más sanas y equilibradas

45% ✕

ATENCIÓN A INFO NUTRICIONAL

Presto más atención que antes a la información nutricional de las comidas y bebidas

51% ✕



RE

In
el co

"Intento reducir el consumo de…"

Carne Margarina Grasas Saturadas Harinas

LATAM trata de reducir su consumo de grasas saturadas, sal y frituras!!!

? ¡MEJOR EN LA

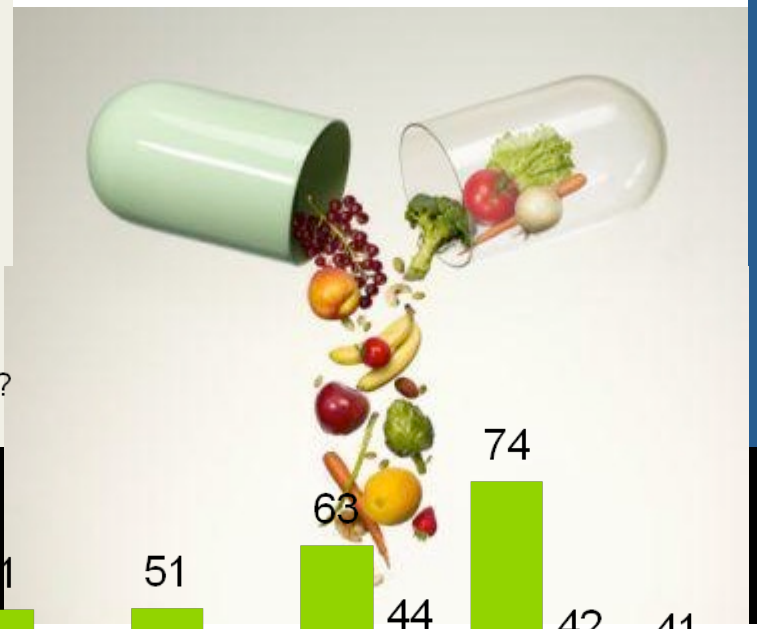
MÉXICO CON EL MENOR CONSUMO

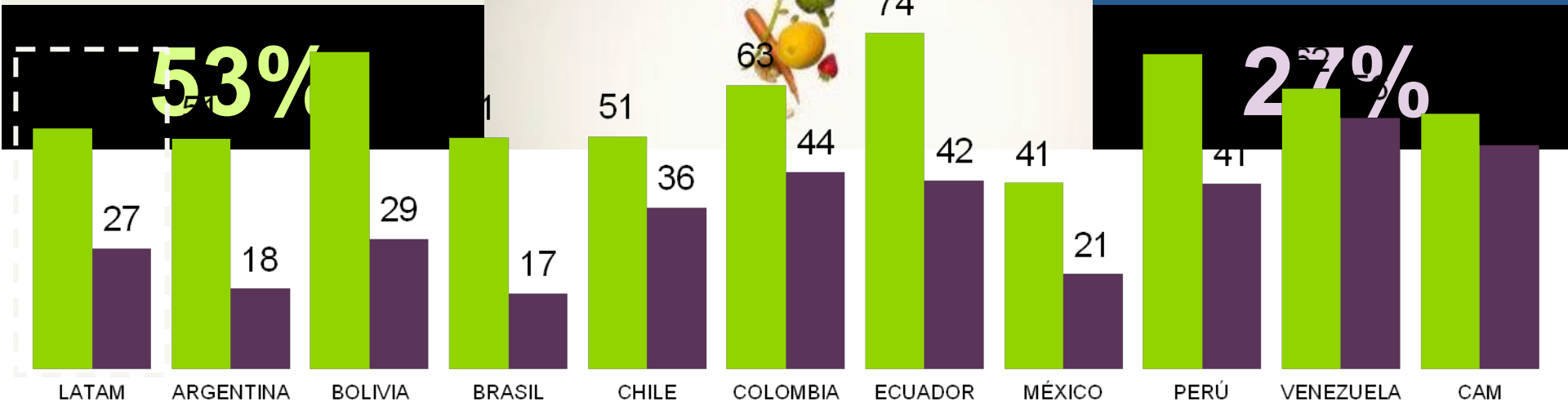
EN ECUADOR 7 DE CADA 10 LOS SUMA AL CESTO

VENEZOLANOS: ALIMENTOS + VITAMINAS

BRASILEROS: PREFIEREN EN LA COMIDA

VITAMÍNICOS?





Fuente: Kantar Worldpanel Latam - ConsumerWatch 2011

Top 2Box (Completamente de acuerdo + De acuerdo)

85% DE LOS MÉXICANOS NO CONSUMEN PRODUCTOS LIGHT

PRINCIPALMENTE NO CREEN EN EL BENEFICIO NI GUSTA EL SABOR

40% México **33%** LATAM

No creo en el beneficio

37% México **34%** LATAM

No me gusta el sabor

36% México **44%** LATAM

Prefiero alimentos más naturales

18% México **29%** LATAM

Su precio es más caro

Fuente: Kantar Worldpanel Latam - ConsumerWatch 2011 Base de cálculo: Base 100 aquellos que NO consumen productos light

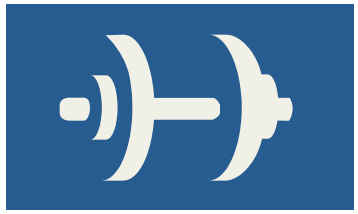
OXIGENARSE ES PRIORIDAD: CAMINAR, CORRER AL AIRE LIBRE Y AERÓBICOS SON LAS PREFERIDAS

% ACTIVIDAD FÍSICA POR ACTIVIDAD

Solo el 38% de los Mexicanos declaran que les gusta mantener su cuerpo en forma, por debajo de LATAM en Pesas y Natación

	CAMINAR	CORRER	BICICLETA	GIMNASIO / CLASES
LATAM	35.8%	5.4%	6.7%	4.7%
MÉXICO	40.9%	6.2%	4.5%	4.6%



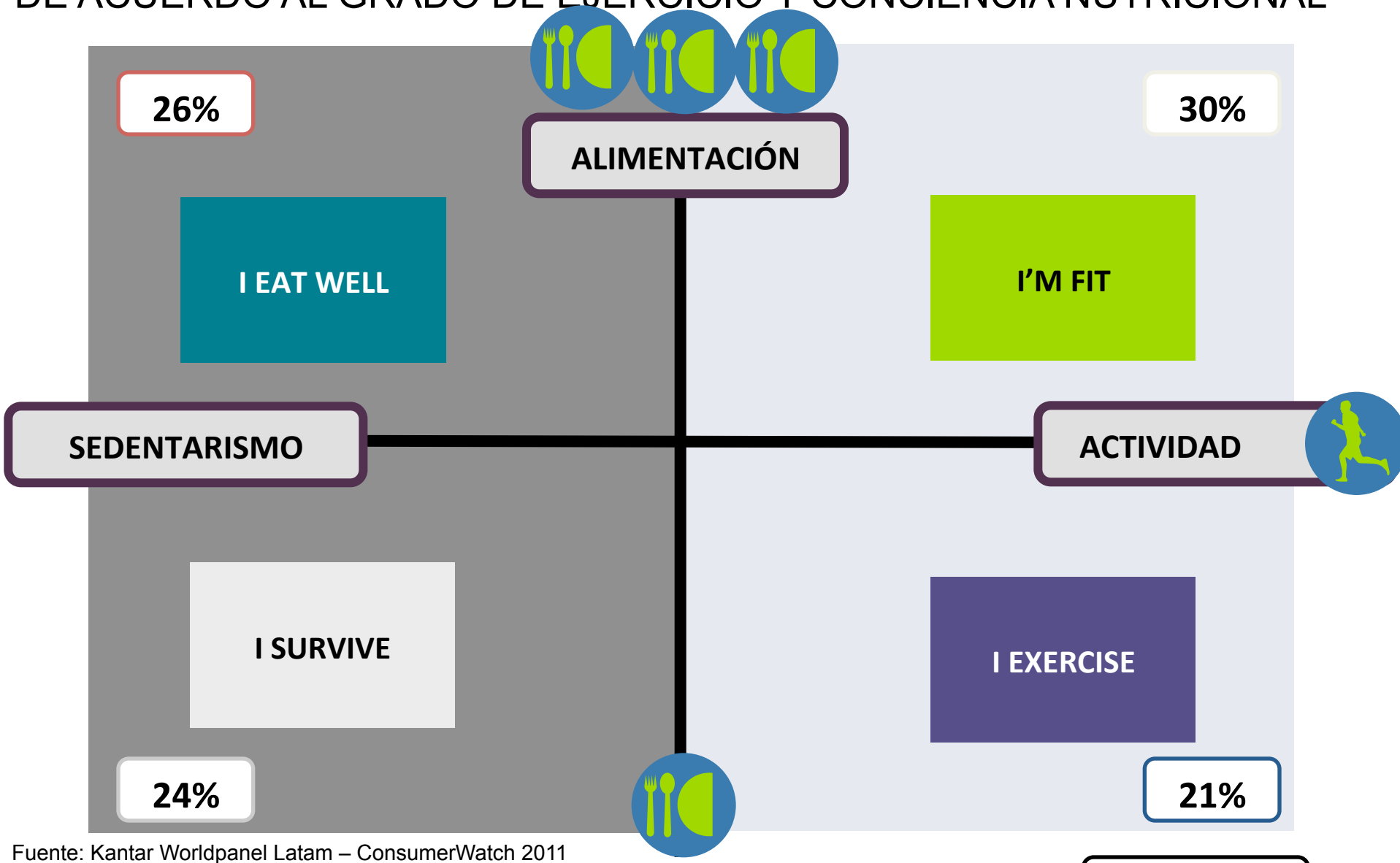



¿QUÉ ACTITUD ADOPTAN LOS LATINOS RESPECTO A SU SALUD?

NOS ENCONTRAMOS CON 4 ESTEREOTIPOS

I´M FIT	I EXERCISE	I EAT WELL	I SURVIVE

4 PROTOTIPOS DE CONSUMIDORES
DE ACUERDO AL GRADO DE EJERCICIO Y CONCIENCIA NUTRICIONAL



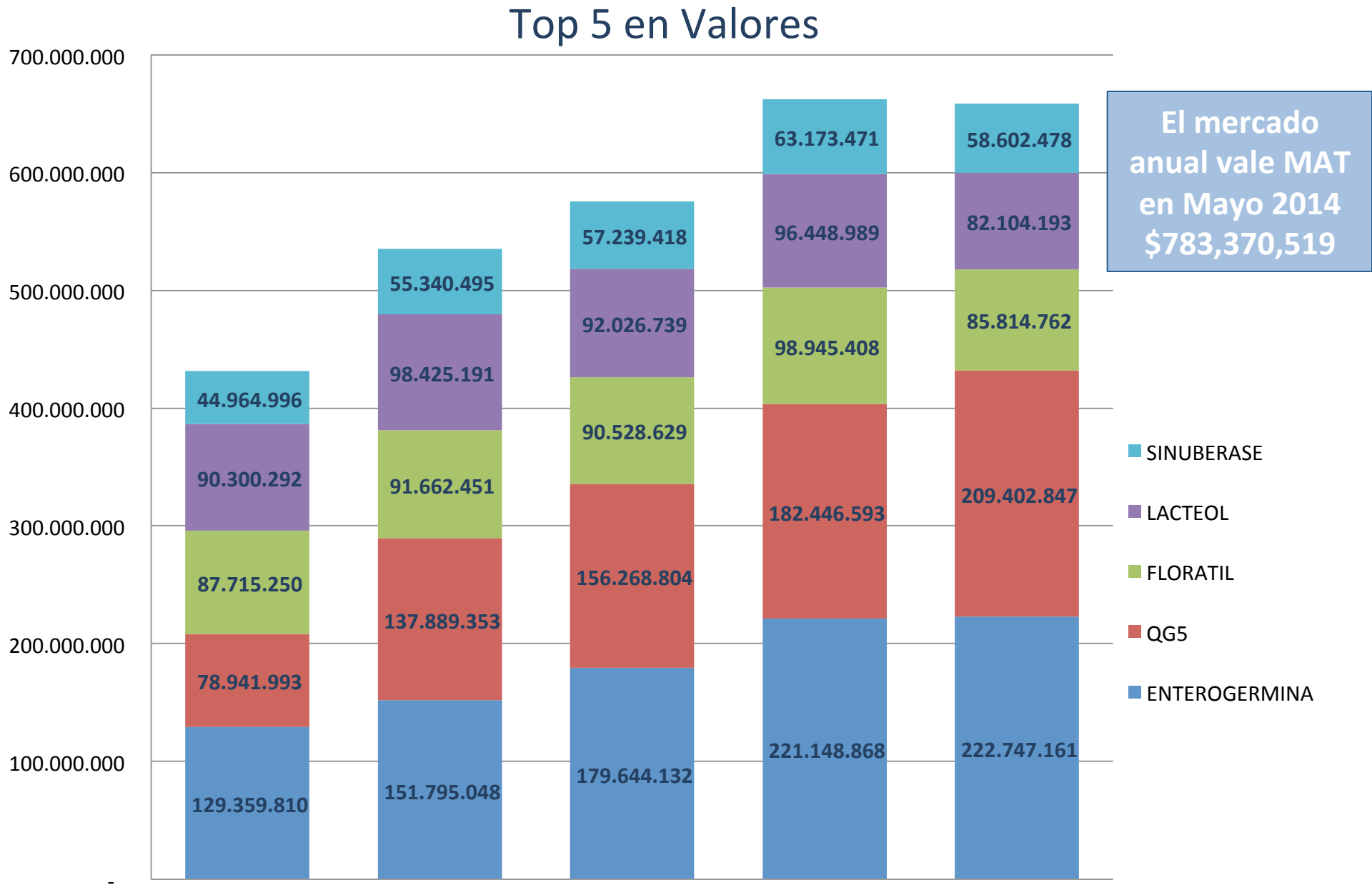
Fuente: Kantar Worldpanel Latam – ConsumerWatch 2011

Datos México

Datos de Mercado Mexicano varios segmentos

Probióticos

Potenciales competidores de probióticos



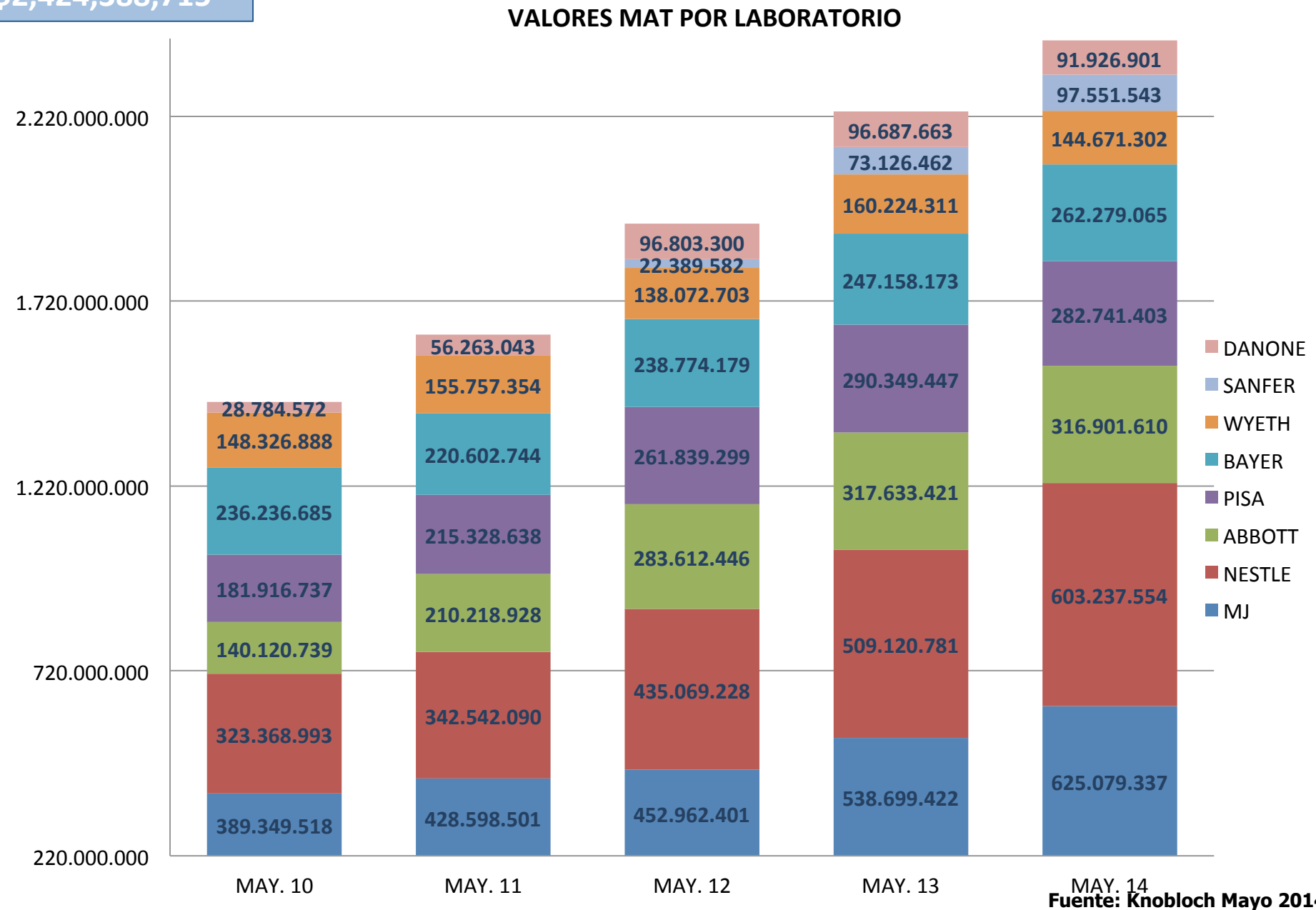
El mercado anual vale MAT en Mayo 2014 $783,370,519

Fuente: Knobloch Mayo 2014

Fórmulas infantiles

El mercado total en Mayo 2014 vale $2,424,388,715

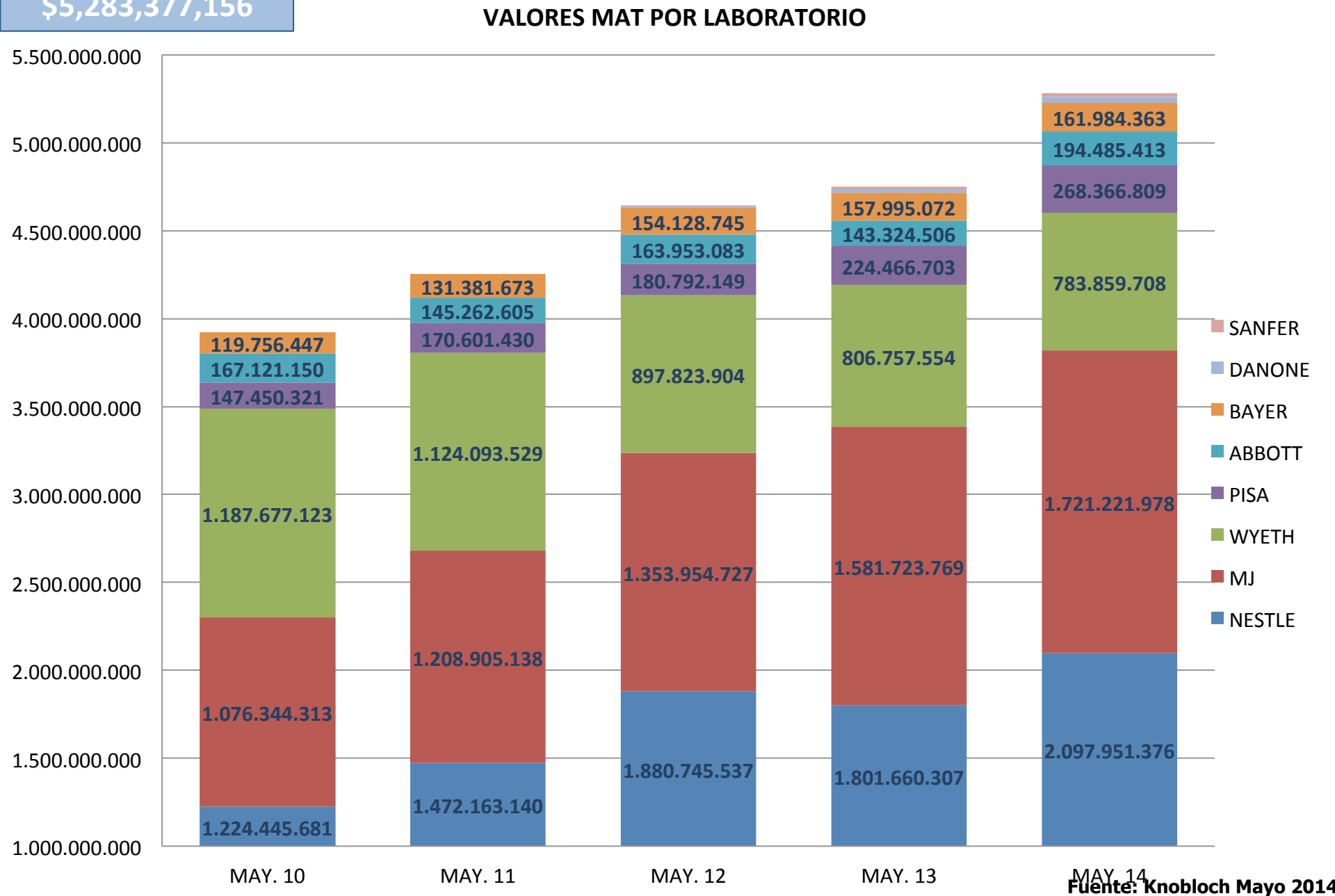
Fórmulas Infantiles de Tolerancia



Fuente: Knobloch Mayo 2014

El mercado total en Mayo 2014 vale $5,283,377,156

Fórmulas Infantiles de Rutina



Fuente: Knobloch Mayo 2014

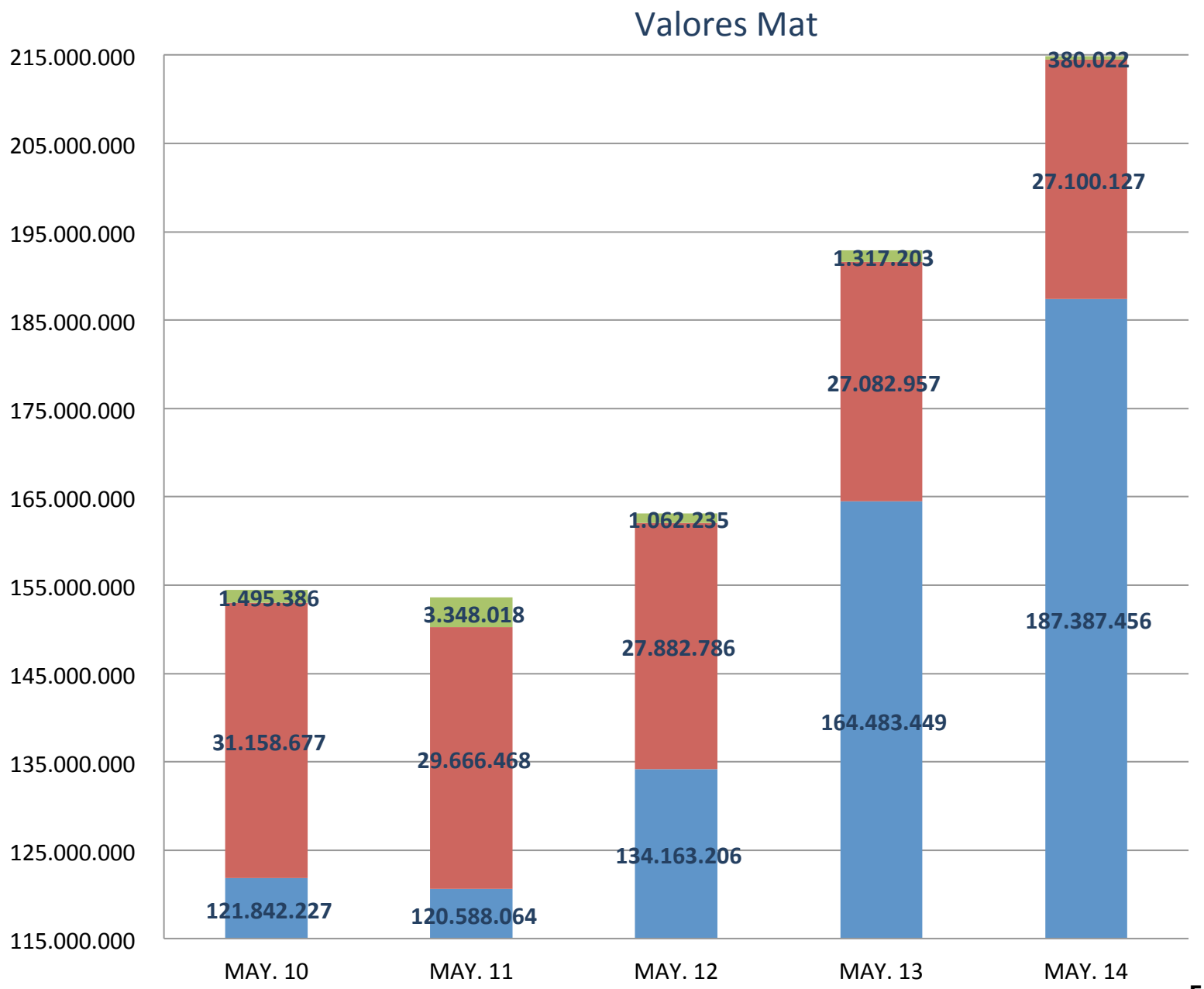
Suplementos alimenticios para personas con diabetes

Abbott
- Glucerna
- Glucerna Diabetes
- Glucerna SR

Pisa
- Enterex Diabetic

Industrias Suanca S.A de C.V
- Glucopromin

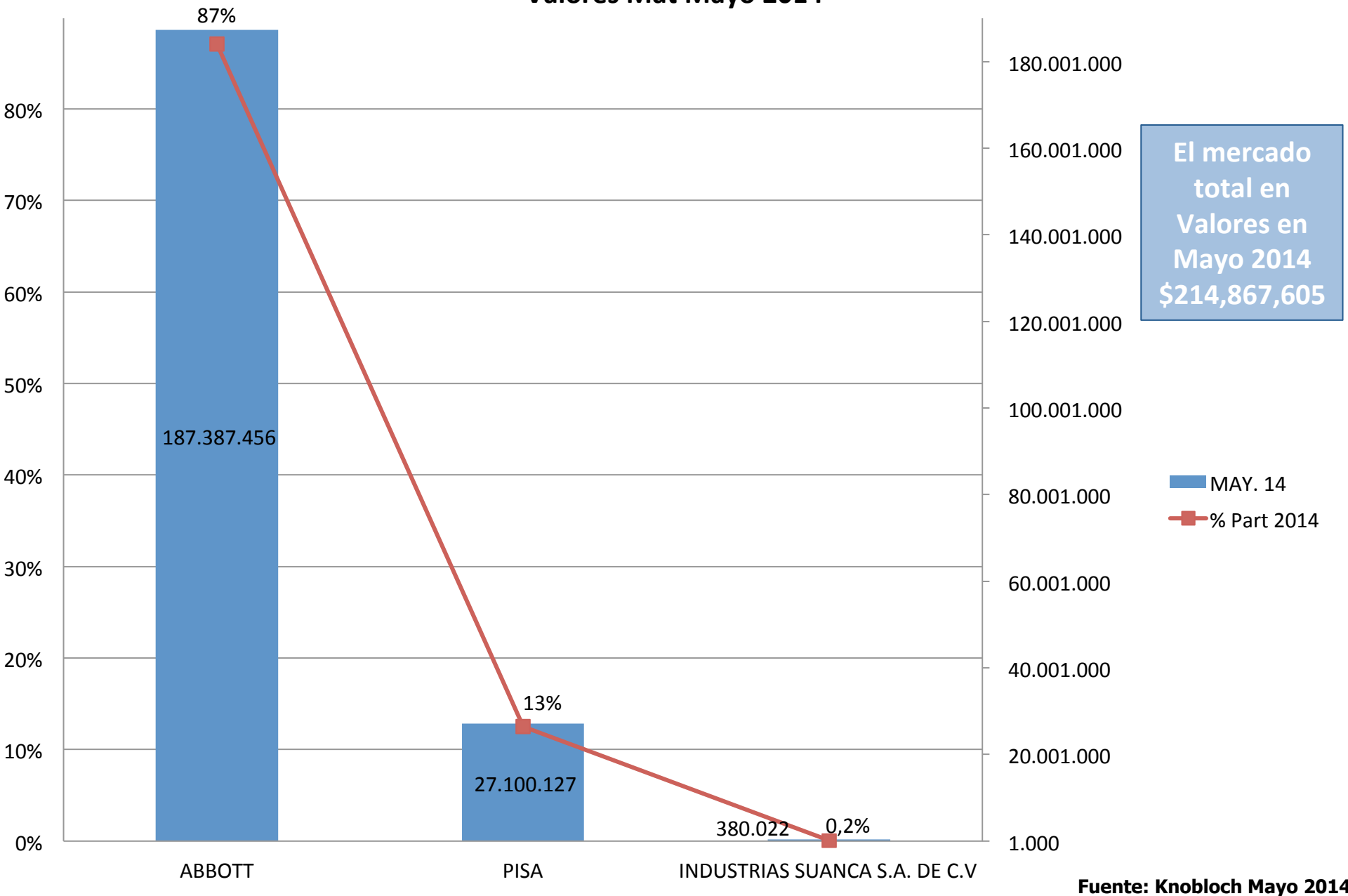
Suplementos alimenticios para personas con diabetes (Valores Mat)



El mercado total $878,958,281 en Mayo 2014 $214,867,605

Fuente: Knobloch Mayo 2014

Suplementos alimenticios para personas con diabetes (Valores Mat Mayo 2014)

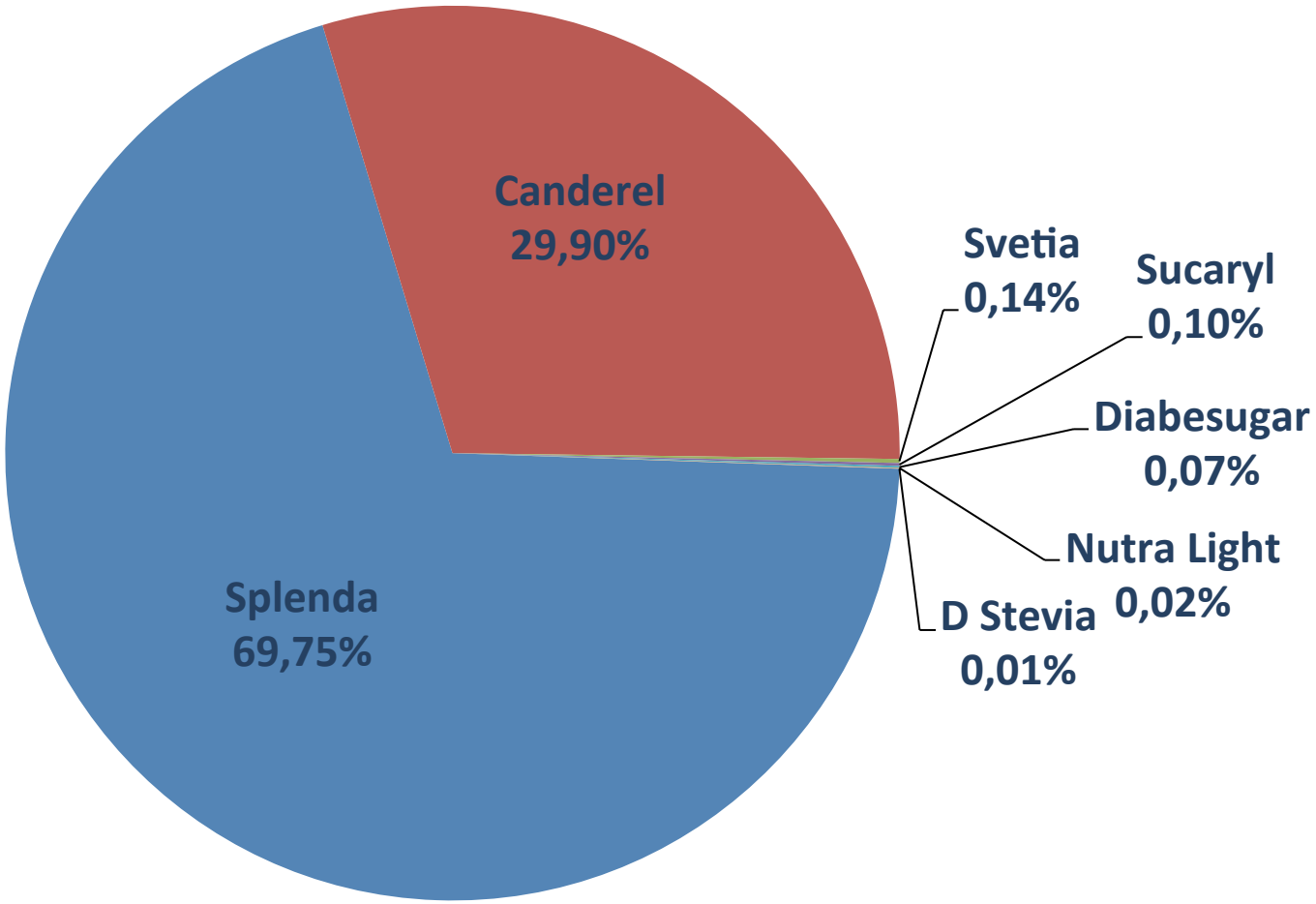


Fuente: Knobloch Mayo 2014

Edulcorantes

% Participación de Mercado de edulcorantes Mat Octubre 2013

El mercado total Mat Oct 2013 en valor es de $33,452,451



Fuente: Knobloch Mayo 2014

Situaciones al lanzar nuevos productos al mercado

Lanzamiento de productos al mercado

MINTEL

Government Ministries and Agencies who have participation in Mexican Regulation

SALUD
SECRETARÍA DE SALUD

Comisión Federal para la Protección contra Riesgos Sanitarios → **HEALTH**

SAGARPA
SECRETARÍA DE AGRICULTURA, GANADERÍA, DESARROLLO RURAL, PESCA Y ALIMENTACIÓN

Senasica → **AGRICULTURE**

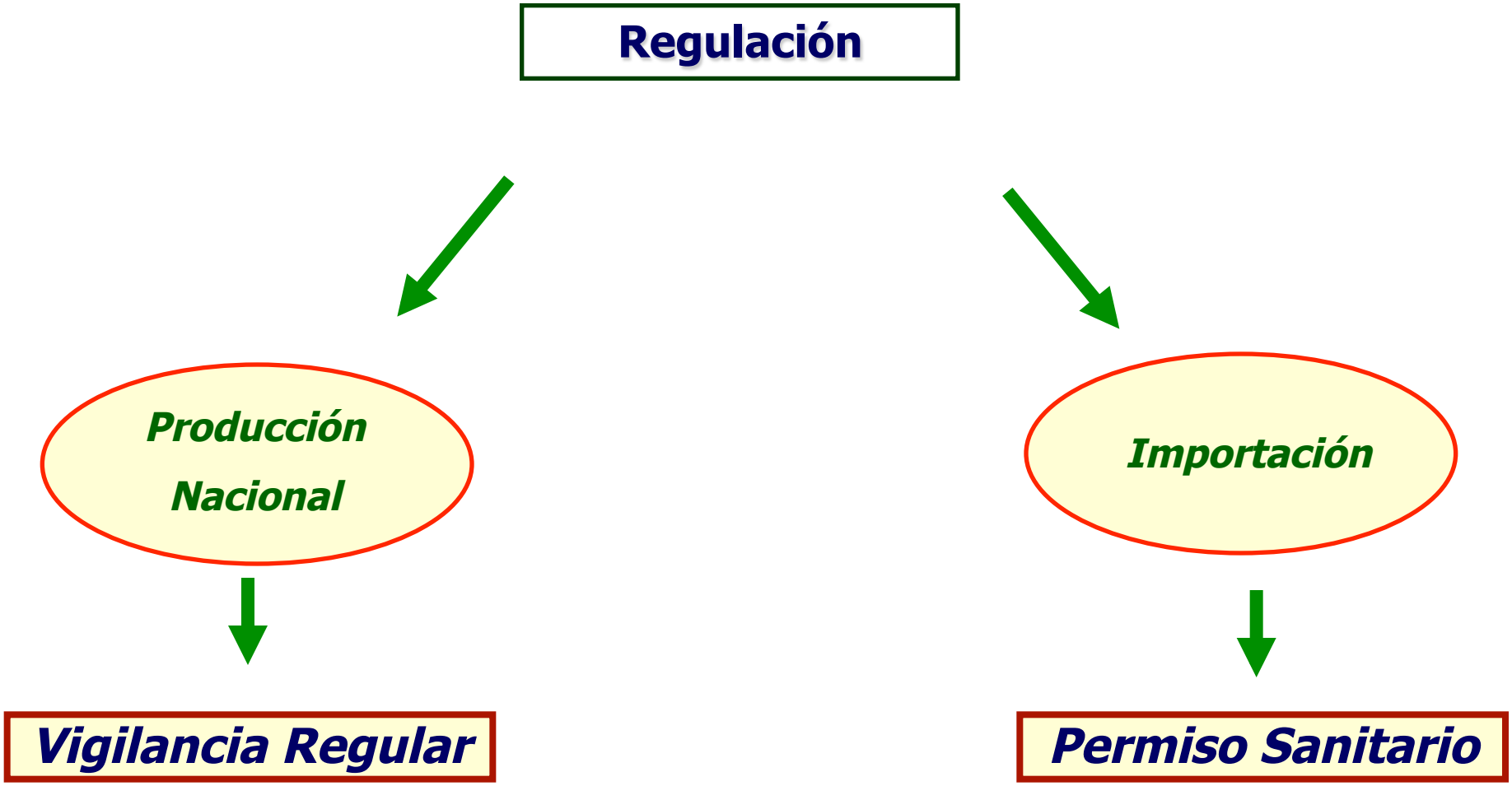
SEMARNAT
SECRETARÍA DE MEDIO AMBIENTE Y RECURSOS NATURALES

PROFEPA
PROCURADURÍA FEDERAL DE PROTECCIÓN AL AMBIENTE → **ENVIRONMENT**

SE
MINISTRY OF THE ECONOMY

Profeco
PROCURADURÍA FEDERAL DEL CONSUMIDOR → **COMERCIAL PUBLICITY AND LABELLING**

Vías de Regulación


Regulación
Producción Nacional
Importación
Vigilancia Regular
Permiso Sanitario

Vigilancia regular

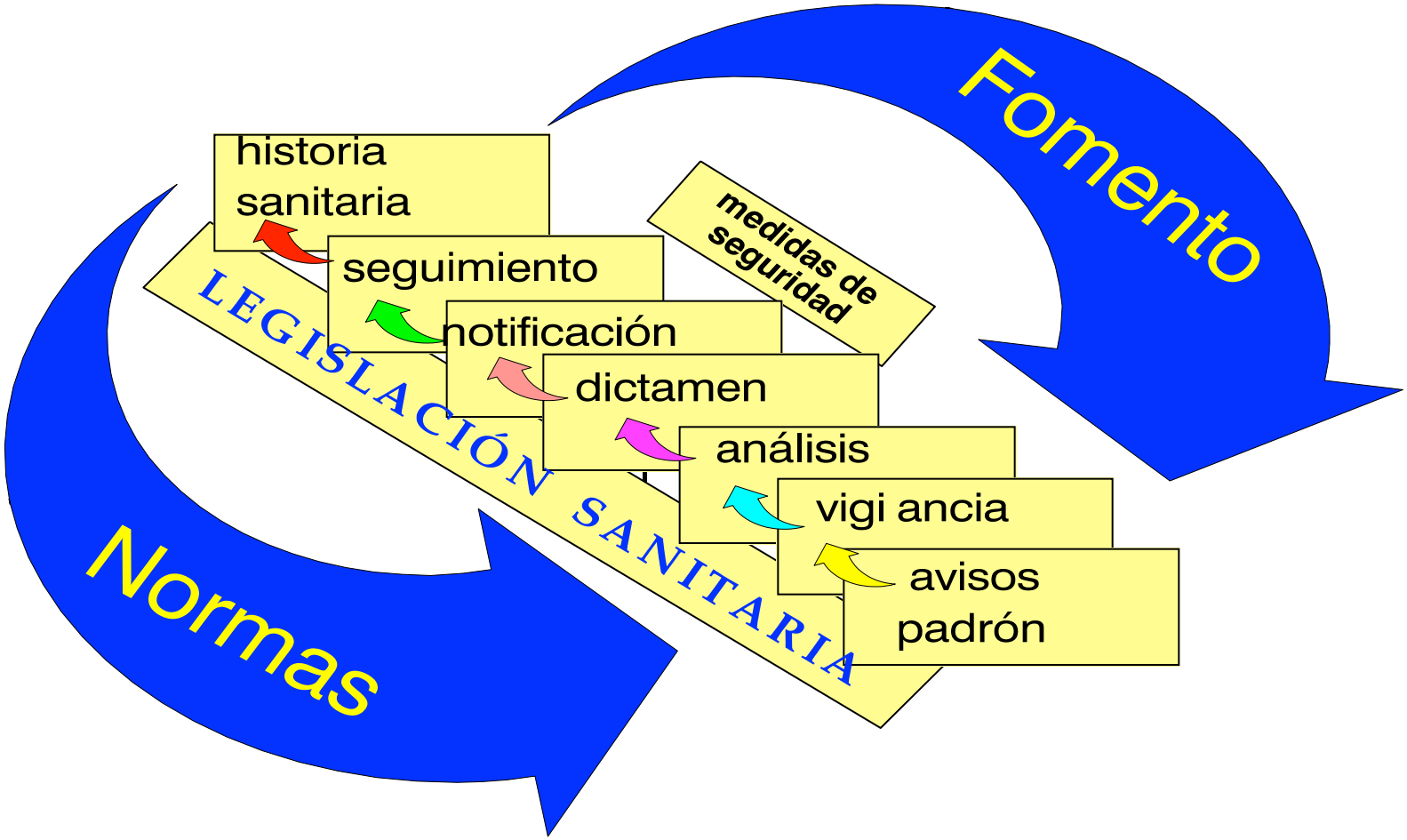
SÍNTESIS DE LAS DISPOSICIONES REGULATORIAS

ESTABLECIMIENTOS

LOS COMERCIALIZADORES Y PRODUCTORES DE SUPLEMENTOS ALIMENTICIOS ESTAN OBLIGADOS A PRESENTAR AVISO DE FUNCIONAMIENTO ANTE LA SECRETARIA DE SALUD

ACUERDO NO. 141 POR EL QUE SE DETERMINAN LOS ESTABLECIMIENTOS SUJETOS A AVISO DE FUNCIONAMIENTO D.O.F. JULIO 22, 1997.

MODELO DE CONTROL SANITARIO



MEDIDAS DE SEGURIDAD

FUNDAMENTADO EN EL ARTICULO 404 Y 414 DE LA LEY GENERAL DE SALUD.

Buenas prácticas sanitarias

NOM-251 Prácticas de higiene y sanidad para el proceso de alimentos, bebidas no alcohólicas y alcohólicas

2 ejemplos de introducción de productos en el mercado mexicano

Fórmulas infantiles (Food category)

Infant formula (Food category) I

Start of a new project

Manufacturing Documents Request → **QQ, BOM, MS, PTI, MAIT**

70 aniversario Silanes

1 week

RA Assessment vs. Regulation → **Norm 131, Norm 051. Trade Mark Registration**

3 days

Infant formula (Food category) II

Custom Code Classification → **Import Department**

2 weeks

Business Decision

Label Development →

1.- Development of the Legal Texts.
2.- Development of the Label.
3.- Ask for the Legal Certificate

NORMEX

2 months

Infant formula (Food category) III

COFEPRIS Notification → **Requires to fullfil Cofepris format**

Cofepris
Comisión Federal para la Protección contra Riesgos Sanitarios

1 day

Documents needed for Importation Process → **RA and Import Department. Pending on the origin of the products.**

2 months

Request for Tin Plates → **1st production. We need this for the import process**

70 aniversario
Silanes

Infant formula (Food category) IV

Request for Import Permit to Cofepris in Food category

We submit:
- **COFS**
- **COA**
- **Sanitary Certificate ***
- **Label**

Cofepris
Comisión Federal para la Protección contra Riesgos Sanitarios

10 days

Achieve the Import Permit

- **Sometimes COFEPRIS orders an Inspection at warehouse**
- **In the case of a local overlabel Normex makes an Inspection at warehouse**

Cofepris
Comisión Federal para la Protección contra Riesgos Sanitarios

NORMEX

3 days

Infant formula (Food category) V

WE CAN SALE THE PRODUCTS

The products in the market are candidates for a local Inspection

- **COFEPRIS**
- **PROFECO**

Cofepris
Comisión Federal para la Protección contra Riesgos Sanitarios

Profeco
Procuraduría Federal del Consumidor

Alimentación Especializada (Drug category)

(Specialized Nutrition – Drug category-) I

Start of a new project

Manufacturing Documents Request → **QQ, BOM, MS, PTI, MAIT**

1 week

RA Assessment vs. Regulation → **Trade Mark Registration**

3 days

Glucerna (Specialized Nutrition – Drug category-) II

Custom Code Classification

→ **Import Department ***

2 weeks

Business Decision

Dossier

→
1.- Clinical information
2.- Technical information
3.- Scientific information
4.- Legal Texts (Label project)
5.- Therapeutic indication
6.- Legal information

3 months

Glucerna (Specialized Nutrition – Drug category-) III

Submit for Registration in COFEPRIS → **We submit the Dossier**

Cofepris — Comisión Federal para la Protección contra Riesgos Sanitarios

12/18 months

Label Development → **Label development using the Legal Texts**

iQ innovative Quality

1 month

We achieve registration letter

Glucerna (Specialized Nutrition – Drug category-) IV

Glucerna (Specialized Nutrition – Drug category-) V

WE CAN SALE THE PRODUCTS

The product's in the market are candidates to a local Inspection

- **COFEPRIS**
- **PROFECO**

Cofepris
Comisión Federal para la Protección contra Riesgos Sanitarios

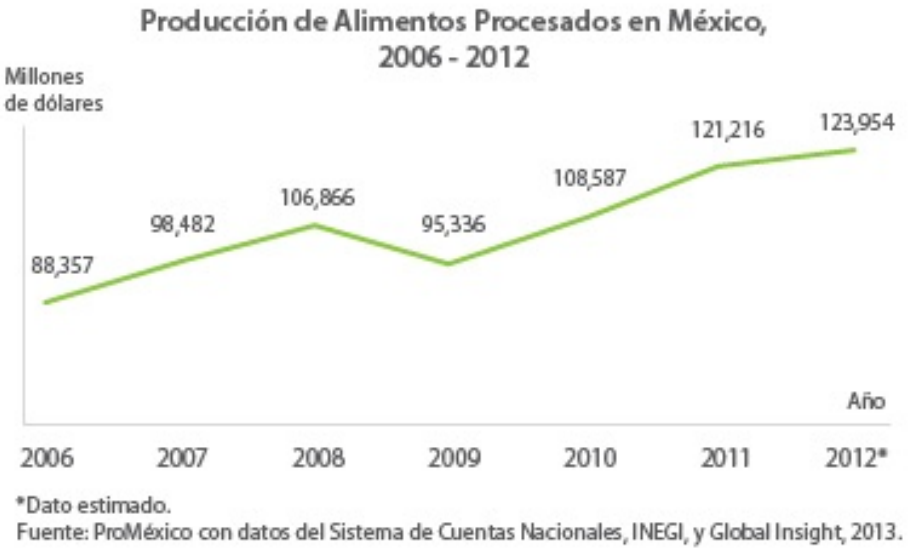
Profeco
Procuraduría Federal del Consumidor

MODELO ACTUAL DEL MERCADO MEXICANO

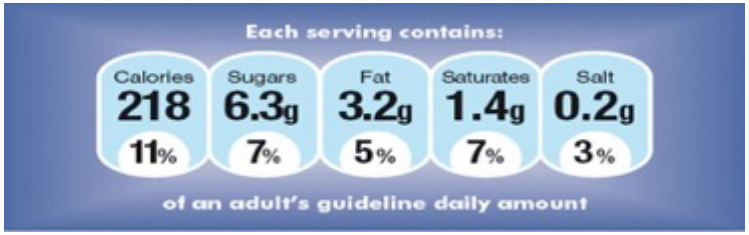
Esquema actual de la Distribución en México

Distribuidor	% PARTICIP.
NADRO S.A.P.I. de C.V.	38.38%
CASA MARZAM S.A. de C.V.	17.16%
FARMACIA GUADALAJARA S.A. de C.V.	14.89%
SERVICIOS EN PUERTOS Y TERMINALES	7.61%
FARMACOS NACIONALES S.A.de C.V.	6.78%
RAMA FARMACEUTICA S.A. C.V.	6.24%
CASA SABA S.A. de C.V.	4.22%
ALMACEN DE DROGAS S.A. de .C.V.	2.22%
DISTRIBUIDORA DEKAFARMA	1.33%
FARMACIA PARIS S.A. DE C.V.	0.89%
SUPERMERCADOS INTERNACIONALES	0.47%
DROGUEROS S.A. de C.V.	0.12%
PROVEEDORA DE MEDICAMENTOS	-0.06%
GRUPO CASA SABA S.A. de C.V.	-2.23%

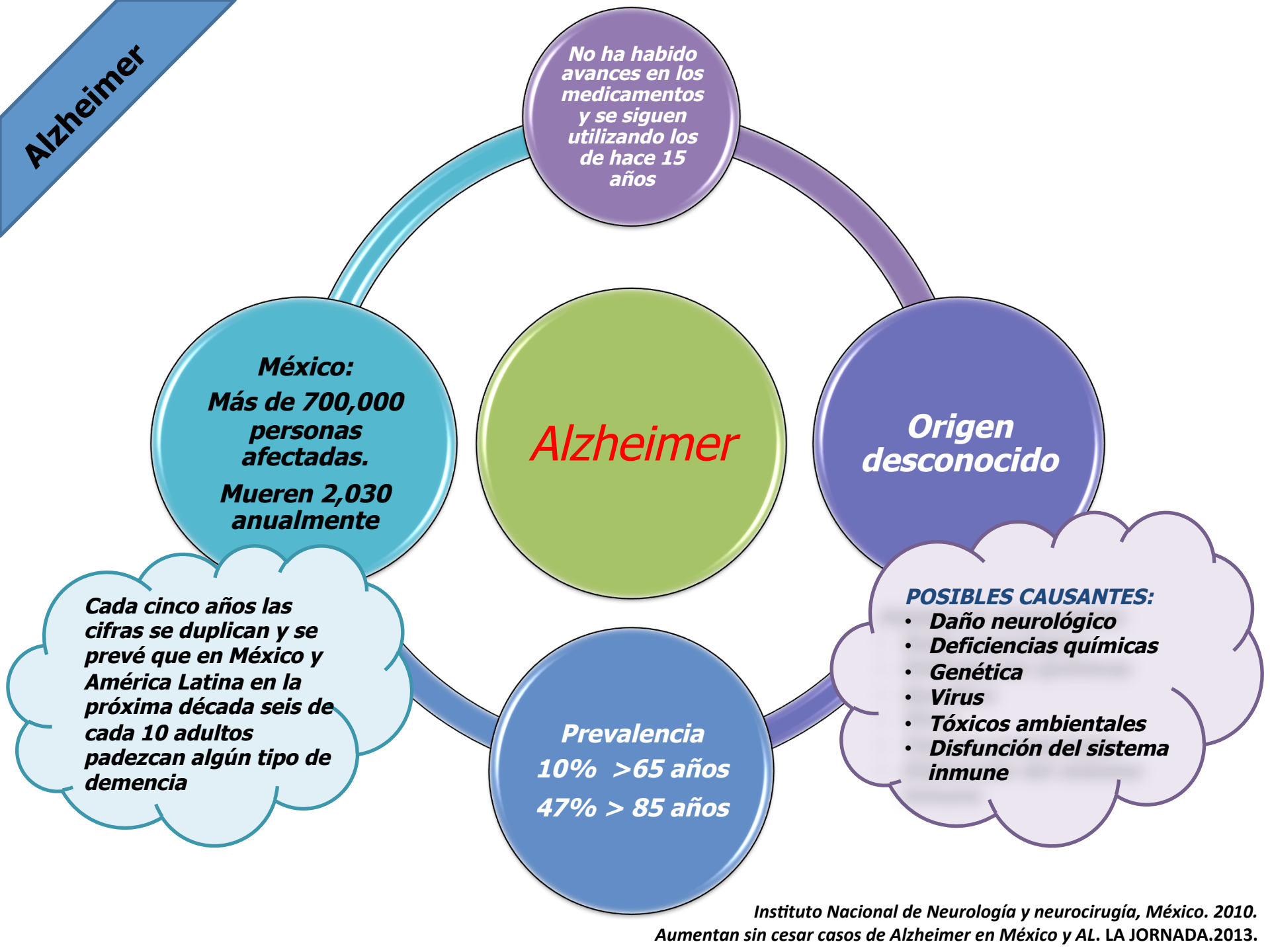
Esquema actual de la Industria de Consumo en México



*Dato estimado.
Fuente: ProMéxico con datos del Sistema de Cuentas Nacionales, INEGI, y Global Insight, 2013.



Tendencias futuras de productos



Instituto Nacional de Neurología y neurocirugía, México. 2010.

Aumentan sin cesar casos de Alzheimer en México y AL. LA JORNADA.2013.

Alzheimer

[Intervention Review]

Omega 3 fatty acid for the prevention of cognitive decline and dementia

Emma Sydenham[1], Alan D Dangour[2], Wee-Shiong Lim[3]

[1]Cochrane Injuries Group, London School of Hygiene & Tropical Medicine, London, UK. [2]Department of Nutrition and Public Health Intervention Research, London School of Hygiene & Tropical Medicine, London, UK. [3]Department of Geriatric Medicine, Tan Tock Seng Hospital, Singapore, Singapore

Contact address: Emma Sydenham, Cochrane Injuries Group, London School of Hygiene & Tropical Medicine, Room 180, Keppel Street, London, WC1E 7HT, UK. emma.sydenham@Lshtm.ac.uk.

Editorial group: Cochrane Dementia and Cognitive Improvement Group.
Publication status and date: New search for studies and content updated (conclusions changed), published in Issue 6, 2012.
Review content assessed as up-to-date: 6 April 2012.

Citation: Sydenham E, Dangour AD, Lim WS. Omega 3 fatty acid for the prevention of cognitive decline and dementia. *Cochrane Database of Systematic Reviews* 2012, Issue 6. Art. No.: CD005379. DOI: 10.1002/14651858.CD005379.pub3.

Menos grasas saturadas y más Omega-3 para una memoria efectiva

Neurology
www.neurology.org

doi: 10.1212/WNL.0b013e3182904f69
Neurology April 30, 2013 vol. 80 no. 18 1684-1692

Article

Adherence to a Mediterranean diet and risk of incident cognitive impairment

Georgios Tsivgoulis, MD, Suzanne Judd, PhD, Abraham J. Letter, MS, Andrei V. Alexandrov, MD, George Howard, DrPH, Fadi Nahab, MD, Frederick W. Unverzagt, PhD, Claudia Moy, PhD, Virginia J. Howard, PhD, Brett Kissela, MD and Virginia G. Wadley, PhD

+ SHOW AFFILIATIONS | + SHOW FULL DISCLOSURES
Correspondence to Dr. Tsivgoulis: tsivgoulisgiorg@yahoo.gr

doi: 10.1212/WNL.0b013e3182904f69
Neurology April 30, 2013 vol. 80 no. 18 1684-1692

Alzheimer

EDITORIAL

Editorials represent the opinions of the authors and *JAMA* and not those of the American Medical Association.

Vitamin E, Memantine, and Alzheimer Disease

Denis A. Evans, MD; Martha Clare Morris, ScD; Kumar Bharat Rajan, PhD

The report by Dysken et al[1] in this issue of *JAMA* raises interesting issues about drug therapy for Alzheimer disease (AD) and emphasizes the importance of closely following this rapidly evolving field. In this randomized clinical trial, older veterans (97% men) with AD and Mini-Mental State Examination (MMSE) scores of 12 to 26 who were receiving acetylcholinesterase inhibitors were assigned to 1 of 4 treatment groups: receiving synthetic vitamin E (alpha tocopherol, 2000 IU/d); memantine, 20 mg/d; both agents; or placebo.

Related article page 33

As in almost all trials of therapy in AD, death was frequent (128 of 613 study participants), medication adherence was moderate, and loss to follow-up was greater than optimal, reflecting the practical challenges in conducting randomized trials among people with this disease of older age.

The primary trial outcome was score on the Alzheimer's Disease Cooperative Study/Activities of Daily Living (ADCS-ADL) Inventory; secondary outcomes included scores on the MMSE and the Alzheimer's Disease Assessment Scale-Cognitive subscale (ADAS-cog). Compared with individuals assigned to placebo, those assigned to vitamin E alone experienced to support its use because the comparison of the group assigned to memantine with the group assigned to placebo suggested no differences in either the primary ADCS-ADL outcome or in the secondary cognitive outcomes. The negative interaction between alpha tocopherol treatment, which was significantly beneficial alone, and memantine treatment in predicting the primary trial outcome is of concern and deserves further investigation. No formal comparison of the primary outcome was reported between the group assigned to alpha tocopherol alone and the group assigned to the combination of alpha tocopherol and memantine; the statement in the "Discussion" that "... the combination of alpha tocopherol and memantine had less effect than either alpha tocopherol alone or memantine alone" is difficult to support in the absence of such a comparison with significance testing.

For vitamin E, the results of this trial are more encouraging because of the significant difference from the placebo group observed for the primary outcome and the absence of severe adverse effects. A previous trial[4] among individuals with moderate to severe AD found delayed disease progression with 2000 IU/d of alpha tocopherol both alone and in combination with selegiline. The results of a trial[5] of vitamin E therapy among

Alzheimer

ELSEVIER

Archives of Medical Research 43 (2012) 699–704

Archives of Medical Research

REVIEW ARTICLE

Can Nutraceuticals Prevent Alzheimer's Disease? Potential Therapeutic Role of a Formulation Containing Shilajit and Complex B Vitamins

Carlos Carrasco-Gallardo,[a,b] Gonzalo A. Farías,[a] Patricio Fuentes,[c] Fernando Crespo,[d] and Ricardo B. Maccioni[a,b]

[a]*International Center for Biomedicine, ICC, Santiago, Chile*

[b]*Laboratory of Cellular and Molecular Neuroscience, Faculty of Science, Universidad de Chile, Santiago, Chile*

[c]*Cognitive Neurology and Dementia Unit, Neurology Service, Hospital del Salvador & Geriatrics Section, Medicine Department, Clinical Hospital of Universidad de Chile, Santiago, Chile*

[d]*Faculty of Engineering, Industrial Engineering School, Universidad de Valparaíso, Santiago, Chile*

Received for publication September 28, 2012; accepted October 18, 2012 (ARCMED-D-12-00546).

Alzheimer's disease (AD) is a brain disorder displaying a prevalence and impact in constant expansion. This expansive and epidemic behavior is concerning medical and public opinion while focusing efforts on its prevention and treatment. One important strategy to prevent this brain impairment is based on dietary changes and nutritional supplements, functional foods and nutraceuticals. In this review we discuss the potential contributions of shilajit and complex B vitamins to AD prevention. We analyze the status of biological studies and present data of a clinical trial developed in patients with mild AD. Studies suggest that shilajit and its active principle fulvic acid, as well as a formula of shilajit with B complex vitamins, emerge as novel nutraceutical with potential uses against this brain disorder.

Key Words: Alzheimer's disease, Tau protein, Nutraceuticals, Functional foods, Shilajit, Fulvic acid.

Alzheimer

J Sci Food Agric. 2013 Nov 8. doi: 10.1002/jsfa.6473. [Epub ahead of print]

Effect of flavonoids on learning, memory and neurocognitive performance: relevance and potential implications for Alzheimer's disease pathophysiology.

Vauzour D.

Author information

Abstract

Recent evidence has indicated that a group of plant-derived compounds known as flavonoids may exert particularly powerful actions on mammalian cognition and may reverse age-related declines in memory and learning. In addition, growing evidence is also suggestive that flavonoids may delay the development of Alzheimer's disease-like pathology, suggestive of potential dietary strategies in dementia. Although these low-molecular-weight phytochemicals are absorbed to only a limited degree, they have been found to counteract age-related cognitive declines possibly via their ability to interact with the cellular and molecular architecture of the brain responsible for memory. However, the majority of the research has been carried out at rather supraphysiological concentrations and only a few studies have investigated the neuromodulatory effects of physiologically attainable flavonoid concentrations. This review will summarize the evidence for the effects of flavonoids and their metabolites in age-related cognitive decline and Alzheimer's disease. Mechanisms of actions will be discussed and include those activating signalling pathways critical in controlling synaptic plasticity, reducing neuroinflammation and inducing vascular effects potentially capable of causing new nerve cell growth in the hippocampus. Altogether, these processes are known to be important in maintaining optimal neuronal function, to limit neurodegeneration and to prevent or reverse age-dependent deteriorations in cognitive performance. © 2013 Society of Chemical Industry.

Alzheimer

Presse Med. 2013 Oct;42(10):1398-404. doi: 10.1016/j.lpm.2013.07.012. Epub 2013 Sep 18.

[Vitamin D and neurology].

[Article in French]

Thouvenot É, Camu W.

Author information

Abstract

Vitamin D deficiency is associated with a higher risk of multiple sclerosis and also with a higher relapse rate as well as a higher number of MRI lesions. Elders with vitamin D deficiency have worse cognitive performance. Vitamin D deficiency is a risk factor for developing Alzheimer's disease. Ischemic stroke are more frequent and more severe in patients with low vitamin D levels. Carotid atherosclerosis is more frequent and more severe in patients with vitamin D deficiency. Vitamin D deficiency is associated with a higher risk and worse prognosis of Parkinson's disease. In the different neurological disorders discussed herein, gene polymorphisms that could alter vitamin D metabolism are also associated with a higher incidence or a worse disease prognosis. Despite the links between vitamin D deficiency and the risks of developing neurological disorders, there is, to date, no proof that supplementation could alter the course of these diseases.

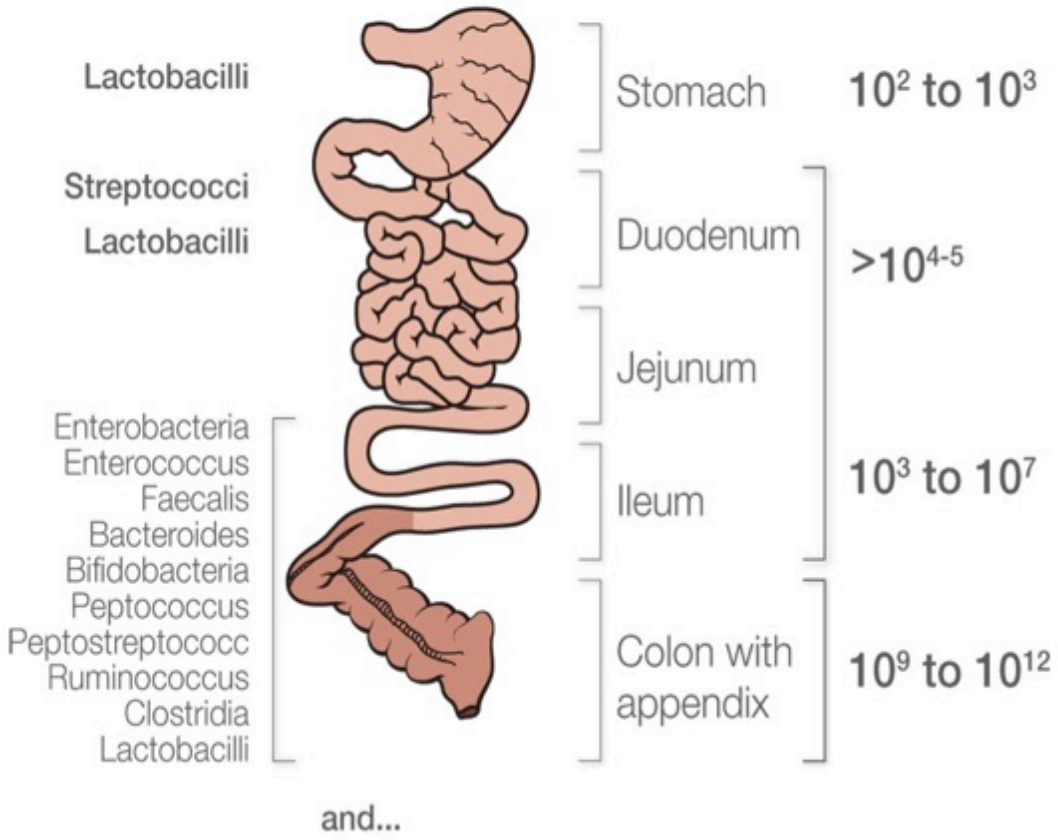
ILSI EUROPE CONCISE MONOGRAPH SERIES

PROBIOTICS, PREBIOTICS AND THE GUT MICROBIOTA

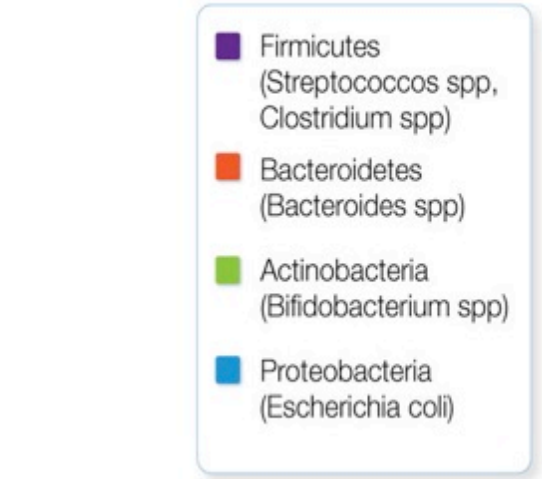
ILSI Europe
International Life Sciences Institute

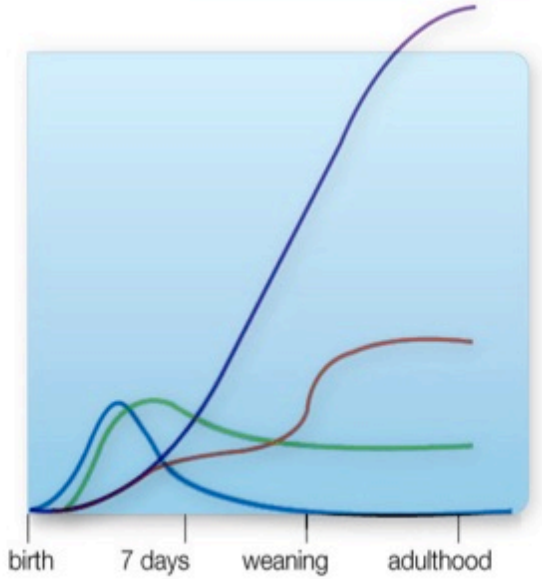
Probióticos y ProMeTe

Intestinal microflora
10^{14} micro-organisms, >500 different species



and...





Christopher Reinhaldt & Fredrik Backed. JPGN 2009;48:249-256

Probióticos

Review Article

Probiotics: Health benefits in the mouth

IVA STAMATOVA, MD & JUKKA H. MEURMAN, MD, PHD

ABSTRACT: Probiotics or health-beneficial bacteria have only recently been introduced in dentistry and oral medicine after years of successful use in mainly gastro-intestinal disorders. The concept of bacteriotherapy and use of health-beneficial micro-organisms to heal diseases or support immune function was first introduced in the beginning of the 20th century. Later the concept lead to the development of modern dairy industry and even today most probiotic strains are lactobacilli or bifidobacteria used in milk fermentation. The mechanisms of probiotic action are mainly unknown but the inter-microbial species interactions are supposed to play a key role in this together with their immuno-stimulatory effects. The introduction of probiotic bacteria in the mouth calls for ascertainment of their particular safety. Since acid production from sugar is detrimental to teeth, care must be taken not to select strains with high fermentation capacity. The first randomized controlled trials have nevertheless shown that probiotics may control dental caries in children due to their inhibitory action against cariogenic streptococci. Less evidence exists on their role in periodontal disease or oral yeast infections. Furthermore the best vehicles for oral probiotic applications need to be assessed. So far mainly dairy products have been investigated but other means such as probiotics in chewing gums or lozenges have also been studied. From the clinical practitioner's point of view direct recommendations for the use of probiotics cannot yet be given. However, scientific evidence so far indicates that probiotic therapy may be a reality also in dentistry and oral medicine in the future. (*Am J Dent* 2009;22:329-338)

CLINICAL SIGNIFICANCE: From the clinical practitioner's point of view direct recommendations for the use of probiotics cannot yet be given. However, scientific evidence so far indicates that probiotic therapy may be a reality also in dentistry and oral medicine in the future.

✉: Dr. Iva Stamatova, 3 Christo Botev Blvd., Faculty of Dental Medicine, 4000 Plovdiv, Bulgaria. E-✉: iva.stamatova@abv.bg

Probióticos

HY-796; No. of Pages 8

ARTICLE IN PRESS

ELSEVIER

Pathophysiology xxx (2014) xxx–xxx

ISP PATHOPHYSIOLOGY

www.elsevier.com/locate/pathophys

Commensal and probiotic bacteria may prevent NEC by maturing intestinal host defenses

Brett M. Jakaitis, Patricia W. Denning *

Emory University School of Medicine, Department of Pediatrics, Division of Neonatology, Atlanta, GA, United States

Abstract

Necrotizing enterocolitis (NEC) is a devastating disease of prematurity with significant morbidity and mortality. Immaturity of intestinal host defenses predisposes the premature infant gut to injury. An abnormal bacterial colonization pattern with a deficiency of commensal bacteria may lead to a further breakdown of these host defense mechanisms, predisposing the infant to NEC. The presence of probiotic and commensal bacteria within the gut has been shown to mature the intestinal defense system through a variety of mechanisms. We have shown that commensal and probiotic bacteria can promote intestinal host defenses by reducing apoptotic signaling, blocking inflammatory signaling, and maturing barrier function in immature intestinal epithelia. Future studies aimed at elucidating the mechanisms by which probiotic and commensal bacteria exert their effects will be critical to developing effective preventive therapies for NEC.
Published by Elsevier Ireland Ltd

Keywords: LGG; *Lactobacillus rhamnosus* GG; Probiotics; Microbiota; Commensal bacteria; Inflammation; Apoptosis; Tight junctions; ROS; Reactive oxygen species; Innate immune system; Intestinal epithelial cell; IL-10

Probióticos

CURRENT OPINION

Irritable bowel syndrome, inflammatory bowel disease and the microbiome

Giles Major and Robin Spiller

Purpose of review
The review aims to update the reader on current developments in our understanding of how the gut microbiota impact on inflammatory bowel disease and the irritable bowel syndrome. It will also consider current efforts to modulate the microbiota for therapeutic effect.

Recent findings
Gene polymorphisms associated with inflammatory bowel disease increasingly suggest that interaction with the microbiota drives pathogenesis. This may be through modulation of the immune response, mucosal permeability or the products of microbial metabolism. Similar findings in irritable bowel syndrome have reinforced the role of gut-specific factors in this 'functional' disorder. Metagenomic analysis has identified alterations in pathways and interactions with the ecosystem of the microbiome that may not be recognized by taxonomic description alone, particularly in carbohydrate metabolism. Treatments targeted at the microbial stimulus with antibiotics, probiotics or prebiotics have all progressed in the past year. Studies on the long-term effects of treatment on the microbiome suggest that dietary intervention may be needed for prolonged efficacy.

Summary
The microbiome represents 'the other genome', and to appreciate its role in health and disease will be as challenging as with our own genome. Intestinal diseases occur at the front line of our interaction with the microbiome and their future treatment will be shaped as we unravel our relationship with it.

Keywords
carbohydrates, inflammatory bowel disease, irritable bowel syndrome, metagenomics, microbiota

Probióticos

Critical Reviews in Food Science and Nutrition

Volume 54, Issue 2, 2014

Impact of Probiotics on Risk Factors for Cardiovascular Diseases. A Review

Abstract

Probiotic microorganisms have historically been used to rebalance disturbed intestinal microbiota and to diminish gastrointestinal disorders, such as diarrhea or inflammatory bowel diseases (e.g., Crohn's disease and ulcerative colitis). Recent studies explore the potential for expanded uses of probiotics on medical disorders that increase the risk of developing cardiovascular diseases and diabetes, such as obesity, hypercholesterolemia, arterial hypertension, and metabolic disturbances such as hyperhomocysteinemia and oxidative stress. This review aims at summarizing the proposed molecular and cellular mechanisms involved in probiotic–host interactions and to identify the nature of the resulting beneficial effects. Specific probiotic strains can act by modulating immune response, by producing particular molecules or releasing biopeptides, and by modulating nervous system activity. To date, the majority of studies have been conducted in animal models. New investigations on the related mechanisms in humans need to be carried out to better enable targeted and effective use of the broad variety of probiotic strains.

Probióticos

Symposium: Probiotic Bacteria:
Implications for Human Health

The Role of Probiotic Cultures in the Control of Gastrointestinal Health[1]

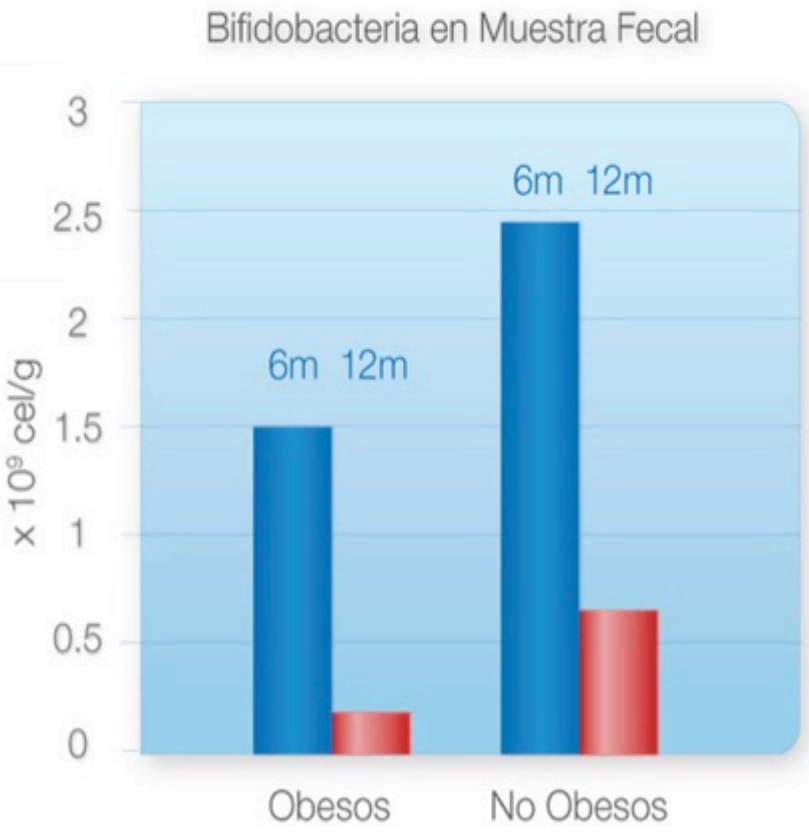
Rial D. Rolfe

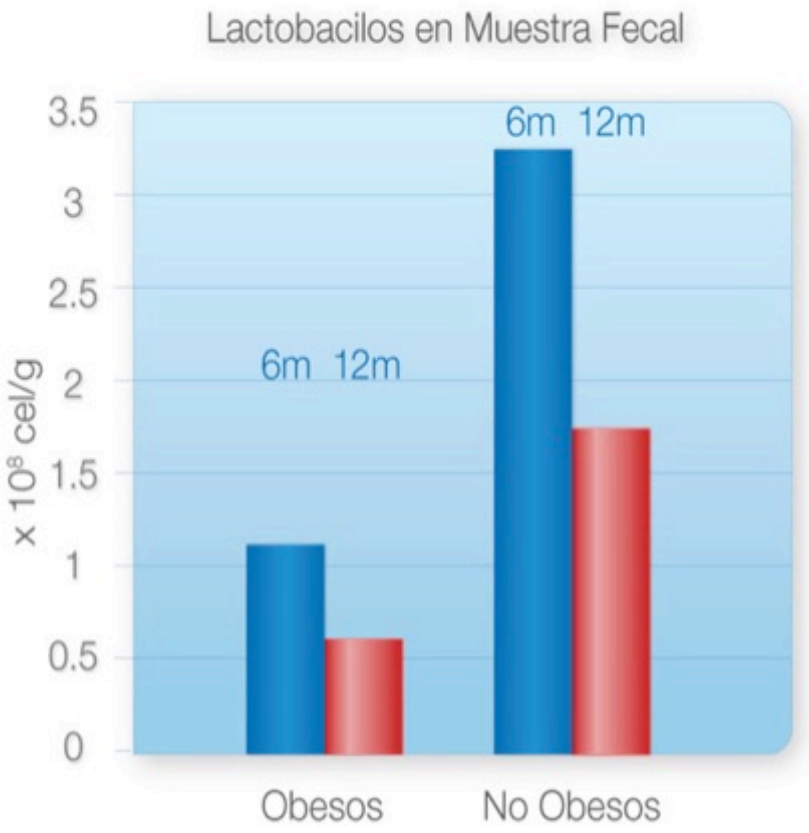
Department of Microbiology and Immunology, Texas Tech University Health Sciences Center, Lubbock, TX 79430

ABSTRACT The use of probiotics to enhance intestinal health has been proposed for many years. Probiotics are traditionally defined as viable microorganisms that have a beneficial effect in the prevention and treatment of specific pathologic conditions when they are ingested. There is a relatively large volume of literature that supports the use of probiotics to prevent or treat intestinal disorders. However, the scientific basis of probiotic use has been firmly established only recently, and sound clinical studies have begun to be published. Currently, the best-studied probiotics are the lactic acid bacteria, particularly *Lactobacillus* sp. and *Bifidobacterium* sp. However, other organisms used as probiotics in humans include *Escherichia coli, Streptococcus* sp., *Enterococcus* sp., *Bacteroides* sp., *Bacillus* sp., *Propionibacterium* sp. and various fungi. Some probiotic preparations contain mixtures of more than one bacterial strain. Probiotics have been examined for their effectiveness in the prevention and treatment of a diverse spectrum of gastrointestinal disorders such as antibiotic-associated diarrhea (including *Clostridium difficile*–associated intestinal disease), infectious bacterial and viral diarrhea (including diarrhea caused by rotavirus, *Shigella, Salmonella,* enterotoxigenic *E. coli, Vibrio cholerae* and human immunodeficiency virus/acquired immunodeficiency disorder, enteral feeding diarrhea, *Helicobacter pylori* gastroenteritis, sucrase maltase deficiency, inflammatory bowel disease, irritable bowel syndrome, small bowel bacterial overgrowth and lactose intolerance. Probiotics have been found to inhibit intestinal bacterial enzymes involved in the synthesis of colonic carcinogens. There are many mechanisms by which probiotics enhance intestinal health, including stimulation of immunity, competition for limited nutrients, inhibition of epithelial and mucosal adherence, inhibition of epithelial invasion and production of antimicrobial substances. Probiotics represent an exciting prophylactic and therapeutic advance, although additional investigations must be undertaken before their role in intestinal health can be delineated clearly. J. Nutr. 130: 396S–402S, 2000.

KEY WORDS: • *probiotics* • *intestinal disorders*

Probióticos y ProMeTe

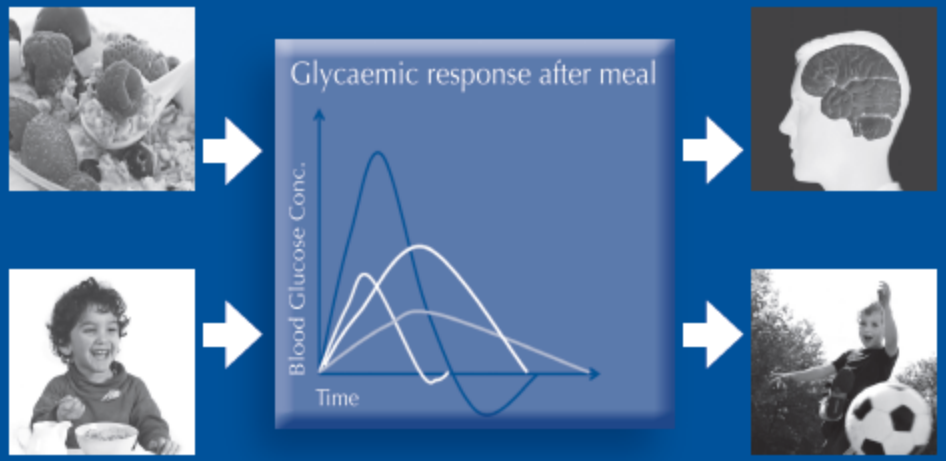




Mario Kalliomaki. Am J Clin Nutr 2008;87:534-538

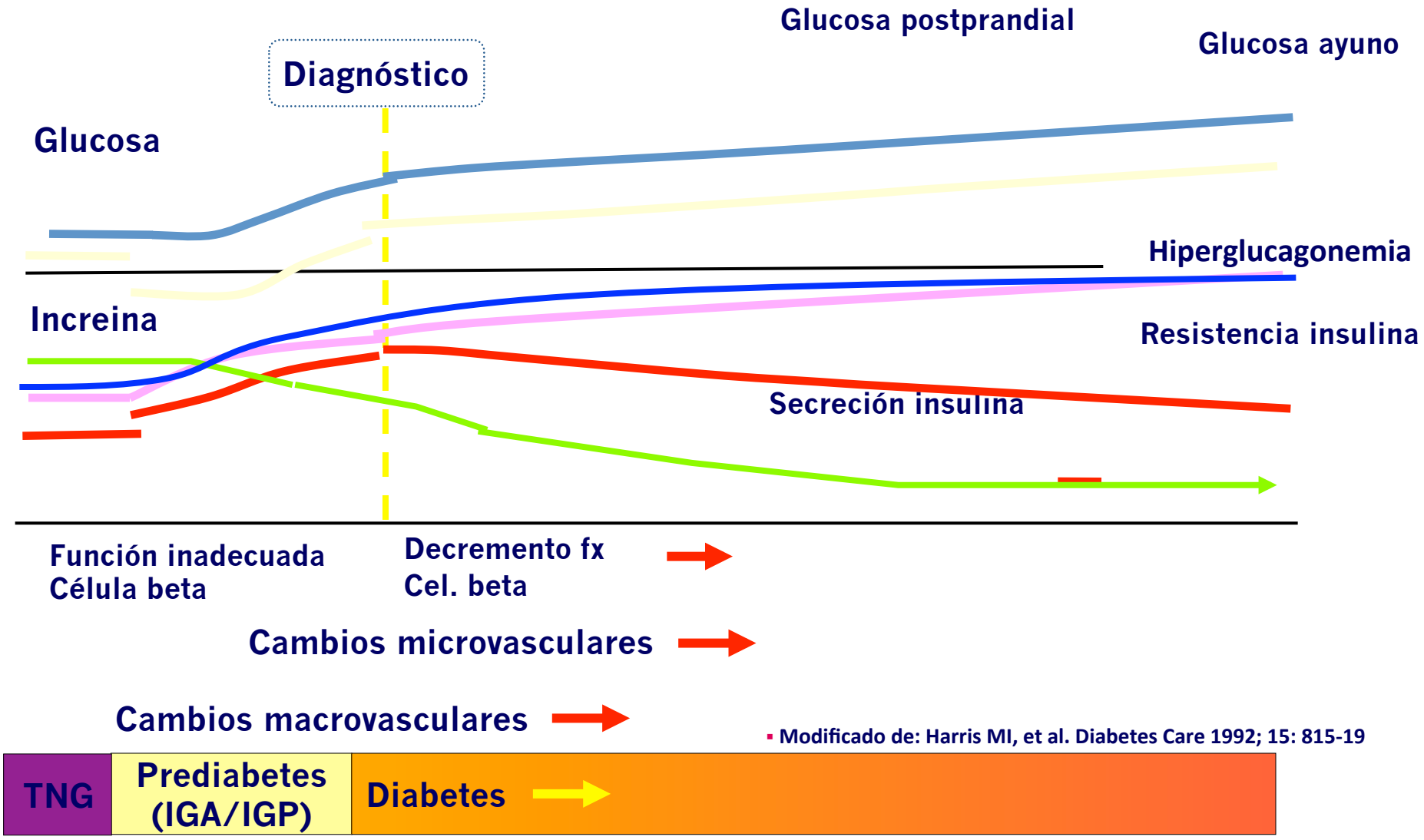
ILSI EUROPE CONCISE MONOGRAPH SERIES

FOOD, GLYCAEMIC RESPONSE AND HEALTH



ILSI Europe
International Life Sciences Institute

La función de los islotes se deteriora con el tiempo provocando la progresión de la enfermedad

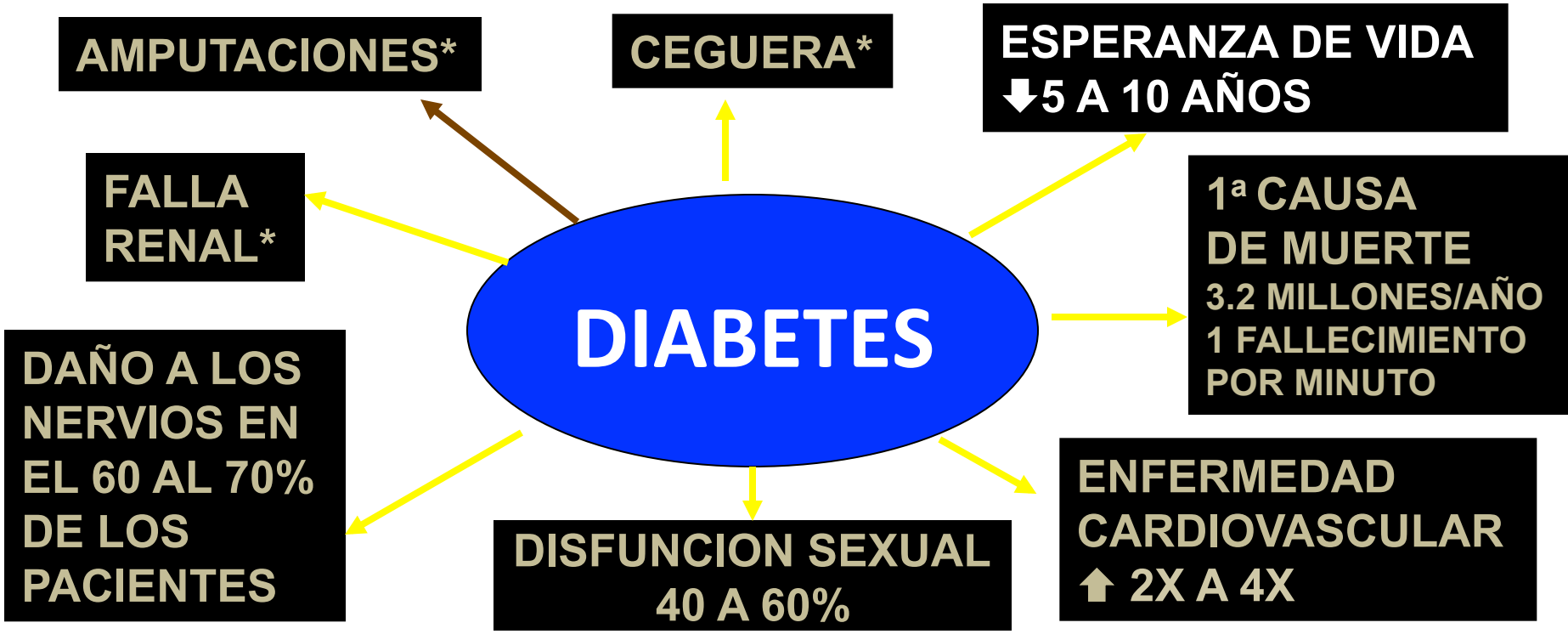


▪ Modificado de: Harris MI, et al. Diabetes Care 1992; 15: 815-19

Diabetes

IMPACTO A LA SALUD

- RESPONSABLE DE PERDIDAS DE VIDASY RECURSOS
- $ 7´784,000,000.00 US DÓLAR*
- 3´432,000,000 directos
- 4¨352,000,000 Indirectos



* Arredondo A. Comunicación Perasonal Agosto 2012
Diabetes Statistics. Oct.1995 (update 1997) NIDDK publication NIH 96-3926.
Harris MI. In: Diabetes in America 2nd. Ed. 1995: 1-13

Drr. Miguel Peniche Flores

Diabetes

European Journal of Internal Medicine 23 (2012) 465–469

Contents lists available at SciVerse ScienceDirect

European Journal of Internal Medicine

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

Original article

Vitamin D and gestational diabetes: A systematic review and meta-analysis

Y.H.M. Poel [a,*], P. Hummel [b], P. Lips [c], F. Stam [a], T. van der Ploeg [d], S. Simsek [a,c]

[a] Department of Internal Medicine, Gynaecology and Reproductive Medicine, Medical Centre Alkmaar, Alkmaar, The Netherlands
[b] Department of Obstetrics, Gynaecology and Reproductive Medicine, Medical Centre Alkmaar, Alkmaar, The Netherlands
[c] Department of Internal Medicine/Endocrinology, VU University Medical Centre, Amsterdam, The Netherlands
[d] Department of Biostatistics, Medical Centre Alkmaar, Alkmaar, The Netherlands

ARTICLE INFO

Article history:
Received 15 November 2011
Received in revised form 23 January 2012
Accepted 25 January 2012
Available online 21 February 2012

Keywords:
Gestational diabetes
Glucose homeostasis
Pregnancy
Meta-analysis
Systematic review
Vitamin D

ABSTRACT

Background: Conflicting results currently exists on the association between vitamin D and glucose metabolism. The role of maternal vitamin D status in gestational diabetes mellitus (GDM) is not clear. This meta-analysis aimed to examine this role in women with GDM compared with normal glucose tolerance (NGT).
Methods: We performed a systematic review and meta-analysis by searching MEDLINE database, the Cochrane library and *Uptodate® Online* for English-language literature up to September 2011. Summary odds ratios were calculated using a random-effects model meta-analysis.
Results: Seven observational studies were eligible for the meta-analysis, including 2146 participants of whom 433 were diagnosed with GDM. Four studies reported a high incidence of vitamin D deficiency in pregnant women (>50%). Overall vitamin D deficiency (serum 25-hydroxyvitamin D (25OHD)<50 nmol/l) in pregnancy was significantly related to the incidence of GDM with an odds ratio of 1.61 (95% CI 1.19–2.17; $p=0.002$). Serum 25OHD was significant lower in participants with GDM than in those with NGT (−5.33 nmol/l (95% CI −9.73 to −0.93; $p=0.018$).
Conclusions: This meta-analysis indicates a significant inverse relation of serum 25OHD and the incidence of GDM. However, it remains unclear whether this association is causal due to the observational study design of the studies. Clinical trials are needed to examine whether vitamin D supplementation will improve glycemic control in women with GDM.

Diabetes

LETTER

doi:10.1038/nature12198

Gut metagenome in European women with normal, impaired and diabetic glucose control

Fredrik H. Karlsson[1]*, Valentina Tremaroli[2]*, Intawat Nookaew[1], Göran Bergström[2], Carl Johan Behre[2], Björn Fagerberg[2], Jens Nielsen[1] & Fredrik Bäckhed[2,3]

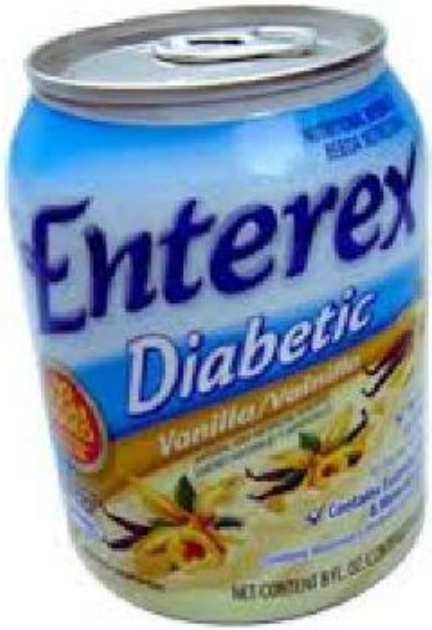
Type 2 diabetes (T2D) is a result of complex gene–environment interactions, and several risk factors have been identified, including age, family history, diet, sedentary lifestyle and obesity. Statistical models that combine known risk factors for T2D can partly identify individuals at high risk of developing the disease. However, these studies have so far indicated that human genetics contributes little to the models, whereas socio-demographic and environmental factors have greater influence[1]. Recent evidence suggests the importance of the gut microbiota as an environmental factor, and an altered gut microbiota has been linked to metabolic diseases including obesity[2,3], diabetes[4] and cardiovascular disease[5]. Here we use shotgun sequencing to characterize the faecal metagenome of 145 European women with normal, impaired or diabetic glucose control. We observe compositional and functional alterations in the metagenomes of women with T2D, and develop a mathematical model based on metagenomic profiles that identified T2D with high accuracy. We applied this model to women with impaired glucose tolerance, and show that it can identify women who have a diabetes-like metabolism. Furthermore, glucose control and medication were unlikely to have major confounding effects. We also applied our model to a recently described Chinese cohort[4] and show

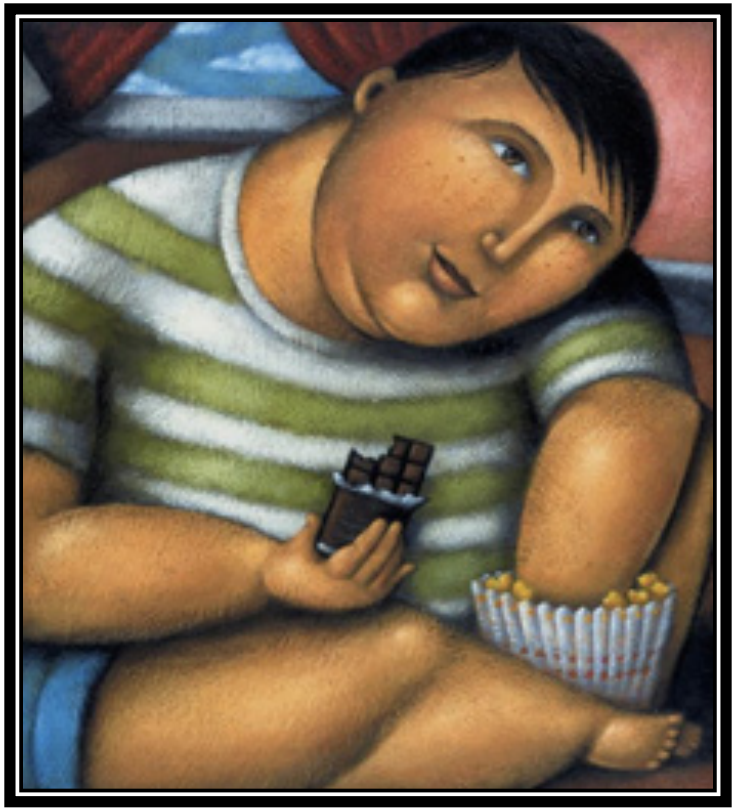
decreases in the abundance of five *Clostridium* species in the T2D group (adjusted $P < 0.05$, Wilcoxon rank sum test) (Supplementary Fig. 1a and Supplementary Table 6). In the total cohort, *Lactobacillus* species correlated positively with fasting glucose and HbA1c (glycosylated haemoglobin), a long-term measure of blood glucose control (adjusted $P < 0.05$, Spearman correlation). By contrast, *Clostridium* species correlated negatively with fasting glucose, HbA1c, insulin, C-peptide and plasma triglycerides, and positively with adiponectin and HDL (Supplementary Fig. 1b and Supplementary Table 7). These correlations are relevant for T2D because high triglycerides and low HDL levels are components of the dyslipidaemia typically found in T2D, whereas serum levels of the insulin-sensitizing hormone adiponectin are reduced in people at risk of T2D (ref. 14). Importantly, these *Lactobacillus* and *Clostridium* species did not correlate with body mass index (BMI), waist circumference or waist-to-hip ratio (WHR) (Supplementary Fig. 1b).

To identify microbial species independently of reference genomes and fully exploit the information contained in the metagenomic data, we performed *de novo* assembly of filtered sequence data. The total length of the assembly was 13.6 Gb, from which 18.6 million genes with a length longer than 100 base pairs (bp) could be predicted. We created a non-redundant gene catalogue for our cohort and merged it


GLUCOSA
Glucerna
TIEMPO


¡NUEVO!
Suplemento Alimenticio
Glucopromin
Formulado para personas con Diabetes
Con FIBRA
Delicioso sabor
CHOCOLATE
CONT. NET. 236 ml


Enterex
Diabetic
Vainilla



OBESIDAD INFANTIL

"El difícil futuro de México"

Peso

Efficacy of conjugated linoleic acid for reducing fat mass: a meta-analysis in humans[1,2]

Leah D Whigham, Abigail C Watras, and Dale A Schoeller

ABSTRACT

Background: Conjugated linoleic acid (CLA) has been shown to be an effective supplement for reducing fat mass in animals, whereas results in humans have been inconsistent.

Objective: This is a meta-analysis of human studies in which CLA was provided as a dietary supplement to test its efficacy in reducing fat mass.

Design: We searched the PubMed database (National Library of Medicine, Bethesda, MD) and references from the resulting search to identify studies in which CLA was provided to humans in randomized, double-blinded, placebo-controlled trials and in which body composition was assessed by using a validated technique.

Results: We identified 18 eligible studies. Of these, 3 were single-isomer studies, and results comparing CLA isomers were inconclusive. We compared the length of treatment by using studies in which a mixture of purified isomers were used and those in which purified *trans*-10,*cis*-12 isomers were used. This comparison indicated that the effect of CLA was linear for up to 6 mo and then slowly approached an asymptote at 2 y. An analysis of the dose effect indicated that fat loss compared with placebo was $-0.024\ \text{kg} \cdot \text{g CLA}^{-1} \cdot \text{wk}^{-1}$ ($P = 0.03$). After adjustment to the median dose of 3.2 g CLA/d, CLA was effective and produced a reduction in fat mass for the CLA group alone (0.05 ± 0.05 kg/wk; $P < 0.001$) and for the CLA group compared with placebo (0.09 ± 0.08 kg/wk; $P < 0.001$)

Conclusion: Given at a dose of 3.2 g/d, CLA produces a modest loss in body fat in humans. *Am J Clin Nutr* 2007;85:1203–11.

animal studies have investigated the effect of CLA on body composition, and although results vary by species, most find that CLA reduces body fat. Mice are most responsive, with treated animals having 60% less total body fat than controls (3). CLA treatment reduced individual fat depots compared with controls by as much as 88% and 61% in retroperitoneal and epididymal fat, respectively, in one study (4) and by ≈50% in each of those depots in another study (5). In pigs, CLA has resulted in 6–25% less total body fat (reviewed in 6). In hamsters, CLA has resulted in 9% (7) to 24% (8) less epididymal fat, 44% less subcutaneous fat (8), and 58% less perirenal fat (9). In rats, some studies have shown no effect of CLA on overall body composition (10, 11), whereas others have shown that feeding CLA from selectively hydrogenated soybean oil resulted in 23% lower total body fat (12).

Animal studies in which specific CLA isomers were used have shown that the effects on body composition are isomer specific. The *t*10, *c*12 isomer has been identified as the one responsible for decreasing body fat (7, 13, 14). Mechanisms by which the *t*10, *c*12 isomer affects body fat include reduction of lipid accumulation by adipocytes mediated through effects on lipoprotein lipase and stearoyl-coenzyme A (Co A) desaturase (reviewed in 15).

On the basis of the effect of CLA in animal studies, there was great potential for CLA to have a beneficial effect on body composition in humans. Of the human CLA trials to date, however,

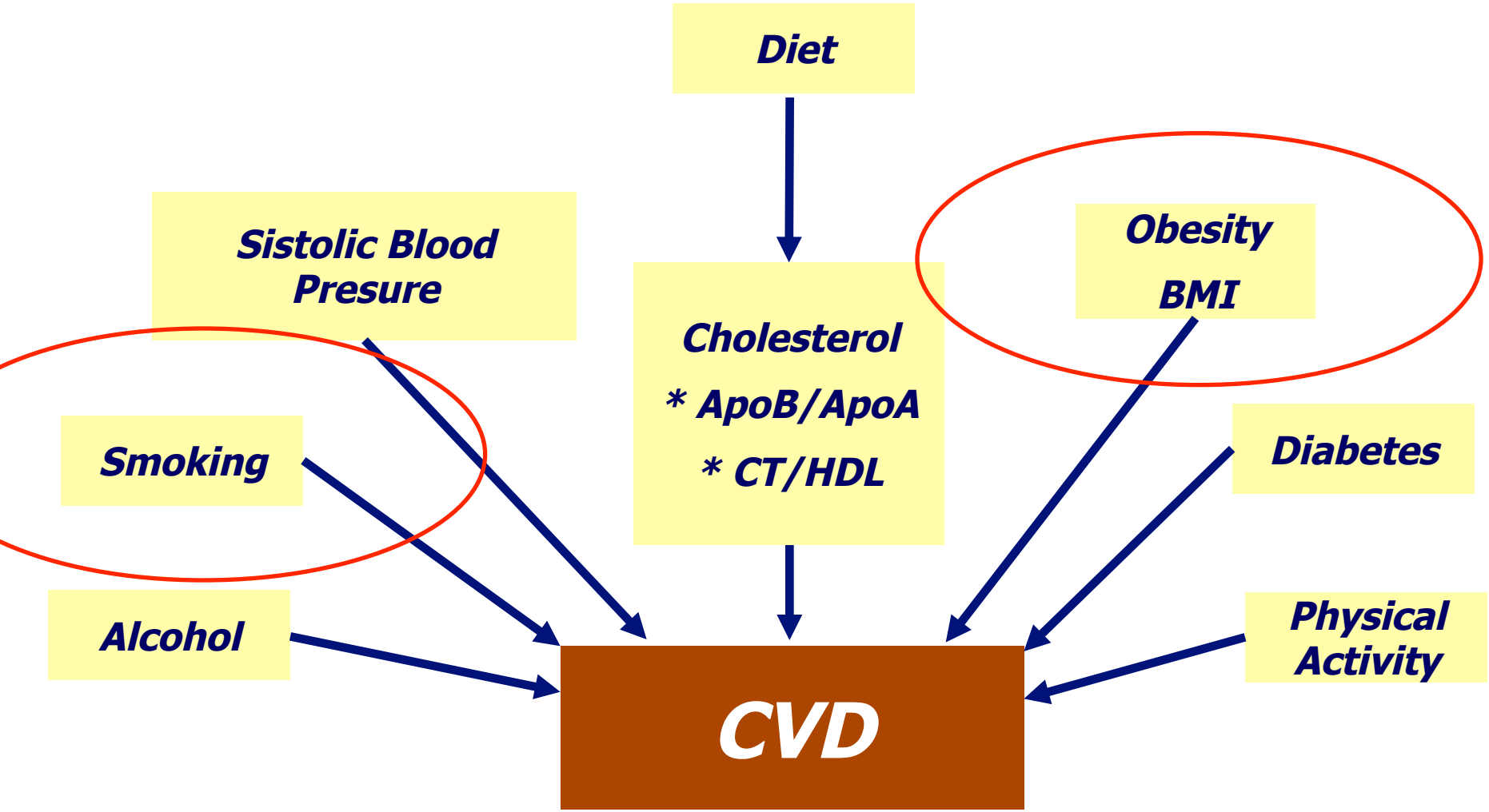
The American Journal of Clinical Nutrition

Quantitative Risk Assessment of Cardiovascular Diseases in Mexican Population

Signorini, M. PhD. Valdés, S. PhD. Luna, J. MsC. Ordoñez, L.

Image in: http://www.google.com/imgres?imgurl=http://topnews.net.nz/images/cardiovascular-diseases.jpg&imgrefurl=http://topnews.net.nz/content/211033-scientists-assess-chances-people-developing-cvds-because-risk-factors&usg=__6q2WtYMo6hUaPYzk59XMStlI564=&h=300&w=400&sz=46&hl=en&start=0&zoom=1&tbnid=44Fppa2E5Z_DPM:&tbnh=157&tbnw=209&ei=SQM2TeatA8mdgQeHx95XCw&prev=/images%3Fq%3Dcardiovascular%2Bdiseases%26hl%3Den%26sa%3DX%26biw%3D1276%26bih%3D837%26gbv%3D2%26tbs%3Disch:10%2C126&itbs=1&iact=hc&vpx=141&vpy=349&dur=3717&hovh=194&hovw=259&tx=166&ty=116&oei=SQM2TeatA8mdgQeHx95XCw&esq=1&page=1&ndsp=24&ved=1t:429,r:12,s:0&biw=1276&bih=837

Model of Risk Factors for CVD in Mexico



Omega 3


PULEVA
PULEVA
OMEGA 3
ÉPA
ÉPA
PULEVA OMEGA 3 ha demostrado científicamente que te ayuda a reducir los niveles de colesterol y triglicéridos, y por ello, a mantener tus arterias y tu corazón sanos. Toma dos vasos diarios, y lo notarás a partir del primer mes. Una dieta equilibrada y un estilo de vida saludable contribuyen a preservar tu salud cardiovascular.


OMACOR
cápsulas blandas
OMACOR
cápsulas blandas


ener ZONA
OMEGA 3 RX
ener ZONA
OMEGA 3 RX

La FDA define “gluten-free” (20 ppm de gluten)

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Food and Drug Administration

21 CFR Part 101

[Docket No. FDA–2005–N–0404]

RIN 0910–AG84

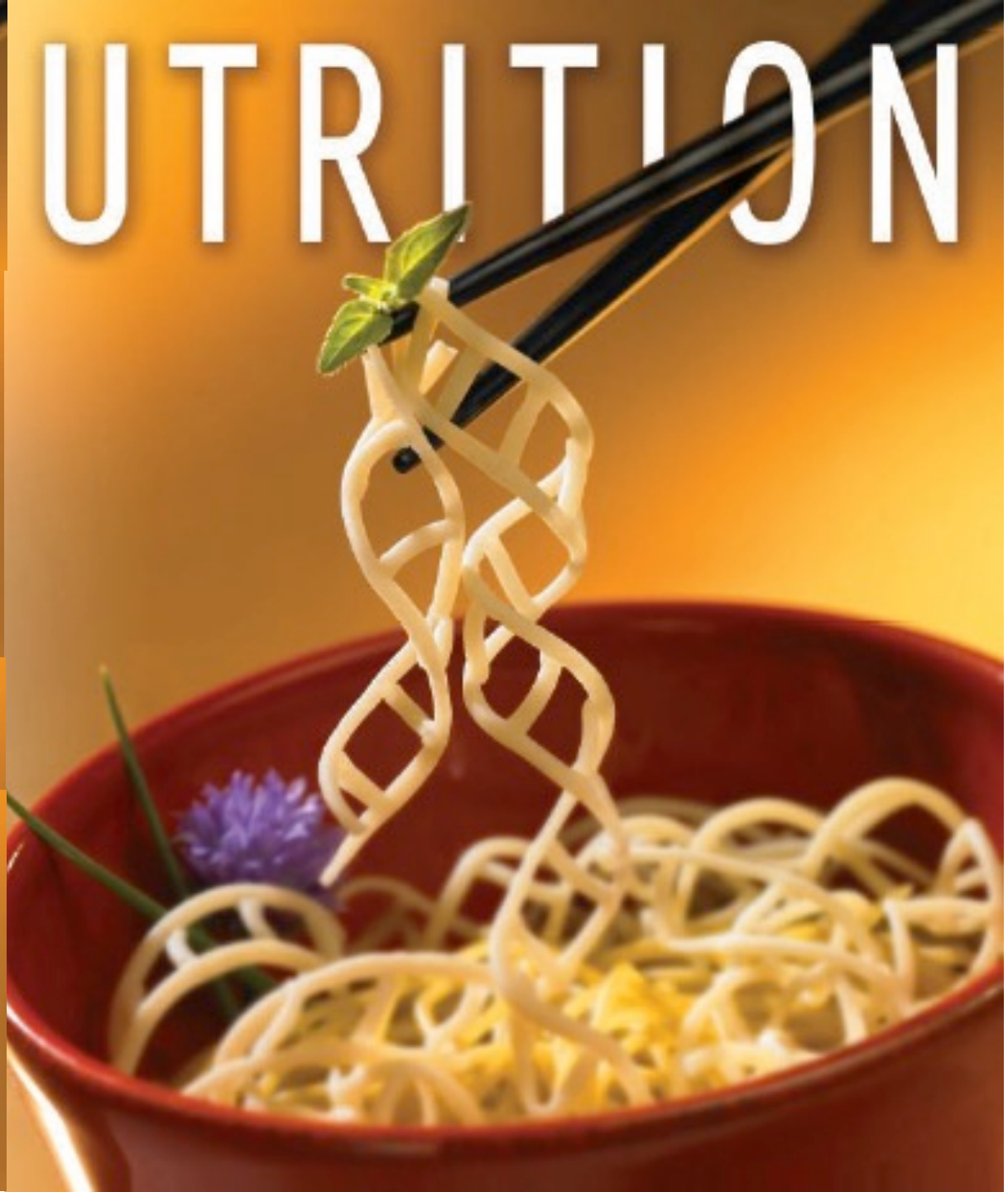
Food Labeling; Gluten-Free Labeling of Foods

AGENCY: Food and Drug Administration, HHS.

ACTION: Final rule.

DESAFÍOS PARA LOS ALIMENTOS FUNCIONALES/NUTRACÉUTICOS

NUTRITION



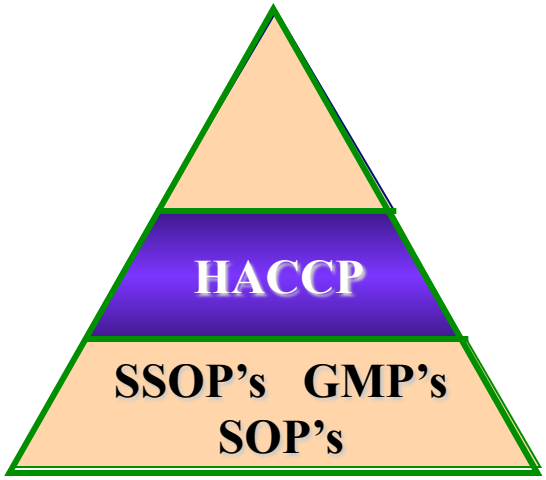
Development of Nutritional Products

Government

Industry

Legislation

Nutritional Products

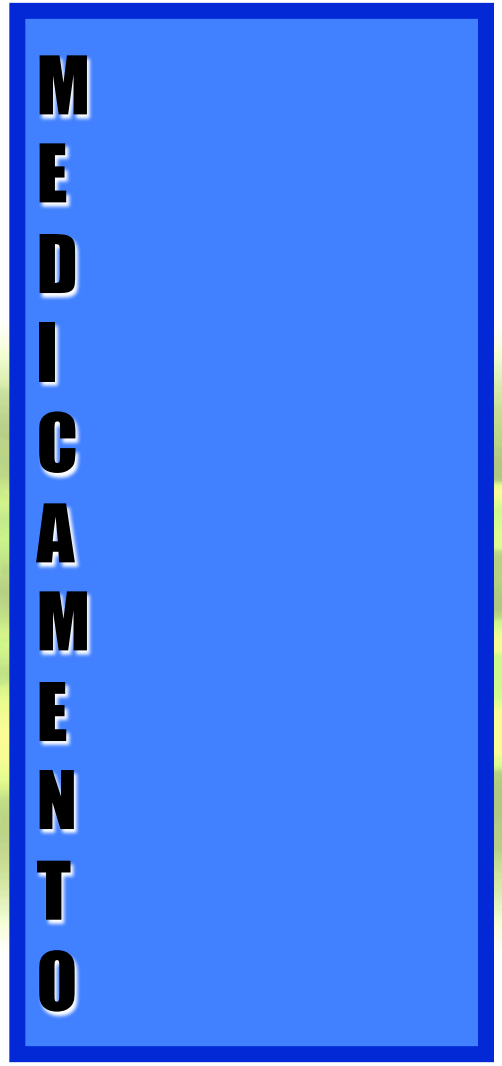


Improve of Legal Framework

Security

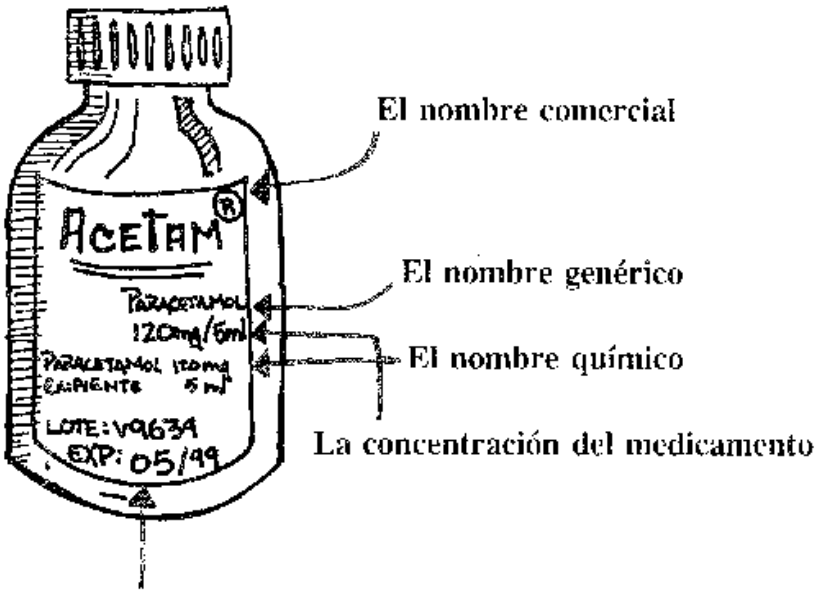
Scientific Development

Academy



PREVENCIÓN

TRATAMIENTO



Fuente: http://www.google.com.mx/imgres?q=etiqueta+antibi%C3%B3tico&um=1&hl=es&sa=N&qscrl=1&nord=1&rlz=1T4AURU_esMX501MX501&biw=1366&bih=641&tbm=isch&tbnid=R1PyU-T2gFPQpM:&imgrefurl=http://vidaynatura.com/salud-y-medicamentos/las-enfermedades-que-si-necesitan-antibioticos-i/&docid=te7VuxZFjYD6kM&imgurl=http://vidaynatura.com/wp-content/uploads/2012/01/antibiotico_small.jpg&w=500&h=333&ei=K8thUL7VE4ra8AS6soCYBw&zoom=1&iact=hc&vpx=695&vpy=188&dur=1074&hovh=183&hovw=275&tx=150&ty=107&sig=105098449763633415402&page=2&tbnh=122&tbnw=183&start=20&ndsp=25&ved=1t:429,r:9,s:20,i:164

3 Rogers DB, Shohat M, Petersen GM, *et al.* Familial Mediterranean fever in Armenians: autosomal recessive inheritance with high gene frequency. *Am J Med Genet* 1989;34:168-72.
4 Barakat MH, Kamik AM, Mareel HWA, El-Sobki NI, Fenech FF. Familial Mediterranean fever (recurrent hereditary polyserositis in Arabs—a study of 175 patients and review of the literature. *Q J Med* 1986;60:837-47.
5 Cook GC. Periodic disease, recurrent polyserositis, familial Mediterranean fever, or simply "FMF." *Q J Med* 1986;60:819-23.
6 Moore PJ, Mansour A, McDonald JD, Kemp A, Kamath KR, Dorney SFA. Familial Mediterranean fever in six Australian children. *Med J Aust* 1989;151:108-10.
7 Gorfu MA, Petitt RM, Perrault J, Kyle RA. Autosomal dominant familial Mediterranean fever-like syndrome with amyloidosis. *Mayo Clin Proc* 1987;62:1095-100.
8 Melamed A, Cabili S, Zakuth V, Sperer Z. The immune regulation in familial Mediterranean fever (FMF). *J Clin Lab Immunol* 1988;26:125-8.
9 Matzner Y, Brzezinski A. C1s-inhibitor deficiency in peritoneal fluids from patients with familial Mediterranean fever. *N Engl J Med* 1984;311:287-90.
10 Shohat M, Korenberg JR, Schwabe AD, Rotter JI. Hypothesis: familial Mediterranean fever—A genetic disorder of the lipocortin family? *Am J Med Genet* 1989;34:163-7.
11 Anon PS, Haines KA, Green W, *et al.* Circulating hydroxy fatty acids in familial Mediterranean fever. *Proc Natl Acad Sci USA* 1985;82:1232-6.
12 Anon PA, Targan SR, Vigna SR, Durham M, Schwabe AD, Shanahan F. Enhanced neutrophil chemiluminescence in familial Mediterranean fever. *J Clin Immunol* 1988;8:148-56.
13 Barakat MH, El-Khawad AO, Gumaa KA, El-Sobki NI, Fenech FF. Metaraminol provocative test: a specific diagnostic test for familial Mediterranean fever. *Lancet* 1984;i:656-7.
14 Barakat MH, Gumaa KA, Malhas LN, El-Sobki NI, Mousa MA, Fenech FF. Plasma dopamine beta-hydroxylase: rapid diagnostic test for recurrent hereditary polyserositis. *Lancet* 1988;ii:1280-3.
15 Hawkins PN, Lavender JP, Pepys MB. Evaluation of systemic amyloidosis by scintigraphy with ^{123}I-labelled serum amyloid P component. *N Engl J Med* 1990;323:508-13.
16 Dinarello CA, Wolff SM, Goldfinger SE, Dale DC, Alling DW. Colchicine therapy for familial Mediterranean fever: a double-blind trial. *N Engl J Med* 1974;291:934-7.
17 Zemer D, Revach M, Pras M, *et al.* A controlled trial of colchicine in preventing attacks of familial Mediterranean fever. *N Engl J Med* 1974;291:932-4.
18 Zemer D, Pras M, Sohar E, Modan M, Cabili S, Gafni J. Colchicine in the prevention and treatment of the amyloidosis of familial Mediterranean fever. *N Engl J Med* 1986;314:1001-5.
19 Anonymous. Colchicine in amyloidosis [Editorial]. *Lancet* 1986;ii:724-5.

The fetal and infant origins of adult disease

The womb may be more important than the home

A hundred years ago, when tuberculosis and rheumatic heart disease were common, the proposition that the childhood environment affects adult health would have been self evident. This proposition may still hold, even though infective disease has given place to degenerative disease.

Studies in Norway, Finland, Britain, and the United States have shown that death rates from cardiovascular disease are inversely related to adult height, and geographical differences in cardiovascular mortality are related to past differences in infant mortality.[1-3] These findings have been interpreted as evidence that adverse living conditions during childhood, such as poor housing and diet, increase the risk of ischaemic heart disease.[4] Case-control studies have generally supported this[4-12]: patients with myocardial infarction have higher infant death rates among their siblings,[4-8] are more likely to come from larger families, and are more likely to have fathers who were unemployed.[10] Now studies in Finland show that men with ischaemic heart disease had worse socioeconomic conditions in childhood (p 1121)[12]—an observation also made in Britain.[9]

The completeness of infant mortality records in England and Wales from 1911 onwards has allowed detailed geographical comparisons of the relation between infant mortality 70 years ago and mortality from cardiovascular disease today. Differences in the death rates from cardiovascular disease among the 212 local authority areas of England and Wales are closely related to past differences in neonatal mortality.[6 13] Most neonatal deaths were associated with low birth weight, and rates were high in areas where mothers had poor health and high death rates during childbirth.[14 15] These findings suggested that research should be redirected towards the intrauterine environment rather than the environment in later childhood—housing, family income, diet, and other influences. The Medical Research Council employed a historian to search for old records of birth and infancy. In Hertfordshire health visitors recorded the birth weight of all babies born in the county from 1911 onwards and visited their homes periodically throughout infancy. Follow up studies of the men and women born 60 and more years ago show that those who weighed more at birth and, if they were breast fed, at 1 year, had lower death rates from ischaemic heart disease and stroke.[16] The differences in death rates were large.

We are beginning to identify processes that link fetal and infant growth with cardiovascular disease. A recent study of 449 men and women aged 50 years who had been born in one hospital in Preston, England, showed that their current blood pressure and risk of hypertension were strongly related to their placental and birth weight.[17] Pressures were highest when birth weight had been lower than expected from placental weight. Discordance between placental and birth weights may be interpreted as fetal growth failure. Its causes are unknown, but maternal nutrition is an obvious suspect.

These epidemiological findings point to the importance of long term programming in early life and parallel findings in clinical and animal research. For example, the composition of infant food has been shown to have an important effect on motor development in preterm babies,[18] and programming of lipid metabolism by early feeding has been shown in baboons.[19] Knowledge of the fetal processes that may determine programming is beginning to emerge.[20] A recent symposium heard evidence that diseases other than cardiovascular disease may also be determined by the maternal environment.[21] Schizophrenia and obstructive lung disease are two examples.

The old model of adult degenerative disease was based on the interaction between genes and an adverse environment in adult life. The new model that is developing will include programming by the environment in fetal and infant life.

D J P BARKER

Director,
MRC Environmental Epidemiology Unit,
University of Southampton,
Southampton General Hospital,
Southampton SO9 4XY

1 Waaler HT. Height, weight and mortality. The Norwegian experience. *Acta Med Scand* 1984;679(suppl):1-56.
2 Nothiola V. *Living conditions in childhood and coronary heart disease in adulthood.* Helsinki: Finnish Society of Science and Letters, 1985.
3 Marmot MG, Shipley MJ, Rose G. Inequalities in death specific explanations of a general pattern? *Lancet* 1984;i:1003-6.
4 Forsdahl A. Are poor living conditions in childhood and adolescence an important risk factor for arteriosclerotic heart disease? *British Journal of Preventive and Social Medicine* 1977;31:91-5.
5 Barker DJP, Osmond C, Golding J. Height and mortality in the counties of England and Wales. *Ann Hum Biol* 1990;17:1-6.
6 Barker DJP, Osmond C. Infant mortality, childhood nutrition and ischaemic heart disease in England and Wales. *Lancet* 1986;i:1077-81.
7 Buck C, Simpson H. Infant diarrhoea and subsequent mortality from heart disease and cancer. *J Epidemiol Community Health* 1982;36:27-30.
8 Rose G. Familial patterns in ischaemic heart disease. *British Journal of Preventive and Social Medicine* 1964;18:75-80.
9 Coggon DNM, Margetts B, Barker DJP, *et al.* Childhood risk factors for ischaemic heart disease and stroke. *Paediatric and Perinatal Epidemiology* 1990;4:464-70.
10 Burr ML, Sweetnam PM. Family size and paternal unemployment in relation to myocardial infarction. *J Epidemiol Community Health* 1980;34:93-5.
11 Hade H. Association between living conditions in childhood and myocardial infarction. *BMJ* 1990;300:512-3.
12 Kaplan GA, Salonen JT. Socioeconomic conditions in childhood are associated with ischaemic heart disease during middle age. *BMJ* 1990;301:1121-3.
13 Barker DJP, Osmond C, Law C. The intra-uterine and early postnatal origins of cardiovascular disease and chronic bronchitis. *J Epidemiol Community Health* 1989;43:237-40.
14 Campbell JM, Cameron D, Jones DM. *High maternal mortality in certain areas.* London: HMSO, 1932. (Ministry of Health reports on public health and medical subjects, No 68).
15 Barker DJP, Osmond C. Death rates from stroke in England and Wales predicted from past maternal mortality. *BMJ* 1986;295:83-6.
16 Barker DJP, Winter PD, Osmond C, Margetts B, Simmonds SJ. Weight in infancy and death from ischaemic heart disease. *Lancet* 1989;ii:577-80.
17 Barker DJP, Bull AR, Osmond C, Simmonds SJ. Fetal and placental size and risk of hypertension in adult life. *BMJ* 1990;301:259-62.
18 Lucas A, Morley R, Cole TJ, *et al.* Early diet in preterm babies and developmental status at 18 months. *Lancet* 1990;335:1477-81.
19 Mott GE, Lewis DS and McGill HC, Jr. Programming of cholesterol metabolism by breast or formula feeding. In: Bock GR, Whelan J, eds. *The childhood environment and adult disease.* Chichester: John Wiley and Sons (in press). (Ciba Foundation symposium No 156.)
20 Dawes GS, Zacutti A, Borruto F, Zacutti A, Jr. *Fetal autonomy and adaptation.* Chichester: John Wiley and Sons, 1990.
21 Bock GR, Whelan J, eds. *The childhood environment and adult disease.* Chichester: John Wiley and Sons (in press). (Ciba Foundation symposium No 156.)

CONSUMIDORES...

British Journal of Nutrition (2007), **98**, 474–484
doi: 10.1017/S000711450778697X

Review Article

Consumer understanding of nutrition and health claims: sources of evidence

Peter D. Leathwood[1], David P. Richardson[2], Peter Sträter[3], Peter M. Todd[4,5] and Hans C. M. van Trijp[6,7]

[1]*Nestlé Research Centre, P.O. Box 44, 1000 Lausanne, Switzerland*
[2]*DPR Nutrition Limited, 34 Grimwade Avenue, Croydon, Surrey, CR0 5DG, UK*
[3]*Südzucker, Wormser Strasse 11, 67283 Mannheim, Germany*
[4]*Indiana University, Department of Psychology, 1101 E. 10th Street, Bloomington, IN 47405, USA*
[5]*Max Planck Institute for Human Development, Lentzeallee 94, 14195 Berlin, Germany*
[6]*Unilever Food and Health Research Institute, Olivier van Noortlaan 120, 3133 AT Vlaardingen, The Netherlands*
[7]*Wageningen University, Hollandseweg 1, 6706 KN Wageningen, The Netherlands*

(Received 19 March 2007 – Revised 18 April 2007 – Accepted 26 April 2007)

Provided that they are scientifically substantiated, nutrition and health (NH) claims linked to food products can help consumers make well-informed food choices. The new European legislation on NH claims made on foods entered into force on 19 January 2007. The law sets out conditions for their use, establishes a system for their scientific evaluation, and will create European lists of authorised claims. An important aspect of this proposed legislation is that it states, in article 5.2, 'the use of nutrition and health claims shall only be permitted if the average consumer can be expected to understand the beneficial effects expressed in the claim'. The present review examines consumer understanding of NH claims from a consumer science perspective. It focuses on the type of data and information that could be needed to provide evidence

Use and understanding of the nutrition information panel of pre-packaged foods in a sample of Mexican consumers

Vanessa De la Cruz-Góngora, MSc,[I] Salvador Villalpando, MD, PhD,[I]
Guadalupe Rodríguez-Oliveros, MBA, PhD,[I] Marcia Castillo-García, MPH,[I]
Verónica Mundo-Rosas, MSc,[I] Sergio Meneses-Navarro, MD, MA.[I]

De la Cruz-Góngora V, Villalpando S, Rodríguez-Oliveros G, Castillo-García M, Mundo-Rosas V, Meneses-Navarro S. Use and understanding of the nutrition information panel of pre-packaged foods in a sample of Mexican consumers. Salud Publica Mex 2012;54:158-166.

Abstract

Objective. To assess the use and understanding of the Nutritional information Panel (NIP) of pre-packaged foods by Mexican consumers. **Materials and methods.** A ques-

De la Cruz-Góngora V, Villalpando S, Rodríguez-Oliveros G, Castillo-García M, Mundo-Rosas V, Meneses-Navarro S. Uso y comprensión del etiquetado nutrimental posterior de los alimentos pre-empaquetados en una muestra de consumidores mexicanos. Salud Publica Mex 2012;54:158-166.

Resumen

Objetivo. Evaluar el uso y comprensión del etiquetado nutricional posterior (NIP, por sus siglas en inglés) de alimentos preempacados por consumidores mexicanos.

CONCLUSIONES