

La nostra contaminació interna: alimentació, salut i societat.

Bellaterra,
3 d'abril de 2019



Miquel Porta Serra

Institut d'Investigació Mèdica Hospital del Mar
Universitat Autònoma de Barcelona
University of North Carolina at Chapel Hill



La nostra contaminació interna: ja fem el que sí és factible?

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3 d'abril de 2019



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Clinical and Molecular Epidemiology of Cancer


Scientific documents



- ⦿ **B. Conferencia: "¿Deberíamos analizar los compuestos tóxicos persistentes que tenemos en la sangre?"**
- ⦿ **Accumulation of genetic and epigenetic alterations: a key causal process between the environment and the occurrence of cancer**
- ⦿ Integrating lifecourse, environmental, molecular and epigenetic epidemiology
- ⦿ Environmental toxic substances: exposed individuals and exposed populations
- ⦿ **Between molecules and the environment: keeping patients in the picture**

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Contaminación interna

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ciberesp

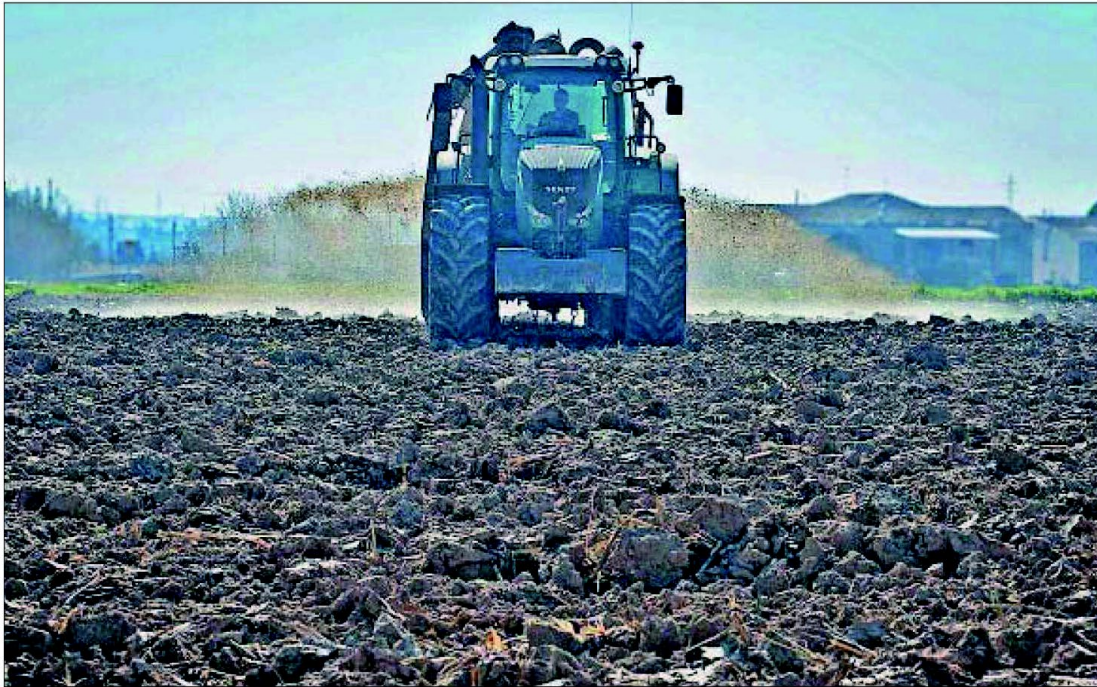
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Epidemiología y Salud Pública

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Facultat de Medicina

CATALUÑA



Una cisterna vierte purín en un campo de Alcarràs (Segrià). / JUAN BARBOSA

El 41% de los acuíferos está contaminado por nitratos

DAVID GARCÍA, **Barcelona**

El problema de la contaminación del agua subterránea no remite. El emponzoñamiento por nitratos afecta ya al 41% de los acuíferos catalanes. Si a finales de 2013, 20 de las 58 masas de agua dulce subterráneas (un 34,5%) tenían exceso de este con-

taminante, proveniente en su mayoría del uso de deyecciones ganaderas para abonar los campos, la Agencia Catalana del Agua (ACA) asegura que 24 superan ya los 50 miligramos por litro, límite que la Organización Mundial de la Salud recomienda no superar para el consumo humano.

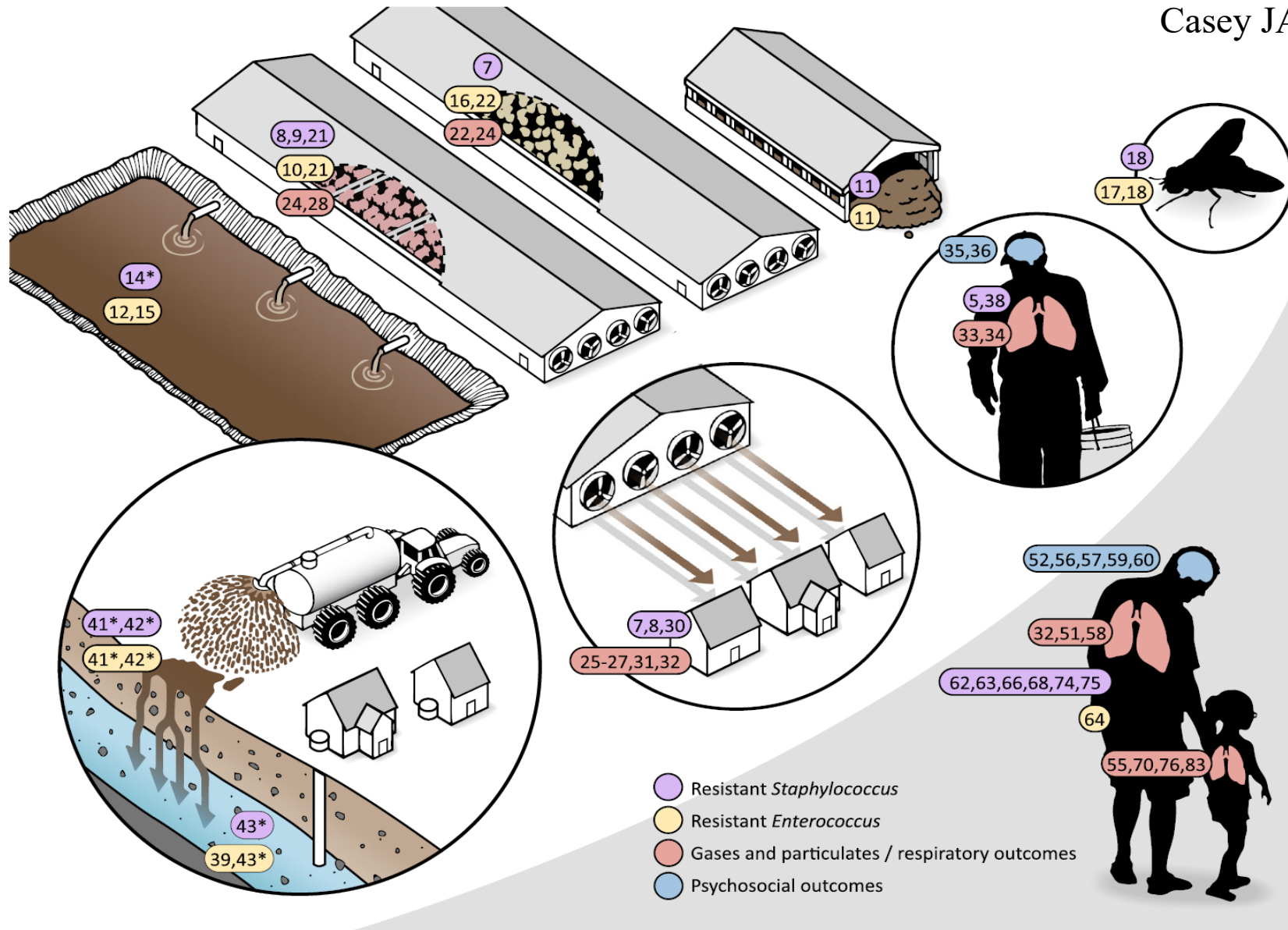


Fig. 1 Studies that document selected hazards associated with IFAP, illustrating potential pathways through the environment, and adverse health outcomes in nearby populations. *Numbers* indicate study

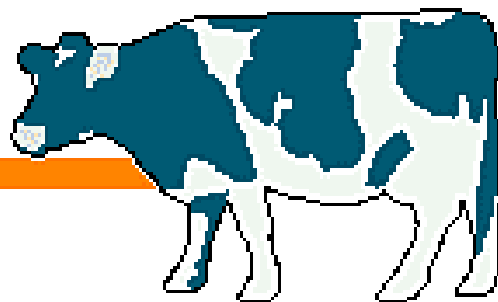
citations. *Study investigated the presence of resistance genes, which could be acquired by *S. aureus*, *Enterococcus*, or other pathogens

El ciclo de la dioxina

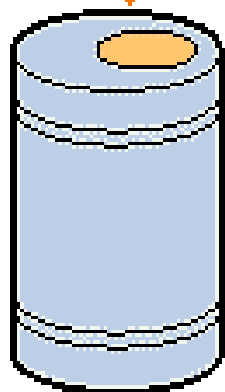
• La contaminación de la cadena alimenticia

18 y 19 de enero.

Un depósito de almacenamiento se contamina con dioxina.



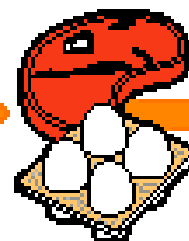
El depósito es usado para almacenar grasas animales.



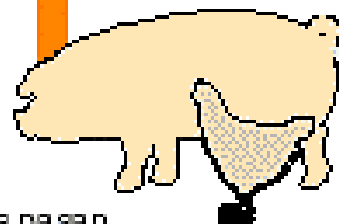
La grasa animal del depósito contaminado se procesa para fabricar alimentos para animales.

La dioxina se acumula en las grasas del cuerpo y se transmite directamente a través de la comida contaminada.

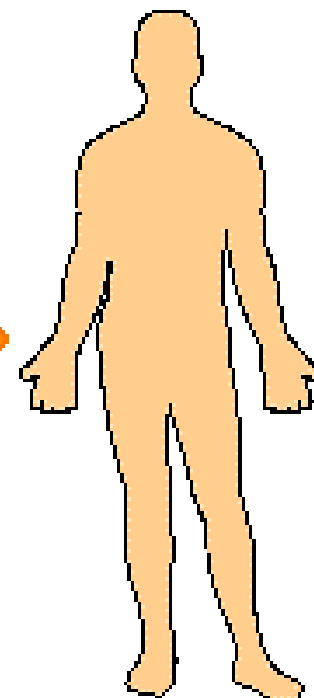
Carnes



Huevos



Las dioxinas pasan al organismo de los animales que consumen alimentos contaminados.



Las dioxinas presentes en productos contaminados de animales son consumidas por humanos.

Disruptores hormonales



Fenoles

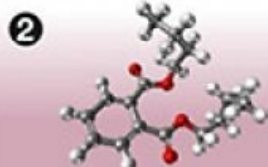
Bisfenol A, Parabenos, Benzofenoles, Triclosán

Se utiliza para para

Protección de envases alimentarios
Dan forma y resistencia a los plásticos

Se encuentran en

Tickets, botellas de agua, conservas, juguetes.
Cosméticos, productos capilares, protector solar.
Detergentes, textiles, productos de limpieza, material escolar.



Ftalato

Se utiliza para
Incrementar la flexibilidad de los plásticos

Se encuentran en

Embalajes, productos de higiene personal, juguetes, cosméticos, insecticidas, aspirina, aparatos médicos.

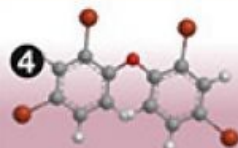


Bifenilos policlorados (PCB)

Se utiliza para
Antiinflamable, aislante térmico, estabilizador

Se encuentran en

Pintura, productos eléctricos, extintores, pigmentos, tinta, papel de albarán.



Bromados (BFR)

Se utiliza para
Retardantes de llama, inhiben la combustión

Se encuentran en

Aislantes, muebles, alfombras, polvo, sillas de bebé, piezas de plástico de equipo electrónico.



Perfluorados

Se utiliza para
Aplicaciones industriales

Se encuentran en

Envases y papel, textiles, cuero, fotografía, limpieza, cosméticos, pescado, marisco, bolsas de palomitas de microondas



A lo largo de la vida, grupos muy amplios de la población acumulan o están constantemente expuestos a mezclas de tóxicos a concentraciones altas y bajas. Con numerosas interacciones.

Tales concentraciones de tóxicos *parecen* bajas en la mayoría de la población sólo **cuando miramos a cada tóxico por separado.**

Es erróneo afirmar que la mayoría de la población tiene concentraciones bajas de tóxicos: **la mayoría de la población tiene concentraciones altas de algunos tóxicos y bajas de la mayoría de ellos.**

Preventive Medicine 55 (2012)

Human contamination by environmental chemical pollutants:
Can we assess it more properly?

Miquel Porta

3 tipos de razones por las que los tóxicos nos importan:

1. La mayoría de personas acumulamos mezclas de contaminantes a lo largo de la vida, tanto a concentraciones bajas como altas;

Contaminantes ambientales:

su papel en las enfermedades humanas.

¿por qué molestarse?

3 tipos de razones por las que los tóxicos nos importan:

1. La mayoría de personas acumulamos mezclas de contaminantes a lo largo de la vida, tanto a concentraciones bajas como altas;

2. Existe mucho conocimiento (básico, toxicológico, clínico, epidemiológico) sobre **los efectos conjuntos de tales mezclas** (por ej., alteraciones sobre funciones metabólicas y expresión génica, papel que los tóxicos tienen en la acumulación de alteraciones genéticas y epigenéticas, etc.); y

3. Desconocemos las causas de muchas enfermedades (cuando no tenemos en cuenta **1 y 2**).

por eso.

¿por qué molestarse?

Los tóxicos a dosis 'bajas', constantes y a lo largo de toda la vida contribuyen a causar:

- infertilidad, endometriosis
- malformaciones congénitas
- problemas de desarrollo, de aprendizaje
- trastornos hormonales
- trastornos en lípidos e hipertensión arterial
- diabetes tipo 2. ¿síndrome metabólico?
- algunos tipos de cáncer
- enfermedades neurológicas
- enfermedades inmunológicas, autoinmunes
- genotoxicidad indirecta y epigenética

ENDOCRINE FACTS AND FIGURES

FIRST EDITION

THYROID

ENDOCRINE
SOCIETY



State of the Science of Endocrine Disrupting Chemicals - 2012

Edited by
Åke Bergman, Jerrold J. Heindel, Susan Jobling,
Karen A. Kidd and R. Thomas Zoeller



ENDOCRINE FACTS AND FIGURES

ENDOCRINE
SOCIETY



HEALTH CONDITIONS ▾



Bone & Mineral
(Available Now)



Cardiovascular & Lipids
(Available Now)



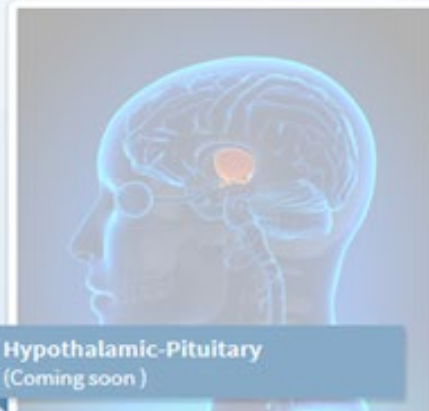
Diabetes
(Available now)



Obesity
(Available now)



Thyroid
(Available now)



Hypothalamic-Pituitary
(Coming soon)

Executive Summary to **EDC-2: The Endocrine Society's Second Scientific Statement on Endocrine-Disrupting Chemicals**

A. C. Gore, V. A. Chappell, S. E. Fenton, J. A. Flaws, A. Nadal, G. S. Prins, J. Toppari, and R. T. Zoeller

This Executive Summary to the Endocrine Society's second Scientific Statement on environmental endocrine-disrupting chemicals (EDCs) provides a synthesis of the key points of the complete statement. The full Scientific Statement represents a comprehensive review of the literature on **seven topics for which there is strong** mechanistic, experimental, animal, and epidemiological **evidence** for endocrine disruption, namely: **obesity and diabetes, female reproduction, male reproduction, hormone-sensitive cancers in females, prostate cancer, thyroid, and neurodevelopment and neuroendocrine systems**. EDCs such as **bisphenol A, phthalates, pesticides, persistent organic pollutants such as polychlorinated biphenyls, polybrominated diethyl ethers, and dioxins** were emphasized because these chemicals had the greatest depth and breadth of available information. The Statement also included thorough coverage of studies of **developmental exposures** to EDCs, especially in the fetus and infant, because these are critical life stages during which perturbations of hormones can increase the probability of a disease or dysfunction later in life. A conclusion of the Statement is that publications over the past 5 years have led to a much **fuller understanding of the endocrine principles by which EDCs act, including nonmonotonic dose-responses, low-dose effects, and developmental vulnerability**. These findings will prove useful to researchers, physicians, and other healthcare providers in translating the science of endocrine disruption to improved public health. (*Endocrine Reviews* 36: 0000–0000, 2015)

Table 1. Classifications, Histories, Chemical Properties, and Physiological Effects of Common EDCs

EDC	Group	Introduction date	Restricted/ Ban Date	Route of Exposure	Sources	Half-Life	Effects/Body Burden
Phthalates	Plasticizers	1920s	Restricted 2009	Ingestion, inhalation, dermal absorption	Contaminated food, PVC plastics and flooring, personal care products, medical devices and tubing	~12 h	Carcinogen, liver damage, reproductive and developmental effects, asthma, obesogen

Gore et al
(*Endocrine Reviews* 2015)

Cardenas, Oken. JAMA 2018.

Liu, Sun. PLoS Med 2018.

PFAS ↑ peso.

ENVIRONMENTAL HEALTH

(2015) 14:54

Open Access

Parma consensus statement on metabolic disruptors

Jerrold J. Heindel^{1*}, Frederick S. vom Saal², Bruce Blumberg³, Patrizia Bovolin⁴, Gemma Calamandrei⁵, Graziano Ceresini⁶, Barbara A. Cohn⁷, Elena Fabbri⁸, Laura Gioiosa⁹, Christopher Kassotis², Juliette Legler¹⁰,

Abstract

A multidisciplinary group of experts gathered in Parma Italy for a workshop hosted by the University of Parma, May 16–18, 2014 to address concerns about the potential relationship between environmental metabolic disrupting chemicals, obesity and related metabolic disorders. The objectives of the workshop were to: 1. Review findings related to the role of environmental chemicals, referred to as “metabolic disruptors”, in obesity and metabolic syndrome with special attention to recent discoveries from animal model and epidemiology studies; 2. Identify conclusions that could be drawn with confidence from existing animal and human data; 3. Develop predictions based on current data; and 4. Identify critical knowledge gaps and areas of uncertainty. The consensus statements are intended to aid in expanding understanding of the role of metabolic disruptors in the obesity and metabolic disease epidemics, to move the field forward by assessing the current state of the science and to identify research needs on the role of environmental chemical exposures in these diseases. We propose broadening the definition of obesogens to that of metabolic disruptors, to encompass chemicals that play a role in altered susceptibility to obesity, diabetes and related metabolic disorders including metabolic syndrome.

Keywords: Metabolic disruptor, Obesogen, Obesity, Diabetes, Metabolic syndrome, Developmental Programming

Chemical threat to brain development

BPA

Till receipts and tin cans



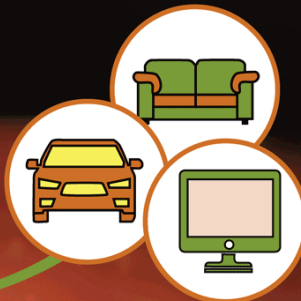
Phthalates/ BFRs

Household dust



BFRs

Car upholstery, sofas, screens



BPA/PFCs

Food containers like Pizza boxes



Pesticides

Agricultural pesticide sprays



PFCs

Waterproof clothing



Arsenic
Lead
Methylmercury
Perchlorate
What next...?

For more details: www.chemtrust.org.uk/brain



CHEMTrust

Protecting humans and wildlife from harmful chemicals

No Brainer

The impact of chemicals on children's brain development: a cause for concern and a need for action





OPINION

The secret danger of everyday things

RICK SMITH AND BRUCE LOURIE
SPECIAL TO THE GLOBE AND MAIL
PUBLISHED FEBRUARY 8, 2019



The coating on cash-register receipts is an unlikely source where people may be exposed to BPA and a related substance, BPS.

A review of dietary and non-dietary exposure to bisphenol-A

Tinne Geens^{a,k}, Dominique Aerts^{b,k}, Carl Berthot^{c,k}, Jean-Pierre Bourguignon^d
Philippe Lecomte^{f,k}, Guy Maghuin-Rogister^{g,k}, Anne-Madeleine Pironnet^{h,k},
Marie-Louise Scippo^{g,k}, Joris Van Loco^{j,k}, Adrian Covaci^{a,k,*}

Due to the large number of applications of bisphenol-A (BPA), the human exposure routes are multiple. We aimed to review shortly the food and non-food sources of BPA, and to evaluate their contribution to the human exposure. Food sources discussed here include epoxy resins, polycarbonate and other applications, such as paperboard and polyvinylchloride materials. Among the non-food sources, exposures through dust, thermal paper, dental materials, and medical devices were summarized. Based on the available data for these exposure sources, it was concluded that the exposure to BPA from non-food sources is generally lower than that from exposure from food by at least one order of magnitude for most studied subgroups. The use of urinary concentrations from biomonitoring studies was evaluated and the back-calculation of BPA intake seems reliable for the overall exposure assessment. In general, the total exposure to BPA is several orders of magnitude lower than the current tolerable daily intake of 50 µg/kg bw/day. Finally, the paper concludes with some critical remarks and recommendations on future human exposure studies to BPA.

Table 1Overview of **BPA** in canned food samples and canned beverages.

Country	Sample size	Detection freq. (%)	Range	Refs.
Canned food (ng/g)				
US	78	91	<2–730	Noonan et al. (2011)
US	97	59	<0.2–65	Schechter et al. (2010)
Canada	78	99	<0.6–534	Cao et al. (2010)
Japan	48	92	<1–842	Sajiki et al. (2007)
Korea	61	64	<3–136	Lim et al. (2009a)
Belgium	21	100	0.2–169	Geens et al. (2010)
Beverage cans (ng/mL)				
Spain	11	64	<0.05–0.61	Gallart-Ayala et al. (2010)
Canada	69	100	0.03–4.5	Cao et al. (2009a)
Belgium	45	91	<0.02–8.1	Geens et al. (2010)
Portugal	30	70	<0.01–4.7	Cunha et al. (2011)

Table 2

Migration of BPA from polycarbonate baby bottles.

Table 3

Estimated intake of BPA in children and adults.

Table 4

Overview of BPA in thermal paper.

Table 5

Overview of the most recent worldwide biomonitoring studies in urine.

Table 6

Overview of the estimated intake of BPA through multiple exposure pathways

Source	Country	Daily intake of BPA	Contribution to exposure scenario
Adults Total Food		1560–10453 ng/day	>90%
Canned food	New-Zealand	570 ng/day (average)–6900 (99th percentile)	
Canned food and beverages	Belgium	1050 ng/day (average)–6050 ng/day (95th percentile)	>90%
Dust	Eastern USA	8.44–109 ng/day (median – 95th percentile)	<1%
Dust	Belgium	29–244 ng/day (median – 95th percentile)	<5%
Thermal paper	USA-Japan-Korea-Vietnam	17.4–541 ng/day (median – 95th percentile)	<5%
		1303 – 40590 ng/day (median – 95th percentile)	





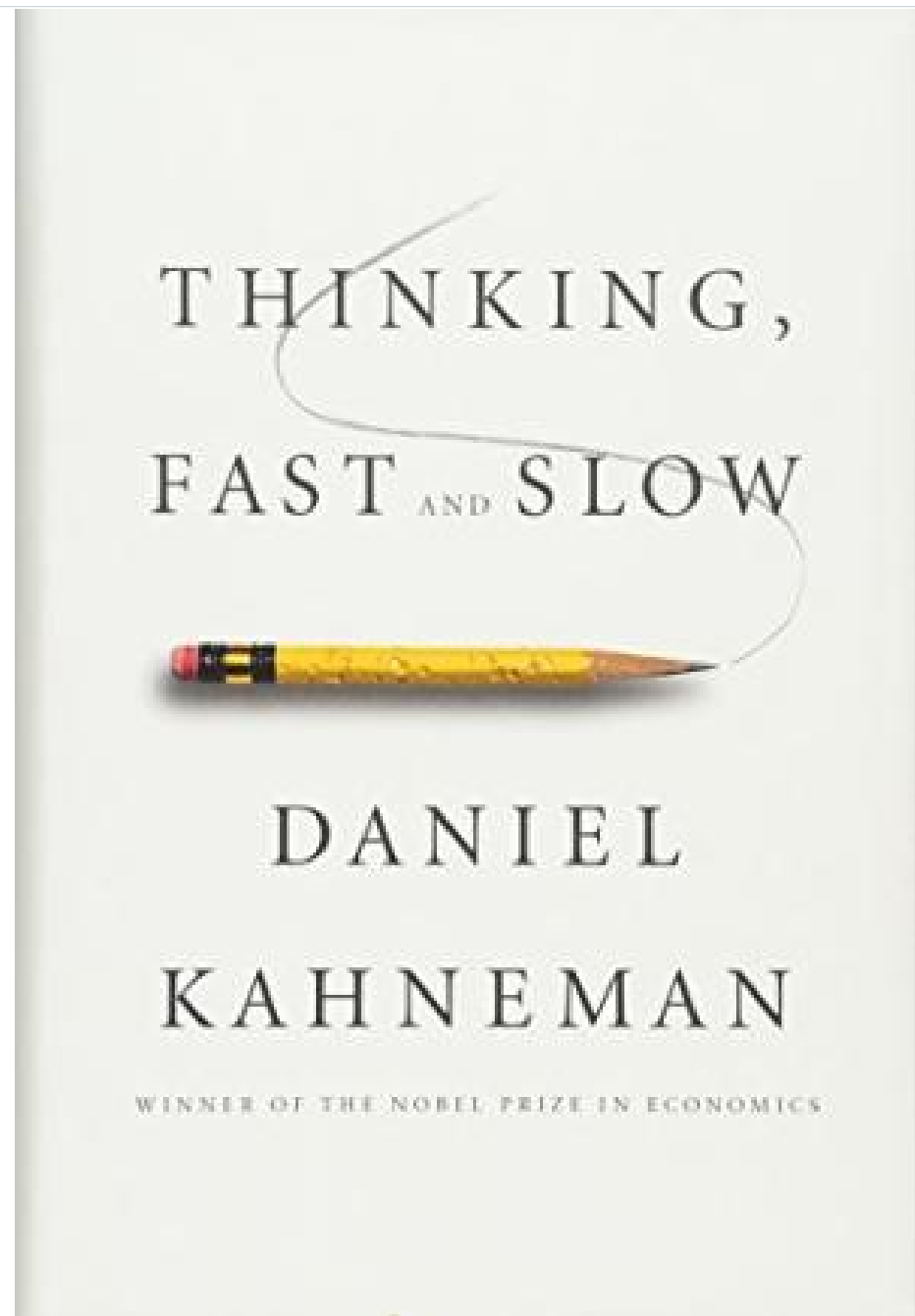
OPINION

The secret danger of everyday things

RICK SMITH AND BRUCE LOURIE
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The coating on cash-register receipts is an unlikely source where people may be exposed to BPA and a related substance, BPS.



Convivimos con problemas 'vintage' y problemas 'postmodernos'

- Algunos solo piensan en el daño que a **corto plazo** pueden hacer los **agentes infecciosos**, lo cual es importante, pero insuficiente.
 - Actúan así, en parte, porque las leyes y los ciudadanos no les pedimos más, y porque su formación ha omitido los **problemas infecciosos y no infecciosos a largo plazo**.
- Debemos y podemos actuar mejor para disminuir el sufrimiento humano y los costes económicos que causan los **agentes infecciosos y no infecciosos a corto y largo plazo**.

Conocimiento científico

– ¿Entre la legalidad y la competencia,
entre la normativa y las ventas...

...en el ingente trabajo de cada semana...

podéis hacer más o [supongo que]

ya **estamos haciendo todo el caso posible**

a los conocimientos científicos existentes y

emergentes con sus luces y sus incertidumbres?

– Hace años que la prensa de calidad informa. Y seguirá.

[datos ≠ información ≠ conocimiento]

Conocimiento científico (imperfecto, claro)

- Una parte de las muertes, trastornos e incapacidades que sufrimos por trastornos y enfermedades crónicas o degenerativas como el cáncer, infertilidad, diabetes o Alzheimer se debe a las mezclas de contaminantes químicos artificiales que tenemos en el cuerpo durante años.
- Los tenemos por 2 motivos:
 - 1) porque estamos expuestos a ellos cotidianamente o
 - 2) porque nuestro cuerpo no los excreta o elimina y se nos acumulan.
- Desde el vientre materno y durante la vida, tales contaminantes contribuyen a que acumulemos múltiples alteraciones genéticas y epigenéticas (lesionan nuestro ADN o hacen que los genes funcionen mal) o son dañinos para el sistema nervioso o actúan como “hormonas artificiales” o por otros mecanismos.
- La principal vía de entrada en nuestro cuerpo de tales contaminantes químicos artificiales son los alimentos y sus envases, el aire que respiramos, el agua que bebemos y otros numerosos bienes de consumo.

Paloma Alonso-Magdalena, Ivan Quesada and Angel Nadal

Endocrine disruptors in the etiology of type 2 diabetes mellitus

Abstract | The etiology of type 2 diabetes mellitus involves the induction of insulin resistance along with the disruption of pancreatic β -cell function and the loss of β -cell mass. In addition to a genetic predisposition, lifestyle factors seem to have an important role. Epidemiological studies indicate that the increased presence of endocrine disrupting chemicals (EDCs) in the environment may also play an important part in the incidence of metabolic diseases. Widespread EDCs, such as dioxins, pesticides and bisphenol A, cause insulin resistance and alter β -cell function in animal models. These EDCs are present in human blood and can accumulate in and be released from adipocytes. After binding to cellular receptors and other targets, EDCs either imitate or block hormonal responses. Many of them act as estrogens in insulin-sensitive tissues and in β cells, generating a pregnancy-like metabolic state characterized by insulin resistance and hyperinsulinemia. Adult exposure in mice produces insulin resistance and other metabolic alterations; in addition, during pregnancy, EDCs alter glucose metabolism in female mice, as well as glucose homeostasis and endocrine pancreatic function in offspring. Although more experimental work is necessary, evidence already exists to consider exposure to EDCs as a risk factor in the etiology of type 2 diabetes mellitus and other diseases related to insulin resistance.

Role of Environmental Chemicals in Diabetes and Obesity: A National Toxicology Program Workshop Review

Kristina A. Thayer,¹ Jerrold J. Heindel,² John R. Bucher,³ and Michael A. Gallo⁴

Overall, the existing literature was judged to provide plausibility, varying from suggestive to strong, that exposure to environmental chemicals may contribute to the epidemic of diabetes and/or obesity.

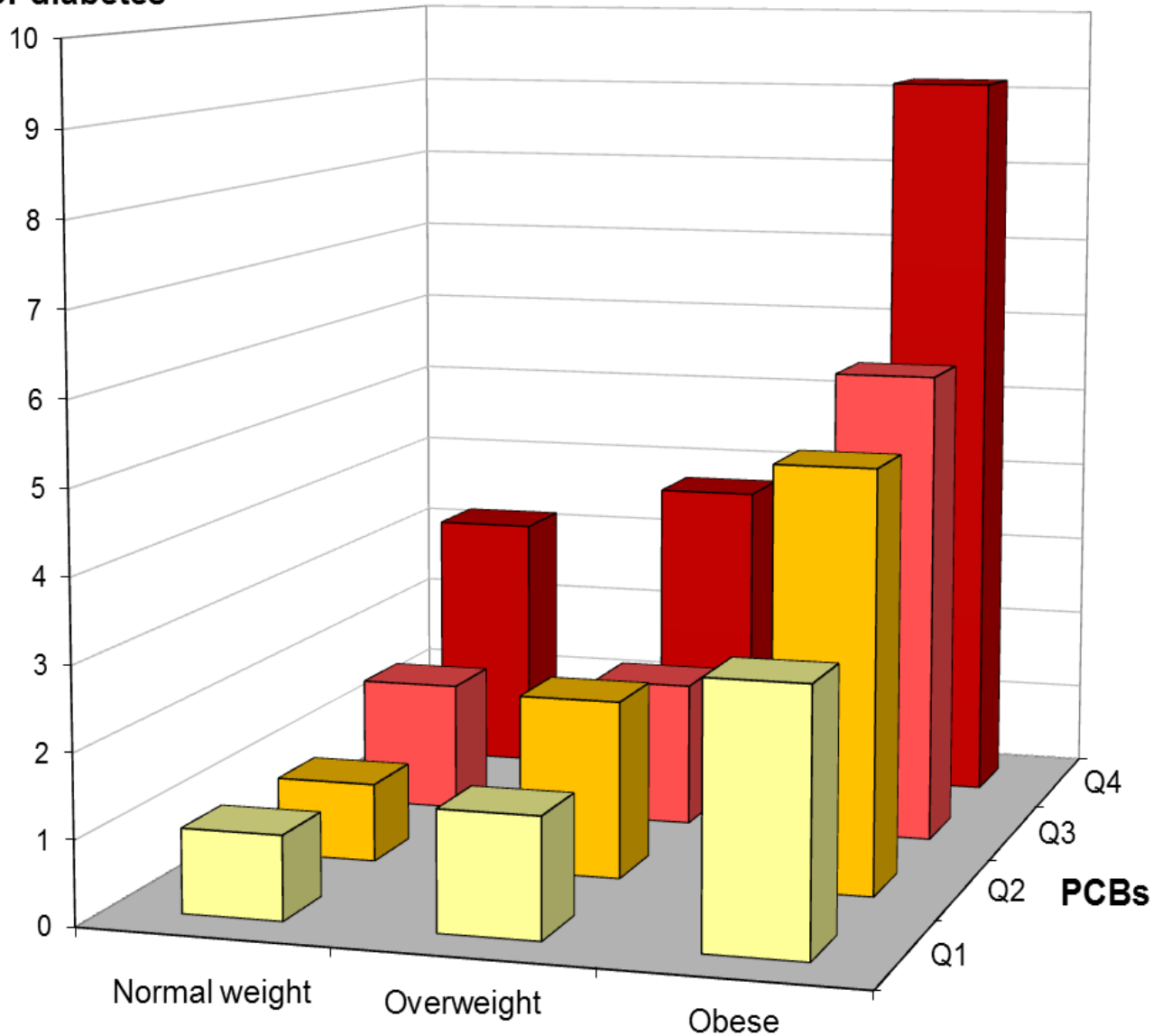
Persistent organic pollutants and the burden of diabetes

Studies from the USA^{1,2} have drawn attention to the possibility that persistent organic pollutants might contribute to cause diabetes.³⁻⁶

Because they contaminate virtually all people, even if they confer only a low individual risk of diabetes, these pollutants might have a substantial overall population effect.

Catalonia 2002. The prevalence of diabetes increases with Body Mass Index and with serum concentrations of PCBs (N = 684).

**Adjusted OR
for diabetes**



PCBs: Sum of orders of PCBs 118, 138, 153, 180.

All odds ratios (OR) are computed with Q1 and normal weight as the reference category, with models adjusted by age, sex, total cholesterol and triglycerides.



ACCEPTED MANUSCRIPT



Blood Concentrations of Persistent Organic Pollutants and Unhealthy Metabolic Phenotypes in Normal-weight, Overweight and Obese Individuals

Magda Gasull ✉, Conxa Castell, Natàlia Pallarès, Carme Miret, José Pumarega, María Téllez-Plaza, Tomàs López, Jordi Salas-Salvadó, Duk-Hee Lee, Albert Goday Miquel Porta

Published: 30 June 2017 **Article history** ▼



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ABSTRACT

Factors underlying metabolic phenotypes, such as the metabolically healthy but obese phenotype, remain unclear. Differences in metabolic phenotypes –particularly, among individuals with a similar body mass index– could be related to concentrations of persistent organic pollutants (POPs). No studies have analyzed POPs and metabolic phenotypes in normal-weight persons. The



Los tóxicos y tú: tanto que hacer

El investigador Miquel Porta acaba de publicar el libro 'Vive más y mejor. Reduciendo tóxicos y contaminantes ambientales' (Grijalbo)

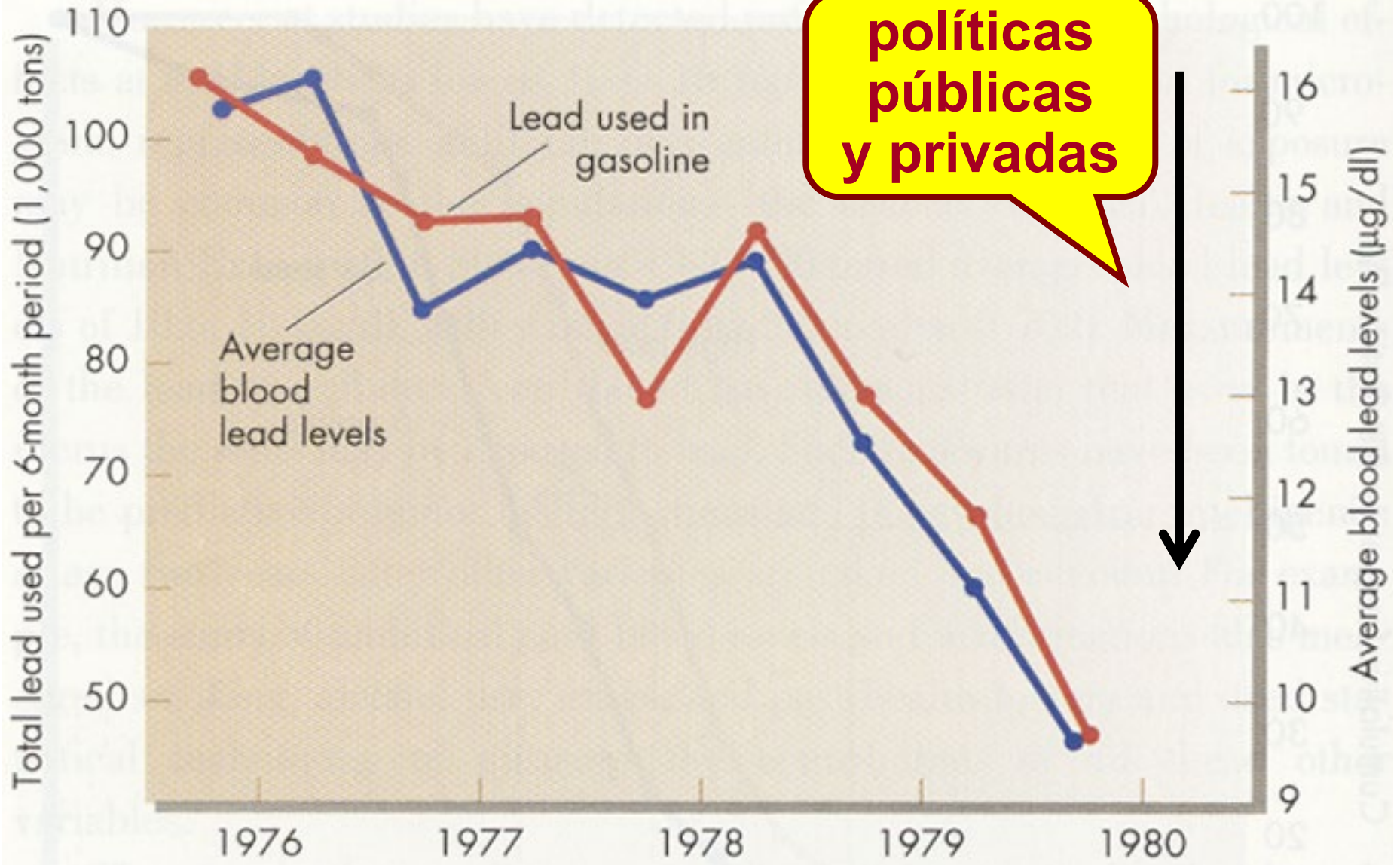
MIQUEL PORTA

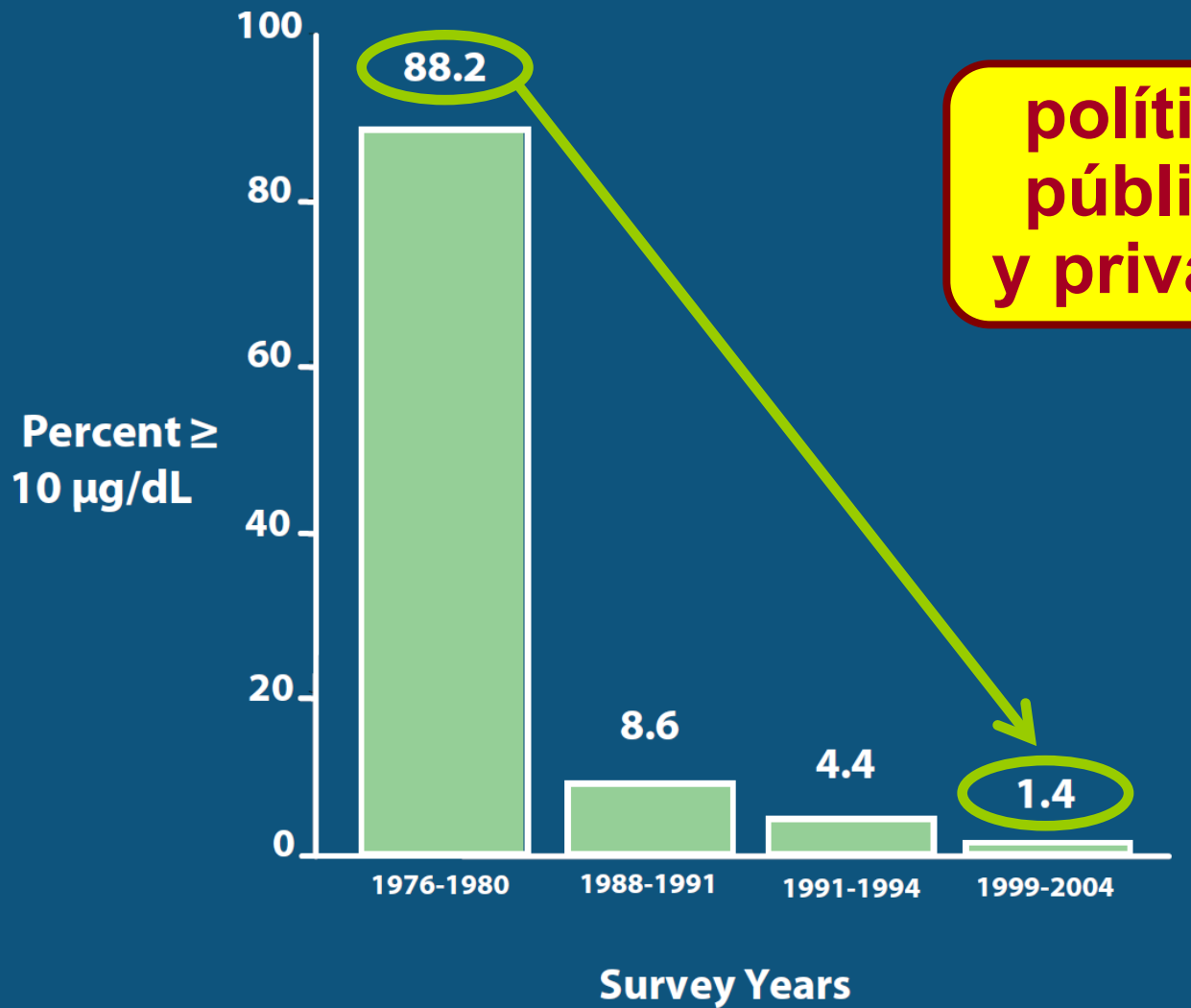
1 OCT 2018 - 10:30 CEST



Tras eliminar el plomo de la gasolina, la reducción espectacular en los niveles corporales de plomo de millones de personas desde la mitad de la década de 1980 es un caso ejemplar de **cómo políticas pública y privadas externas al sistema sanitario han mejorado nuestro bienestar y nuestra salud.** Hoy la inmensa mayoría de los fetos ya no están prácticamente expuestos a él. Lo demuestran miles de análisis en sangre de cordón umbilical. Igual ocurre en todas las otras edades. Los beneficios biofísicos, cognitivos y económicos han sido bien cuantificados. Los beneficios humanos e inmateriales son percibidos por quien lo intenta.

<https://bit.ly/2Nc8DFK>





políticas
públicas
y privadas

Figure 1. Percentage of children 1-5 years old in the U.S. population with elevated blood lead levels ($\geq 10 \mu\text{g}/\text{dL}$).¹

Economic Gains Resulting from the Reduction in Children's Exposure to Lead in the United States

Scott D. Grosse,¹ Thomas D. Matte,¹ Joel Schwartz,² and Richard J. Jackson¹

¹National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta, Georgia, USA; ²School of Public Health, Harvard University, Cambridge, Massachusetts, USA

In this study we quantify economic benefits from projected improvements in worker productivity resulting from the reduction in children's exposure to lead in the United States since 1976. We calculated the decline in blood lead levels (BLLs) from 1976 to 1999 on the basis of nationally representative National Health and Nutrition Examination Survey (NHANES) data collected during 1976 through 1980, 1991 through 1994, and 1999. The decline in mean BLL in 1- to 5-year-old U.S. children from 1976–1980 to 1991–1994 was 12.3 $\mu\text{g}/\text{dL}$, and the estimated decline from 1976 to 1999 was 15.1 $\mu\text{g}/\text{dL}$. We assumed the change in cognitive ability resulting from declines in BLLs, on the basis of published meta-analyses, to be between 0.185 and 0.323 IQ points for each 1 $\mu\text{g}/\text{dL}$ blood lead concentration. These calculations imply that, because of falling BLLs, U.S. preschool-aged children in the late 1990s had IQs that were, on average, 2.2–4.7 points higher than they would have been if they had the blood lead distribution observed among U.S. preschool-aged children in the late 1970s. We estimated that each IQ point raises worker productivity 1.76–2.38%. With discounted lifetime earnings of \$723,300 for each 2-year-old in 2000 dollars, the estimated economic benefit for each year's cohort of 3.8 million 2-year-old children ranges from \$110 billion to \$319 billion.

Biomonitoring exposure assessment to contemporary pesticides in a school children population of Spain

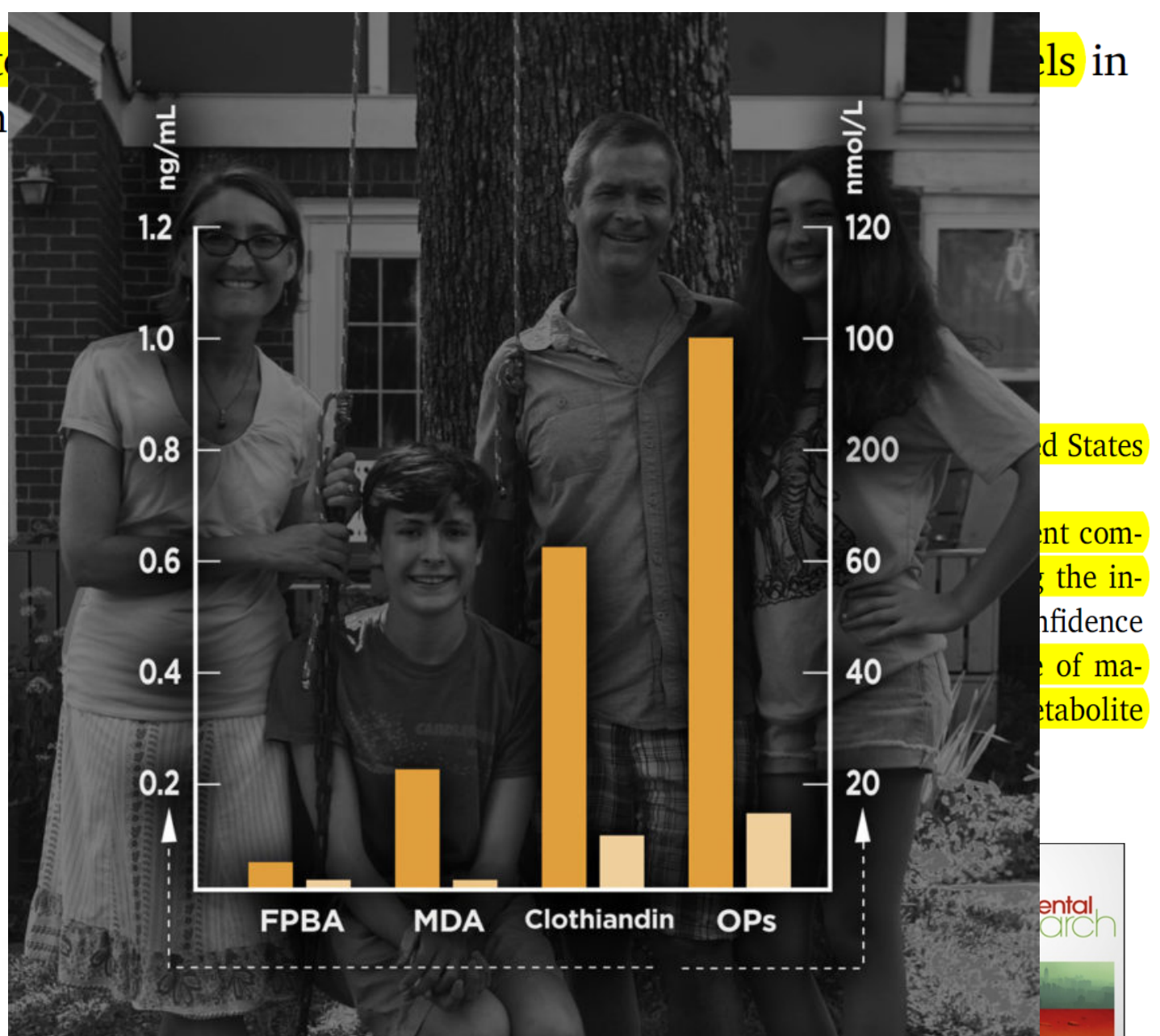
Marta Roca^{a,b}, Ana Miralles-Marco^{a,b}, Joan Ferré^d, Rosa Pérez^a, Vicent Yusà^{a,b,c,*}

The exposure to pesticides amongst school-aged children (6–11 years old) was assessed in this study. One hundred twenty-five volunteer children were selected from two public schools located in an agricultural and in an urban area of Valencia Region, Spain. Twenty pesticide metabolites were analyzed in children's urine as biomarkers of exposure to organophosphate (OP) insecticides, synthetic pyrethroid insecticides, and herbicides. These data were combined with a survey to evaluate the main predictors of pesticide exposure in the children's population. A total of 15 metabolites were present in the urine samples with detection frequencies (DF) ranging from 5% to 86%. The most frequently detected metabolites with DF > 53%, were 3,5,6-trichloro-2-pyridinol (TCPy, metabolite of chlorpyrifos), diethyl phosphate (DEP, generic metabolite of OP insecticides), 2-isopropyl-4-methyl-6-hydroxypyrimidine (IMPY, metabolite of diazinon) and para-nitrophenol (PNP, metabolite of parathion and methyl parathion). The calculated geometric means ranged from 0.47 to 3.36 µg/g creatinine, with TCPy and IMPY showing the higher mean concentrations. Statistical significant differences were found between exposure subgroups (Mann–Whitney test, $p < 0.05$) for TCPy, DEP, and IMPY. Children living in the agricultural area had significantly higher concentrations of DEP than those living in the urban area. In contrast, children aged 6–8 years from the urban area, showed statistically higher IMPY levels than those from agricultural area. Higher levels of TCPy were also found in children with high consumption of vegetables and higher levels of DEP in children whose parents did not have university degree studies. The multivariable regression analysis showed that age, vegetable consumption, and residential use of pesticides were predictors of exposure for TCPy, and IMPY; whereas location and vegetable consumption were factors associated with DEP concentrations. Creatinine concentrations were the most important predictors of urinary TCPy and PNP metabolites.

Organic diet intake in U.S. children and

Carly Hyland^a, Asa
Robert B. Gunier^a, et al.

Methods: We collected urine samples before and after an organic diet intervention.
Results: We observed significant reductions in pesticide residues representing 10 different pesticides. The introduction of an organic diet resulted in a 95% reduction in the concentration of malathion (95% CI: -95.0%; 95% CI: -95.0%) and a 60% reduction in the concentration of chlorpyrifos (-60.0%; 95% CI: -60.0%; 95% CI: -60.0%).



The pesticide residues present in the Boyd family's urine samples while eating a conventional (dark orange) and organic (light orange) diet.



The Organic Effect



CoopSverige

Subscriu-m'hi 3.991

5.167.004

+ Afegeix a... Comparteix Més

5.836 1.288

Data de publicació: 4 maig 2015

Want to know what happens in your body when you switch from eating conventional food to organic? Watch this! The study was conducted by the Swedish Environmental Research Institute IVL, and the full report is available here: <https://www.coop.se/organiceffect>



The Organic Effect



CoopSverige



The Organic Effect

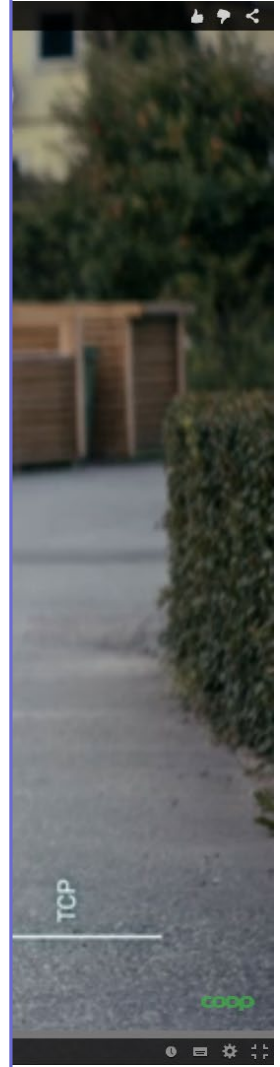


Human exposure to pesticides from food

A pilot study

For Coop Sverige AB

Jörgen Magnér, Petra Wallberg, Jasmin Sandberg, Anna Palm Cousins



TCP

coop

Obesity, Diabetes, and Associated Costs of Exposure to Endocrine-Disrupting Chemicals in the European Union

Juliette Legler, Tony Fletcher, Eva Govarts, Miquel Porta, Bruce Blumberg, Jerrold J. Heindel, and Leonardo Trasande

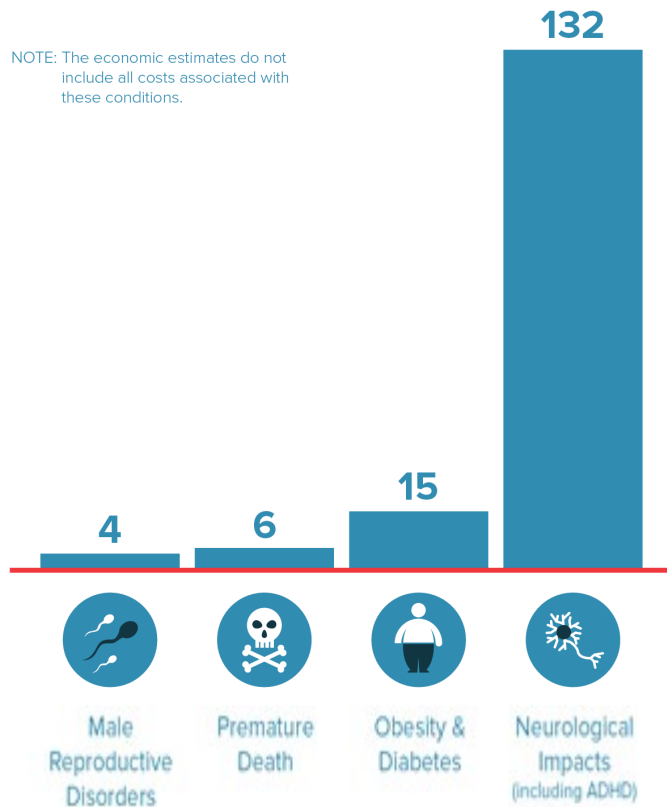
Results: The panel identified a 40% to 69% probability of dichlorodiphenyldichloroethylene causing 1555 cases of overweight at age 10 (sensitivity analysis: 1555–5463) in 2010 with associated costs of €24.6 million (sensitivity analysis: €24.6–86.4 million). A 20% to 39% probability was identified for dichlorodiphenyldichloroethylene causing 28 200 cases of adult diabetes (sensitivity analysis: 28 200–56 400) with associated costs of €835 million (sensitivity analysis: €835 million–16.6 billion). The panel also identified a 40% to 69% probability of phthalate exposure causing 53 900 cases of obesity in older women and €15.6 billion in associated costs. Phthalate exposure was also found to have a 40% to 69% probability of causing 20 500 new-onset cases of diabetes in older women with €607 million in associated costs. Prenatal bisphenol A exposure was identified to have a 20% to 69% probability of causing 42 400 cases of childhood obesity, with associated lifetime costs of €1.54 billion.

Conclusions: EDC exposures in the EU contribute substantially to obesity and diabetes, with a moderate probability of >€18 billion costs per year. This is a conservative estimate; the results emphasize the need to control EDC exposures. (*J Clin Endocrinol Metab* 100: 1278–1288, 2015)

HEALTH EFFECTS FROM ENDOCRINE DISRUPTING CHEMICALS COST THE EU 157 BILLION EUROS EACH YEAR.

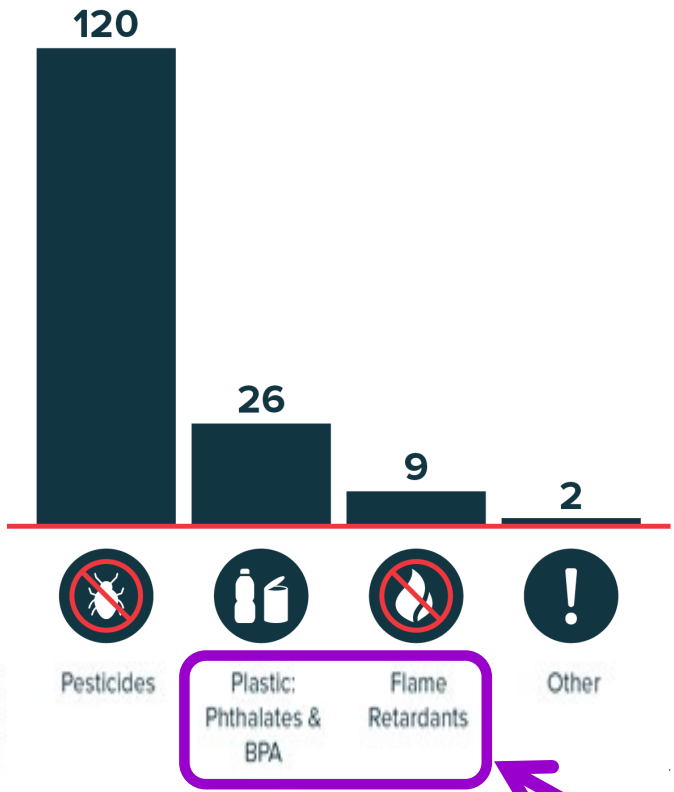
This is the tip of the iceberg: Costs may be as high as €270B.

€157B Cost by Health Effect



NOTE: The economic estimates do not include all costs associated with these conditions.

€157B Cost by EDC Type



Endocrine Disrupting Chemicals (EDCs) interfere with hormone action to cause adverse health effects in people.

“THE TIP OF THE ICEBERG”

The data shown to the left are based on fewer than 5% of likely EDCs. Many EDC health conditions were not included in this study because key data are lacking. Other health outcomes will be the focus of future research.

SOME EDC-RELATED HEALTH OUTCOMES NOT INCLUDED:

- Breast Cancer
- Prostate Cancer
- Immune Disorders
- Female Reproductive Disorders
- Liver Cancer
- Parkinson's Disease
- Osteoporosis
- Endometriosis
- Thyroid Disorders

SOME EDCs NOT INCLUDED:

- Atrazine
- 2, 4-D
- Styrene
- Triclosan
- Nonylphenol
- Polycyclic Aromatic Hydrocarbons
- Bisphenol S
- Cadmium
- Arsenic
- Ethylene glycol

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Health

Hormone-disrupting chemicals ‘cost billions’

By James Gallagher

Health editor, BBC News website, San Diego

 6 March 2015 | [Health](#)

Common chemicals that disrupt human hormones could be costing more than €150bn (\$165.4bn; £108.5bn) a year in damage to human health in Europe, a series of studies claims.

**Sólo un 5,9% de recién nacidos
tuvieron >2µg/dL de plomo en sangre!**

Table 2

Distribution of cord blood lead levels in the 4 cohorts participating in the INMA project (µg/dL).

	N	N>LOQ (2 µg/dL)	%> LOQ (CI)	AM ^a	SD ^a	GM ^a	Maximum
Asturias	341	14	4.1 (2.3–6.8)	1.12	1.06	1.05	19.0
Gipuzkoa	527	46	8.7 (6.5–11.5)	1.14	0.50	1.08	5.0
Sabadell	297	7	2.3 (0.9–4.8)	1.04	0.26	1.02	4.0
Valencia	301	20	6.6 (4.1–10.1)	1.13	0.61	1.07	7.0
Total	1466	87	5.9 (4.8–7.3)	1.11	0.67	1.06	19.0

N: sample size.

LOQ: limit of determination (2 µg/dL).

CI: confidence intervals.

AM: arithmetic mean.

SD: standard deviation.

GM: geometric mean.

P95: 95th percentile.

^a Non-detected levels were counted with half of LOQ.

**Un 64% de recién nacidos
tuvieron niveles de mercurio en sangre
superiores a los recomendados por la EPA... (!)**

Cord blood total mercury concentrations ($\mu\text{g/L}$) by area. INMA study, Spain, 2004–8.

T-Hg ($\mu\text{g/L}$)	n	Mean	GM	%<LOD= 2 $\mu\text{g/L}$	%> EPA RfD ^a	Percentiles				Max
						25th	50th	75th	90th	
Valencia	554	13.1	9.5	4.2	68.4	5.3	9.5	18.0	26.5	66.0
Sabadell	460	8.2	6.3	7.6	49.1	4.1	6.4	10.0	16.0	60.0
Asturias	340	13.9	10.8	3.2	75.6	6.6	12.0	18.8	25.9	69.0
Gipuzkoa	529	9.3	7.5	3.8	64.7	5.1	8.1	12.0	17.0	50.0
All cohort	1883	11.0	8.2	4.7	63.9	5.0	8.5	14.0	22.0	69.0

GM: geometric mean; LOD: limit of determination; EPA: Environmental Protection Agency, RfD: reference dose.

^a 6.4 $\mu\text{g/L}$ as the T-Hg equivalent to the EPA RfD for methylmercury (5.8 $\mu\text{g/L}$) assuming that methylmercury accounts for $\geq 90\%$ of total mercury (Mahaffey, 2005).

Prenatal mercury exposure in a multicenter cohort study in Spain

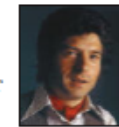
R. Ramon et al. / Environment International 37 (2011)



sociedad
El CSIC patrocinó estudios racistas en la era colonial



sociedad
Multa a Danone por exagerar el beneficio del yogur



cultura
El rico legado de Enrique Morente

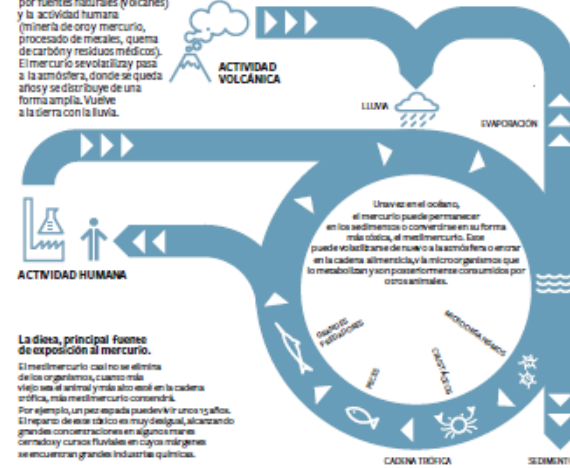


pantallas
El pecé pierde militantes en favor de la tableta



El ciclo del mercurio en la naturaleza

El mercurio se libera al entorno por fuentes naturales (volcánicas) y la actividad humana (minería de oro y mercurio, procesamiento de metales, quema de carbón y residuos médicos). El mercurio se volatiliza y pasa a la atmósfera, donde se queda años y se distribuye de una forma amplia. Vuelve a la tierra con la lluvia.



La dieta, principal fuente de exposición al mercurio.
El metilmercurio, casi no se elimina de los organismos, cuanto más viejo sea el animal y más alto está en la cadena trófica, más metilmercurio contendrá. Por ejemplo, un pez espada puede vivir unos 15 años. El reparto de este tóxico es muy desigual, alcanzando grandes concentraciones en algunas marisacas y cursos fluviales en cuyos márgenes se encuentran grandes industrias químicas.

Alimentos y metales pesados
El mercurio se encuentra en más alimentos de los señalados a continuación, pero son estos en los que más se detecta y los que más contribuyen a la entrada en nuestro organismo.

PISCADOS
(en especial el grupo de los predadores, como el salmón o el emperador, y el marisco)
Anémico, cadmio, mercurio, plomo, cinc, níquel, arsénico, bifenilos, heptaclorociclo y sabateno.

ARROZ
Pequeñas cantidades de arsénico.
Café.

ACEITES Y GRASAS
(también derivados lácteos)
Plomo, cinc, níquel, arsénico, hidrocarburos aromáticos, heptaclorociclo y sabateno, entre otros.

PAN Y CEREALES
Dioxina, cadmio, arsénico e hidrocarburos aromáticos.
CARNE
Hidrocarburos aromáticos y heptaclorociclo.

- ▶ La contaminación del pescado cuestiona la recomendación de consumo regular
- ▶ Compuestos cotidianos afectan al desarrollo cognitivo o al sistema reproductor

Los científicos confirman un descenso de contaminantes como el plomo en el ambiente. Preocupan más el pescado y el marisco porque las concentraciones de compuestos no bajan.

La mayoría de alertas de la Agencia Española fueron por estos animales

Cuanto más viejos y grasos sean, más afectan al organismo humano

La cosmética es otra fuente contaminante, según los científicos

No es fácil vincular a estos agentes con la aparición de enfermedades

Se han relacionado los clorados con alteraciones en la función cognitiva

“El cuerpo tiene compuestos que nunca antes tuvo”, dice un científico

Chicas embarazadas:

Porcentajes de detección >40%, >70%, >90% de compuestos tóxicos persistentes.

Organochlorine levels during the first trimester of pregnancy in the Gipuzkoa ($n = 628$) and Sabadell ($n = 631$) cohorts. measured in the serum of 1259 pregnant women.

Organochlorine compounds (ng g^{-1} lipid)	Gipuzkoa		Sabadell	
	% Detected ^b	GM (95% CI) ^a	% Detected	GM (95% CI) ^a
PCB 138	92	29.3 (27.7–31.0)	76	16.5 (15.7–17.3)
PCB 180	95	36.0 (34.1–37.9)	82	20.3 (19.2–21.4)
PCB 153	97	50.1 (47.5–52.8)	92	30.7 (29.1–32.3)
HCB	90	32.0 (30.1–34.1)	90	35.1 (32.9–37.5)
β -HCH	47	12.0 (11.2–12.8)	89	30.3 (28.5–32.3)
p,p' -DDE	98	95.8 (89.8–102.2)	100	126.1 (118.1–134.6)

^a Geometric mean and 95% confidence interval.

^b Percentage of samples with levels above the detection limit.

Sociodemographic, reproductive and dietary predictors compounds levels in pregnant women in Spain

J. Ibarluzea et al. / Chemosphere 82 (2011)

pero las concentraciones han estado bajando

Baja la 'contaminación interior'

Por primera vez disminuyen los niveles en sangre de los compuestos tóxicos persistentes ● Aun así solo el 4% de la población tiene cantidades reducidas



La contaminación atmosférica y la alimentación son las dos vías de entrada al organismo de los compuestos tóxicos persistentes. / TEJEDERAS

Baja la 'contaminación interior'

Por primera vez disminuyen los niveles en sangre de los compuestos tóxicos persistentes ● Aun así solo el 4% de la población tiene cantidades reducidas

MIGUEL PORTA

Los niveles sanguíneos de compuestos tóxicos persistentes (CTP) descendieron de forma significativa en los habitantes de Barcelona entre 2002 y 2006, según un estudio de la Agencia de Salud Pública de esa ciudad. Los policlorobifenilos (PCB, productos de origen industrial que suelen contaminar a dosis bajas los alimentos grasos) disminuyeron más de un 30%; el DDT (plaguicida prohibido hace más de 30 años), descendió un 39%, mientras que los niveles de su principal metabolito, el DDE, lo hicieron un 53%. El hexa-clorobenceno (un fungicida) y el beta-hexaclorociclohexano (un compuesto relacionado con el insecticida lindano) disminuyeron un 53% y un 50%, respectivamente. Los resultados, en líneas generales, deberían ser extrapolables al resto de España, aunque habría que estudiarlo y tener en cuenta los condicionantes de cada lugar. La magnitud del descenso en los niveles corporales de los tóxicos fue similar en mujeres y hombres, algo mayor en las personas más jóvenes, y mayor en las personas obesas.

Es la primera vez que una ciudad española analiza las concentraciones sanguíneas de compuestos tóxicos persistentes en dos momentos diferentes: tampoco lo ha hecho ninguna comunidad autónoma. Barcelona integró tales análisis en los sondeos

o encuestas de salud que se realizaron en muestras representativas de su población en 2002 y 2006. Otras ciudades llevan a cabo encuestas de salud, pero ninguna ha analizado nunca la contaminación interna por CTP. En los dos años mencionados los métodos epidemiológicos y químicos fueron idénticos, garantizando así la validez de la comparación. Nuestro estudio lo publica la revista *Science of the Total Environment*.

Las razones de la disminución no están claras. Lo más verosímil es que se deba primordialmente a las políticas de control de los CTP en alimentos desarrolladas durante décadas por las autoridades y empresas que operan en la ciudad.

Las mezclas de CTP que habitualmente se detectan en las poblaciones del planeta tienen efectos inmunosupresores, inflamatorios, neurotóxicos, metabólicos, endocrinos, epigenéticos o cancerígenos. Existen amplios conocimientos científicos —aunque no siempre concluyentes, como es habitual en ciencia— de que estos compuestos aumentan el riesgo de diversos cánceres (como los linfomas no-Hodgkin), infertilidad, asma, parkinson, diabetes, problemas tiroideos o de aprendizaje. Es pues plausible que la contaminación por CTP explique una parte relevante de la carga de enfermedad que sufrimos.

Entre los ciudadanos existen grandes diferencias en su contaminación; por ejemplo, el nivel sanguíneo más alto de DDE hallado en una persona (8.227 nanogramos/gramo, ng/g) fue 1.100 veces superior al de quien tuvo menos (7 ng/g); las concentraciones de otros compuestos son en unas personas centenares de veces superiores a las de otras. Las

El control de la producción de alimentos favorece el descenso

Las sustancias se acumulan en la grasa de los animales

razones de las diferencias individuales en la impregnación corporal por tóxicos no están claras, aunque esta suele aumentar con la edad y el peso. Ocho de los 19 CTP analizados se detectaron en la mayoría de la población barcelonesa: el DDT se detectó en la sangre del 97% de los participantes y el DDE en el 100%, mientras que el hexa-clorobenceno y el beta-hexaclorociclohexano se detectaron en un 98% y un 97%, respectivamen-

te. El número mínimo de contaminantes que se detectó en una persona fue de cinco, y el máximo, 15. Por tanto, la totalidad de la población almacena estos compuestos. El 72% de los barceloneses acumula en su cuerpo 10 o más tóxicos; ese porcentaje era el 90% en 2002. Los datos son en buena medida extrapolables a otras poblaciones españolas. Los 19 contaminantes analizados son una cifra modesta en relación a las decenas de compuestos que pueden detectarse en un ciudadano medio.

Muchos estudios analizan los niveles de cada CTP individualmente, no conjuntamente; observan entonces que una mayoría de la población tiene niveles muy inferiores a los de una relativa minoría. Este hecho —y la proverbial ceguera ante lo obvio, que también afecta a los científicos— ha hecho que durante años se creyese que apenas nadie tiene concentraciones altas de tóxicos. Lo que ha resultado ser falso, como ha puesto de relieve otro estudio nuestro, basado en una muestra representativa de la población de Cataluña, de inminente publicación en la revista *Environment International*.

El punto de partida es la ignorancia existente a nivel mundial acerca de una cuestión muy simple: ¿todos los individuos con niveles corporales bajos de algunos contaminantes tienen concentraciones asimismo bajas de

dos; el 34% de la población tiene niveles altos de tres o más tóxicos. Entre las mujeres de 60 a 74 años, el 48% tiene concentraciones altas de seis o más compuestos. Tan solo el 4% de la población catalana tiene concentraciones bajas de todos los CTP analizados. Por tanto, algunos subgrupos de ciudadanos acumulan mezclas de CTP a concentraciones altas.

Las componentes del sistema

Los tóxicos ingeridos provocan la aparición de cánceres

Solo al medirlos todos juntos se obtiene una idea real de su extensión

económico global causantes de la actual crisis generaron numerosas prácticas y productos financieros que —con curiosa sinceridad— se denominan tóxicos. La metáfora tiene la virtud de aludir a la naturaleza venenosa de las causas de la crisis y a sus perniciosos efectos. Pero entre estos no contabiliza la carga de sufrimiento, enfermedad y muerte que el sistema vigente contribuye a causar. Es más, todos sabemos que esta no es una crisis exclusivamente financiera o económica, sino una crisis de ciertos modelos de economía, política y cultura. Disminuir la contaminación humana por compuestos que no son tóxicos metafóricamente, sino literalmente, exige que promovamos otros sistemas de economía, cultura y sociedad. Debemos promover políticas públicas y privadas más humanas, saludables y socialmente eficientes —otras políticas sobre ganadería y agricultura, consumo y seguridad alimentaria, condiciones laborales, energía, medio ambiente y salud pública—.

Es tiempo de que las organizaciones ciudadanas hagan aumentar el cumplimiento de las normas jurídicas autonómicas, estatales e internacionales —como la Ley General de Salud Pública y el Convenio de Estocolmo— que establecen que los Gobiernos (central, autonómicos y municipales) deben vigilar y controlar la contaminación interna por compuestos ambientales. Sería toda una señal de que ya funcionan los nuevos valores, conocimientos y políticas que necesitamos para salir auténticamente de la crisis, por tantos motivos en verdad tóxica.

Miguel Porta Serra es médico y epidemiólogo.

Las razones de la disminución no están claras. Lo más verosímil es que se deba primordialmente a las políticas de control de los CTP en alimentos desarrolladas durante décadas por las autoridades y empresas que operan en la ciudad.

p,p' -DDE ($\mu\text{g/L}$)

Paediatric and Perinatal Epidemiology, 19, 31–35 2005

Gerd M. Lackmann

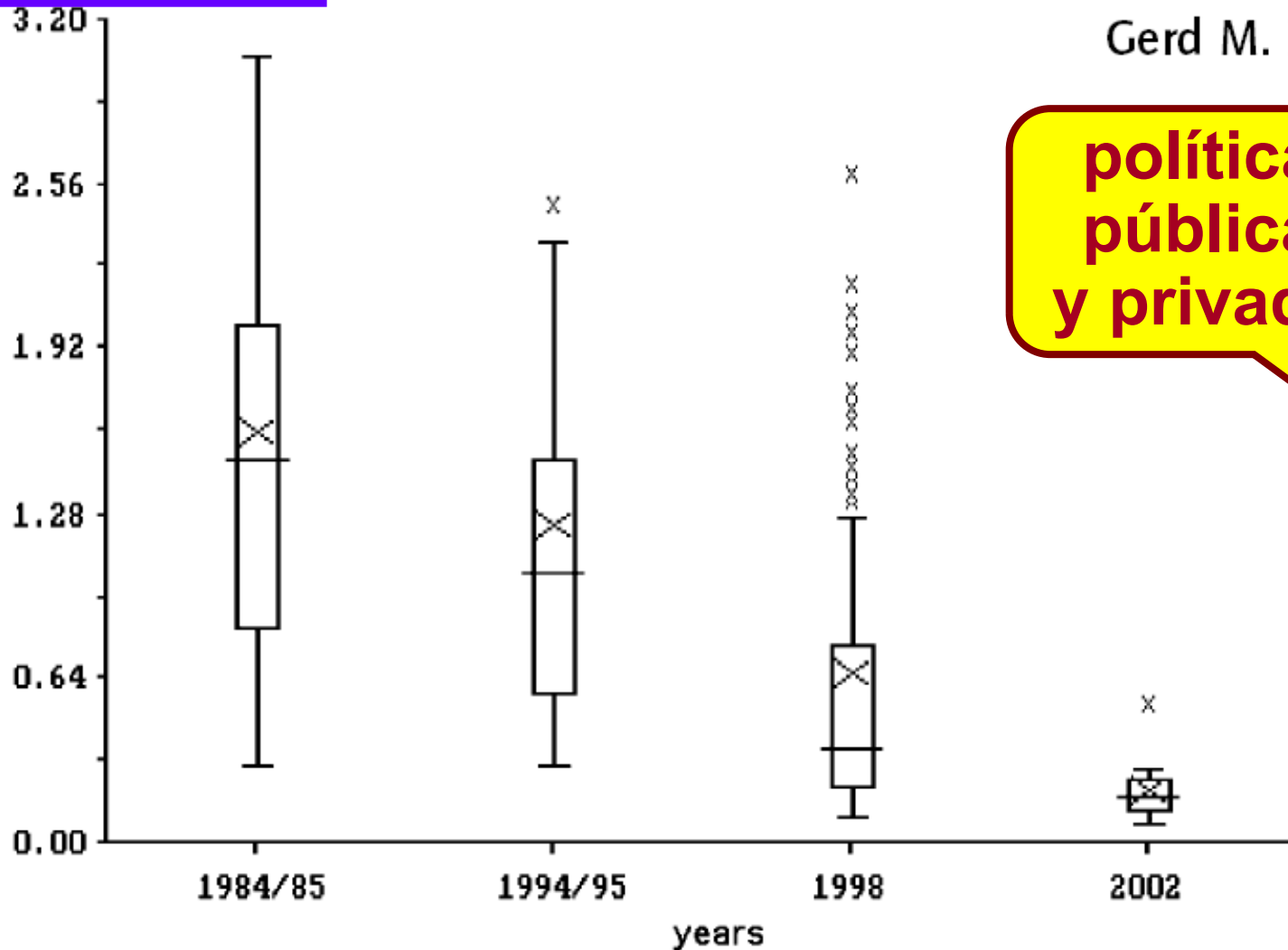


Figure 1. Box & Whisker-diagram of p,p' -DDE concentrations ($\mu\text{g/L}$) in full-term German neonates from the mid-1980s to 2002.

Determinants of organochlorine levels detectable in the amniotic fluid of women from Tenerife Island (Canary Islands, Spain)☆

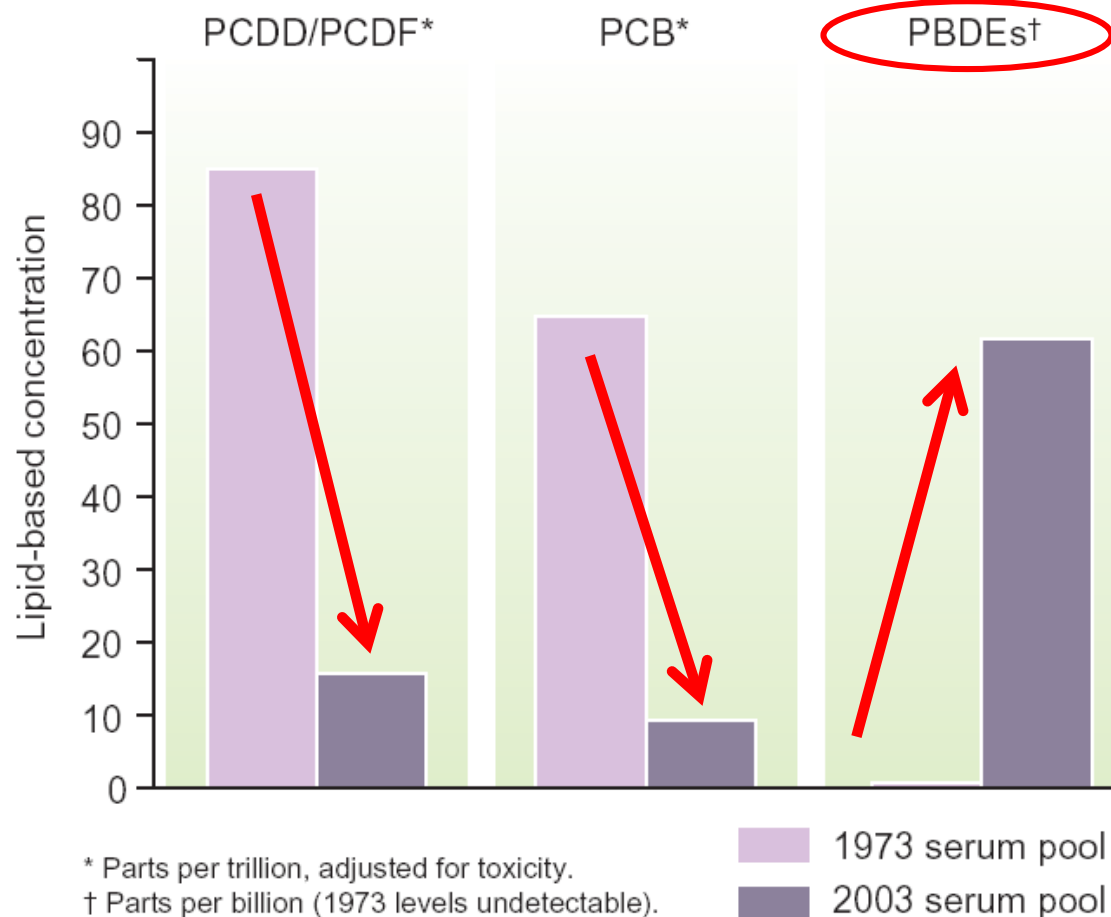
Octavio P. Luzardo^{a,d}, Vikesh Mahtani^b, Juan M. Troyano^b, Margarita Álvarez de la Rosa^b, Ana I. Padilla-Pérez^b, Manuel Zumbado^{a,d}, Maira Almeida^{a,d}, Guillermo Burillo-Putze^c, Carlos Boada^c, Luis D. Boada^{a,d,*}

Environmental Research 109 (2009)

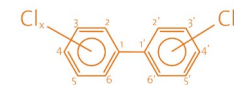
Organochlorines (OCs) tend to accumulate in human tissues and can be measured in amniotic fluid (AF). The detection of OCs in AF samples reflects intrauterine exposure of human beings to these persistent organic pollutants. The present study was performed to evaluate the level of contamination of AF by OCs in 100 pregnant women from Tenerife Island (Canary Islands, Spain). Gas chromatography/mass spectrometry (GC/MS) was used to identify and quantify the analytes, including 7 polychlorobiphenyl (PCB) congeners and 18 OC pesticides and metabolites. The majority of the AF samples (67%) showed some detectable OC-residue, hexachlorobenzene (HCB) being the most frequently detected compound (66% of the samples) and at the highest concentration (median 0.023 ng/ml). Lindane was also detected in 28% of the samples. Inverse associations were found between previous lactation and hexachlorocyclohexane isomers (HCH) and cyclodienes in the group of younger women ($p = 0.037$ and $p = 0.027$, respectively). Unexpectedly, serum values of HCB ($r = -0.414$; $p = 0.04$), γ -HCH ($r = -0.294$; $p = 0.035$), and \sum OCs ($r = -0.350$; $p = 0.014$) were negatively related to age. Even more, women with detectable levels of HCH isomers were younger (33.9 ± 4.9 years) than women with undetectable levels of them (36.1 ± 4.9 years; $p = 0.035$). We conclude that approximately one in two fetuses in the Canary Islands is exposed to OCs *in utero*, and that, therefore, the exposure of young women from these Islands to some HCH isomers persists nowadays. Because prenatal exposure to these chemicals may be a causative factor in adverse health trends, further studies are required to enhance preventive measures.

TOXICOLOGY

Exposure to Flame Retardants On the Rise



Burning issue Body burden of dioxins and furans and PCBs has declined since their use was banned; exposure to PBDEs has climbed steeply.



3.2.6 Avaluació del risc

A la taula 33 es presenta la ingesta diària de bifenils policlorats (PCB) per quilogram de pes corporal. La ingesta estimada per a un home adult és de 2,15 pg/kg de pes corporal/dia (OMS-TEQ), que representa un 53,5% del rang màxim de seguretat establert per la UE per a dioxines i PCB amb efecte dioxina. En el cas dels nens i nenes, la ingesta diària per quilo de pes corporal supera el rang de seguretat establert per l'OMS.

Taula 33. Ingesta diària de bifenils policlorats relativa al pes corporal

Grups de població	Ingesta diària de PCB (pg OMS-TEQ/kg/dia)
Homes	2,15
Dones	2,27
Nens i nenes	4,63
Adolescents	2,31
Persones més grans de 65 anys	1,95

CQEDTC 2000-2002

En el cas dels nens la ingesta diària per quilo de pes corporal supera el rang de seguretat establert per l'OMS.

Phthalate metabolites in 24-h urine samples of the German Environmental Specimen Bank (ESB) from 1988 to 2015 and comparison with US NHANES data from 1999 to 2012

Holger M. Koch^{a,*}, Maria Rüther^b, André Schütze^a, André Conrad^b,
Petra Apel^b, Thomas Brüning^a, Marike Kolossa-Gehring^b

The German Environmental Specimen Bank (ESB) continuously collects 24-h urine samples since the early 1980s in Germany. In this study we analyzed 300 urine samples from the years 2007 to 2015 for 21 phthalate metabolites (representing exposure to 11 parent phthalates) and combined the data with two previous retrospective measurement campaigns (1988 to 2003 and 2002 to 2008). The combined dataset comprised 1162 24-h urine samples spanning the years 1988 to 2015. With this detailed set of human biomonitoring data we describe the time course of phthalate exposure in Germany over a time frame of 27 years. For the metabolites of the endocrine disrupting phthalates di(2-ethylhexyl) phthalate (DEHP), di-*n*-butyl phthalate (DnBP) and butylbenzyl phthalate (BBzP) we observed a roughly ten-fold decline in median metabolite levels from their peak levels in the late 1980s/early 1990s compared to most recent levels from 2015. Probably, bans (first enacted in 1999) and classifications/labelings (enacted in 2001 and 2004) in the European Union lead to this drop. A decline in di-isobutyl phthalate (DiBP) metabolite levels set in only quite recently, possibly due to its later classification as a reproductive toxicant in the EU in 2009. In a considerable number of samples collected before 2002 health based guidance values (BE, HBM I) have been exceeded for DnBP (27.2%) and DEHP (2.3%) but also in recent samples some individual exceedances can still be observed (DEHP 1.0%). A decrease in concentration for all low molecular weight phthalates, labelled or not, was seen in the most recent years of sampling. For the high molecular weight phthalates, DEHP seems to have been substituted in part by di-isononyl phthalate (DiNP), but DiNP metabolite levels have also been declining in the last years. Probably, non-phthalate alternatives increasingly take over for the phthalates in Germany.

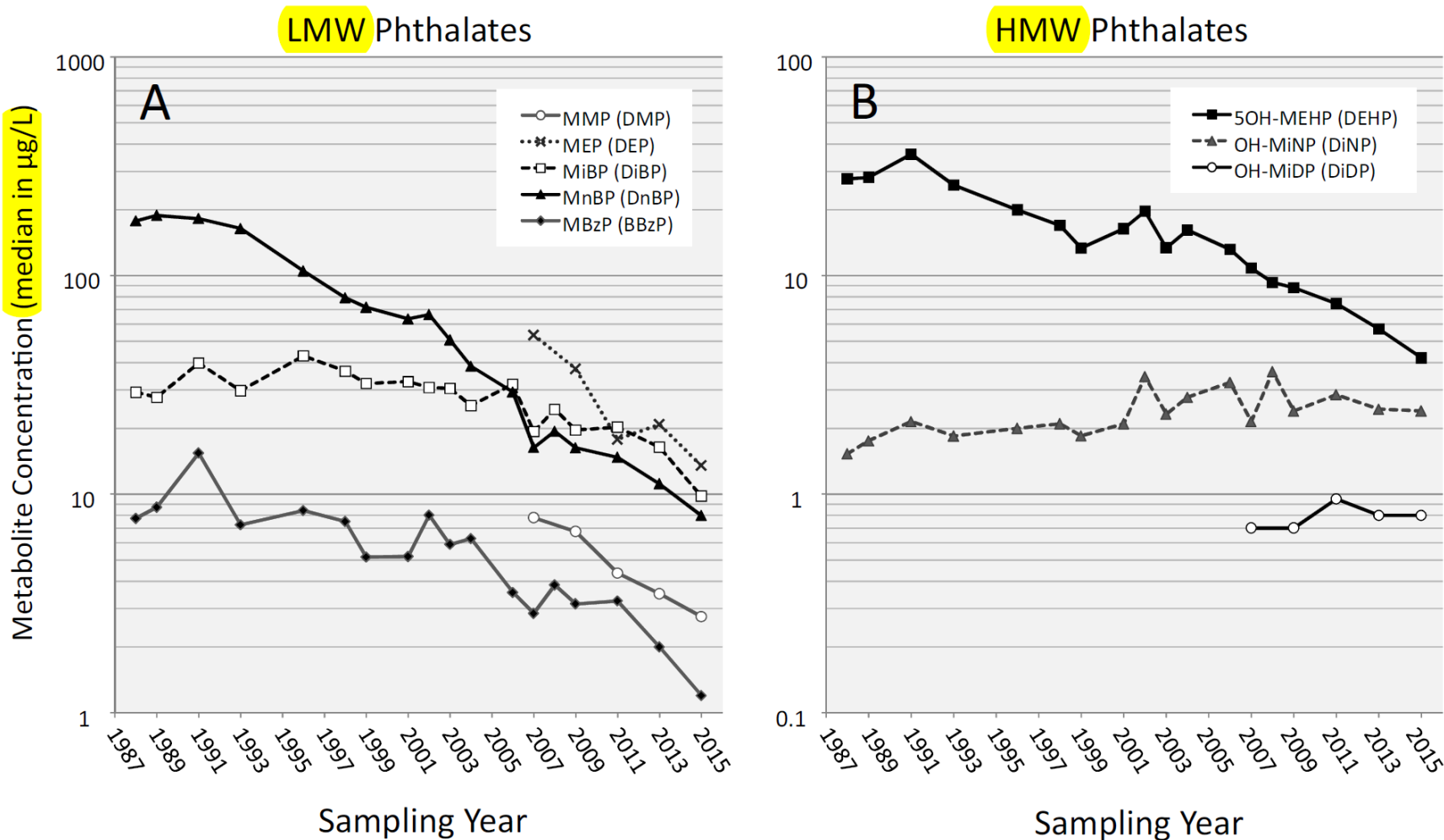
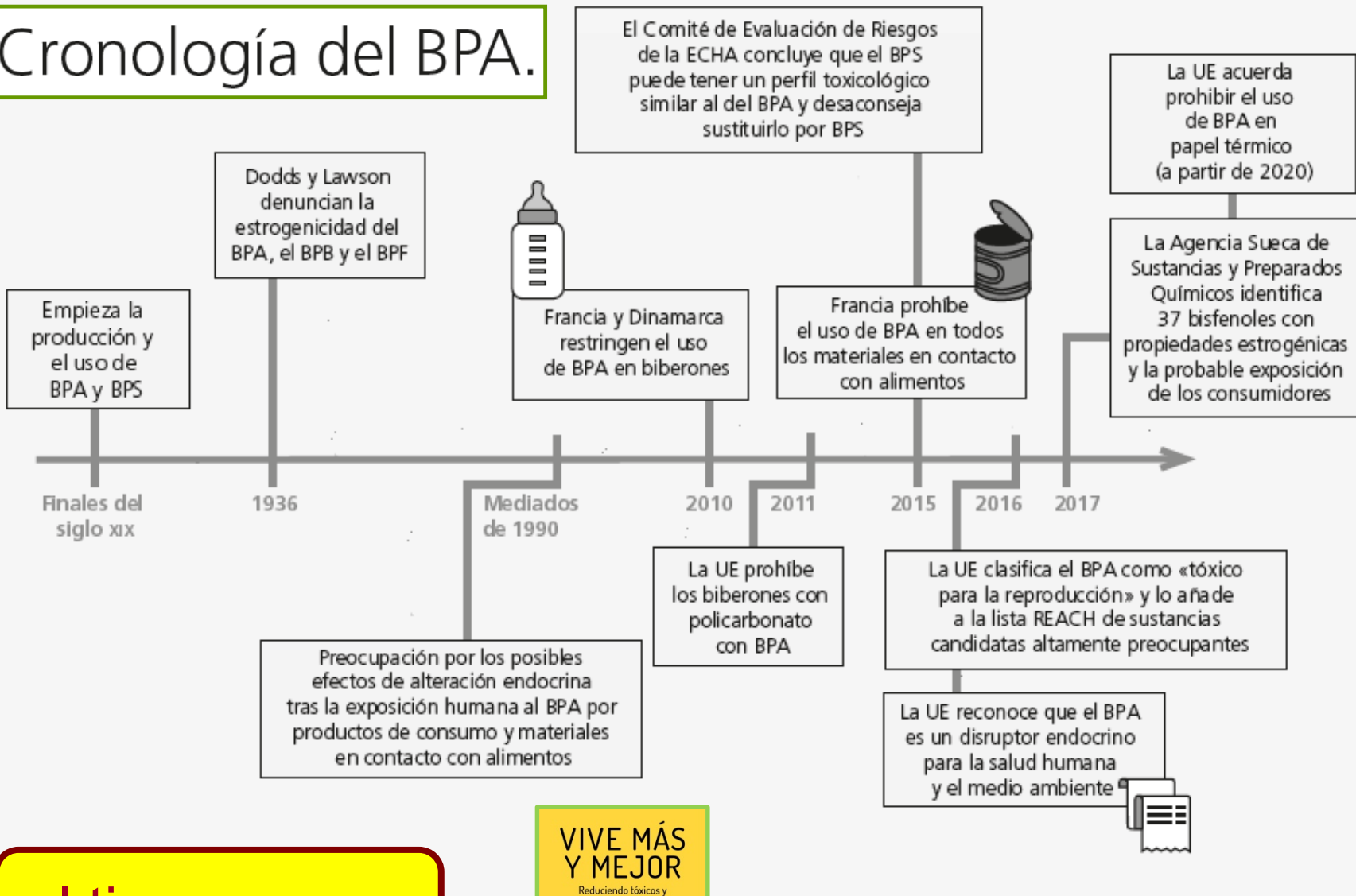
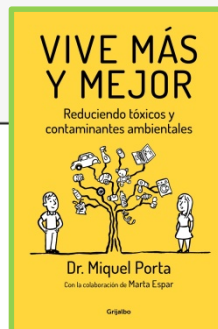


Fig. 1. Concentrations of key LMW and HMW phthalate metabolites (median, in $\mu\text{g/L}$) in the German ESB over the years 1988–2015.

Cronología del BPA.



el tiempo pasa



<http://www.chemtrust.org/food-contact/>

In-utero and childhood chemical exposome in six European mother-child cohorts

Methods: We relied on the Early-Life Exposome project, HELIX, a collaborative project across six established population-based birth cohort studies in Europe. In 1301 subjects, biomarkers of exposure to 45 contaminants (i.e. organochlorine compounds, polybrominated diphenyl ethers, *per*- and polyfluoroalkyl substances, toxic and essential elements, phthalate metabolites, environmental phenols, organophosphate pesticide metabolites and cotinine) were measured in biological samples from children (6–12 years) and their mothers during pregnancy, using highly sensitive biomonitoring methods.

Results: Most of the exposure biomarkers had high detection frequencies in mothers (35 out of 45 biomarkers with > 90% detected) and children (33 out of 45 biomarkers with > 90% detected). Concentrations were significantly different between cohorts for all compounds, and were generally higher in maternal compared to children samples. For most of the persistent compounds the correlations between maternal and child concentrations were moderate to high (Spearman $Rho > 0.35$), while for most non-persistent compounds correlations were considerably lower (Spearman $Rho < 0.15$). For mercury, PFOS and PFOA a considerable proportion of the samples of both mothers and their children exceeded the HBM I value established by The Human Biomonitoring Commission of the German Federal Environment Agency.

Discussion: Although not based on a representative sample, our study suggests that children across Europe are exposed to a wide range of environmental contaminants in fetal life and childhood including many with potential adverse effects. For values exceeding the HBM I value identification of specific sources of exposure and reducing exposure in an adequate way is recommended. Considerable variability in this “chemical exposome” was seen between cohorts, showing that place of residence is a strong determinant of one's personal exposome. This extensive dataset comprising > 100,000 concentrations of environmental contaminants in mother-child pairs forms a unique possibility for conducting epidemiological studies using an exposome approach.

Estudio DemoCophes (Env Health Persp 2015):

Españoles: se detectó BPA en el 95.8% de los niños de 5-12 años el 96.5% de las madres.

MEP (ftalato de monoetilo): 6 veces superior que la media Europea.

España: niveles de mercurio también superiores a la media europea.

Nicolás Olea | Director del Instituto de Investigación Biosanitaria ibs.Granada

«Todos los españoles orinamos plástico»

El catedrático de Radiología, que participó en la Escuela Salud Pública del Llazeret, denuncia la inacción de los poderes públicos para frenar las patologías derivadas de los químicos tóxicos del medio ambiente

ciando la inacción de los poderes públicos para frenar las patologías derivadas de los tóxicos del medio ambiente. Después de más de 30 años de investigación, el doctor Olea se pregunta por qué con los datos de los trabajos realizados por su grupo, las administraciones siguen más empeñadas en administrar fármacos que en evitar las enfermedades. Así lo expuso la semana pasada en la XXIX Escuela Salud Pública, en el Llazeret.

¿Hasta qué punto pone en duda el papel de la Administración en este problema?

—Desconfío completamente de la española, de la europea y de las multinacionales, porque cuando deciden de un año a otro que algo es malo es porque han visto otra oportunidad de negocio que, seguro, es igual de fraudulenta que la anterior.

Los envases de plástico para el agua, por ejemplo.

—El peor uso es el reuso, cuanto más viejo es el plástico más libera sus componentes. La molécula PET (politereftalato de etileno) es un derivado puro del petróleo, viene de la acetona y el fenol. El que hace la botella tiene un catálogo de 600 compuestos químicos para hacerla, el número de aditivos que lleva el plástico es inmenso y desconocido para el comprador.

¿Cómo llegan a esta conclusión?

—Hemos analizado 30 hoteles diferentes del mercado del sur de España, y vimos que el 83 por ciento del agua que llevan es hormonalmente activa, estrogénica y un 42 por ciento antiestrogénica. El lobby del PET nos dijo que era inerte, y es cierto, pero nos explicaron que los contaminantes serían algunos de los 600 posibles aditivos que le añade el fabricante de la botella. Les dimos que los identificarán y eliminarán los que causan problemas. Luego movilizaron a sus amigos, un buen laboratorio de Barcelona, para medir todos los contaminantes de plástico de una botella y asegurar y jurar que todo está dentro de la legalidad, y lo está, pero mal fundamentada porque no considera los efectos biológicos del conjunto de químicos.

Entonces...



Nicolás Olea, durante su intervención en la Escuela Salud Pública del Llazeret. Foto: DAVID ARQUIMBAU

—Dan a entender que todo está bajo control, la Administración satisfecha porque los productores cumplen con sus obligaciones pero el resultado no es el esperable.

¿Cuál ese es final?

—Elegimos una cohorte INMA (Infancia y Medio Ambiente), es decir, grupo de personas, 3.600 mujeres sanas con sus hijos, seguidas durante 17 años en Asturias, País Vasco, Menorca, Ribera del Ebro, Palencia y Granada. Se les han tomado muestras de orina anualmente, desde el embarazo. Vamos entonces que el cien por cien, es decir, todos los españoles orinamos plástico.

¿Por qué Menorca en el estudio?

—Aquí está uno de los mejores epidemiólogos de España, Matías Torment.

¿Y esa conclusión no ha motivado ninguna intervención?

—Dijeron que los valores eran normales, pero cómo va a ser normal orinar plástico? Se dedicaron a comparar los valores con los de los otros países y todo el mundo orina plástico. Cuando viene esta gente con tan buen corazón y denuncia que un pez sufre el plástico del mar, yo digo que es mucho más grave que todos estemos orinando plástico. Parece que intentan desviar la atención. Si hay una mancha de plástico en el pacífico, mala suerte, pero quítale el plástico a la gente en su ingesta diaria.

¿Por qué no actúan las adminis-

das con factores ambientales.

(C)

—La medicina más vendida en España con receta es la hormona tiroidea y es porque está muy relacionada con la exposición química porque muchos de esos compuestos interfieren en la glándula tiroidea y nadie hace nada. La hormona es muy barata y está en el mercado. Si la medicina se pone una solución parece que está todo resuelto, pero no es así, hay que ir a la raíz, ¿por qué cambian tanto los patrones de enfermedad? Hay una situación enorme de impunidad pero en la Unión Europea, que es donde se decide todo, no hacen nada.

A pesar de los datos de sus investigaciones...

—Dirijo un equipo de investigación de medicina medioambiental de la Universidad de Granada pero estamos integrados en varios grupos. Nos lo han financiado todo, incluso en la crisis tuvimos más proyectos de los que podíamos atender con dinero público. Supongo que esta gente se leerá nuestros trabajos porque las decisiones se toman en la Unión Europea. Ahora vamos a presentarnos en Viena con otro proyecto de 75 millones de euros para medir la exposición de cuantos tóxicos orinan varios grupos de edades. ¿Pero cuántas veces vamos a medirla, si ya lo sabemos?

¿Qué pautas debemos seguir para evitar contaminarnos?

—Esa es la clave. Primero el asociacionismo local que presione y exija medidas locales, luego el asociacionismo a gran escala participando en Europa. Y a nivel particular recomendaciones para disminuir la exposición como, por ejemplo, no comprar un coche nuevo si vais un tener un niño porque el plástico evaporado influye en la embarazada, no pintar la habitación ni comprar muebles nuevos para evitar plásticos y barnices sintéticos. Airear la casa quince minutos por la mañana y quince por la tarde y aspirar y no barrer porque los polímeros vienen de los circuitos eléctricos, tv, ordenadores, evitar el papel térmico de las cajas de los supermercados como se hace en Francia y aquí no se aplicará hasta el 2020, aunque parezca increíble.

Las consecuencias

—Pues las cargas de enfermedad.

¿Aunque la esperanza de vida esté cada vez más alta?

—Uno de cada tres menorquines, por ejemplo, tendrá cáncer si llega a los setenta años, y una de cada cuatro mujeres. Si le añades el cán-



«Somos unos sumideros que nos metemos al día de todo, desde la cosmética hasta la cena»

«Nos siguen envenenando de forma agravada porque no se pone remedio»

traciones?

—No hay, dicen, demostración de la causalidad de la enfermedad ante esta exposición. Son exposiciones tóxicas y hormonalmente activas que provocan, por ejemplo, esterilidad. En los centros de donación de semen hace diez años rechazaban a un voluntario de cada diez y ahora se dan con un carrito en los disetes si sirve uno de cada diez. El gran problema de la fecundidad de las parejas es que la solución, en lugar de ser buscar la causa, es la creación de centros de fecundación invitro. La exposición maternofantil al plástico y a los componentes hormonales de los contaminantes en los cuarenta días posteriores a la fecundación condicionan la calidad seminal de ese hombre a los 34 años.

A tan largo plazo la demostración causa-efecto es muy difícil.

—Claro. Son exposiciones crónicas en momentos críticos del desarrollo con efectos a largo plazo: fertilidad, cáncer de mama, hormodependiente, de preñata, desarrollo neuroconductor... Todo está demostrado epidemiológicamente porque no se permite investigar con humanos.

Usted decía en 2014 que nos estaban envenenando impunemente. ¿Lo siguen haciendo, entonces?

—Sí, y de forma agravada porque no se pone remedio. En el siglo XXI sigue dándose la cifra mágica para la toxicidad individual de ese compuesto cuando un tomate de Almería, por ejemplo, ya tiene siete compuestos. Las cifras no deberían ser individuales para cada pesticida sino para el conjunto de los siete que puede tener para saber si juntos superan los valores normales, pero no se atreven a hacerlo. Somos unos sumideros que nos metemos al día de todo, desde la cosmética de la mañana, hasta la cena y el resultado es una exposición global indeseable porque todo aparece en la orina, no es lo normal.

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The New York Times

By Roni Caryn Rabin

July 12, 2017



Potentially harmful chemicals that were banned from children's teething rings and rubber duck toys a decade ago may still be present in high concentrations in your child's favorite meal: macaroni and cheese mixes made with powdered cheese.

The chemicals, called phthalates, can disrupt male hormones like testosterone and have been linked to genital birth defects in infant boys and learning and behavior problems in older children. The chemicals migrate into food from packaging and equipment used in manufacturing and may pose special risks to pregnant women and young children.

Recent **Fast Food Consumption** and **Bisphenol A and Phthalates** Exposures among the U.S. Population in NHANES, 2003–2010

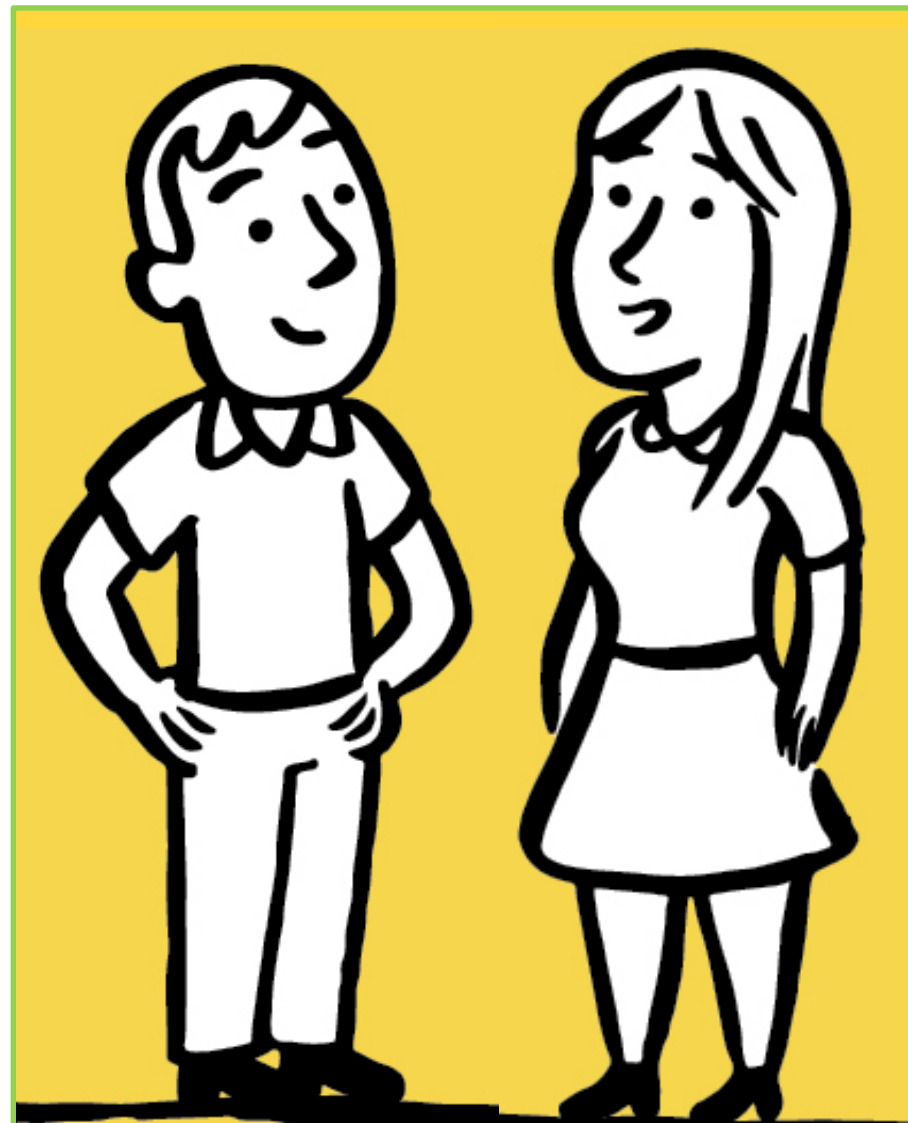
Ami R. Zota, Cassandra A. Phillips, and Susanna D. Mitro

Environmental Health Perspectives • October 2016

METHODS: We combined data on 8,877 participants from the National Health and Nutrition Examination Survey (NHANES 2003–2010). Using 24-hr dietary recall data, we quantified: *a*) fast food intake [percent of total energy intake (TEI) from fast food]; *b*) fast food-derived fat intake (percent of TEI from fat in fast food); and *c*) fast food intake by food group (dairy, eggs, grains, meat, and other). We examined associations between dietary exposures and urinary chemical concentrations using multivariate linear regression.

RESULTS: We observed evidence of a positive, dose–response relationship between fast food intake and exposure to phthalates (p -trend < 0.0001) but not BPA; participants with high consumption ($\geq 34.9\%$ TEI from fast food) had 23.8% (95% CI: 11.9%, 36.9%) and 39.0% (95% CI: 21.9%, 58.5%) higher levels of Σ DEHPm and DiNPm, respectively, than nonconsumers. Fast food-derived fat intake was also positively associated with Σ DEHPm and DiNPm (p -trend < 0.0001). After adjusting for other food groups, Σ DEHPm was associated with grain and other intake, and DiNPm was associated with meat and grain intake.

CONCLUSION: Fast food may be a source of exposure to DEHP and DiNP. These results, if confirmed, could inform individual and regulatory exposure reduction strategies.



sustancias cuya toxicidad es objeto de preocupación razonada en las sociedades más avanzadas.



Los tóxicos y tú: tanto que hacer

De modo que cada día vivimos hechos íntimamente conectados con esta idea elemental: podemos estar razonablemente contentos (o no) con la calidad de vida que tenemos –y disfrutar o no de muchas cosas maravillosas de la vida– y a la vez reconocer que debemos y podemos mejorar otras muchas cosas.

Reconocer las ventajas que nos aporta la química sintética y, a la vez, que muchos de sus usos nos cobran una factura onerosa: enfermedades y muertes prematuras con unos elevados costes humanos y económicos, por ejemplo.

Hacer nuestra esa realidad tóxica y reaccionar, cada uno a su manera, es perfectamente compatible con seguir disfrutando sin miedo de la vida. Parte de ella.

<https://bit.ly/2Nc8DFK>

Convivimos con problemas 'vintage' y problemas 'postmodernos'

- Algunos solo piensan en el daño que a **corto plazo** pueden hacer los **agentes infecciosos**, lo cual es importante, pero insuficiente.
- Actúan así, en parte, porque las leyes y los ciudadanos no les pedimos más, y porque su formación ha omitido los **problemas infecciosos y no infecciosos a largo plazo**.

– Debemos y podemos actuar mejor para disminuir el sufrimiento humano y los costes económicos que causan los **agentes infecciosos y no infecciosos a corto y largo plazo**.

- Debemos actuar no sólo para minimizar nuestra exposición a **tóxicos químicos**, sino también a **microbios resistentes a los antibióticos**, **hormonas**, **antibióticos** y otras sustancias dañinas a **medio y largo plazo** para la **salud humana**, la **salud animal** y el **medio ambiente**.
- **Los ciudadanos debemos valorar y apoyar más** a las administraciones, empresas y organizaciones que mejor hacen estas cosas.

Ver lo que nos sale a cuenta

Los beneficios socioeconómicos de la salud pública son reales, generales, a largo plazo, pero difíciles de cuantificar y, a menudo, invisibles. Hay que dar mayor valor a estas inversiones



EVA VÁZQUEZ

Casi nadie ve a la salud pública como un sector de inversión y de creación de riqueza

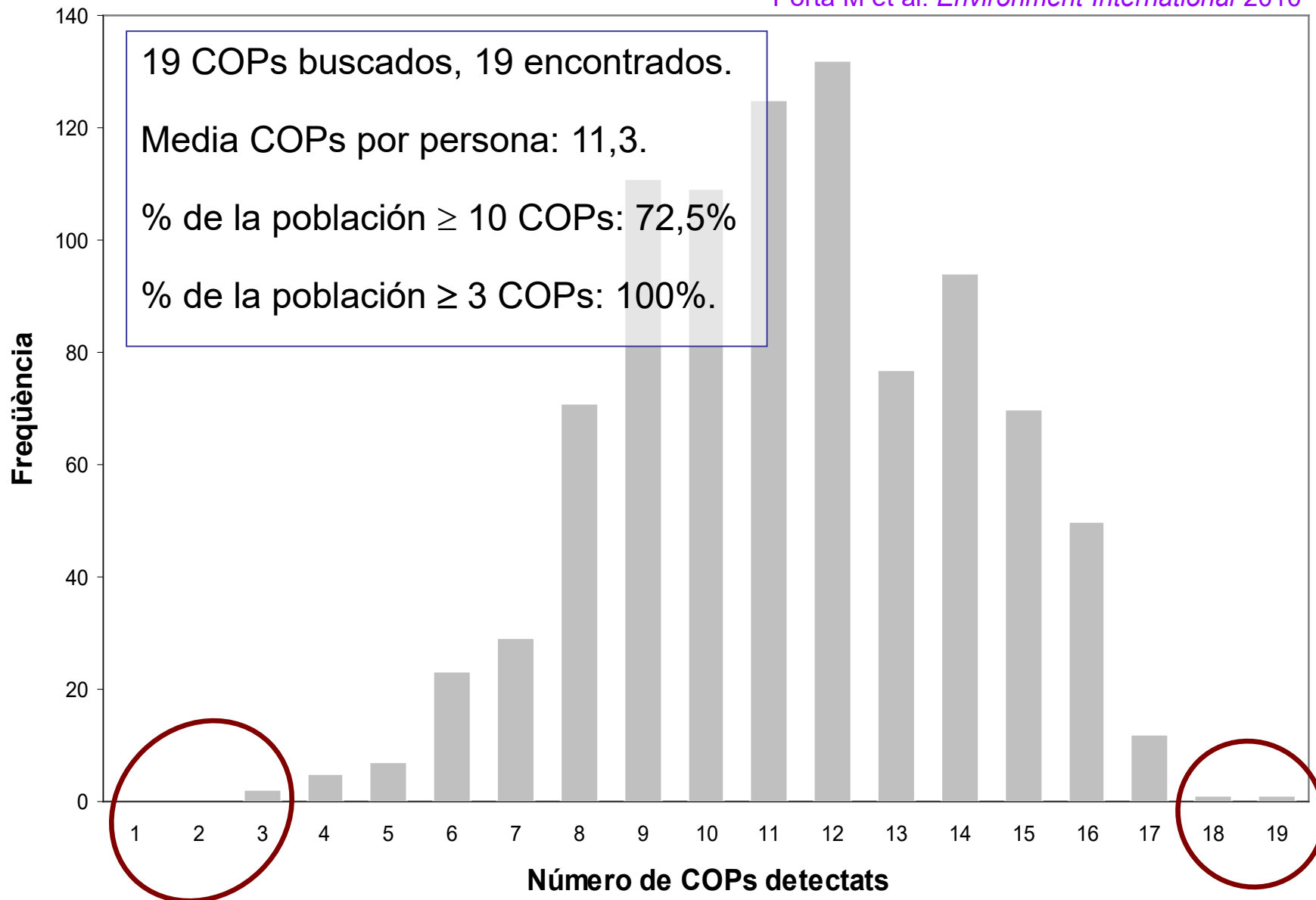
¿Por qué no crear negocios que actúen sobre las causas de enfermar y rindan beneficios?

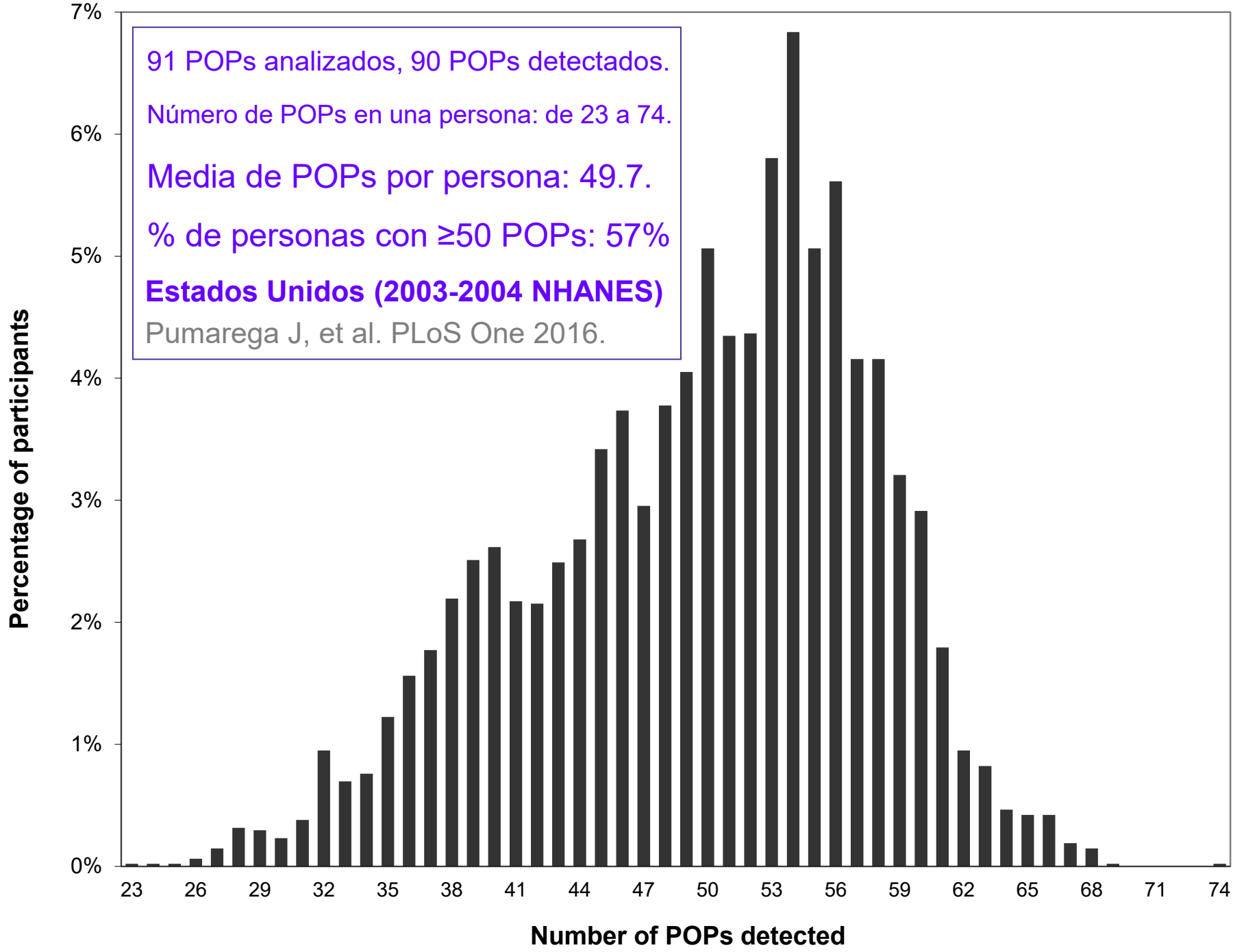
Conocimiento científico imperfecto

- Una parte de las muertes, trastornos e incapacidades que sufrimos por trastornos y enfermedades crónicas o degenerativas como el cáncer, infertilidad, diabetes o Alzheimer se debe a las mezclas de contaminantes químicos artificiales que tenemos en el cuerpo durante años.
- Los tenemos por 2 motivos:
 - 1) porque estamos expuestos a ellos cotidianamente o
 - 2) porque nuestro cuerpo no los excreta o elimina y se nos acumulan.
- Desde el vientre materno y durante la vida, tales contaminantes contribuyen a que acumulemos múltiples alteraciones genéticas y epigenéticas (lesionan nuestro ADN o hacen que los genes funcionen mal) o son dañinos para el sistema nervioso o actúan como “hormonas artificiales” o por otros mecanismos.
- La principal vía de entrada en nuestro cuerpo de tales contaminantes químicos artificiales son los alimentos y sus envases, el aire que respiramos, el agua que bebemos y otros numerosos bienes de consumo.

Número de COP detectados en una muestra representativa de la población general de Cataluña (número de personas: 919)

Porta M et al. *Environment International* 2010







How can we decrease exposure to phthalates?

Food & Beverage

- Common source of exposure to phthalates from processing and packaging materials that come into contact with foods and beverages.
- We can make food and beverage choices to reduce exposure.
 - ❖ Reduce use of processed and packaged foods
 - ❖ Increase use of fresh foods
 - ❖ Reduce storing and heating foods and beverages in plastic containers

Perfumes & Personal Care Products

- Phthalates may be found in some lotions, soaps, makeup, nail polish.
- Products with “fragrance” listed may contain phthalates.
 - ❖ Use “phthalate-free” lotions and soaps
 - ❖ Reduce use of products with “fragrance” by opting for “fragrance-free” choices
 - ❖ Use nail polish brands that advertise “No Di-Butyl Phthalate” or “No DBP”

Household Goods

- Flooring, blinds, shower curtains, electronics, and other PVC products can be a source of DEHP.
- Scented cleaning products, laundry detergent, synthetic air fresheners can contain phthalates.
 - ❖ Use PVC-free products: replace with cotton, bamboo or polyethylene vinyl acetate (PEVA)
 - ❖ Use “fragrance-free” cleaning and laundry products



Exposure to phthalates may raise risk of pregnancy loss, gestational diabetes

DEHP is a plasticizer—it helps make polyvinyl chloride (PVC) plastics flexible and durable. It's found in items such as medical equipment, building materials, shower curtains, plastic window blinds, and headphone cords. PVC plastics with DEHP are also used in manufacturing, such as in tubing or conveyer belts in food processing plants. Because DEHP is only loosely chemically bonded in plastic products, it can easily leach out, and people can be exposed to DEHP through air, water, food, intravenous fluids, or skin contact with DEHP-containing plastics.

One study followed a group of 256 women at Massachusetts General Hospital Fertility Center from 2004 to 2014 who were undergoing medically assisted reproduction, such as in-vitro fertilization. The researchers measured concentrations of 11 phthalate metabolites in the women's urine around the time of conception. Women with the highest concentrations of a type of phthalate called di-(2-ethylhexyl) phthalate, or DEHP, were 60% more likely to lose a pregnancy prior to 20 weeks than those with the lowest concentrations, the study found.



	Phthalates	Triclosan	BHA	Formaldehyde	Parabens
On Label & Hazard Score* ①-②: Low ③-⑥ Moderate ⑦-⑩: High *ewg.org/skindeep	(pronounced thal-ates) ⑩ DBP or Dibutyl phthalate ⑩ DEHP or Di-2-ethylhexyl phthalate ⑦ DEP or Diethyl Phthalate ⑧ Fragrance "Fragrance" is proprietary. A fragrance may contain 3,000 + chemicals, including phthalates.	⑦ Triclosan	⑩ BHA or Butylated hydroxyanisole	⑩ Formaldehyde ⑩ Formalin <i>Occurs as an impurity in:</i> ⑧-⑩ Bronopol (2-Bromo-2-Nitropropane-1,3-Diol) ⑦-⑧ DMDM hydantoin ⑥ Quaternium 15 ⑤ Diazolidinyl urea ⑤ Imidazolidinyl urea	⑤ butylparaben* ⑤ methylparaben ⑤ propylparaben* ⑤ polyparaben ④ ethylparaben ③ isobutylparaben
Found in	Nail products, deodorants, fragrances, hair spray, soap, shampoo, lotion, makeup	Liquid soap, bar soap, deodorant, face wash, toothpaste, mouthwash, acne treatments	Makeup, moisturizers, and food products	Eye cosmetics, nail care, shower gel, shampoo, conditioner, liquid soap, bubble bath, baby wipes	Thousands of products, including lotion/moisturizers, makeup, anti-aging products
Purpose	Help cosmetics penetrate skin. Prevent chipping in nail polish. Fixatives in fragrances. Emulsifiers	Anti-bacterial, Registered pesticide	Preservative	Preservative, Biocide, Nail Hardener	Preservative
Health Effects	Hormone Disruptors DBP: Cancer and Birth Defects (Testicular atrophy, structural defects of penis, reduced sperm count) in laboratory animals. DEHP: Cancer: "Probable Human Carcinogen" (U.S. EPA), "Reasonably Anticipated Human Carcinogen" (U.S. National Toxicology Program's (NTP) 12 th Report on Carcinogens (ROC)) DBP & DEHP: Banned in Europe (classified as mutagens causing genetic damage)	Hormone Disruptor: Disrupts thyroid, estrogen, androgen hormones (EPA). Cancer: Reacts with chlorine in tap water to create chloroform ("Probable Human Carcinogen" (U.S. EPA)). Allergies in children, Anti-bacterial resistance: Overuse is linked to these. Restricted in Canada & Japan	Hormone Disruptor: Strong evidence as endocrine disruptor. (European Commission on Endocrine Disruption). Cancer: "Reasonably Anticipated Human Carcinogen" (U.S. NTP's 12 th ROC). Banned in Europe	Cancer: "Known Human Carcinogen" (U.S. NTP's 12 th ROC). Irritant: to the eyes, nose, throat, and skin. Banned in Japan & Sweden	Hormone Disruptor, Cancer: Parabens act like estrogen—thus raise risks for certain cancers, impaired fertility, and altered development of a fetus or young child. Found in cancerous breast tumors. *Banned in Denmark in cosmetic products for children under 3 years
Alternatives					
Look for Instead	DBP-Free nail polish. Cosmetics, perfume, or cologne scented with plant-derived essential oils.	Proper hygiene. Use castile or mild soap & warm water. Rub hands for 30 seconds. Rinse.	Preservatives that cause the least irritation and fewest allergic reactions according to The Safe Shopper's Bible by David Steinman and Samuel S. Epstein: Grapefruit seed extract Sorbic acid Potassium sorbate	Vitamin A (retinyl) Vitamin C (ascorbic acid) Tocopherol (vitamin E)	

Canned Soup Consumption and Urinary Bisphenol A: A Randomized Crossover Trial

Results. Of 84 volunteers, 75 (89%) completed the study. Median age was 27 years and 51 (68%) were female; median treatment adherence was 100% (TABLE). Bisphenol A was detected in 77% (n=58) of samples after fresh soup consumption and 100% (n=75) of samples after canned soup consumption. The SG-adjusted geometric mean concentration of BPA was 1.1 $\mu\text{g/L}$ (95% CI, 0.9-1.4 $\mu\text{g/L}$) after fresh soup consumption (unadjusted: 0.9 $\mu\text{g/L}$; 95% CI, 0.7-1.2 $\mu\text{g/L}$) and 20.8 $\mu\text{g/L}$ (95% CI, 17.9-24.1 $\mu\text{g/L}$) after canned soup consumption (unadjusted: 17.5 $\mu\text{g/L}$; 95% CI, 14.1-21.8 $\mu\text{g/L}$). Stratification by treatment sequence revealed similar values (FIGURE). Following canned soup consumption, SG-adjusted urinary BPA concentrations were, on average, 22.5 $\mu\text{g/L}$ higher (95% CI, 19.6-25.5 $\mu\text{g/L}$) than those measured after a week of fresh soup consumption ($P < .001$), representing a 1221% increase.

lata
↓
comida
↓
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orina

hay
migración

Chemical contaminants in food



- | | |
|----------|------------------------|
| NATURAL | MAN MADE |
| • Plants | • Residues |
| • Fungi | • Unwanted by-products |
| | • Pollutants |

Metals

empleados para tratar a los animales durante la cría.

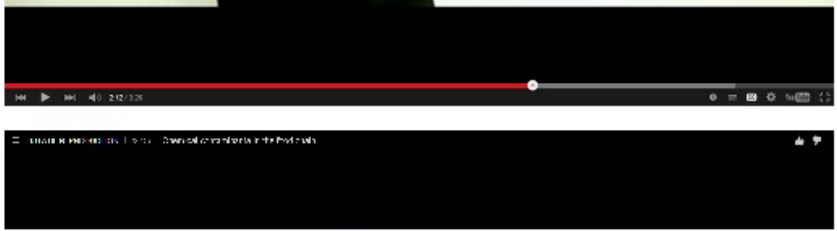
Chemical contaminants in food



- | | |
|----------|------------------------|
| NATURAL | MAN MADE |
| • Plants | • Residues |
| • Fungi | • Unwanted by-products |
| | • Pollutants |

Metals

Los residuos de los materiales en contacto con los alimentos



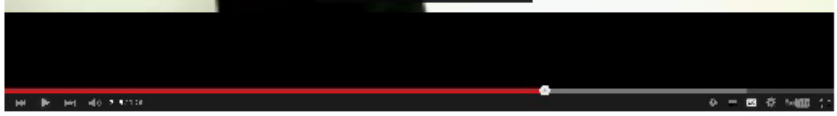
Chemical contaminants in food



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|----------|------------------------|
| NATURAL | MAN MADE |
| • Plants | • Residues |
| • Fungi | • Unwanted by-products |
| | • Pollutants |

Metals

también pueden pasar a nuestros alimentos.



Los efectos de los metales pesados en la evaluación de riesgos químicos

¿Qué son los plaguicidas y cómo llegan a nuestros alimentos?

Los plaguicidas son un grupo muy amplio de sustancias que se utilizan para controlar las plagas de plantas y animales. Sin embargo, su uso puede ser perjudicial para la salud humana y el medio ambiente. Es importante conocer cómo se regulan y cómo se pueden reducir los riesgos de exposición a estos productos.

[LAURA TARDÓN](#) > Madrid

SALUD PÚBLICA Informe anual

Actualizado: 12/03/2015 16:10 horas

Casi la mitad de los alimentos en Europa contienen restos de pesticidas

□ De este porcentaje, el 2,6% supera los niveles legales establecidos



Manzanas, uno de los alimentos examinados por la Agencia Europea de Seguridad Alimentaria (EFSA). | EL MUNDO



ecologistas
en acción



Directo
a tus hormonas
Guía de alimentos
disruptores

**Residuos de plaguicidas con capacidad
de alterar el sistema endocrino
en los alimentos españoles**

Fluorinated substances in paper packaging passed on to the cake table

18. mar 2016

Af Niels Søndergaard | Translation: Pia Saxild

A new test from the Danish Consumer Council THINK Chemicals establishes the presence of problematic fluorinated substances in paper packaging for cakes such as frangipane tarts, raspberry Swiss rolls, carrot cakes and muffins. Manufacturers declare that they will phase out the substances.



Food packaging and migration of food contact materials: will epidemiologists rise to the neotoxic challenge?

Jane Muncke,¹ John Peterson Myers,
Miquel Porta⁵

Lifelong, low-dose exposure to FCMs is of concern for several reasons. First, acknowledged toxicants are legally used in FCMs in Europe, the USA and other regions (notably, China). In the USA, several types of asbestos are authorised as indirect food additives for use in rubber.¹⁹ Formaldehyde, another known carcinogen, is widely present at low levels in plastic bottles made of polyethylene terephthalate²⁰; formaldehyde also migrates from melamine formaldehyde tableware.²¹ Considering how widely beverages are consumed from polyethylene terephthalate soda bottles, this may amount to a significant, yet unrecognized, exposure of the population.

Food safety

Chemicals leaching into food from packaging raise safety concerns

Sarah Boseley, health editor

Wednesday 19 February 2014
00.01 GMT



Shares 12,518
Comments 442

Save for later

Food & drink

Concerns over chemicals in food packaging misplaced, say scientists

Journal article's warning that packaging is significant source of chemical food contamination is roundly rejected



Sarah Boseley, health editor

Wednesday 19 February 2014 19.23 GMT



Shares 300

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Bisfenol A hasta en la sopa



Plato de sopa. | El Mundo

More Chemicals Show Epigenetic Effects across Generations

Epigenetic changes occur when the function of a gene is altered by various mechanisms although its DNA sequence remains stable. **Transgenerational effects** result from a mother's exposure and are inherited through successive generations in the absence of direct exposure of the offspring.² **Such environmentally induced effects have been demonstrated in people, rodents, birds, fish, insects, worms, plants, and microbes,** in some cases lasting dozens of generations.³

Transgenerational Actions of Environmental Compounds on Reproductive Disease and Identification of Epigenetic Biomarkers of Ancestral Exposures

Mohan Manikkam¹, Carlos Guerrero-Bosagna¹, Rebecca Tracey, Md. M. Haque, Michael K. Skinner*

Center for Reproductive Biology, School of Biological Sciences, Washington State University, Pullman, Washington, United States of America

Environmental factors during fetal development can induce a permanent epigenetic change in the germ line (sperm) that then transmits epigenetic transgenerational inheritance of adult-onset disease in the absence of any subsequent exposure. The epigenetic transgenerational actions of various environmental compounds and relevant mixtures were investigated with the use of a pesticide mixture (permethrin and insect repellent DEET), a plastic mixture (bisphenol A and phthalates), dioxin (TCDD) and a hydrocarbon mixture (jet fuel, JP8). After transient exposure of F0 gestating female rats during the period of embryonic gonadal sex determination, the subsequent F1–F3 generations were obtained in the absence of any environmental exposure. The effects on the F1, F2 and F3 generations pubertal onset and gonadal function were assessed. The plastics, dioxin and jet fuel were found to promote early-onset female puberty transgenerationally (F3 generation). Spermatogenic cell apoptosis was affected transgenerationally. Ovarian primordial follicle pool size was significantly decreased with all treatments transgenerationally. Differential DNA methylation of the F3 generation sperm promoter epigenome was examined. Differential DNA methylation regions (DMR) were identified in the sperm of all exposure lineage males and found to be consistent within a specific exposure lineage, but different between the exposures. Several genomic features of the DMR, such as low density CpG content, were identified. Exposure-specific epigenetic biomarkers were identified that may allow for the assessment of ancestral environmental exposures associated with adult onset disease.



Cornwall W. *Science* 2017

RULES OF EVIDENCE

Are evidence standards used by chemical regulators excluding solid science?

Ichemical safety. He noticed that the synthetic chemical bisphenol A (BPA) weakly mimics the human hormone estrogen. In the decades that followed, BPA became a ubiquitous ingredient in epoxy resins and polycarbonate plastic, used by the millions of tons every year for everything from dental sealants to plastic water bottles. But BPA doesn't stay put. In the 1990s, Stanford University researchers realized that tiny amounts can

with the endocrine system—the symphony of hormones in the human body—wondered whether those traces of BPA were doing any harm.

By now tests have found the chemical in more than 90% of Americans. But the risks of BPA contamination are still in dispute. One reason: Studies have produced conflicting or inconclusive results, in part because alterations in the endocrine system can be subtle and hard to pin down. Another is a

of chemicals.

In 2014, the U.S. Food and Drug Administration (FDA) vetted 161 new studies about the potential health effects of BPA. The goal was to see whether science could make a definitive judgment about the compound's safety and light a clear path for regulators. The compilation of evidence included reams of papers published in peer-reviewed journals, many of which found evidence suggesting tiny amounts of BPA could tin-



**Baby bottles are now free of BPA,
but it is still present in other food containers and packaging.**



Cornwall W. *Science* 2017



Chlorinated Persistent Organic Pollutants, Obesity, and Type 2 Diabetes

Duk-Hee Lee, Miquel Porta, David R. Jacobs Jr., and Laura N. Vandenberg

Persistent organic pollutants (POPs) are lipophilic compounds that travel with lipids and accumulate mainly in adipose tissue. Recent human evidence links low-dose POPs to an increased risk of type 2 diabetes (T2D). Because humans are contaminated by POP mixtures and POPs possibly have nonmonotonic dose-response relations with T2D, critical methodological issues arise in evaluating human findings. This review summarizes epidemiological results on chlorinated POPs and T2D, and relevant experimental evidence. It also discusses how features of POPs can affect inferences in humans. The evidence as a whole suggests that, rather than a few individual POPs, background exposure to POP mixtures—including organochlorine pesticides and polychlorinated biphenyls—can increase T2D risk in humans. Inconsistent statistical significance for individual POPs may arise due to distributional differences in POP mixtures among populations. Differences in the observed shape of the dose-response curves among human studies may reflect an inverted U-shaped association secondary to mitochondrial dysfunction or endocrine disruption. Finally, we examine the relationship between POPs and obesity. There is evidence in animal studies that low-dose POP mixtures are obesogenic. However, relationships between POPs and obesity in humans have been inconsistent. Adipose tissue plays a dual role of promoting T2D and providing a relatively safe place to store POPs. Large prospective studies with serial measurements of a broad range of POPs, adiposity, and clinically relevant biomarkers are needed to disentangle the interrelationships among POPs, obesity, and the development of T2D. Also needed are laboratory experiments that more closely mimic real-world POP doses, mixtures, and exposure duration in humans. (*Endocrine Reviews* 35: 557–601, 2014)

Published online: 06 February 2019

Evidence indicates that obesity can be promoted by chemical 'obesogens' that drive adiposity, hunger, inflammation and suppress metabolism. Dioctyl sodium sulfosuccinate (DOSS), a lipid emulsifier and candidate obesogen *in vitro*, is widely used in processed foods, cosmetics and as stool softener medicines commonly used during pregnancy. *In vivo* testing of DOSS was performed in a developmental origins of adult obesity model. Pregnant mice were orally administered vehicle control or DOSS at times and doses comparable to stool softener use during human pregnancy. All weaned offspring consumed only standard diet. Adult male but not female offspring of DOSS-treated dams showed significantly increased body mass, overall and visceral fat masses, and decreased bone area. They exhibited significant decreases in plasma adiponectin and increases in leptin, glucose intolerance and hyperinsulinemia. Inflammatory IL-6 was elevated, as was adipose Cox2 and Nox4 gene expressions, which may be associated with promoter DNA methylation changes. Multiple significant phospholipid/sterol lipid increases paralleled profiles from long-term high-fat diet induced obesity in males. Collectively, developmental DOSS exposure leads to increased adult adiposity, inflammation, metabolic disorder and dyslipidemia in offspring fed a standard diet, suggesting that pharmaceutical and other sources of DOSS taken during human pregnancy might contribute to long-term obesity-related health concerns in offspring.

Increased adiposity, inflammation, metabolic disruption and dyslipidemia in adult male offspring of DOSS treated C57BL/6 dams

Alexis M. Temkin¹, Robert R. Bowers², Candice Z. Ulmer³, Kayla Penta⁴, John A. Bowden³, Jennifer Nyland⁵, John E. Baatz⁶ & Demetri D. Spyropoulos^{1,2}

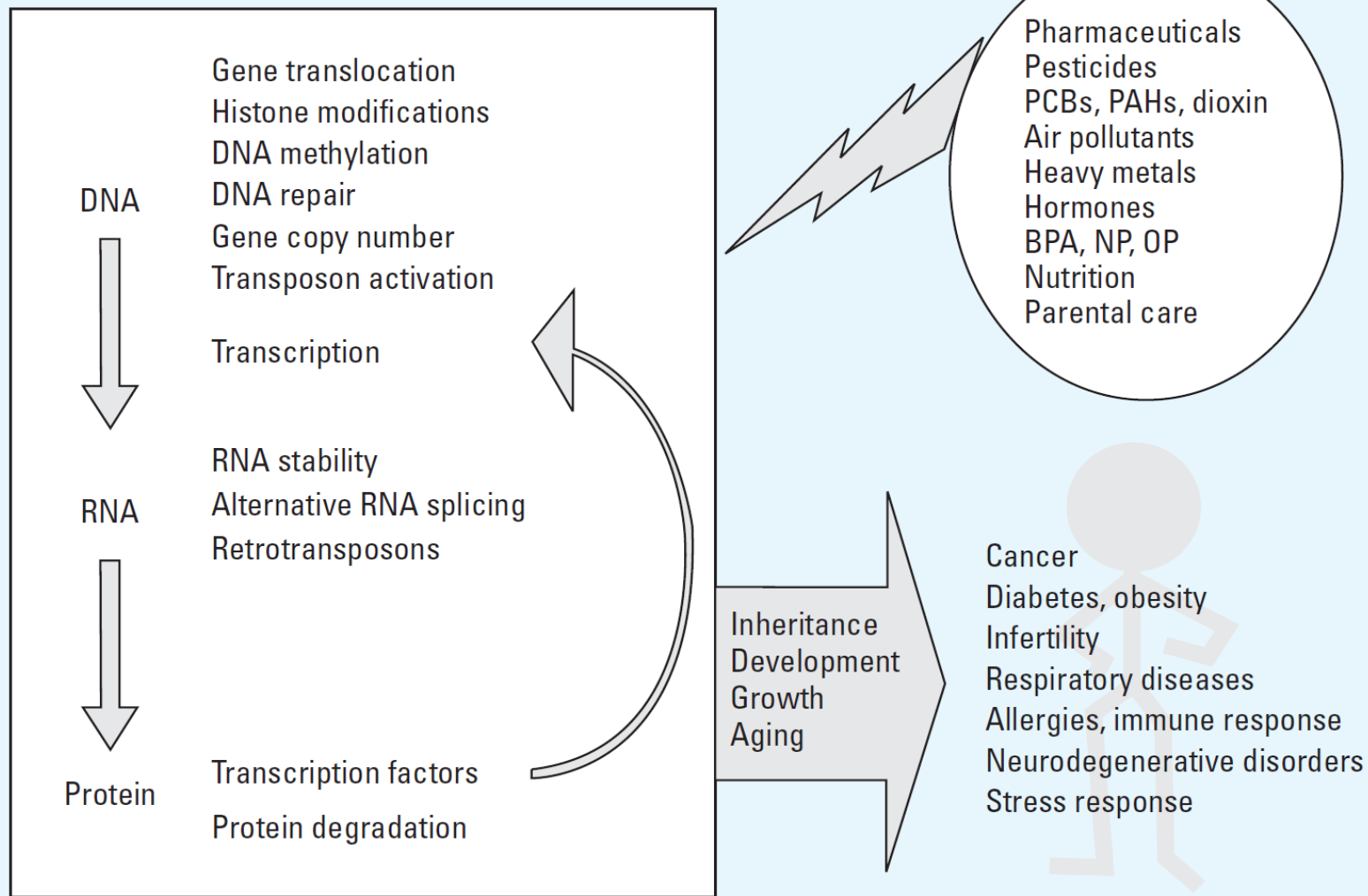
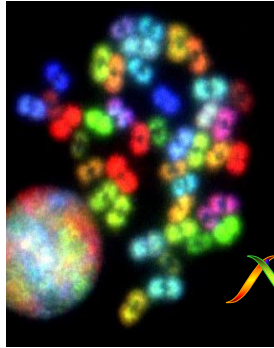


Figure 1. Summary of gene regulatory mechanisms affected by environmental exposures, with disease implications. Abbreviations: BPA, bisphenol A; NP, 4-nonylphenol; PAHs, polycyclic aromatic hydrocarbons, PCBs, polychlorinated biphenyls; OP, 4-*tert*-octylphenol.

Chemical exposure

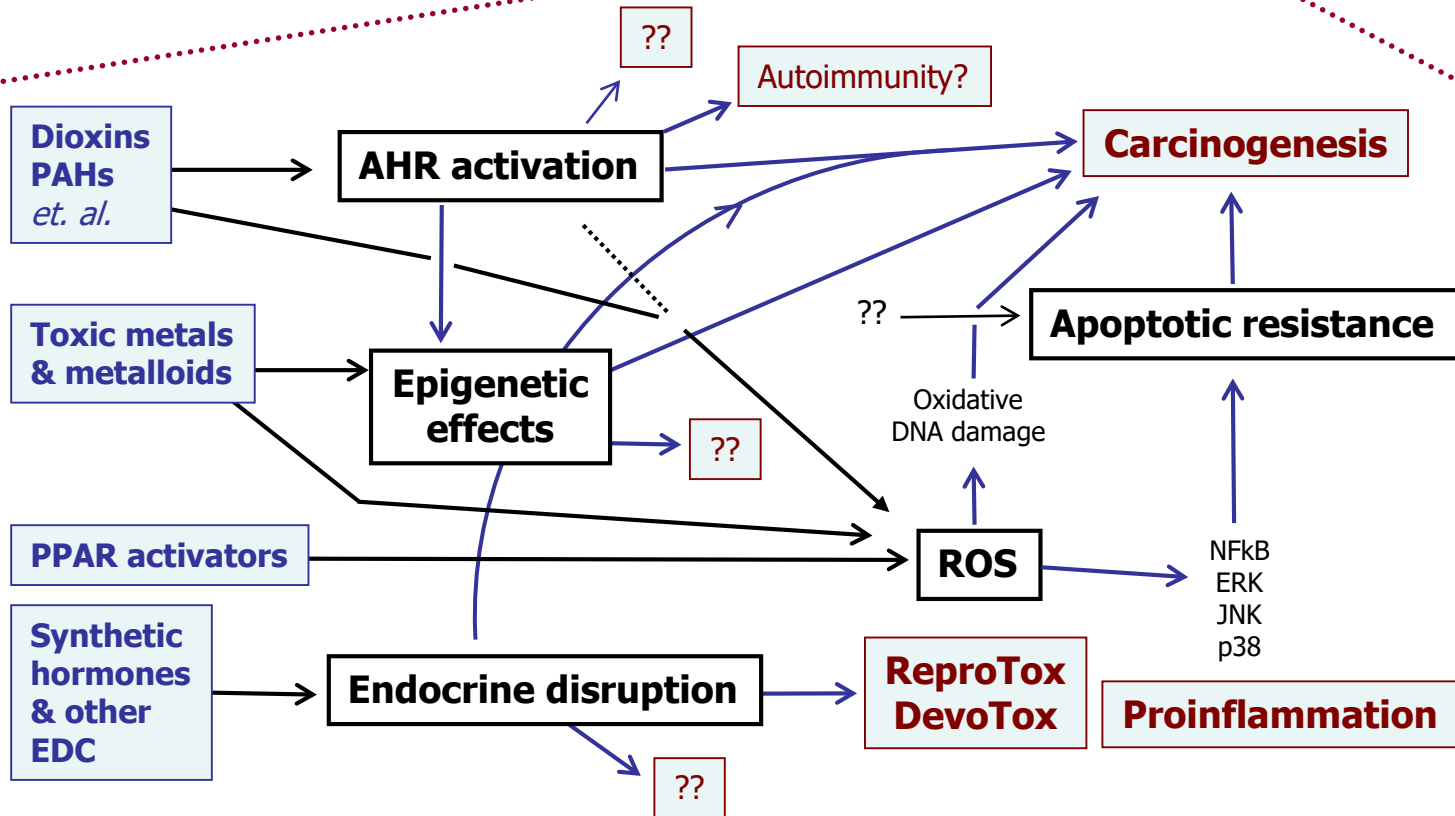
Henkler & Luch
JECH 2010



Genotoxic Damage

- Mutagenicity
- Clastogenicity
- Aneugenicity

Nongenotoxic mechanisms



Área de Seguridad Alimentaria

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Inspectors find phthalates in toys and asbestos in second-hand products

ECHA/PR/18/04

In an EU/EEA-wide project of ECHA's Enforcement Forum, inspectors found hundreds of consumer products with illegal amounts of restricted chemicals. Every fifth toy inspected contained high levels of restricted phthalates.

Helsinki, 13 February 2018 - The project report shows a relatively high number of products on the European market containing chemicals that are restricted under REACH. Inspectors in 27 European countries checked 1 009 mixtures, 4 599 articles and 17 substances. Overall, out of 5 625 targeted product checks, 18 % did not comply with the restrictions.

Basel Convention

Rotterdam Convention

Stockholm Convention

Synergies



**STOCKHOLM
CONVENTION**

Protecting human health and the environment
from persistent organic pollutants



Short-chain chlorinated paraffins (SCCPs)

Technically feasible alternatives are commercially available for all known uses of SCCPs.

PFOS Information submitted



Danish Ministry of the Environment
Environmental Protection Agency

Hazardous substances in plastics

Survey of chemical substances in consumer products No. 132, 2014

Que grandes multinacionales de todo el mundo se pongan de acuerdo para **eliminar plásticos** es, desde luego, una iniciativa que hay que aplaudir. En concreto, una treintena de ellas se han unido en la [Alliance to End Plastic Waste](#) (AEPW) y destinarán más de 1.500 millones de dólares (unos **1.316,7 millones de euros**) en los próximos cinco años para luchar contra este problema, en particular en los océanos.

Entre esas grandes compañías figuran [Henkel](#), [BASF](#), [Procter & Gamble](#), [Braskem](#), [Berry Global](#) o [Mitsubishi Chemical Holdings](#). Aunque inicialmente no están otras de las principales corporaciones como [Coca-Cola](#), [Danone](#), [Mondelez](#), [Unilever](#), [Mars Incorporated](#) o [Colgate-Palmolive](#) si es verdad que estas firmas están **comprometidas con este desafío**. Así, por ejemplo, Coca-Cola o Danone han firmado recientemente el 'Compromiso global para la nueva economía de plásticos', una iniciativa de la Fundación MacArthur.

El presidente de la AEPW y director ejecutivo de Procter & Gamble, David Taylor, ha hecho un llamamiento a todas las empresas, grandes o pequeñas de "todas las regiones y sectores" a unirse a la iniciativa.

SABER MÁS

Aumenta el rechazo a los envases plásticos en Europa

Un packaging para latas que respeta la vida marina

OUR COMMITMENT



\$1.5 Billion
over the next
five years

03
JUN

Pop Stop: Denmark retailer stops sale of microwave popcorn amid health fears

WRITTEN BY ZOE LAVENTHOL - PERMALINK

<http://greensciencepolicy.org/pop-stop/>



Some love it, some hate it: that overwhelming buttery, salty smell that fills the house every time you throw a bag of popcorn in the microwave. However, **larger concerns have led Danish retailer Coop Denmark to pull microwave popcorn from the shelves of their 1,200 stores.** Highly fluorinated chemicals like those used in food packaging have been linked to immune system problems, **increased risk of miscarriage** in women, and several types of **cancer**.



Highly fluorinated chemicals are commonly used in microwave popcorn bags and other food packaging products because they help make surfaces grease resistant. They also have water-resistant, grease-resistant and other useful properties and are used in waterproof jackets, stain-resistant carpets, nonstick cookware and more. However, many fluorinated compounds are also bioaccumulative, meaning

they build up in the body over time, and **persistent**, meaning they remain in the environment for as long as millions of years.

Claudia&Julia

Utensilios para una cocina tradicional y saludable



Si te interesa saber más sobre este tema, primero de todo, vamos a aclarar **qué es el teflón y su composición**. El teflón es una marca comercial que designa un compuesto usado para fabricar el recubrimiento antiadherente de las sartenes. Este compuesto se divide en dos componentes:

1. Un polímero denominado **PTFE** (politetrafluoroetileno), que es antiadherente, y
2. una sustancia llamada **PFOA** (ácido perfluorooctanoico), que sirve para adherir este polímero a la sartén.

Esta sustancia (el PFOA), que se usaba en el proceso de fabricación de las sartenes, se ha relacionado con cáncer y defectos congénitos en los estudios de laboratorio. Por este motivo, ya desde el 2014 ningún fabricante de sartenes u otros utensilios de cocina medianamente conocido usa el PFOA y **su uso se prohibió por completo en el 2015**.

En cambio, **el PTFE no es un producto tóxico** por sí mismo, incluso se usa en medicina para prótesis y creación de tejidos artificiales. La única consideración a tener presente es no calentarlo a más de 250° ya que se descompone emitiendo gases tóxicos. De ahí **nuestra recomendación** de usar las sartenes antiadherentes siempre para cocciones a baja temperatura y las sartenes de hierro para cocciones a fuego fuerte.

Claudia&Julia

Utensilios para una cocina tradicional y saludable

hola, entiendo que la sartén antiadherente de le Creuset... 1) no contiene PFOA, cierto? 2) sí tiene PTFE, cierto? 3) cuál es la temperatura que recomendáis no superar? y 4) a qué número del 1 al 9 corresponde esa temperatura en una placa de inducción Bosch comprada hace poco?

Hola, buenas tardes.

Claudia&Julia

hola

Así es! Sobre la temperatura, no aconsejamos que pase del 5 o 6 como mucho. Es importante calentarla más bajo aún y a medida que esté caliente, se puede aumentar hasta 5 o 6 como mucho.

Claudia&Julia

vale, gracias.

Gracias a ti!

Claudia&Julia

FOOD PACKAGING FORUM SCIENTIFIC STUDY

A scientific publication by the Food Packaging Forum reviews migration of substances of very high concern legally used in contact with food

[Read more](#)

FPP WEBINAR ON MIGRATION OF SVHCS

Join the Food Packaging Forum's webinar discussing its new scientific study on the migration of substances of very high concern from food contact materials, March 13, 2017!

Plastic is a substance the earth cannot digest.

DONATE

REFUSE SINGLE-USE PLASTIC

Resources



THE PROBLEM



SCIENCE



TAKE ACTION



EDUCATION

THE FACTS

Plastic never goes **away**.

Plastic spoils our **groundwater**.

Plastic attracts other **pollutants**.

Plastic threatens **wildlife**.

Plastic piles up in the **environment**.

Plastic poisons our **food chain**.

Plastic costs **billions** to abate.

Plastic affects human health.

Chemicals leached by plastics are in the blood and tissue of nearly all of us. Exposure to them is linked to cancers, birth defects, impaired immunity, endocrine disruption and other ailments.



Consumers' Guide to Highly Fluorinated Chemicals

“Stain-resistant, nonstick, waterproof and lethal” is how journalist Callie Lyons describes a highly fluorinated chemical called C8. This chemical leaked into the water supply near production facilities in West Virginia and Ohio. Hundreds of thousands of people were found to have C8 in their bodies and a wide range of **health problems** associated with this exposure.

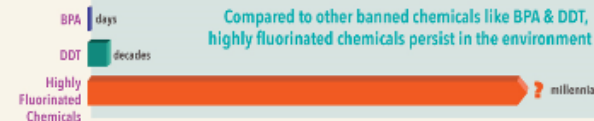
But such exposure is not just a problem for people living near chemical plants. This affects all of us, because we are exposed to highly fluorinated chemicals like C8 from a variety of consumer products we commonly use, such as clothing, carpets, cosmetics, and more.

LEARN MORE ABOUT:

- Why are highly fluorinated chemicals harmful?
- How are we exposed?
- What can you do?
- Resources
- In the Media

Many non-stick, waterproof, and stain-resistant products contain **highly fluorinated chemicals**. Though these products are convenient, they can harm our health and our environment.

HOW LONG DOES **NON-STICK** STICK AROUND?



Why is that a problem? The few highly fluorinated chemicals that have been studied are linked with:

cancer
obesity
liver malfunction
thyroid disruption

high cholesterol
hormonal changes
low birth weight & size
ulcerative colitis

What are they in? Highly fluorinated chemicals can be found in:



How can I avoid them?

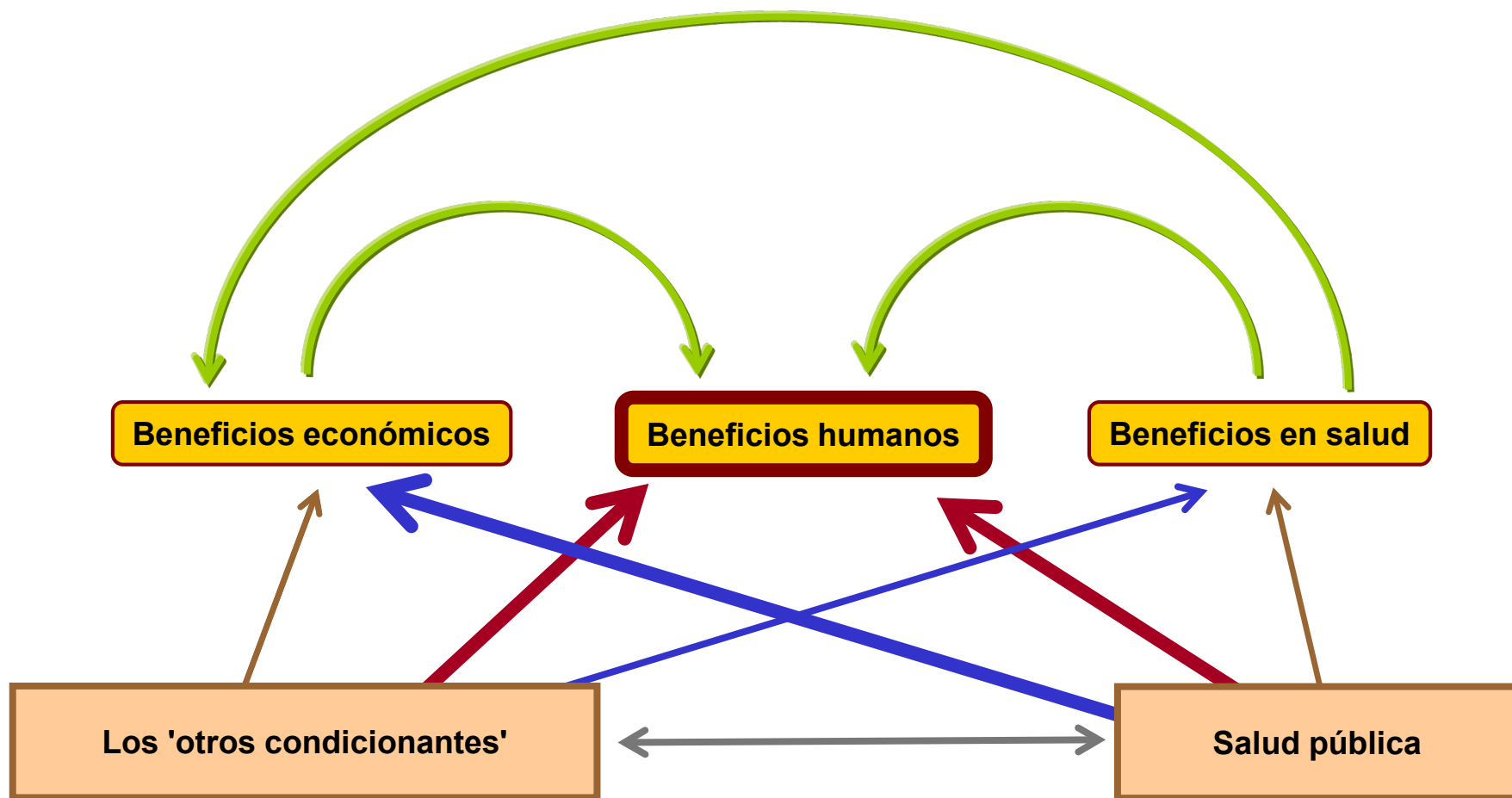
- AVOID** "perfluor-" "polyfluor-" "PTFE" in ingredient lists
- CONSIDER** the harm before buying products with these chemicals
- ASK** manufacturers for products without fluorinated chemicals
- MORE TIPS** at GreenSciencePolicy.org/highly-fluorinated-chemicals

Chemical Evaluation report: Achievements, challenges and recommendations after a decade of REACH

🕒 8 February 2019

Dossier evaluation checks whether the information provided by industry in the registration dossiers is compliant with the legal information required by REACH. ECHA performed Dossier evaluations on over 2,000 dossiers covering 700 substances. Dossier evaluation revealed that 70% of the dossiers is not compliant with the legal information requirements of REACH.

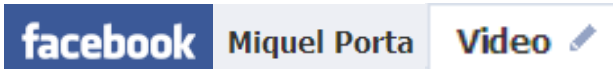
Ver lo que nos sale a cuenta



Economía, educación, trabajo, alimentación,
medio ambiente, transporte, energía...



**MOLTES GRÀCIES
PER LA VOSTRA ATENCIÓ.**



Miquel Porta (miquelporta)

Contaminación interna



miquel_porta



DOUBT IS THEIR PRODUCT

How Industry's Assault on Science
Threatens Your Health

David Michaels



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Cuidado con el catedrático

➤ "Debemos y podemos clarificar los intereses, los conocimientos y el papel de divulgador o de investigador científico que en cada noticia tiene el supuesto experto que la valora", defiende Porta.

Miquel Porta  Seguir a @miquelporta - Catedrático de Salud Pública del IMIM y Universidad Autónoma de Barcelona

23/10/2015 - 20:58h



SIXTH EDITION

A DICTIONARY OF
EPIDEMIOLOGY

EDITED BY

MIQUEL PORTA



A HANDBOOK SPONSORED BY THE IEA

OXFORD

A
Dictionary
of
Epidemiology

Sixth Edition

Edited for the
International Epidemiological Association

by

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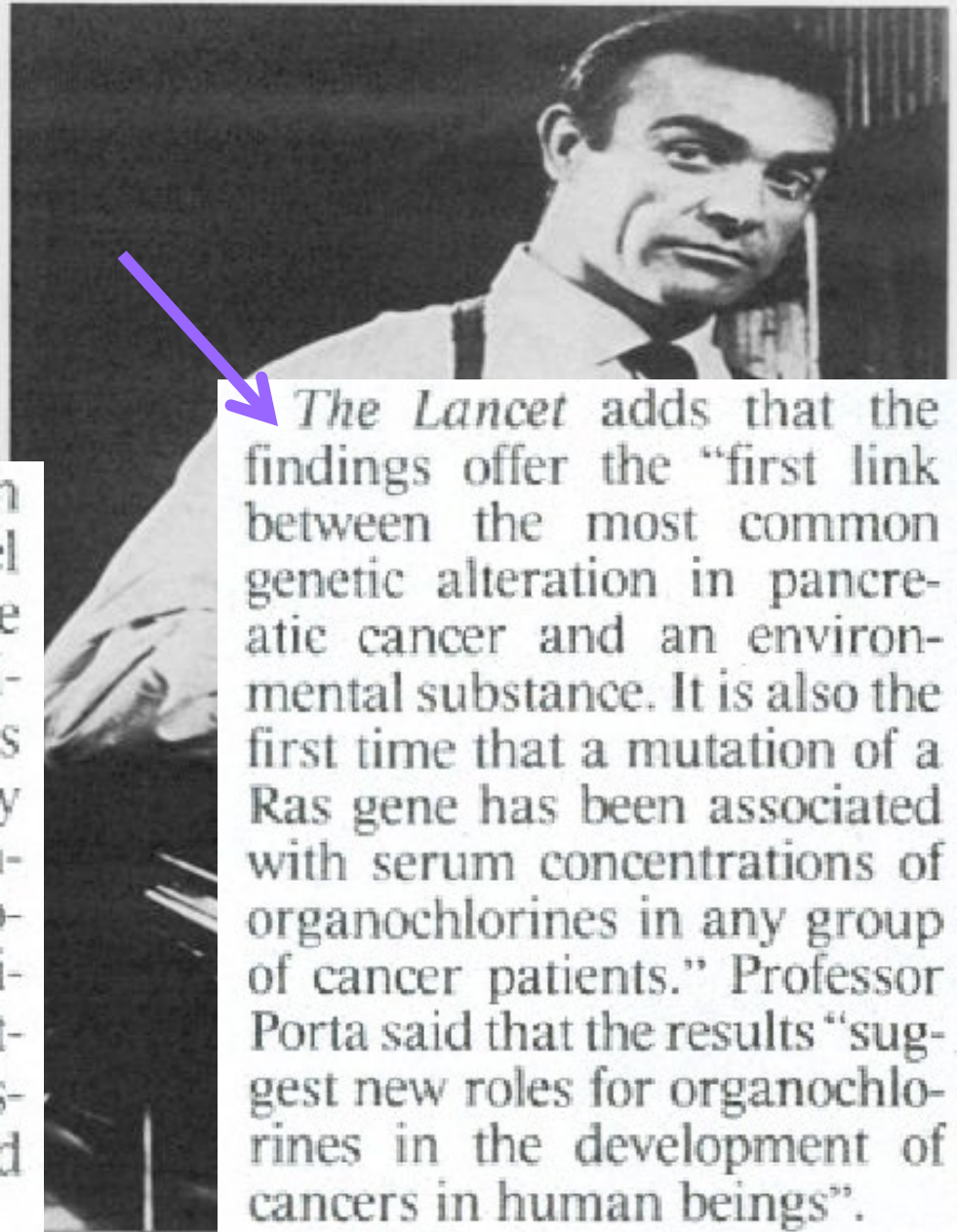
Assistant Editor

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OXFORD
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Pesticide pollution is linked to cancer

The cancer link has been made by Professor Miguel Porta and colleagues at the Municipal Institute of Medical Research and Autonomous University in Barcelona. They have been studying compounds known as organochlorines, including the pesticide DDT, and polychlorinated biphenyls or PCBs, used historically for insulating and cooling electrical equipment.



The Lancet adds that the findings offer the “first link between the most common genetic alteration in pancreatic cancer and an environmental substance. It is also the first time that a mutation of a Ras gene has been associated with serum concentrations of organochlorines in any group of cancer patients.” Professor Porta said that the results “suggest new roles for organochlorines in the development of cancers in human beings”.

EARLY REPORT

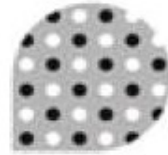
2125 **Serum concentrations of organochlorine compounds and K-ras mutations in exocrine pancreatic cancer**

M Porta and others, for the PANKRAS II Study Group

Volume 354, Number 9196 • Founded 1823 • Published weekly • Saturday 18/25 December 1999

Several organochlorine compounds can act as carcinogens and tumour promoters.³⁻⁸ Some modulate the expression of oncogenes, including *ras* genes.^{9,10} DDT and some PBCs have endocrine effects.^{1,2,11,12} Although presumably weak, such effects may be enhanced by environmental biodegradation, the long half-lives of the compounds (about 10 years for DDE, 30 years or more for some PCBs), and their concentrations in target tissues (100-fold to 350-fold higher in adipose tissue than in blood).^{1,5,6}

Trace Elements and Pancreatic Cancer Risk



A new study has found that high bodily levels of the trace elements nickel and selenium may be associated with reduced risk for pancreatic cancer, and that high levels of arsenic, cadmium and lead may increase the risk.

The study, published online Dec. 19 in the journal *Gut*, included 118 pancreatic cancer patients and 399 patients with other diagnoses at several hospitals in Spain. Researchers analyzed toenail samples with plasma mass spectrometry, a highly sensitive technique for detecting trace elements.

After controlling for age, sex, smoking, diabetes and other factors, the scientists found that the

subjects with the highest levels of arsenic were at twice the risk for pancreatic cancer, compared with those with the lowest concentrations. Those with high levels of cadmium were at three times the risk for pancreatic cancer, while those with the highest levels of lead were at six times the risk.

Those with the highest levels of nickel and selenium, on the other hand, were at significantly lower risk for pancreatic cancer.

Dr. Núria Malats, an epidemiologist at the Spanish National Cancer Research Center and the senior author of the new study, said that it was the first to provide these kinds of results with trace elements, and that it did not mean that people should take dietary supplements.

“All the News
That’s Fit to Print”

311130489
The New York Times

PRESS
RELEASE

ORIGINAL ARTICLE

Pancreatic cancer risk and levels of trace elements

André F S Amaral,¹ Miquel Porta,^{2,3} Debra T Silverman,⁴ Roger L Milne,¹ Manolis Kogevinas,⁵ Nathaniel Rothman,⁴ Kenneth P Cantor,⁶ Brian P Jackson,⁷ José A Pumarega,^{2,3} Tomàs López,^{2,3} Alfredo Carrato,^{8,9} Luisa Guarner,¹⁰ Francisco X Real,^{11,12} Núria Malats¹

Gut 2012

► Additional supplementary tables are published online only. To view these files please visit the journal online (<http://dx.doi.org/10.1136/gutjnl-2011-301086>).

¹Genetic and Molecular Epidemiology Group, Spanish National Cancer Research Centre (CNIO), Madrid, Spain
²Institut Municipal d'Investigació Mèdica (IMIM), Universitat Autònoma de Barcelona, Barcelona, Spain

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⁶KP Cantor Environmental LLC, Silver Spring, Maryland, USA

⁷Trace Element Analysis Core, Department of Earth Sciences, Dartmouth College, Hanover, New Hampshire, USA

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⁹Hospital General de Elche, Alicante, Spain

¹⁰Hospital de la Vall Hebron, Barcelona, Spain

¹¹Epithelial Carcinogenesis Group, Spanish National Cancer Research Centre (CNIO), Madrid, Spain

ABSTRACT

Background and Aims Knowledge on the aetiology of exocrine pancreatic cancer (EPC) is scant. The best established risk factor for EPC is tobacco smoking. Among other carcinogens, tobacco contains cadmium, a metal previously associated with an increased risk of EPC. This study evaluated the association between concentrations of trace elements in toenails and EPC risk.

Methods The study included 118 EPC cases and 399 hospital controls from eastern Spain. Levels of 12 trace elements were determined in toenail samples by inductively coupled plasma mass spectrometry. OR and 95% CI, adjusted for potential confounders, were calculated using logistic regression.

Results Significantly increased risks of EPC were observed among subjects whose concentrations of cadmium (OR 3.58, 95% CI 1.86 to 6.88; $p_{\text{trend}}=5 \times 10^{-6}$), arsenic (OR 2.02, 95% CI 1.08 to 3.78; $p_{\text{trend}}=0.009$) and lead (OR 6.26, 95% CI 2.71 to 14.47; $p_{\text{trend}}=3 \times 10^{-5}$) were in the highest quartile. High concentrations of selenium (OR 0.05, 95% CI 0.02 to 0.15; $p_{\text{trend}}=8 \times 10^{-11}$) and nickel (OR 0.27, 95% CI 0.12 to 0.59; $p_{\text{trend}}=2 \times 10^{-4}$) were inversely associated with the risk of EPC.

Conclusion Novel associations are reported of lead, nickel and selenium toenail concentrations with pancreatic cancer risk. Furthermore, the results confirm previous associations with cadmium and arsenic. These novel findings, if replicated in independent studies, would point to an important role of trace elements in pancreatic carcinogenesis.

Significance of this study

What is already known about the subject?

- Little is known about the aetiology of pancreatic cancer.
- Some trace elements, such as arsenic and cadmium, are carcinogenic for humans and may enter the organism through different routes.
- A few studies have found a link between exposure to arsenic and cadmium and pancreatic cancer risk.

What are the new findings?

- Individuals with the highest levels of selenium or nickel in toenails present a lower risk of pancreatic cancer.
- The study confirms the increased risk of pancreatic cancer among subjects with the highest levels of arsenic or cadmium in toenails.
- Besides arsenic and cadmium, high levels of lead may also be a risk factor for pancreatic cancer.

How might it impact on clinical practice in the foreseeable future?

- Selenium intake might be tested in clinical trials as a chemopreventive intervention in individuals at high risk of pancreatic cancer. Understanding the role of trace elements in pancreatic cancer pathogenesis could lead to preventive measures or treatments.

La nostra contaminació interna: alimentació, salut i societat.

Juneda,
4 d'abril de 2019



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