HASSENFELD CHILDREN'S HOSPITAL AT NYU LANGONE

# The Plastic Paradox: Why Plastic is an Urgent Threat to Human Health

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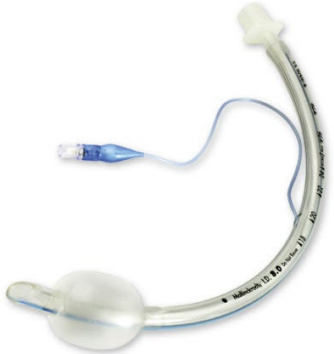
# Plastics have transformed health care.



# Plastics have transformed the world.

2 million tons produced each year in 1950s

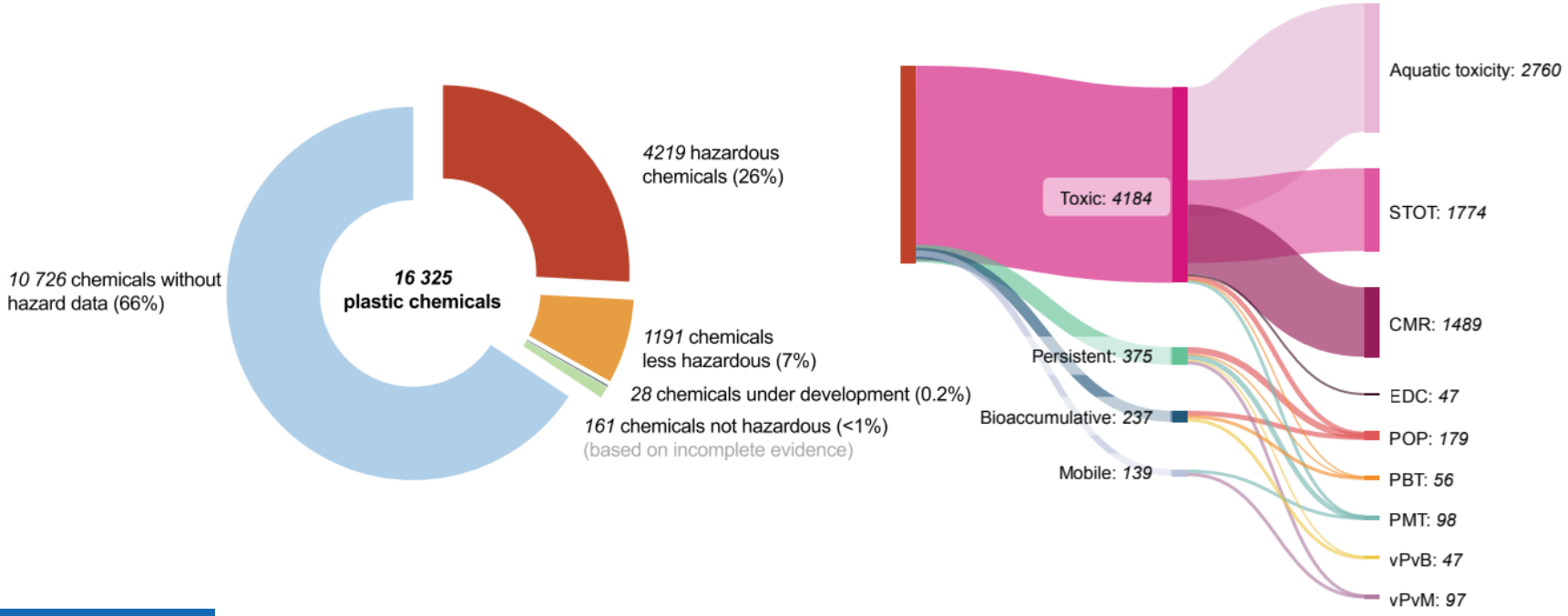
Today: 400 million tons produced each year

Growing demand from emerging economies

Of the virgin plastics produced globally in 2020, 52% produced in Asia (32% in China), followed by North America (19%), Europe (17%), the Middle East and Africa (7%), and Latin America (4%)

# Plastics have created a crisis.

- Ecosystem effects
- Climate change
- Chemicals and human health




PlastChem

Wagner et al (2024)

Winning isn’t everything; it’s the only thing.

– UCLA Coach Henry Russell Sanders
(not Vince Lombardi)

The endocrine system isn’t everything; it’s the only thing.

# What are endocrine disrupting chemicals?

Endocrine disrupting chemicals (EDCs) interfere with hormonal signaling systems

- Mimic, block, or modulate the synthesis, release, transport, metabolism, binding, or elimination of natural hormones
- Brain, pituitary, gonads, thyroid, and other components of the endocrine system

| | Representative EDCs |
|---|---|
| Pharmaceuticals | Trenbolone acetate, ethinylestradiol, dexamethasone, levonorgestrel, rosiglitazone |
| Cosmetics, personal care products | DBP, benzophenones, parabens, triclosan, DEET |
| Pesticides, herbicides, fungicides | Chlorpyrifos, glyphosate, pyraclostrobin, DDT, atrazine |
| Industrial chemicals | BPA, PCBs, triphenyl phosphate, PBDEs |
| Metals | Lead, cadmium, mercury, arsenic |
| Synthetic and naturally occurring hormones | Progesterone, testosterone, cortisol, oestrone |

Representative EDCs from diverse functional use categories. EDC=endocrine-disrupting chemical. DBP=dibutyl phthalate. DEET=N,N-diethyl-m-toluamide. DDT=dichlorodiphenyltrichloroethane. BPA=bisphenol A. PCB=polychlorinated biphenyl. PBDE=polybrominated diphenyl ether.

***Table 1:*** **List of representative EDCs in use**

## Endocrine-Disrupting Chemicals: An Endocrine Society Scientific Statement

Evanthia Diamanti-Kandarakis, Jean-Pierre Bourguignon, Linda C. Giudice, Russ Hauser, Gail S. Prins, Ana M. Soto, R. Thomas Zoeller, and Andrea C. Gore

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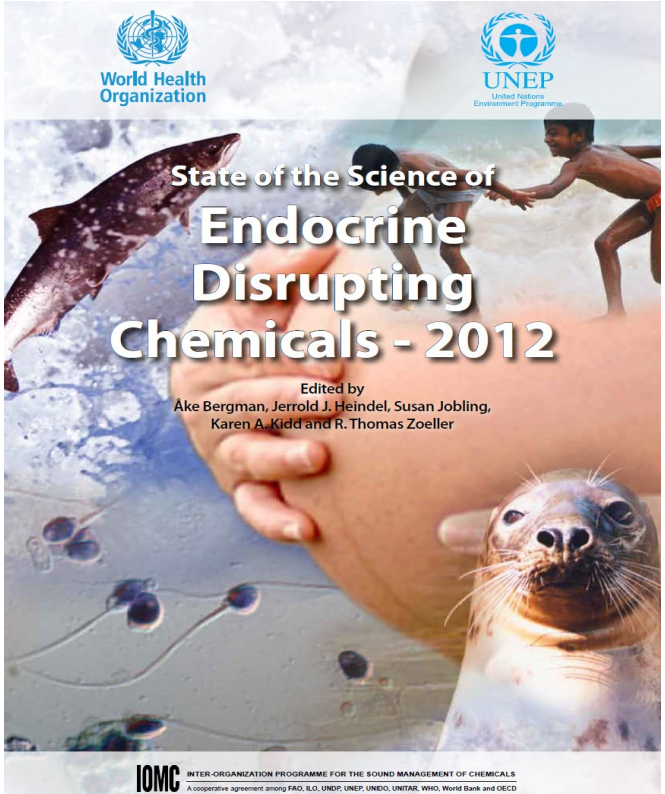
There is growing interest in the possible health threat posed by endocrine-disrupting chemicals (EDCs), which are substances in our environment, food, and consumer products that interfere with hormone biosynthesis, metabolism, or action resulting in a deviation from normal homeostatic control or reproduction. In this first Scientific Statement of The Endocrine Society, we present the evidence that endocrine disruptors have effects on male and female reproduction, breast development and cancer, prostate cancer, neuroendocrinology, thyroid, metabolism and obesity, and cardiovascular endocrinology. Results from animal models, human clinical observations, and epidemiological studies converge to implicate EDCs as a significant concern to public health. The mechanisms of EDCs involve divergent pathways including (but not limited to) estrogenic, antiandrogenic, thyroid, peroxisome proliferator-activated receptor γ, retinoid, and actions through other nuclear receptors; steroidogenic enzymes; neurotransmitter receptors and systems; and many other pathways that are highly conserved in wildlife and humans, and which can be modeled in laboratory *in vitro* and *in vivo* models. Furthermore, EDCs represent a broad class of molecules such as organochlorinated pesticides and industrial chemicals, plastics and plasticizers, fuels, and many other chemicals that are present in the environment or are in widespread use. We make a number of recommendations to increase understanding of effects of EDCs, including enhancing increased basic and clinical research, invoking the precautionary principle, and advocating involvement of individual and scientific society stakeholders in communicating and implementing changes in public policy and awareness. (***Endocrine Reviews*** **30: 293–342, 2009)**



# Response to WHO/UNEP Report

WHO/UNEP report (2012) “welcomed” by all participant countries at 2015 Strategic Alliance for International Chemicals Management


## 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

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The Endocrine Society’s first Scientific Statement in 2009 provided a wake-up call to the scientific community about how environmental endocrine-disrupting chemicals (EDCs) affect health and disease. Five years later, a substantially larger body of literature has solidified our understanding of plausible mechanisms underlying EDC actions and how exposures in animals and humans—especially during development—may lay the foundations for disease later in life. At this point in history, we have much stronger knowledge about how EDCs alter gene-environment interactions via physiological, cellular, molecular, and epigenetic changes, thereby producing effects in exposed individuals as well as their descendants. Causal links between exposure and manifestation of disease are substantiated by experimental animal models and are consistent with correlative epidemiological data in humans. There are several caveats because differences in how experimental animal work is conducted can lead to difficulties in drawing broad conclusions, and we must continue to be cautious about inferring causality in humans. In this second Scientific Statement, we reviewed the literature on a subset of topics for which the translational evidence is strongest: 1) obesity and diabetes; 2) female reproduction; 3) male reproduction; 4) hormone-sensitive cancers in females; 5) prostate; 6) thyroid; and 7) neurodevelopment and neuroendocrine systems. Our inclusion criteria for studies were those conducted predominantly in the past 5 years deemed to be of high quality based on appropriate negative and positive control groups or populations, adequate sample size and experimental design, and mammalian animal studies with exposure levels in a range that was relevant to humans. We also focused on studies using the developmental origins of health and disease model. No report was excluded based on a positive or negative effect of the EDC exposure. The bulk of the results across the board strengthen the evidence for endocrine health-related actions of EDCs. Based on this much more complete understanding of the endocrine principles by which EDCs act, including nonmonotonic dose-responses, low-dose effects, and developmental vulnerability, these findings can be much better translated to human health. Armed with this information, researchers, physicians, and other healthcare providers can guide regulators and policymakers as they make responsible decisions. (***Endocrine Reviews*** **36: 0000–0000, 2015)**

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# This is mainstream science.


POLICY STATEMENT Organizational Principles to Guide and Define the Child Health Care System and/or Improve the Health of all Children

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™

## Food Additives and Child Health

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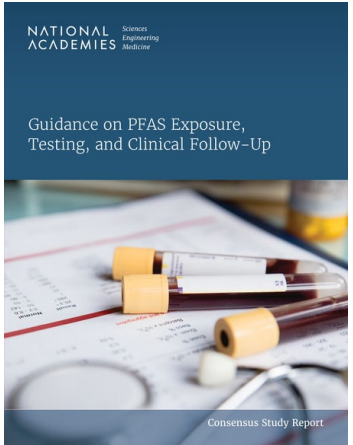
COUNCIL ON ENVIRONMENTAL HEALTH

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ETIOLOGY AND PATHOPHYSIOLOGY

OBESITY Reviews WILEY

## Endocrine-disrupting chemicals and obesity risk: A review of recommendations for obesity prevention policies

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**Summary**

Emerging evidence indicates that industrially produced endocrine-disrupting chemicals (EDCs) may be as obesogenic as poor dietary patterns and should be considered in obesity prevention policies. The authors conducted two reviews: (a) a systematic search of four electronic databases for papers published since January 2010 to identify the policy recommendations contained in scientific reviews of EDC exposure and obesity risk and (b) a narrative review of obesity policy documents published since January 2012 to identify the recommendations of national and international agencies. A search of four electronic databases found 63 scientific reviews with policy recommendations, of which 26 suggested individual responsibility to avoid exposure, 11 suggested medical interventions to counter the effects of exposure, and 42 suggested regulatory control of hazardous chemicals. Of sixty policy documents examined, six mentioned pollutants as a possible risk factor for obesity, and only one made explicit reference to strategies for reducing exposure to EDCs. The UN Sustainable Development Goals include targets to prevent ill health from hazardous chemicals (Targets 3.9 and 12.4) and to remove unsafe industrial chemicals from the environment (Targets 6.3, 11.6, 12.4, and 14.1). The authors suggest these should be explicitly linked to World Health Assembly targets to halt the rise in obesity.

# Plastics are a crucial source of EDCs

Bisphenols (polycarbonate plastics, aluminum can linings)

Phthalates (food packaging)

Per- and polyfluoroalkyl substances (PFAS, nonstick cooking and fluoropolymer plastics)

Brominated and organophosphorus flame retardants (additives to reduced flammability)

Burning plastics → dioxins

# Endocrine disruption and the developing brain

Thyroid hormone has long been known to be critical to early brain development

- Predictable outcomes of its disruption: IQ deficits, autism spectrum disorder (ASD), and attention-deficit hyperactivity disorder (ADHD).

- Brominated flame retardants have chemical structure similar to thyroid hormone itself (may directly inhibit its function and reduce its production)

# Brominated flame retardants

Four well-designed longitudinal birth cohorts have examined PBDE effects on child neurodevelopment

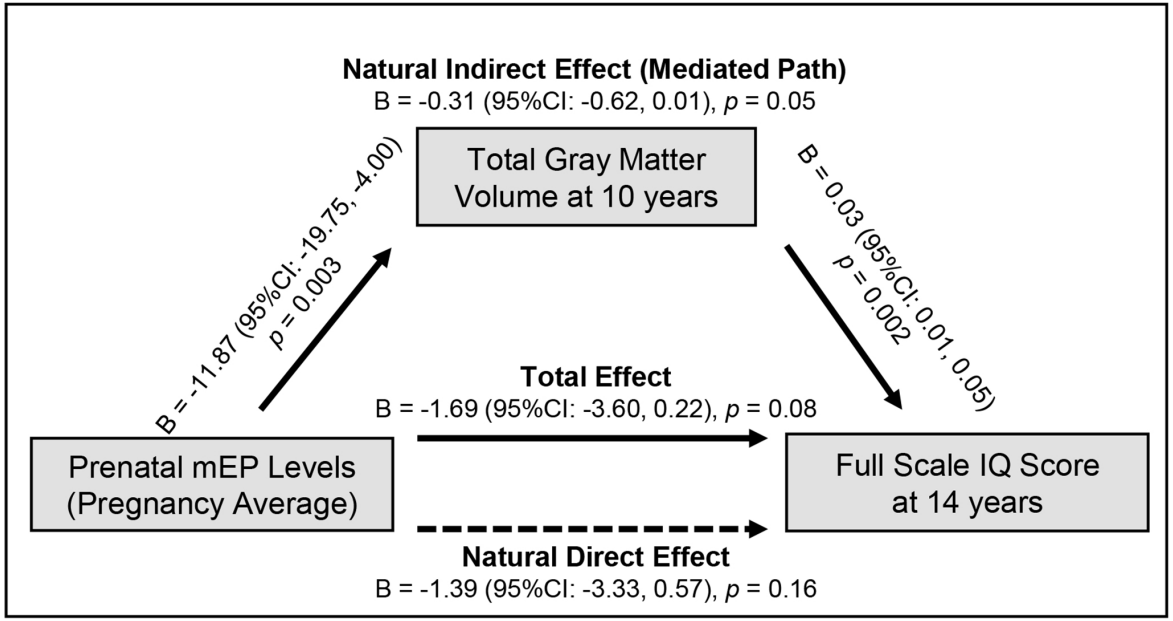
- Three (all US) identified consistent, exposure-response relationships with IQ, with carefully collected data on many potential confounders.

Chen et al EHP 2014; Eskenazi et al EHP 2013; Herbstman et al EHP 2010

- Fourth (from Spain), despite modest sample size with few detectable PBDE levels, showed substantial directionality towards cognitive and motor dysfunction at age 4.

Gascon et al Environment International 2011

# Reductions in total gray matter explain lower IQs due to phthalates




Ghassabian et al Mol Psychiatry. 2023 Nov; 28(11): 4814–4822.

# Chemical exposure and thyroid function in pregnancy

| | Free T4 (ng/dL) beta [95% CI] | Total T4 (ng/dL) beta [95% CI] | Free T4/Free T3 (pg/mL) beta [95% CI] | TSH (mU/L) beta [95% CI] | TSH/Free T4 (ng/dL) OR [95% CI] |
|---|---|---|---|---|---|
| ∑Bisphenols | -0.01 [-0.02, 0.00] | -0.16 [-0.32, -0.00]* | 0.00 [-0.00, 0.00] | 0.05 [-0.03, 0.13] | 0.06 [-0.03, 0.14] |
| OP pesticides ∑DM | 0.01 [-0.01, 0.02] | 0.12 [-0.06, 0.29] | 0.00 [-0.00, 0.01] | -0.08 [-0.16, 0.01] | -0.12 [-0.23, -0.01]* |
| OP pesticides ∑DE | 0.01 [-0.01, 0.02] | 0.06 [-0.12, 0.23] | 0.00 [-0.00, 0.01] | -0.08 [-0.16, 0.01] | -0.13 [-0.24, -0.02]* |
| OP pesticides ∑DAP | 0.01 [-0.01, 0.02] | 0.14 [-0.04, 0.31] | 0.01 [0.00, 0.01]* | -0.09 [-0.18, -0.00]* | -0.15 [-0.26, -0.04]* |
| ∑PAHs | -0.01 [-0.02, 0.01] | 0.04 [-0.14, 0.22] | 0.00 [-0.01, 0.01] | -0.03 [-0.12, 0.06] | -0.03 [-0.14, 0.09] |
| Phthalates ∑LMW | -0.01 [-0.02, 0.00] | -0.08 [-0.24, 0.08] | -0.00 [-0.01, 0.00] | -0.01 [-0.09, 0.07] | -0.02 [-0.11, 0.06] |
| Phthalates ∑HMW | -0.02 [-0.03, -0.00]* | -0.22 [-0.38, -0.06]* | -0.00 [-0.01, 0.00] | -0.01 [-0.09, 0.06] | 0.00 [-0.08, 0.09] |
| Phthalates ∑DEHP | -0.02 [-0.03, -0.01]* | -0.32 [-0.48, -0.16]* | -0.00 [-0.01, 0.00] | -0.04 [-0.11, 0.04] | -0.01 [-0.10, 0.08] |
| Phthalates ∑DnOP | -0.02 [-0.03, -0.00]* | -0.23 [-0.39, -0.08]* | -0.00 [-0.01, 0.00] | 0.07 [-0.01, 0.15] | 0.09 [0.01, 0.18]* |
| Phthalates ∑DiNP | -0.00 [-0.02, 0.01] | -0.03 [-0.19, 0.12] | -0.00 [-0.01, 0.00] | 0.03 [-0.04, 0.11] | 0.05 [-0.04, 0.13] |

Models were adjusted for age, educational levels, race and ethnicity, parity, insurance type, environmental exposure to tobacco, depressive symptoms, and gestational age at the time of thyroid measurement

# Phthalates and preterm birth

Phthalic acid, diisodecyl phthalate (DiDP), di-n-octyl phthalate (DnOP), and diisononyl phthalate (DiNP) were most strongly associated with gestational age, birth length, and birthweight, especially compared with DEHP or other metabolite groupings.

Although DEHP was associated with preterm birth (odds ratio 1·45 [95% CI 1·05–2·01]), the risks per $\log_{10}$ increase were higher for phthalic acid (2·71 [1·91–3·83]), DiNP (2·25 [1·67–3·00]), DiDP (1·69 [1·25–2·28]), and DnOP (2·90 [1·96–4·23]).

We estimated 56 595 (sensitivity analyses 24 003–120 116) phthalate-attributable preterm birth cases in 2018 with associated costs of US$3·84 billion (sensitivity analysis 1·63– 8·14 billion).



Trasande et al Lancet Planet Health
. 2024 Feb;8(2):e74-e85. doi: 10.1016/S2542-5196(23)00270-X.

# Phthalates and bisphenols in Generation R First: fetal and postnatal growth

Pregnancy-averaged phthalic acid (PA, end metabolite of all phthalates; -0.08 SD: 95% CI -0.14, -0.02) and LMW (-0.09 SD: 95% CI -0.16, -0.02): lower fetal weight through 40 weeks postpartum.

First trimester BPS: decreased bone mineral density (6.13 mg/cm$^2$; 95% CI: -10.02, -2.23) and content (0.12 g, 95% CI: -0.20, -0.04) at age 10 (no sex specific differences, endured multiple testing). (van Zwol-Janssens et al Env Res 2020)

# Phthalates and bisphenols in Generation R First: fat mass, insulin resistance and BP

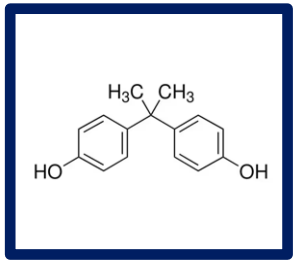
First trimester PA: 0.13 (95% CI: 0.04, 0.21) SD deviation score increase in pericardial fat index (stronger among boys, significant after multiple comparison). (Sol et al Int J Obesity 2020)

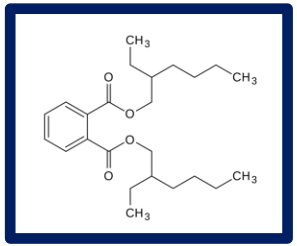
Second trimester maternal urine total bisphenol and BPA: higher systolic BP (0.13; 95% CI 0.03, 0.23 and 0.14; 95% CI 0.04; 0.23) among boys.

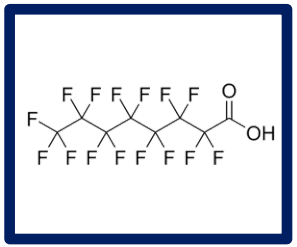
Third trimester PA: 0.20 (95% CI 0.07, 0.34) SD higher triglycerides among boys at age 10.

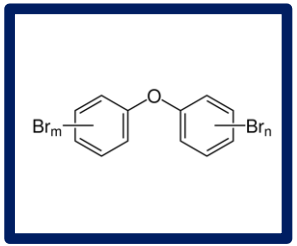
Some opposite (beneficial) sexually-dimorphic effects (Sol et al Env Int 2020)

# Plastics: Diabetes Impact



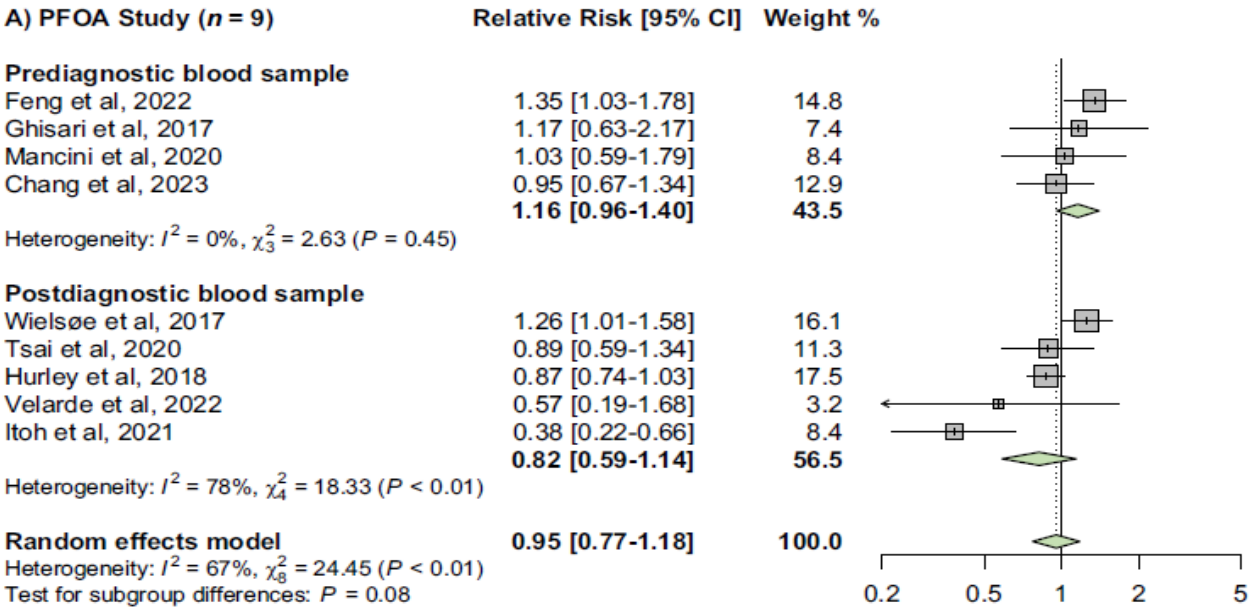




**Recent Meta-Analyses:**

- **Bisphenol A: OR of 1.28 (95%CI: 1.14-1.44) for T2DM among those with highest vs. lowest exposure.** (Hwang et al., *BMC Endo Dis*, 2018)
- **Phthalates: OR of 2.15 (95%CI: 1.18-4.85) for diabetes for the summation of DEHP metabolites.** (Zhang et al., *Environ Res*, 2022)
- **PFAS: OR of 1.10 (95%CI: 1.01-1.19) for GDM.** (Yao et al., *Chemosphere*, 2023)
- **PBDE: OR of 1.32 (95%CI: 1.15-1.53) for GDM.** (Yan et al., *Environ Health*, 2022)
- **Dioxins: OR of 1.91 (95%CI: 1.44-2.54) for T2DM.** (Song et al., *J Diabetes*, 2016)

# PFAS and Breast Cancer




Chang CJ et al. Exposure to per- and polyfluoroalkyl substances and breast cancer risk: a systematic review and meta-analysis of epidemiologic studies. AJE 2024

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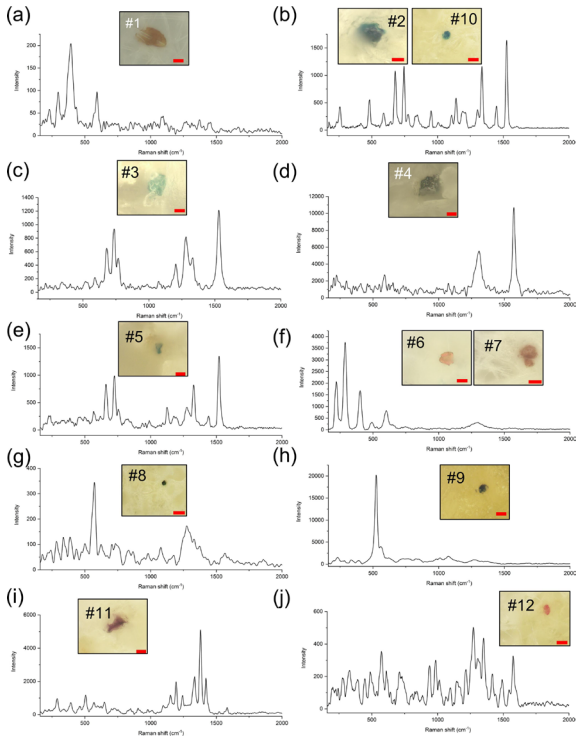
# Microplastics in humans

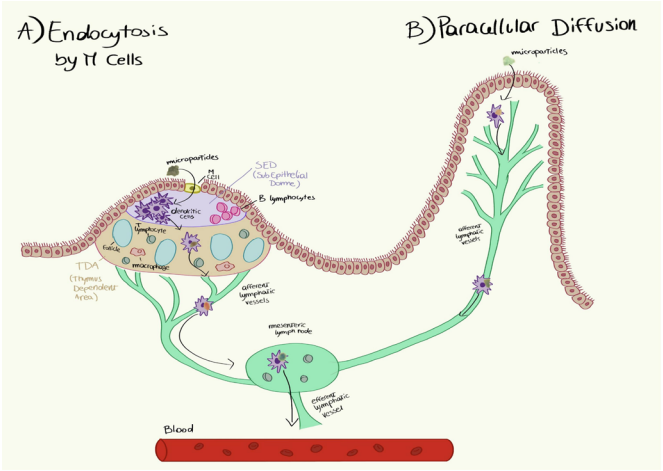
Widely detected in human samples to date

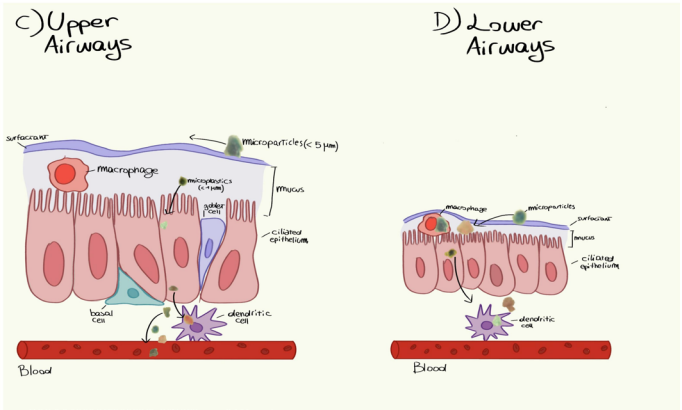
One study of adults and kids found polyethylene and polycarbonate microplastics in most (PET) or all (PC) adult stool samples but at concentrations an order of magnitude lower than in infants for PET MPs (<LOQ–16,000 ng/g, median, 2600 ng/g).

The estimated mean daily exposures from the diet of infants to PET and PC MPs were 83,000 and 860 ng/kg body weight per day, respectively, which were significantly higher than those of adults (PET: 5800 ng/kg-bw/day; PC: 200 ng/kg-bw/d).

# Plasticenta



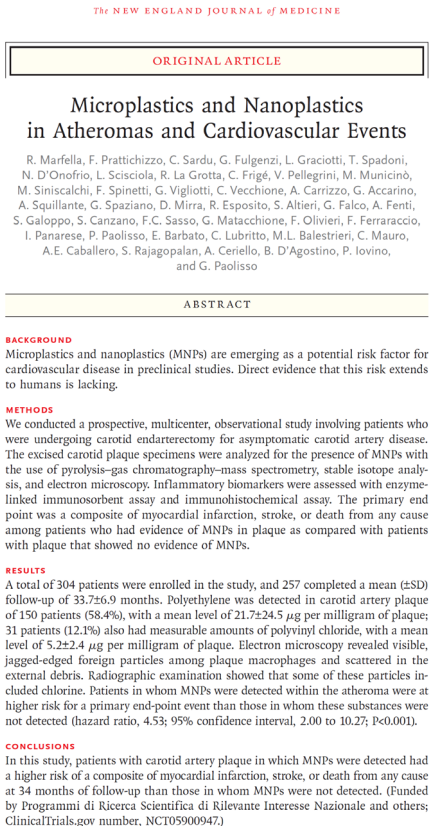

Ragusa et al 2021 Jan:146:106274. doi: 10.1016/j.envint.2020.106274. Epub 2020 Dec 2.

# What do we know about microplastics and human health?

Early findings raise serious concerns

Cautions

Particle size
Assays not harmonized yet
Chemicals on lining of microplastics may vary



The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

## Microplastics and Nanoplastics in Atheromas and Cardiovascular Events

R. Marfella, F. Prattichizzo, C. Sardu, G. Fulgenzi, L. Graciotti, T. Spadoni, N. D'Onofrio, L. Scisciola, R. La Grotta, C. Frigé, V. Pellegrini, M. Municinò, M. Siniscalchi, F. Spinetti, G. Vigliotti, C. Vecchione, A. Carrizzo, G. Accarino, A. Squillante, G. Spaziano, D. Mirra, R. Esposito, S. Altieri, G. Falco, A. Fenti, S. Galoppo, S. Canzano, F.C. Sasso, G. Matacchione, F. Olivieri, F. Ferraraccio, I. Panarese, P. Paolisso, E. Barbato, C. Lubritto, M.L. Balestrieri, C. Mauro, A.E. Caballero, S. Rajagopalan, A. Ceriello, B. D'Agostino, P. Iovino, and G. Paolisso

ABSTRACT

BACKGROUND
Microplastics and nanoplastics (MNPs) are emerging as a potential risk factor for cardiovascular disease in preclinical studies. Direct evidence that this risk extends to humans is lacking.

METHODS
We conducted a prospective, multicenter, observational study involving patients who were undergoing carotid endarterectomy for asymptomatic carotid artery disease. The excised carotid plaque specimens were analyzed for the presence of MNPs with the use of pyrolysis–gas chromatography–mass spectrometry, stable isotope analysis, and electron microscopy. Inflammatory biomarkers were assessed with enzyme-linked immunosorbent assay and immunohistochemical assay. The primary end point was a composite of myocardial infarction, stroke, or death from any cause among patients who had evidence of MNPs in plaque as compared with patients with plaque that showed no evidence of MNPs.

RESULTS
A total of 304 patients were enrolled in the study, and 257 completed a mean (±SD) follow-up of 33.7±6.9 months. Polyethylene was detected in carotid artery plaque of 150 patients (58.4%), with a mean level of 21.7±24.5 μg per milligram of plaque; 31 patients (12.1%) also had measurable amounts of polyvinyl chloride, with a mean level of 5.2±2.4 μg per milligram of plaque. Electron microscopy revealed visible, jagged-edged foreign particles among plaque macrophages and scattered in the external debris. Radiographic examination showed that some of these particles included chlorine. Patients in whom MNPs were detected within the atheroma were at higher risk for a primary end-point event than those in whom these substances were not detected (hazard ratio, 4.53; 95% confidence interval, 2.00 to 10.27; P<0.001).

CONCLUSIONS
In this study, patients with carotid artery plaque in which MNPs were detected had a higher risk of a composite of myocardial infarction, stroke, or death from any cause at 34 months of follow-up than those in whom MNPs were not detected. (Funded by Programmi di Ricerca Scientifica di Rilevante Interesse Nazionale and others; ClinicalTrials.gov number, NCT05900947.)

N Engl J Med 2024;390:900-10.
DOI: 10.1056/NEJMoa2309822

# BPA → cardiovascular mortality

BPA associated with:

- increased carotid intima-media thickness of 12-30 and >70 year olds

  Lin et al Atherosclerosis 2015, Lind et al Atherosclerosis 2011)

- BPA associated with severity of coronary artery disease in angiography

  Melzer et al PLOS One 2012

- Reduced heart rate variability in adults

  Bae et al Hypertension 2012

- All-cause mortality, and cardiovascular disease mortality

  Bao et al JAMA Network Open 2020

# Phthalates → cardiovascular mortality

Low T either predictor of or marker of cardiovascular mortality in adult men

High molecular weight phthalates were associated with lower total, free, and bioavailable testosterone among men age ≥60.

Attina et al Lancet Diab Endo 2016; Hauser et al JCEM 2015

Cardiovascular mortality was significantly increased in relation to a prominent DEHP metabolite, mono-(2-ethyl-5-oxohexyl)phthalate. Extrapolating to the population of 55-64 year old Americans, 50,200 attributable deaths and $23.4 billion in lost economic productivity.

Trasande et al Env Pollution 2021

# PFAS restrict fetal growth and increase childhood obesity

Meta-analysis of 24 studies: −10·5 g (95% CI: −16·7, −4·4) birth weight per ng/ml increase in maternal or cord blood PFOA

Steenland et al Epidemiology 2018

A later study including newer cohorts from the ECHO consortium still confirmed the findings, with multiple PFAS associated with lower birthweight-for-gestational-age Z-score, and a failure of perceived stress to modify the observed associations.

Padula et al EHP 2024

Meta-analysis of 10 studies: 25% increase in childhood overweight and 0.10 unit increase in BMI z-score per ng/mL PFOA in maternal blood

Liu et al 2018

# PFAS contribute to adult weight gain/diabetes

Diabetes Prevention Program lifestyle intervention trial:

- Total PFAS were associated with increased weight gain exclusively among the control group.

Cardenas et al 2018

Follow-up of the successful POUNDS LOST trial:

- Perfluorooctane sulfonate (PFOS) and perfluorononanoic acid (PFNA), were associated with reductions in resting metabolic rate.

Liu et al 2018

PIVUS (Sweden), Nurses (US), DPPOS (US):

- PFAS associated with incident diabetes

Cardenas et al 2019, Sun et al 2018, Lind et al 2014

# EDCs in plastic reduce fertility

Fertility is a condition of a couple, where reproductive health of both sexes plays a role

Louis et al 2013

Fetal exposure to phthalates with reduced infant anogenital distance (AGD)

- Shortened adult AGD is associated with reduced semen quality and testosterone level

Swan et al EHP 2005, Bornehag et al EHP 2014

Multiple studies have identified reduced male fertility and poor semen quality with multiple EDCs, including phthalates, bisphenol A, and polyfluorinated chemicals

Juul et al Nat Rev Endo 2014

# What is the burden of disease burden and are the health costs due to plastics?

Expert panels identified conditions where the evidence is strongest for causation due to EDCs

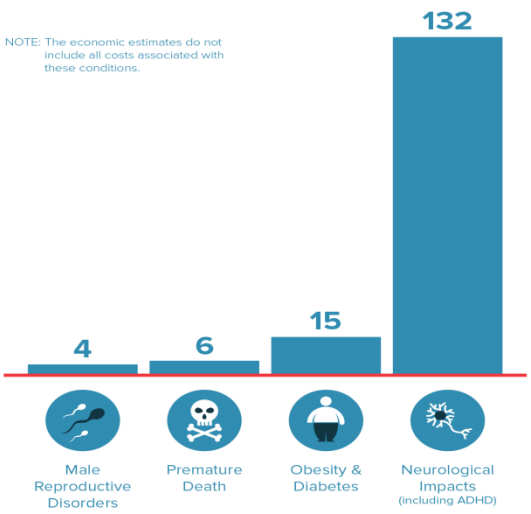
- Developed ranges for fractions of disease burden that can be attributed for EDCs
- Adapted GRADE Working Group and WHO criteria for evaluating epidemiologic evidence
- Adapted IPCC approach to integrate epidemiology and toxicology evidence and estimate probability of causation

Trasande et al J Clin Endo Metab 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




**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

## €157B Cost by EDC Type


**SOME EDCs NOT INCLUDED:**

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

NYU School of Medicine
NYU LANGONE MEDICAL CENTER

**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.


See Trasande et al. The Journal of Clinical Endocrinology & Metabolism
**http://press.endocrine.org/edc**

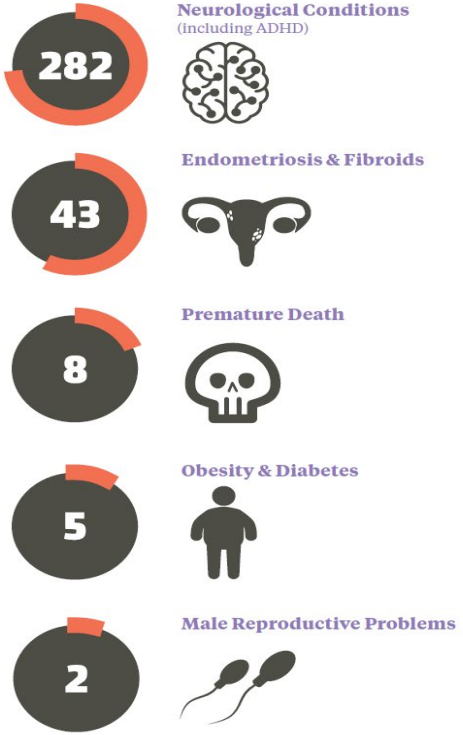

HASSENFELD CHILDREN'S HOSPITAL OF NEW YORK AT NYU LANGONE

## Health Effects From Endocrine Disrupting Chemicals Cost The U.S.

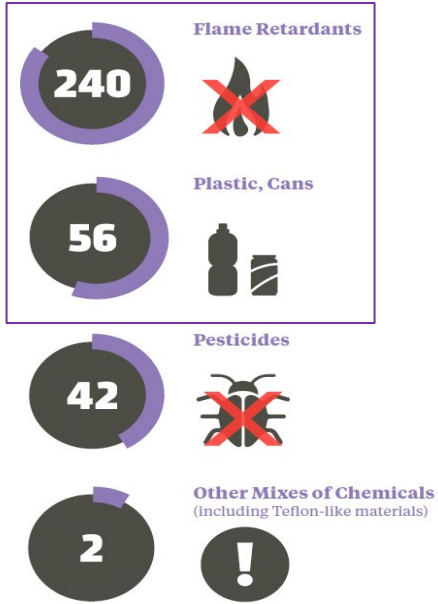
**$340 Billion Annually**

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

### $340 Billion by Health Effect




### $340 Billion by EDC Type




Attina et al Lancet Diab Endo 2016

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# How much disease burden is related to plastic?

- 97.5% for bisphenol A (96.25-98.75% for sensitivity analysis)
- 98% (96%-99%) for di-2-ethylhexylphthalate
- 100% (71%-100%) for butyl phthalates and benzyl phthalates
- 98% (97%-99%) for PBDE-47
- 93% (16%-96%) for PFAS



## Chemicals Used in Plastic Materials: An Estimate of the Attributable Disease Burden and Costs in the United States

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**Abstract**

**Context:** Chemicals used in plastics have been described to contribute to disease and disability, but attributable fractions have not been quantified to assess specific contributions. Without this information, interventions proposed as part of the Global Plastics Treaty cannot be evaluated for potential benefits.

**Objective:** To accurately inform the tradeoffs involved in the ongoing reliance on plastic production as a source of economic productivity in the United States, we calculated the attributable disease burden and cost due to chemicals used in plastic materials in 2018.

**Methods:** We first analyzed the existing literature to identify plastic-related fractions (PRF) of disease and disability for specific polybrominated diphenylethers (PBDE), phthalates, bisphenols, and polyfluoroalkyl substances and perfluoroalkyl substances (PFAS). We then updated previously published disease burden and cost estimates for these chemicals in the United States to 2018. By uniting these data, we computed estimates of attributable disease burden and costs due to plastics in the United States.

**Results:** We identified PRFs of 97.5% for bisphenol A (96.25-98.75% for sensitivity analysis), 98% (96%-99%) for di-2-ethylhexylphthalate, 100% (71%-100%) for butyl phthalates and benzyl phthalates, 98% (97%-99%) for PBDE-47, and 93% (16%-96%) for PFAS. In total, we estimate $249 billion (sensitivity analysis: $226 billion-$289 billion) in plastic-attributable disease burden in 2018. The majority of these costs arose as a result of PBDE exposure, though $66.7 billion ($64.7 billion-67.3 billion) was due to phthalate exposure and $22.4 billion was due to PFAS exposure (sensitivity analysis: $3.85-$60.1 billion).

**Conclusion:** Plastics contribute substantially to disease and associated social costs in the United States, accounting for 1.22% of the gross domestic product. The costs of plastic pollution will continue to accumulate as long as exposures continue at current levels. Actions through the Global Plastics Treaty and other policy initiatives will reduce these costs in proportion to the actual reductions in chemical exposures achieved.

- In total, we estimate $249 billion (sensitivity analysis: $226 billion-$289 billion) in plastic-attributable disease burden in 2018.
- The majority of these costs arose as a result of PBDE exposure, though $66.7 billion ($64.7 billion-67.3 billion) was due to phthalate exposure and $22.4 billion was due to PFAS exposure (sensitivity analysis: $3.85-$60.1 billion).

Trasande et al J Endo Soc 2024

# Global costs of plastics

Likely similar as industrializing nations become predominant consumers and producers of chemicals as expected by OECD by 2030

Data from 70 countries suggest exposure to PFOA contributed to approximately 461,635 (95% CI: 57,418-854,645) cases per year of LBW during the past two decades

- Predominantly from Asian regions

Fan et al ES&T 2022

# Reduce, reuse or recycle?

Recycled plastics themselves present health threats.

Recycling itself is energy intensive and more expensive than creating plastic from fossil fuels.

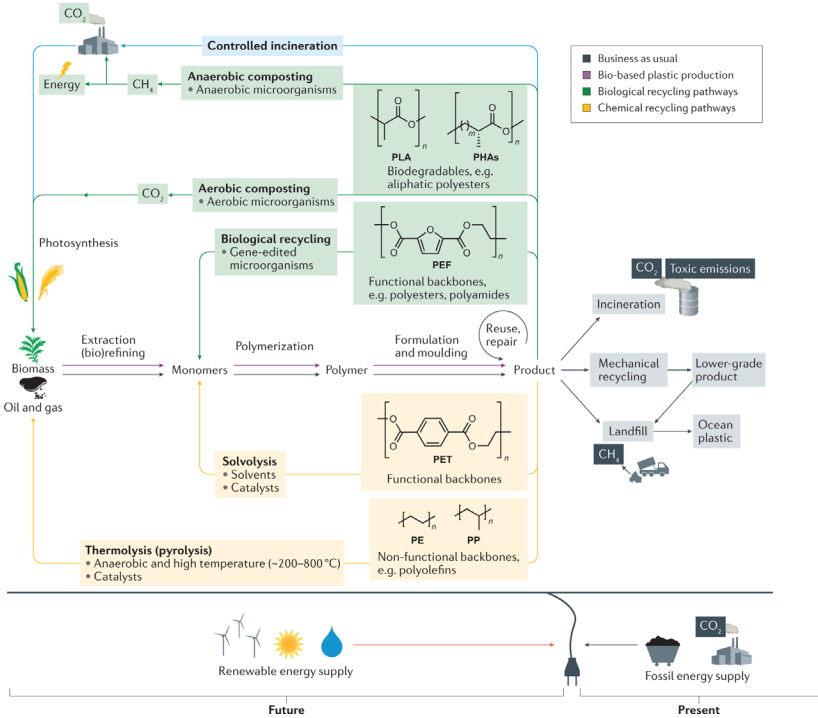
Recycled polyethylene plastic bottles also have been identified to contain higher levels of bisphenols, phthalates and metals than newly-produced bottles due to cross-contamination during disposal, collection and reprocessing.

# Bioplastics?

Plant- and other bio-based plastics have also been touted as a planet-friendly alternative and form of sustainable innovation.

However, they require high temperature to be recycled, and when the high costs force bioplastics into landfills, they produce methane, which is more potent than carbon dioxide in driving climate change.

Laboratory studies also suggest greater oxidative stress and antiandrogenicity of chemicals found in liquids obtained from bioplastic bottles.



# Independent science tells us that we need to:

Reduce plastic production

Recognize hazards posed by recycling and use of bioplastics

Uses hazard rather than risk to evaluate and remove endocrine disrupting chemicals from plastic

Expand biomonitoring globally

Establish an independent scientific body to evaluate hazards of endocrine disrupting chemicals

# Summary

There is clear and extensive evidence of the human health impacts of many chemicals in common plastics.

This isn't solely a waste problem – it's a use problem.  Exposures will continue to increase unless reductions in production and consumption are achieved (complemented by needed increases in recycling).

We need to pivot to essentiality rather than circularity.

# Thank you

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