Progetto sottomisura 16.2. PSR 2014-2020 Regione Toscana

Peter Baby Bio – Nutriamo il futuro

Bio Baby Food

Mangiare sano nei primi mesi di vita…..Cosa significa oggi?

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Fattoria Poggio Alloro – S. Gimignano, 26 gennaio 2017

Livelli di Assunzione di Riferimento dei Nutrienti *(LARN, IV revisione, 2014)*

LARN PER L'ENERGIA					
Età	**Peso corporeo**	**Velocità di crescita**	**DET**	**Energia**	**FABBISOGNO ENERGETICO**
mesi	kg	(g/die)	(kcal/die)	(kcal/die)	(kcal/die)
Maschi					
6	7,9	14,0	581	39	620
7	8,3	11,9	618	18	640
8	8,6	10,5	646	15	660
9	8,9	9,5	674	14	690
10	9,2	8,6	702	23	730
11	9,4	8,1	720	22	740
12	9,6	7,9	739	21	760
Femmine					
6	7,3	13,3	525	49	570
7	7,6	11,5	553	20	580
8	7,9	10,4	581	18	600
9	8,2	9,1	609	16	630
10	8,5	8,2	637	19	640
11	8,7	7,8	655	18	660
12	8,9	7,6	674	18	690

LARN PER LE PROTEINE						
		Peso corporeo	AR Fabbisogno medio		PRI Assunzione raccomandata per la popolazione	
		(kg)	(g/kg×die)	(g/die)	(g/kg×die)	(g/die)
LATTANTI	6-12 mesi	8,6	1,11	9	1,32	11

Pasto	*Alimento*	*kcal*		*Prot. g*	
1° pasto	Latte 220 cc	154		3.0	
2° pasto	Minestrina 25 g	87.2		2.6	
	Omogen. 60 g		48		3.6
	Mela 50 g	26		0.2	
	Olio d'oliva 10 g	88,4		0.0	
3° pasto	Latte 220 cc	154		3.0	
4° pasto	Minestrina 25 g	87.2		2.6	
	Omogen. 60 g		48		3.6
	Mela 50 g	26		0.2	
	Olio d'oliva 10 g	88.4		0.0	
		807.2		18.8	(9.8%)

Nutrients consumption in Europe at 8 - 24 months*

Country	age (mos)	Prot. (g/kg)	Prot. (%)	Lip. (%)	Chol.
Spain	9	4.4	15.7	26.4	58.0
France	10		4.3	15.6	27.1	57.0
Italy	12	5.1	19.	30.5	50.0
Denmark	12-36	3.3	15.0	28.0	57.0

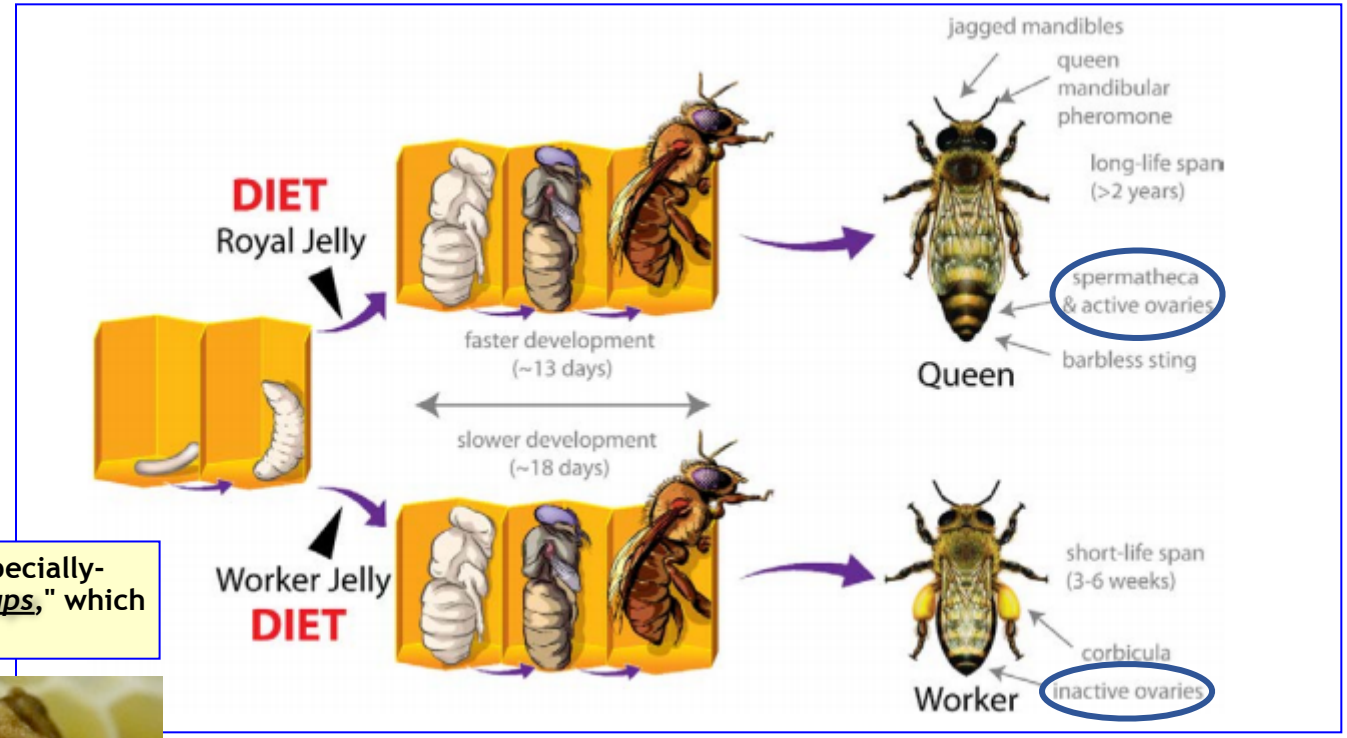
Rolland-Cachera et al, Acta Paediatr 88:365; 1999

**** Protein requirement: 8 - 12 %** (% of calorie needs)*
Agostoni C, et al Int J Obes 29:S8;2005

What Do Studies of Insect Polyphenisms Tell Us about Nutritionally-Triggered Epigenomic Changes and Their Consequences?

Nutrients **2015**, *7*, 1787-1797; doi:10.3390/nu7031787

OPEN ACCESS
nutrients
ISSN 2072-6643
www.mdpi.com/journal/nutrients

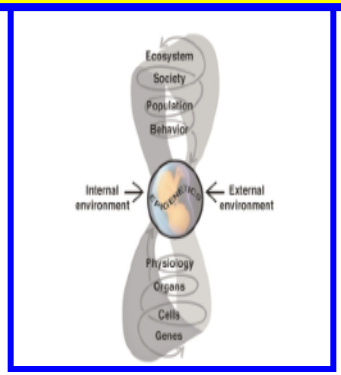


Queen Bee Larvae are raised in specially-constructed cells called "***queen cups***," which are **filled with *royal jelly*.**

The **larvae** that develop into ***workers*** and ***queens*** are **genetically identical**. But **as a result of the royal jelly diet, the queen will develop functional ovaries and a larger abdomen** for egg laying

Kucharski R., Maleszka J., Foret S., Maleszka R. ***Nutritional Control of Reproductive Status in Honeybees via DNA Methylation*** Science (2008) 319: 1827-1830

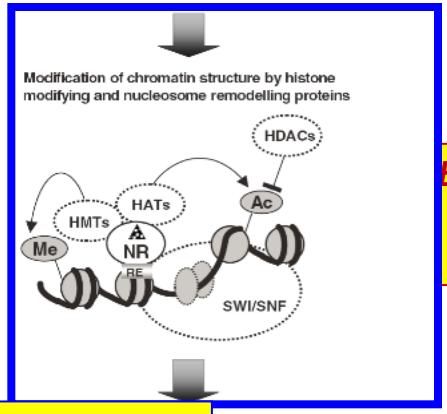
The 7 keywords: from genetics to *epigenetics*



3

*Ontogeny**

4

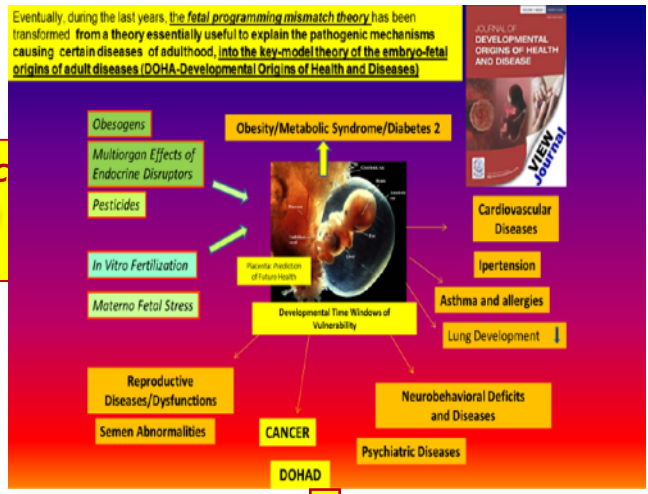


Developmental Plasticity

Evo → Devo

6

Epi-genetic Mismatch DOHaD



Fetal programming

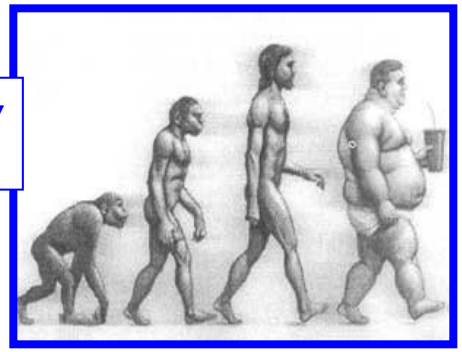
*Phylogeny**

2

Environment

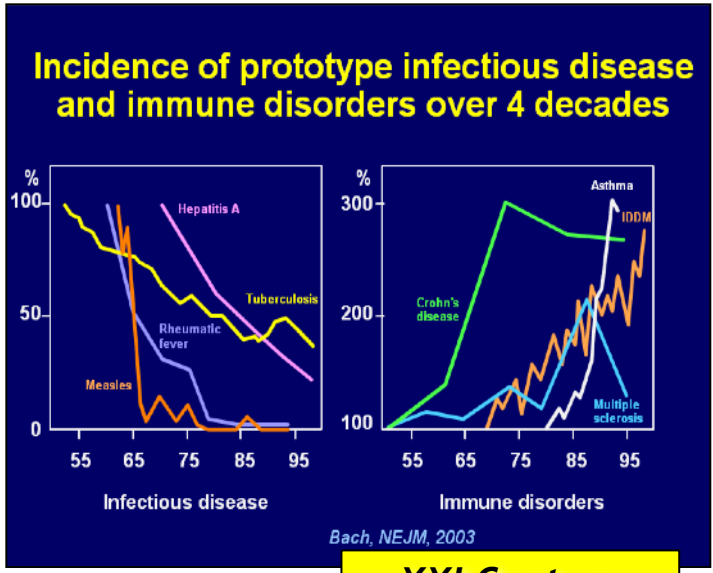
5

Evolutionary Medicine



1

From Genetics to Epigenetics



7

XXI Century Epidemiological Transition

Towards a paradigm shift in biomedicine. Environmental interference with the human (epi)genome

(Cortesia dr. E. Burgio)

van Dijk et al. Clinical Epigenetics (2015) 7:66
DOI 10.1186/s13148-015-0101-5

CLINICAL EPIGENETICS

REVIEW Open Access

Recent developments on the role of epigenetics in obesity and metabolic disease

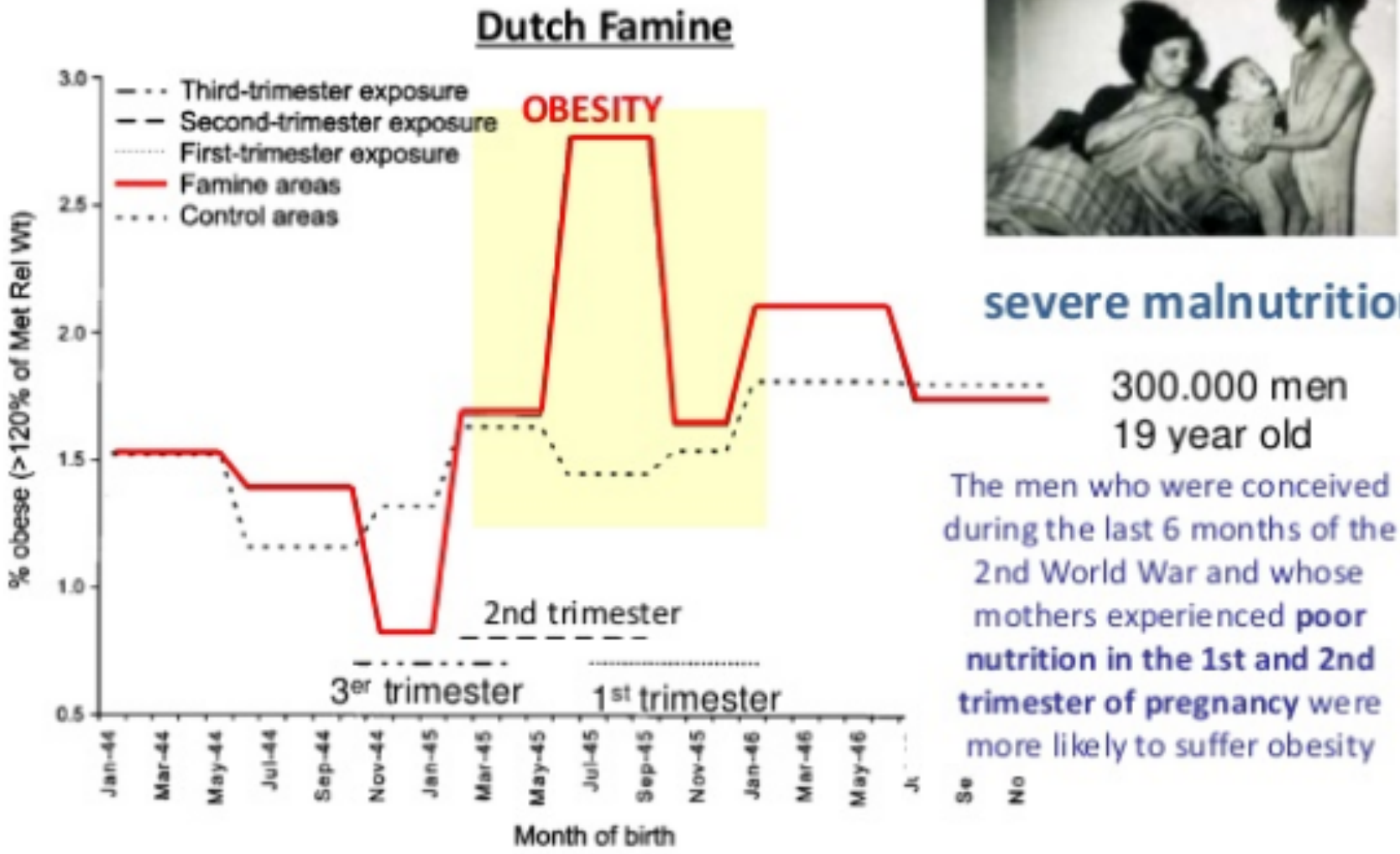
Susan J. van Dijk[1], Ross L. Tellam[2], Janna L. Morrison[3], Beverly S. Muhlhausler[4,5†] and Peter L. Molloy[1*†]

Abstract

The increased prevalence of obesity and related comorbidities is a major public health problem. While genetic factors undoubtedly play a role in determining individual susceptibility to weight gain and obesity, the identified genetic variants only explain part of the variation. This has led to growing interest in understanding the potential role of epigenetics as a mediator of gene-environment interactions underlying the development of obesity and its associated comorbidities. Initial evidence in support of a role of epigenetics in obesity and type 2 diabetes mellitus (T2DM) was mainly provided by animal studies, which reported epigenetic changes in key metabolically important tissues following high-fat feeding and epigenetic differences between lean and obese animals and by human studies which showed epigenetic changes in obesity and T2DM candidate genes in obese/diabetic individuals. More recently, advances in epigenetic methodologies and the reduced cost of epigenome-wide association studies (EWAS) have led to a rapid expansion of studies in human populations. These studies have also reported epigenetic differences between obese/T2DM adults and healthy controls and epigenetic changes in association with nutritional, weight loss, and exercise interventions. There is also increasing evidence from both human and animal studies that the relationship between perinatal nutritional exposures and later risk of obesity and T2DM may be mediated by epigenetic changes in the offspring. The aim of this review is to summarize the most recent developments in this rapidly moving field, with a particular focus on human EWAS and studies investigating the impact of nutritional and lifestyle factors (both pre- and postnatal) on the epigenome and their relationship to metabolic health outcomes. The difficulties in distinguishing consequence from causality in these studies and the critical role of animal models for testing causal relationships and providing insight into underlying mechanisms are also addressed. In summary, the area of epigenetics and metabolic health has seen rapid developments in a short space of time. While the outcomes to date are promising, studies are ongoing, and the next decade promises to be a time of productive research into the complex interactions between the genome, epigenome, and environment as they relate to metabolic disease.

Keywords: Epigenetics, DNA methylation, Obesity, Type 2 diabetes, Developmental programming

Gestational Caloric Restriction and Predisposition of Offspring to Obesity



severe malnutrition

300.000 men
19 year old

The men who were conceived during the last 6 months of the 2nd World War and whose mothers experienced **poor nutrition in the 1st and 2nd trimester of pregnancy** were more likely to suffer obesity

Ravelli GP *et al* (1976) **Obesity in young men after famine exposure in utero and early infancy**. N. Engl. J. Med. 295: 349 –353

Mangiare sano nei primi mesi di vita…..Cosa significa oggi?

1. Rispettare il giusto apporto calorico/ proteico previsto.

CHEMICAL FALL OUT

The gift our mothers never wanted to give us

1. ENDOCRINE DISRUPTORS
dioxin-like moleculles

2. HEAVY METALS

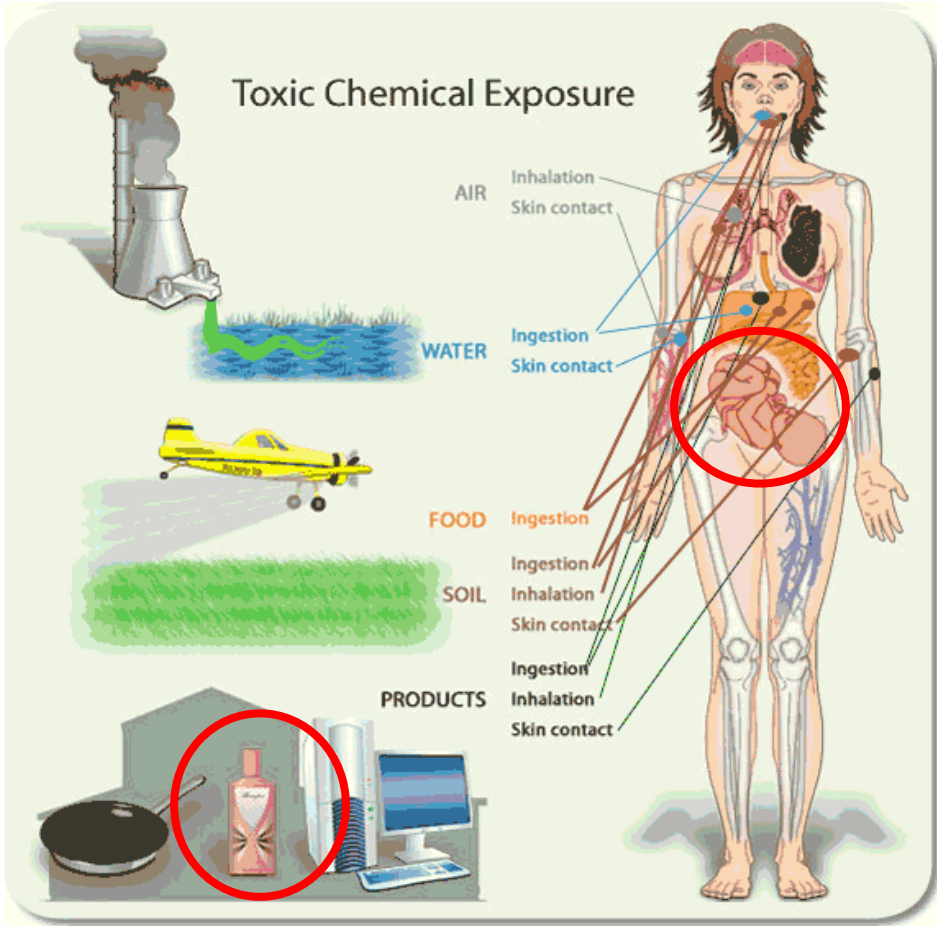
3. ULTRAFINE PARTICLES

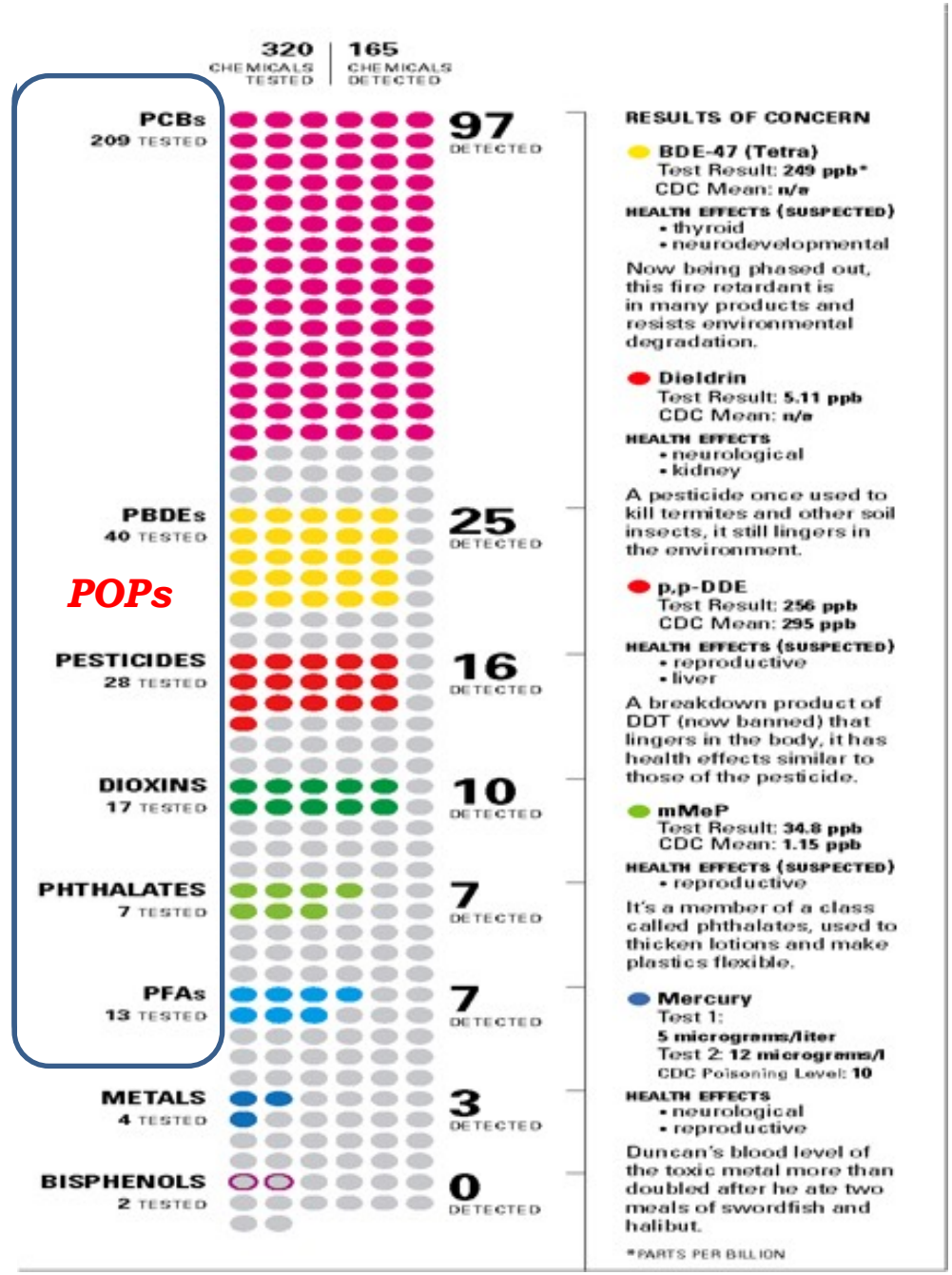
.. at present many studies in various parts of the world are evaluating the ***chemical body burden*** .. especially in women, children, embryos / fetuses, **providing dramatic results.**

http://www.ewg.org/reports/generations/

Monitoring Body-Burdens

700 different synthetic chemicals or heavy metals found in human blood,





(Cortesia dr. E. Burgio)

Neurodevelopmental Disorders and Prenatal Residential Proximity to Agricultural Pesticides: The CHARGE Study

Janie F. Shelton,[1] Estella M. Geraghty *Environ Health Perspect*; DOI:10.1289/ehp.1307044: 23 June 2014

970 participants, **California Pesticide Use Report** (1997-2008) linked to the ***addresses during pregnancy***. *Pounds of active ingredient …* aggregated within 1.25km, 1.5km, and 1.75km buffer distances from the home

- **Organophosphates** higher 3rd trimester expos: **60% increased risk of Autistic Spectrum Disorder (ASD)**
- **Pyrethroid insecticide** just prior to conception or for 3rd trimester at **greater risk for both ASD and DD** (developmental delay)
- **Carbamate**: risk for **DD** increased (Arprocarb: Undene, Propoxur).

Environmental Health

BioMed Central

Review

Environmental Health 2008, **7**:50 doi:10.1186/1476-069X-7-50

Open Access

Potential developmental neurotoxicity of pesticides used in Europe

Marina Bjørling-Poulsen*[1], Helle Raun Andersen[1] and Philippe Grandjean[1,2]

Pesticides used in agriculture are designed to protect crops against unwanted species, such as weeds, insects, and fungus. Many compounds target the nervous system of insect pests. Because of the similarity in brain biochemistry, such pesticides may also be neurotoxic to humans. Concerns have been raised that the developing brain may be particularly vulnerable to adverse effects of neurotoxic pesticides. Current requirements for safety testing do not include developmental neurotoxicity. We therefore undertook a systematic evaluation of published evidence on neurotoxicity of pesticides in current use, with specific emphasis on risks during early development. Epidemiologic studies show associations with neurodevelopmental deficits, but mainly deal with mixed exposures to pesticides. Laboratory experimental studies using model compounds suggest that many pesticides currently used in Europe – including organophosphates, carbamates, pyrethroids, ethylenebisdithiocarbamates, and chlorophenoxy herbicides – can cause neurodevelopmental toxicity. Adverse effects on brain development can be severe and irreversible. Prevention should therefore be a public health priority. The occurrence of residues in food and other types of human exposures should be prevented with regard to the pesticide groups that are known to be neurotoxic. For other substances, given their widespread use and the unique vulnerability of the developing brain, the general lack of data on developmental neurotoxicity calls for investment in targeted research. While awaiting more definite evidence, existing uncertainties should be considered in light of the need for precautionary action to protect brain development.

Research | Children's Health

VOLUME 123 | NUMBER 3 | March 2015 • Environmental Health Perspectives

A Section 508–conformant HTML version of this article is available at http://dx.doi.org/10.1289/ehp.1408133.

Autism Spectrum Disorder and Particulate Matter Air Pollution before, during, and after Pregnancy: A Nested Case–Control Analysis within the Nurses' Health Study II Cohort

Raanan Raz,[1] Andrea L. Roberts,[2] Kristen Lyall,[3,4] Jaime E. Hart,[1,5] Allan C. Just,[1] Francine Laden,[1,5,6] and Marc G. Weisskopf[1,6]

BACKGROUND: Autism spectrum disorder (ASD) is a developmental disorder with increasing prevalence worldwide, yet has unclear etiology.

OBJECTIVE: We explored the association between maternal exposure to particulate matter (PM) air pollution and odds of ASD in her child.

METHODS: We conducted a nested case–control study of participants in the Nurses' Health Study II (NHS II), a prospective cohort of 116,430 U.S. female nurses recruited in 1989, followed by biennial mailed questionnaires. Subjects were NHS II participants' children born 1990–2002 with ASD (n = 245), and children without ASD (n = 1,522) randomly selected using frequency matching for birth years. Diagnosis of ASD was based on maternal report, which was validated against the Autism Diagnostic Interview-Revised in a subset. Monthly averages of PM with diameters ≤ 2.5 μm ($PM_{2.5}$) and 2.5–10 μm ($PM_{10-2.5}$) were predicted from a spatiotemporal model for the continental United States and linked to residential addresses.

RESULTS: $PM_{2.5}$ exposure during pregnancy was associated with increased odds of ASD, with an adjusted odds ratio (OR) for ASD per interquartile range (IQR) higher $PM_{2.5}$ (4.42 μg/m^3) of 1.57 (95% CI: 1.22, 2.03) among women with the same address before and after pregnancy (160 cases, 986 controls). Associations with $PM_{2.5}$ exposure 9 months before or after the pregnancy were weaker in independent models and null when all three time periods were included, whereas the association with the 9 months of pregnancy remained (OR = 1.63; 95% CI: 1.08, 2.47). The association between ASD and $PM_{2.5}$ was stronger for exposure during the third trimester (OR = 1.42 per IQR increase in $PM_{2.5}$; 95% CI: 1.09, 1.86) than during the first two trimesters (ORs = 1.06 and 1.00) when mutually adjusted. There was little association between $PM_{10-2.5}$ and ASD.

CONCLUSIONS: Higher maternal exposure to $PM_{2.5}$ during pregnancy, particularly the third trimester, was associated with greater odds of a child having ASD.

ASDs risk (OR> 50%) increased significantly among mothers exposed to fine particles (PM 2.5)
and not to PM 2.5-10 especially during the **third trimester of pregnancy (Synaptogenesis!)**

Two large case-control studies had already shown this correlation JAMA Psy 2013;70(1):71-7;
EHP 2013;121(3):380-6

Disregolatori endocrini

Composti	Fonti di esposizione (ingestione, contatto, inalazione)
Fitoestrogeni	Dieta, piante edibili (soia, Tofu, lenticchie, fagioli, piselli, finocchio, grano, olio di oliva, ciliegie, mele, pere, sesamo, orzo, riso).
Micotossine (Zearalonone)	Contaminazione mangimi, accumulo nel tessuto adiposo animale
DDT, p,p'-DDE, Lindano, dieldrina	Dieta, pesticidi ed erbicidi anche accumulati nel grasso animale.
PCB (bifenili policlorinati)	Dieta, prodotti intermedi industria elettrica anche accumulati nel grasso animale (componenti elettrici, isolanti).
Bisfenolo A	Resine epossidiche, plastiche in policarbonato.
Ftalati	Materiali in plastica (PVC) (industria elettrica, calzaturiera, tessile, dispositivi medicali, cosmetici, deodoranti, profumi, etc.).
Paraben (esteri alchilici dell'acido p-idrossibenzoico)	Conservanti per prodotti farmaceutici, industria alimentare, cosmetici, schermanti solari.
Diossine (TCDD)	Combustione di rifiuti contenenti PVC; uso di PCB contaminati con diossine; produzione di erbicidi (orange compound)
Alluminio	Cosmetici (ad es. antitraspiranti).

“Epidemie” di telarca prematuro

- Milano, scuola di via Folli, anni 1977 – 1978, *250 casi*, età 3 – 7 aa. (*Sospetta contaminazione di carne di pollo e vitello con estrogeni*. Fara et al., Lancet 2:295; 1979).
- Portorico, anni 1990 – 1995, *1916 casi*, età 6 mesi – 8 aa. (*Elevati livelli sierici di zearalenone e ftalati*. Larriuz-Serrano et al., PR Health Sci J 20:13; 2001).

www.jpeds.com • THE JOURNAL OF PEDIATRICS

Mycoestrogen Pollution of Italian Infant Food

Valentina Meucci, PharmD, PhD, Giulio Soldani, MD, Elisabetta Razzuoli, DVM, Giuseppe Saggese, MD

- Almeno il 28% dei campioni di latte formulato sono risultati contaminati dallo zearalenone e/o dai suoi metaboliti;
- Almeno il 27% dei campioni di alimenti per bambini a base di carne sono risultati contaminati dallo zearalenone e/o dai suoi metaboliti.

Conclusions This study shows the presence of mycoestrogens in infant (milk-based and meat-based) food, and this is likely to have great implications for subsequent generations, suggesting the need to perform occurrence surveys in this type of food. *(J Pediatr 2011;159:278-83).*

Mangiare sano nei primi mesi di vita…..Cosa significa oggi?

1. Rispettare il giusto apporto calorico/ proteico previsto.
2. Utilizzare alimenti da coltivazioni/ alleva-menti non contaminati (bio) e controllati.

Nutrizione e AGEs (Advanced glycation end-products)

- AGEs o glicotossine: gruppo eterogeneo di composti formati attraverso una glicazione non enzimatica di proteine, lipidi, acidi nucleici.
  - Possono avere origine endogena (es. diabete mellito non controllato);
  - *In buona parte originano dagli alimenti, "food AGEs" (**cibi cotti ad alta temperatura**)*;
  - In parte vengono assorbiti (10%) e trattenuti nel corpo o eliminati con le urine.
- Es. di AGEs: CML (carbossimetillisina);

  MG (metilglicossale).

(Goldberg T. et al., J Am Diet Assoc 2004)

Nutrizione e AGEs

AGEs e Stress ossidativo (OS) → disfunzione endoteliale (precursore di aterosclerosi e CVD)

AGEs causano OS tramite: Ossido nitrico (NO)

di mediatori di disfunzione endoteliale

(VCAM, E-Selectina)

adiponectina e leptina

(disfunzione adipociti)

(Stirban A et al., N.Y.Acad Sci 2008)

Original Article

SAGE Open Medicine

Skin advanced glycation end-products evaluation in infants according to the type of feeding and mother's smoking habits

SAGE Open Medicine
Volume 4: 1–7

DOI: 10.1177/2050312116682126
smo.sagepub.com
SAGE

Giovanni Federico[1], Martina Gori[1], Emioli Randazzo[1] and Francesco Vierucci[2]

Abstract

Objectives: This study was conducted to assess whether formula-fed infants had increased skin advanced glycation end-products compared with breastfed ones. We also evaluated the effect of maternal smoke during pregnancy and lactation on infant skin advanced glycation end-products accumulation.

Methods: Advanced glycation end-product–linked skin autofluorescence was measured in 101 infants.

Results: In infants born from non-smoking mothers, advanced glycation end-products were higher in formula-fed subjects than in breastfed subjects (0.80 (0.65–0.90) vs 1.00 (0.85–1.05), $p < 0.001$). Advanced glycation end-products in breastfed infants from smoking mothers were higher than in those from non-smoking mothers (0.80 (0.65–0.90) vs 1.00 (0.90–1.17), $p = 0.009$).

Conclusion: Formula-fed infants had increased amounts of advanced glycation end-products compared with the breastfed ones, confirming that breast milk represents the best food for infants. Breastfed infants from mothers smoking during pregnancy and lactation had increased skin advanced glycation end-products, suggesting that smoke-related advanced glycation end-products transfer throughout breast milk. Moreover, advanced glycation end-products may already increase during gestation, possibly affecting fetal development. Thus, we reinforced that smoking must be stopped during pregnancy and lactation.

nutrients

MDPI

Article

On the Importance of Processing Conditions for the Nutritional Characteristics of Homogenized Composite Meals Intended for Infants

Elin Östman *, Anna Forslund, Eden Tareke and Inger Björck

Published: 3 June 2016

Abstract: The nutritional quality of infant food is an important consideration in the effort to prevent a further increase in the rate of childhood obesity. We hypothesized that the canning of composite infant meals would lead to elevated contents of carboxymethyl-lysine (CML) and favor high glycemic and insulinemic responses compared with milder heat treatment conditions.

It was therefore concluded that intake of commercially canned composite infant meals leads to reduced postprandial insulin sensitivity and increased exposure to oxidative stress promoting agents.

Food Additives and Contaminants
Vol. 26, No. 6, June 2009, 776–785

Taylor & Francis
Taylor & Francis Group

Risk assessment of furan in commercially jarred baby foods, including insights into its occurrence and formation in freshly home-cooked foods for infants and young children

Dirk W. Lachenmeier*, Helmut Reusch and Thomas Kuballa

Chemisches und Veterinäruntersuchungsamt Karlsruhe, Weißenburger Str. 3, Karlsruhe, 76187 Germany

Furan is a possible human carcinogen (IARC group 2B) with widespread occurrence in many types of foods. In this study, a survey of furan contamination in 230 commercially jarred ready-to-eat infant food products was conducted using headspace sampling in combination with gas chromatography and mass spectrometry (HS-GC/MS) with a detection limit of 0.2 $\mu g\,kg^{-1}$. The incidence of furan contamination in jarred infant beverages, cereals and fruits was relatively low, with average concentrations below 10 $\mu g\,kg^{-1}$. Significantly higher concentrations were found in pasta ($34.8 \pm 14.5\,\mu g\,kg^{-1}$), meals containing meat ($28.2 \pm 15.0\,\mu g\,kg^{-1}$), and meals containing vegetables ($31.2 \pm 17.3\,\mu g\,kg^{-1}$). The average exposure of 6-month-old infants to furan was estimated to be 0.2 µg per kg bodyweight per day. The margin of exposure calculated using the T25 dose descriptor would be 2692, which points to a possible public health risk. In contrast to commercially jarred food products, none of 20 freshly home-prepared baby foods contained furan above the limit of detection. Only after re-heating in closed vessels was furan found to have formed. Furan was especially prevalent in reheated foods containing potatoes, with values ranging between 2.3 and 29.2 $\mu g\,kg^{-1}$. The formation of furan in potato-containing baby foods was increased by addition of ascorbic acid, by longer heating times above 1 h and by temperatures above 50°C. Research regarding reduction of furan in commercial baby foods should be conducted, with a priority aimed at reducing this heat-induced contaminant without concomitantly increasing the microbiological risk.

European Food Safety Authority

EFSA Journal 2011; 9(9):2347

SCIENTIFIC REPORT OF EFSA

Update on furan levels in food from monitoring years 2004-2010 and exposure assessment[1]

European Food Safety Authority[2, 3]

European Food Safety Authority (EFSA), Parma, Italy

ABSTRACT

Furan, which can be formed in a variety of heat-treated commercial foods, has been shown to be carcinogenic in animal experiments. The current report provides an update to include all data sampled and analysed between 2004 and 2010 and, in addition to previous reports, presents exposure estimates for different populations. The analysis includes a total of 5,050 analytical results for furan content in food submitted by 20 countries. The highest furan levels were found in coffee with mean values varying between 45 µg/kg for brewed coffee and 3,660 µg/kg for roasted coffee beans. The highest 95th percentile was reported for roasted coffee beans at 6,407 µg/kg. In the non-coffee categories, mean values ranged between 3.2 µg/kg for infant formula and 49 µg/kg for jarred baby food 'vegetables only', the latter also with the highest 95th percentile of 123 µg/kg. Mean furan exposure across surveys was estimated to range between 0.03 and 0.59 µg/kg b.w. per day for adults, between 0.02 to 0.13 µg/kg b.w. per day for adolescents, between 0.04 and 0.22 µg/kg b.w. per day for other children, between 0.05 to 0.31 µg/kg b.w. per day for toddlers and between 0.09 and 0.22 µg/kg b.w. per day for infants. A major contributor to exposure for adults was brewed coffee with an average of 85% of total furan exposure. Major contributors to furan exposure in toddlers and other children were fruit juice, milk-based products and cereal-based products, whereas in addition for toddlers jarred baby foods were major contributors. To reduce uncertainty associated to exposure estimates future testing should preferably target food products where limited results are available.

FOOD ADDITIVES & CONTAMINANTS: PART A, 2016
VOL. 33, NO. 4, 612–622
http://dx.doi.org/10.1080/19440049.2016.1154613

Taylor & Francis
Taylor & Francis Group

Effect of oxygen availability and pH on the furan concentration formed during thermal preservation of plant-based foods

Stijn Palmers, Tara Grauwet, Laura Vanden Avenne, Thomas Verhaeghe, Biniam T. Kebede, Marc E. Hendrickx and Ann Van Loey

Laboratory of Food Technology, Leuven Food Science and Nutrition Research Center (LFoRCe), Department of Microbial and Molecular Systems (M^2S), KU Leuven, Kasteelpark Arenberg 22, Box 2457, B-3001 Heverlee, Belgium

ABSTRACT

Thermally treated fruit- and vegetable-based foods are important contributors to the furan exposure of children and adults. Furan reduction by adding or removing precursors from the product has proven to be challenging because of major food constituents and interactions involved in the reaction pathways leading to furan formation. Instead of intervening at the precursor level, it might be more feasible to influence these formation pathways by adjusting the matrix properties of the product. As opposed to many previous literature sources, the present study investigated the effects of oxygen availability (normal versus reduced) and pH (acid versus low acid) on the furan formation in a real food system. Different combinations of both matrix properties were prepared in a reconstituted potato purée and subjected to a thermal treatment with a pasteurisation or sterilisation intensity. Irrespective of the addition of the furan precursors ascorbic acid, fructose and fatty acids, a considerable furan reduction was observed for the sterilised purées (F_{121}^{10} = 15 min) with either a reduced oxygen availability (0.1–1.8 mg l^{-1}) or at pH 3. The effects of both matrix properties were less pronounced in the pasteurised purées (P_{90}^{10} = 10 min), because of the lower furan concentrations. Even though the mechanisms of furan reduction for both types of matrix properties could not be fully elucidated, the results showed that lowering the oxygen concentration or pH prior to thermal processing offers a powerful, additional strategy for furan mitigation in thermally treated plant-based foods.

Mangiare sano nei primi mesi di vita…..Cosa significa oggi?

1. Rispettare il giusto apporto calorico/ proteico previsto.
2. Utilizzare alimenti da coltivazioni/allevamenti non contaminati (bio) e controllati.
3. Utilizzare alimenti prodotti con processi di trasformazione delle materie prime in grado di abbattere la formazione di eventuali sostanze tossiche.

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Flavor Perception in Human Infants: Development and Functional Significance

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Key Words
Taste · Smell · Flavor · Infant · Child · Food · Imprinting

Abstract
Background: Foods people consume impact on their health in many ways. In particular, excess intake of salty, sweet and fatty foods and inadequate intake of fruits and vegetables have been related to many diseases including diabetes, hypertension, cardiovascular disease and some cancers. The flavor of a food determines its acceptability and modulates intake. It is thus critical to understand the factors that influence flavor preferences in humans. ***Aim:*** To outline several of the important factors that shape flavor preferences in humans. ***Methods:*** We review a series of studies, mainly from our laboratories, on the important role of early experiences with flavors on subsequent flavor preference and food intake. ***Results and Conclusions:*** Some taste preferences and aversions (e.g. liking for sweet, salty and umami; disliking for bitter) are innately organized, although early experiences can modify their expression. In utero events may impact on later taste and flavor preferences and modulate intake of nutrients. Both before and after birth, humans are exposed to a bewildering variety of flavors that influence subsequent liking and choice. Fetuses are exposed to flavors in amniotic fluid modulating preferences later in life and flavor learning continues after birth. Experience with flavors that are bitter, sour or have umami characteristics, as well as volatile flavors such as carrot and garlic, occurs through flavorings in breast milk, infant formula and early foods. These early experiences mold long-term food and flavor preferences which can impact upon later health.

- L'esposizione del feto ai sapori/aromi nel liquido amnio-tico modula le preferenze dei cibi nella vita postnatale, durante cui prosegue l'apprendi-mento degli odori;
- Il contatto con sapori amari, aspri e umami, o aromi volatili come carota e aglio, si verifica con l'allattamento al seno, con latte formulato e con i cibi dei primi mesi;
- Queste esperienze condizionano le preferenze di gusti e cibi nelle epoche

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4. Diversificare gli alimenti anche per favorire lo sviluppo dell'olfatto e del gusto, facilitando l'accettazione dei nuovi cibi.


Grazie per le attenzioni che avrete per farmi mangiare sano