

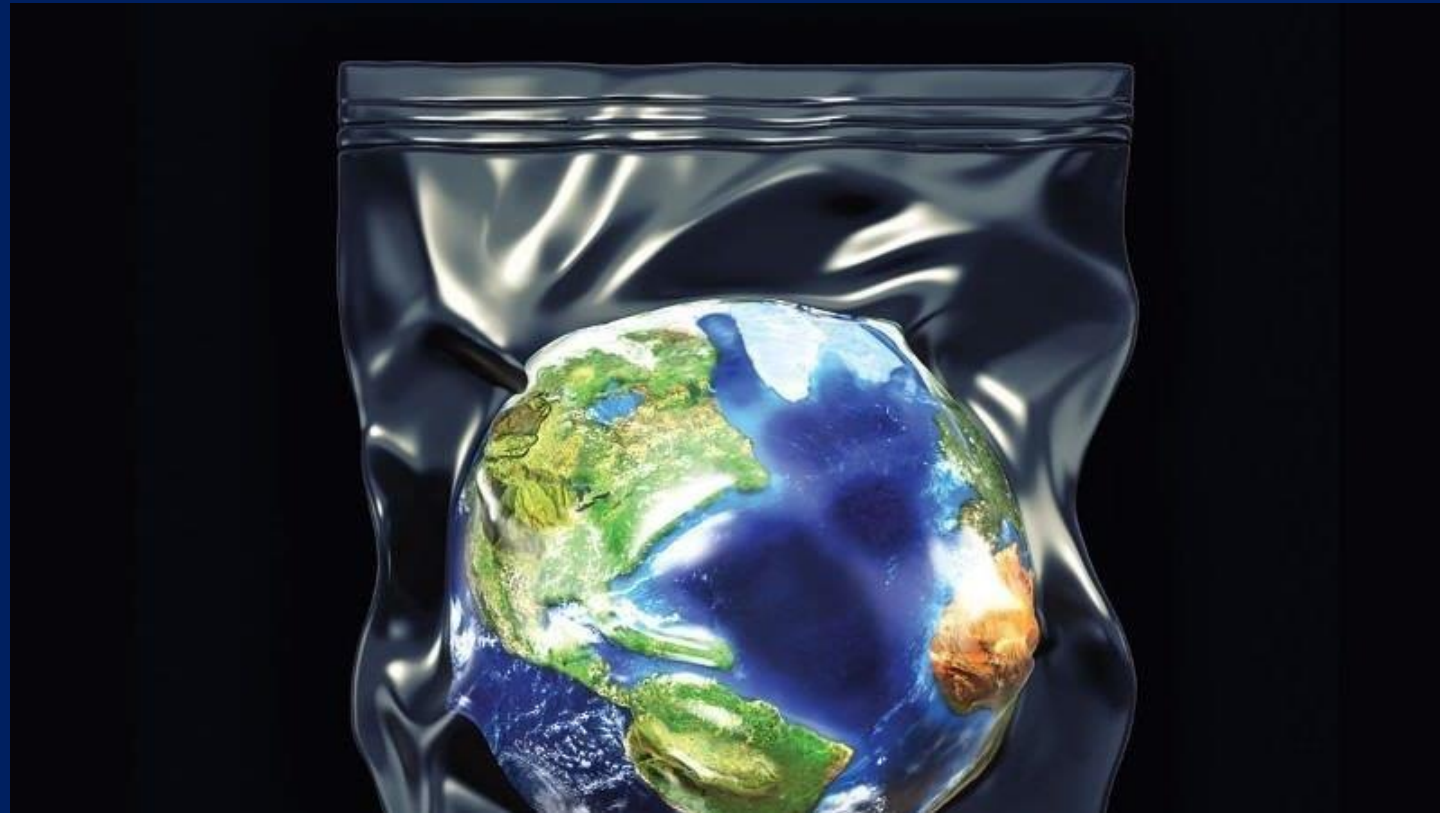
# Health Impacts of Plastic Pollution

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Plastics have created significant changes in everyday life. Currently, their risks have seemed to out way their benefits because of their misuse.



The global plastic production currently exceeds 320 million tonnes (Mt) per year, over 40% of which is used as single-use packaging, resulting in plastic waste. A substantial proportion of the plastic produced each year is lost to and persists in the marine environment, with an estimated accumulative potential of 250 Mt by 2025. (*Wright SL, Kelly FJ 2017*)



Asia accounts for more than 80% of the total leakage of plastics to the ocean. (World Economic Forum, 2016). Half of the land-based, plastic-waste leakage originates in five countries (China, Indonesia, Philippines Thailand, Vietnam) (*Ocean Conservancy & Mckinsey Center for Business and Environment, 2015*)

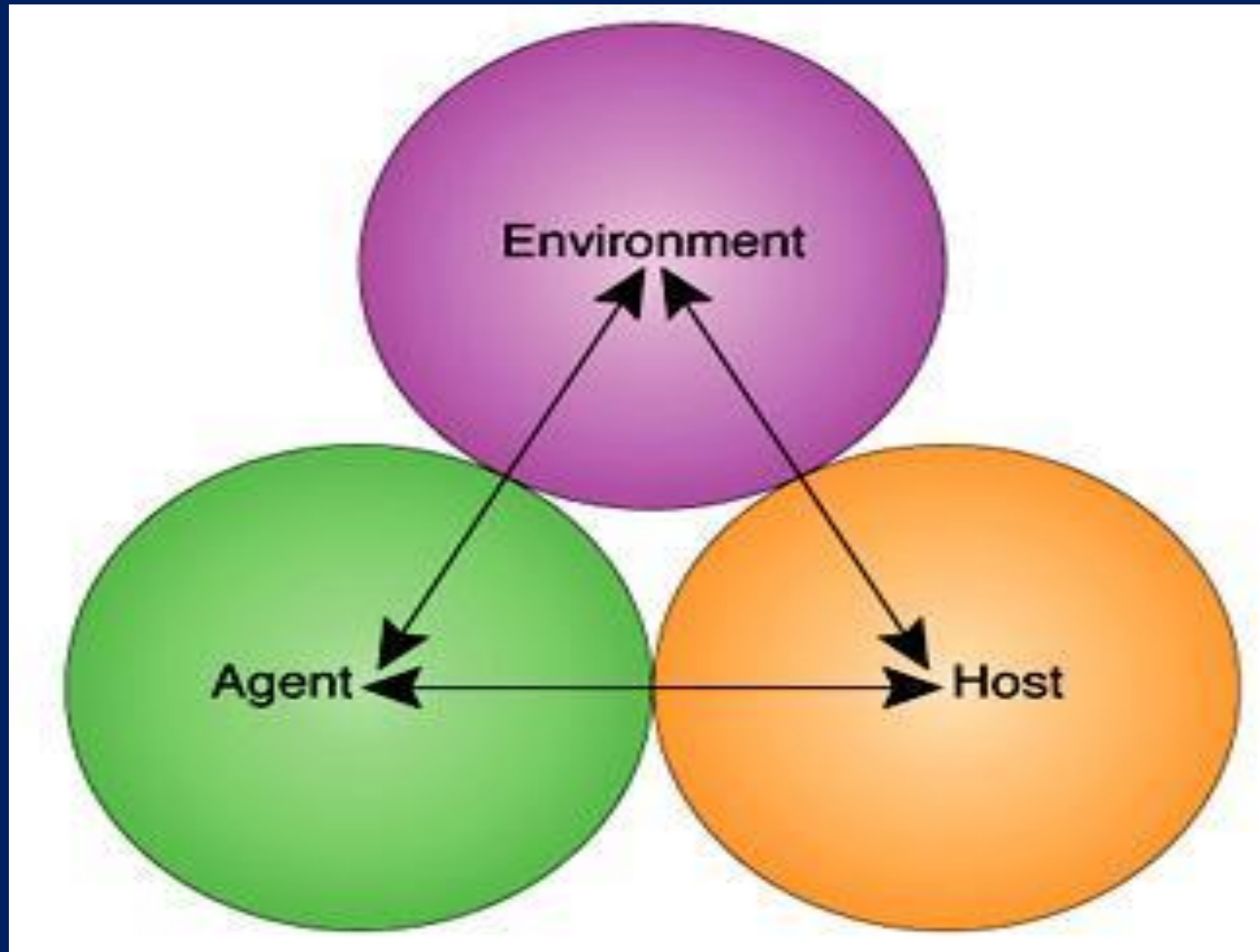
The background is a solid dark blue color. It is decorated with several realistic water droplets of various sizes, scattered across the top and bottom edges. The droplets have highlights and shadows, giving them a three-dimensional appearance.

**WHAT IS THE IMPACT OF PLASTIC  
POLLUTION TO HUMAN HEALTH?**

# OBJECTIVES

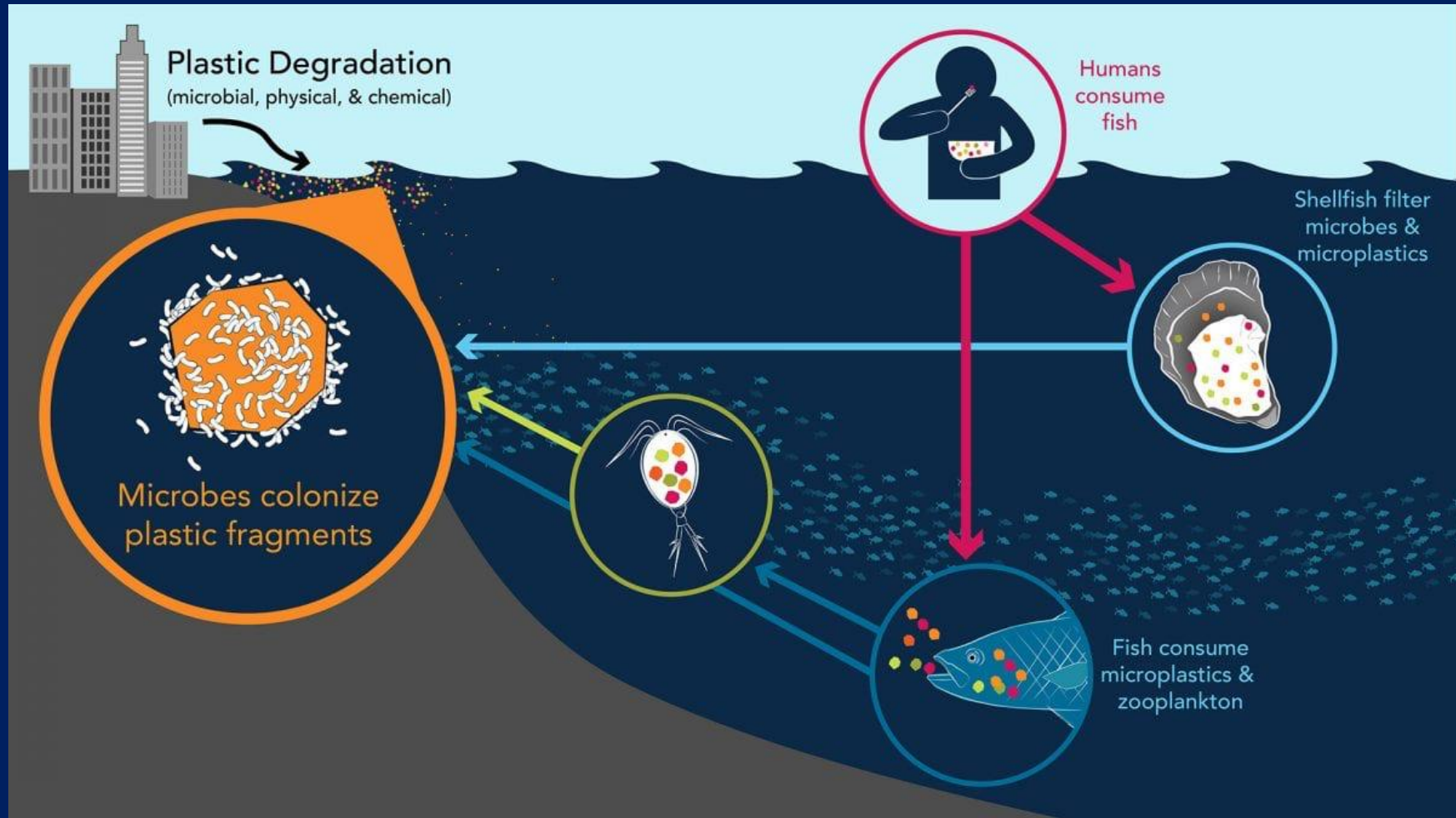
- To present available information regarding chemicals in plastics possessing toxic effects to humans
- To discuss data gaps that are needed in order to conduct proper risk assessment

# FACTORS AFFECTING HEALTH



1

# ENVIRONMENT



# ENVIRONMENT: ROUTE OF EXPOSURE (ORAL)

- Existing evidence (2014-2018 studies) regarding presence of microplastics in fish and shellfish.
- In European countries with high shellfish consumption, consumers ingest up to 11,000 microplastic particles (size range 5-1000  $\mu\text{m}$ ) per year. In countries with low shellfish consumption, consumers ingest an average of 1,800 microplastics per year (*Cauwenberghe and Janssen 2014*).
- Seafood species that are eaten whole pose greater threat to seafood contamination than for example gutted fish or peeled shrimp.



# ENVIRONMENT: ROUTE OF EXPOSURE (INHALATIONAL)

- A 2016 study (*Dris R, et al*) showed evidence for the **presence of microplastics in atmospheric fallout has been reported**. The total atmospheric fallout of microplastics was assessed in a densely populated urban area and a less-dense suburban area in Paris. The majority of particles observed were fibers, approximately 30% of which were confirmed plastic. Periods of heavy rainfall corresponded with some of the highest concentrations observed.
- Other exposures: sludge byproducts used for agricultural purposes
- Air pollution caused by the wearing out of rubber tires

2

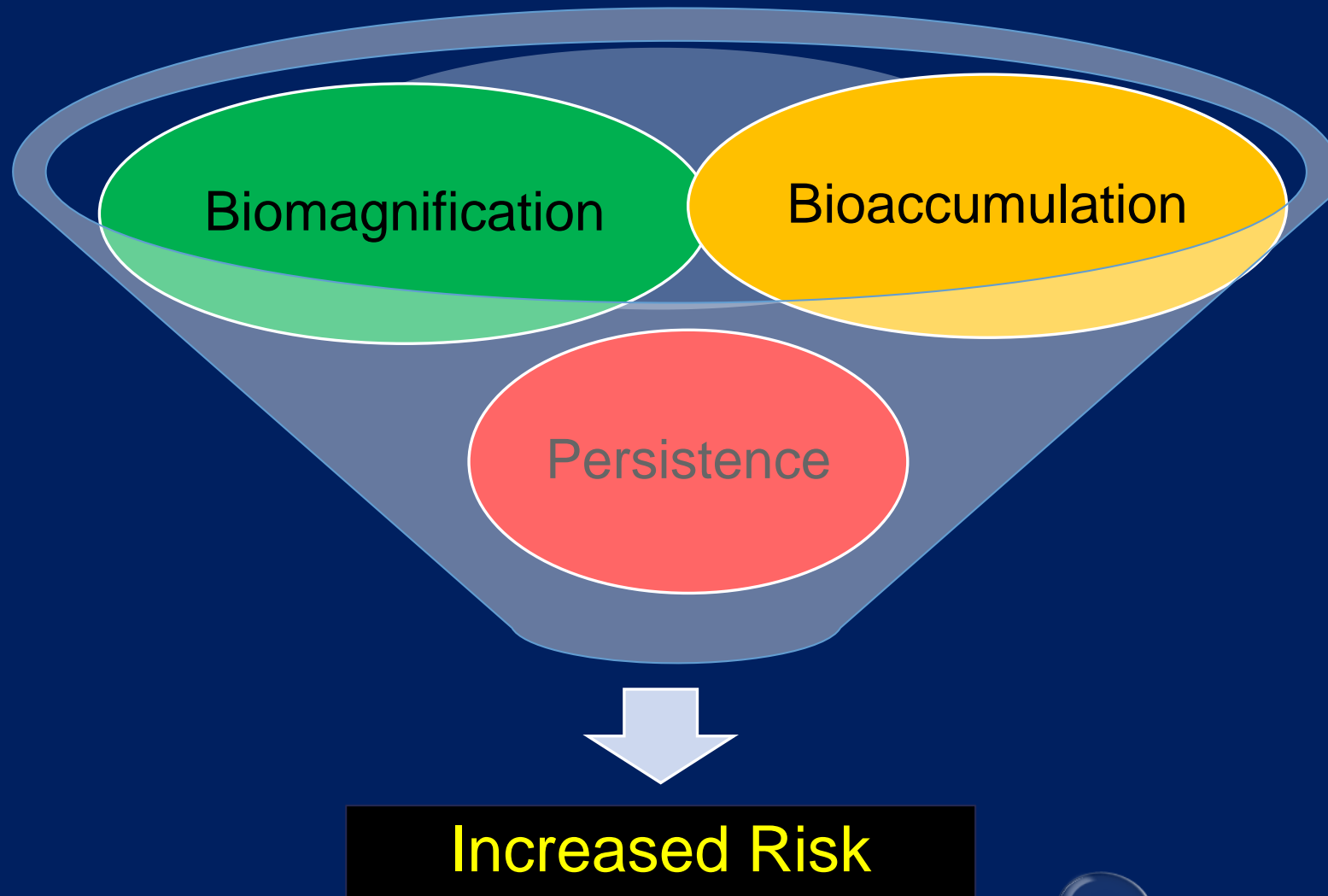
## AGENT

- Microplastics may act as vehicles for **chemicals**, including
  - ✓ Those intentionally added during their **manufacturing process** (**phthalates, styrene, bisphenol A**)
  - ✓ **Environmental contaminants** (because of their hydrophobic surface) that may be absorbed on to their surface during their use and permanence into the environment [polychlorinated biphenyls (PCB) and polycyclic aromatic hydrocarbons (PAHs), dichlorodiphenyltrichloroethane (DDTs), toxic metals (mercury, cadmium, zinc, nickel, lead)]
- **Microbes**: introduction of pathogens and vectors

# AGENT

- Introduction of pathogens and vectors
  - ✓ In the environment, the surface of microplastics becomes rapidly colonized by microbes
  - ✓ Well-developed biofilms establish on the surface of plastic after 7 days in water or sediment
  - ✓ Such biofilms significantly differ from the ambient environment and can include harmful human pathogens such as strains of *Vibrio spp.*

# Agent Characteristics that Increase the Risk ...



# AGENT

## CHEMICAL INTERACTIONS

- ❑ Several studies with marine organisms published in recent years have been showing that microplastics influence the toxicity (increasing, changing the type or the pattern of the effects) of a wide diversity of pollutants, such as polycyclic hydrocarbons, metals and pharmaceuticals.
- ❑ Moreover, temperature variation, especially temperature rise, has been found to influence such toxicological interactions.

# 3

## HOST

- Current 'safe' exposure levels are typically based on the application of traditional toxicological assumptions regarding acute toxicants to calculate daily exposures for chemicals in a range of widely used plastic items.
- The toxicological consequences of such exposures, especially for susceptible subpopulations such as **children and pregnant women**, remain **unclear and warrant further investigation**.
- However, there is evidence of associations between urinary concentrations of some phthalate metabolites and biological outcomes. For example, an inverse relationship has been reported between the concentrations of DEHP metabolites in the mother's urine and anogenital distance, penile width and testicular descent in male offspring.

# HEALTH EFFECTS

- If inhaled or ingested, plastics may accumulate and exert localized particle toxicity by **inducing or enhancing an immune response**.
- Exposure to plastic additives and other endocrine disruptors may cause altered **endocrine activity and reproductive development** through a number of biological mechanisms, which can **target different levels of the hypothalamic– pituitary – gonad/thyroid axis**, ranging from effects on hormone receptors to effects on hormone synthesis, secretion or metabolism.

# HEALTH EFFECTS: BISPHENOL A

- **Routes of exposure:** main exposure is through ingestion of foods and drinks contaminated with BPA from polycarbonate bottles and cans coated with epoxy resins; easily migrates into the food, beverages and into the environment
- Numerous reports indicate that chronic exposure to BPA, even at low doses, can lead to many adverse health effects, such as ischemic heart disease, diabetes, obesity, but also reductions in body and organ weight, as well as other disorders and behavioral abnormalities in children.



# HEALTH EFFECTS: BISPHENOL A

## Obesity:

There are reports in the literature about the problem of obesity in conjunction with BPA exposure in adults, but only few studies are accessible on BPA-related obesity in children

## Diabetes mellitus:

Clinical studies on humans and preclinical studies on in vivo, ex vivo, and in vitro models indicate that BPA, mostly at low doses, may have a role in increasing type 2 diabetes mellitus developmental risk, directly acting on pancreatic cells, in which BPA induces the impairment of insulin and glucagon secretion, triggers inhibition of cell growth and apoptosis, and acts on muscle, hepatic, and adipose cell function, triggering an insulin-resistant state.

# HEALTH EFFECTS: BISPHENOL A

## CARDIOVASCULAR EFFECT:

- ❑ Cross-sectional and longitudinal epidemiological studies
- ❑ Epidemiological studies have shown that higher urinary BPA concentration in humans is associated with various types of CV diseases, including angina, hypertension, heart attack and coronary and peripheral arterial disease.
- ❑ Higher BPA urinary levels associated with LDL and HDL Cholesterol levels
- ❑ Experimental studies suggest that both acute and chronic BPA exposure at environmentally-relevant “low-dose” could affect the physiological functioning of CV system and promote abnormal CV activities such as arrhythmias, cardiac remodeling, atherosclerosis, and altered blood pressure.

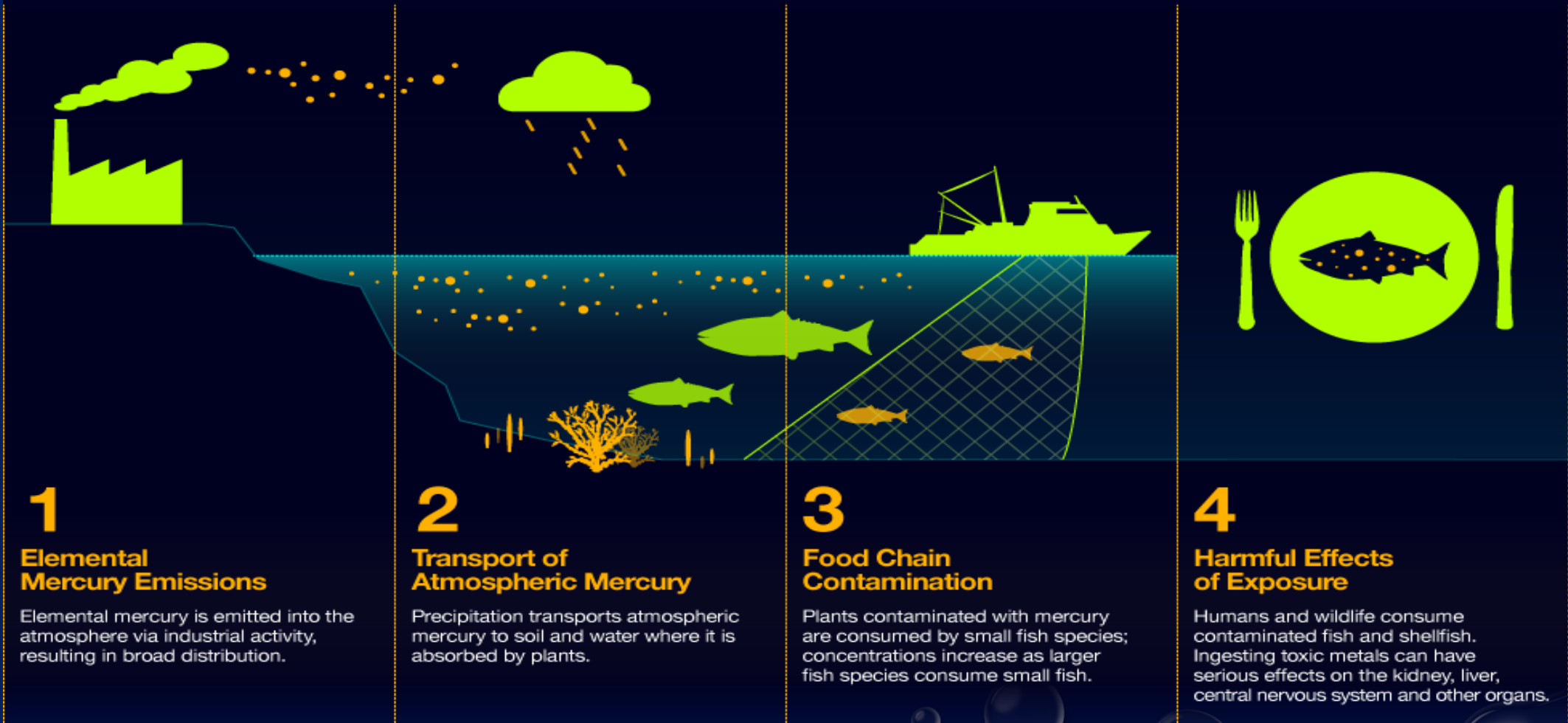
# HEALTH EFFECTS: BISPHENOL A

## CHILDREN'S DEVELOPMENT:

- Birth weight was significantly lower in the children of mothers exposed to BPA than those of the unexposed mothers. The same is true for the children whose fathers were exposed, compared to the children of unexposed fathers. However, the difference was not significant. Since findings from these studies suggest different data, exposure of pregnant women to BPA, especially in the workplace, should be limited. (*Miao et al, 2011*)
- Some studies also found a close relationship between urinary BPA, body weight, and early puberty, which can be explained by the obesogenic effect of BPA itself.

# HEALTH EFFECTS: MERCURY

## Mercury Pollution Cycle



**1**

### Elemental Mercury Emissions

Elemental mercury is emitted into the atmosphere via industrial activity, resulting in broad distribution.

**2**

### Transport of Atmospheric Mercury

Precipitation transports atmospheric mercury to soil and water where it is absorbed by plants.

**3**

### Food Chain Contamination

Plants contaminated with mercury are consumed by small fish species; concentrations increase as larger fish species consume small fish.

**4**

### Harmful Effects of Exposure

Humans and wildlife consume contaminated fish and shellfish. Ingesting toxic metals can have serious effects on the kidney, liver, central nervous system and other organs.

SOURCE:

U.S. Environmental Protection Agency

# HEALTH EFFECTS: MERCURY

- **Nervous system effects**

Tremors, anxiety, emotional lability, forgetfulness, insomnia, anorexia, erethism (abnormal irritation, sensitivity, or excitement), fatigue, and cognitive and motor dysfunction, polyneuropathy (paresthesias, stocking-glove sensory loss, hyperactive tendon reflexes, slowed sensory and motor nerve conduction velocities)

- **Renal impairment:** glomerulonephritis, nephrotic syndrome

- **Immunologic:** Delayed idiosyncratic non-allergic hypersensitivity

# HEALTH EFFECTS: MERCURY

- **Reproductive:** menstrual disturbances miscarriage, spontaneous abortions, stillbirth and low birth weight
- **Endocrine disruptor:** affects insulin, estrogen, testosterone and adrenaline, thyroid hormone

# HEALTH EFFECTS: MERCURY



A Japanese mother bathes her child, one of many such children born with severe birth defects in the town of Minamata due to the parents eating fish contaminated with mercury dumped into Minamata Bay by the Chisso plastics manufacturing company. Recent reports say that Minamata disease (mercury poisoning) is now showing up among Indian villagers in the Amazon due to gold miners using mercury to separate gold from ore.

Says Tsuginori Hamamoto, who grew up eating fish caught in Minamata Bay and now is confined to a wheelchair because of the ravages of mercury poisoning, "Because we have destroyed the environment, human beings have been destroyed. The cause of Minamata disease is industrial effluent from the (Chisso) factory. What lies behind this is the fact that everyone is seeking an easy life. Are you still going to continue to lead an easy life?"

[Photo by W. Eugene Smith]

Infants exposed prenatally to methylmercury in the Minamata Bay catastrophe manifested with:

- Decreased birth weight
- Decreased muscle tone
- Profound developmental delay
- Seizure disorders
- Deafness
- Blindness
- Severe spasticity

Source: <http://oecotextiles.wordpress.com/category/chemicals/mercury>

# HEALTH EFFECTS: MERCURY

- **Developmental effects:**

Although the results of published studies are mixed, a majority of the studies found associations between prenatal mercury exposure and at least one aspect of cognition relevant to language development.

Meta-analysis (*Axelrad and colleagues, 2007*) which combined data from the Seychelles, Faroe Islands, and New Zealand found a significant association as did the large ALSPAC study. Findings from the Faroe islands, Seychelles, and Hong Kong cohorts also suggest that there may be an association between prenatal mercury exposure and verbal working memory. Finally, although the results are inconsistent across studies, language assessments suggest that impairments in verbal comprehension and word knowledge might be associated with prenatal methylmercury exposure.



The background is a solid dark blue color, decorated with several realistic-looking water droplets of various sizes. The droplets are positioned in the corners and along the edges, with some showing highlights and shadows to give them a three-dimensional appearance.

**ESTABLISHING RISK:  
What are existing gaps?**

1



# CURRENT GAPS: Hazard Identification

- **IDENTIFICATION AND DOCUMENTATION OF THE PRESENCE OF CHEMICALS**
  - ✓ Environmental and biologic monitoring levels of plastics/chemicals
  - ✓ Laboratory capabilities

2



## CURRENT GAPS: Hazard Characterization

- **DOSE RESPONSE RELATIONSHIPS**
  - ✓ Effects of low-dose chronic exposure to chemicals or mixtures of chemicals used as plastic monomers or additive?
- **EXISTING REFERENCE DOSES/GUIDELINE VALUES**
  - ✓ BPA tolerable daily intake (oral route) (safe dose for humans) : 0.05 mg/kg BW/day (*Mikolajewska, K, et al 2015*)  
**BUT for what health end point?**

The toxicological tenet that the DOSE makes the POISON may no longer apply.



# CURRENT GAPS: Exposure Assessment

## • AMOUNT INGESTED PARTICLE SIZE

- ✓ Only microplastics with size  $\leq 20\mu\text{m}$  would be able to penetrate into organs while the smallest fraction ( $0.1 > 10\mu\text{m}$ ) would be able to access all organs, cross cell membranes, the blood-brain barrier and the placenta.

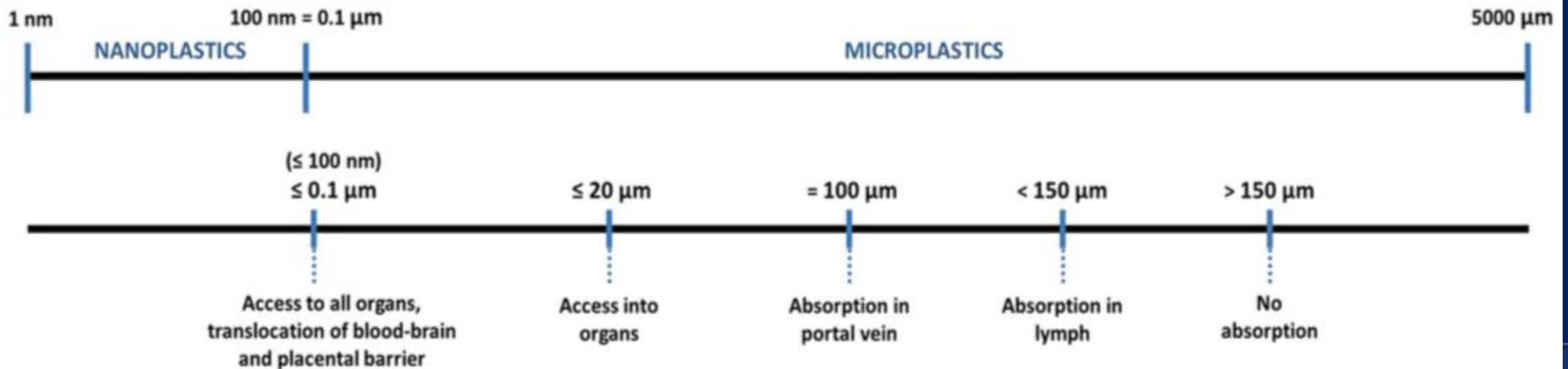


Fig. 1. Fate of micro- and nanoplastics in mammalian bodies (adapted from [Lusher et al., 2017](#)).

# CURRENT GAPS: Exposure Assessment

## EXPOSURE RATE: Limitations in measuring concentrations of chemicals

**Table 2.** Concentrations of bisphenol A (BPA) determined in different matrices

Matrice	Concentration of BPA	Reference
Canned foods	40 µg/kg (70–90 µg/kg food or simulants)	22
Canned beverages	< 7 µg/l simulants	22
Meat products	110 µg/kg meat (17–380 µg/kg meat)	18
Beef	98 µg/kg	19
Fish	< 20–109 µg/kg	19
Beverages	< 10 µg/kg	19
Migration to water from plastic bottles for infants	< 10–20 µg/l	28
Migration to water from plastic bottles for infants (water 100°C)	0.23 µg/l (0.11–0.43 µg/l)	20
Migration to water from plastic bottles for infants after multiple washing in the dishwasher	2.5–17 µg/l	20
Migration to water from plastic bottles for infants after heating in microwave oven	< 0.1–0.7 µg/l	23
Migration to water from plastic bottles for infants after sterilization	2 400–14 300 µg/kg	24

# CURRENT GAPS: Exposure Assessment

## • EXPOSURE RATE:

For BPA, based on the data from over 4,300 adults in New Zealand, it has been estimated that, assuming the average body weight of ca. 75 kg, the average dietary exposure to BPA would be 0.008  $\mu\text{g}$  BPA/kg BW/day. The most severe exposure determined in the study was 0.29  $\mu\text{g}$ /kg BW/Day which was well below the TDI for BPA.

- ✓ ISSUES ON SAMPLE SIZE, EXPOSURES FROM OTHER SOURCES
- ✓ LABORATORY CAPABILITIES

# CURRENT GAPS: Exposure Assessment

- **EXPOSURE RATE:**

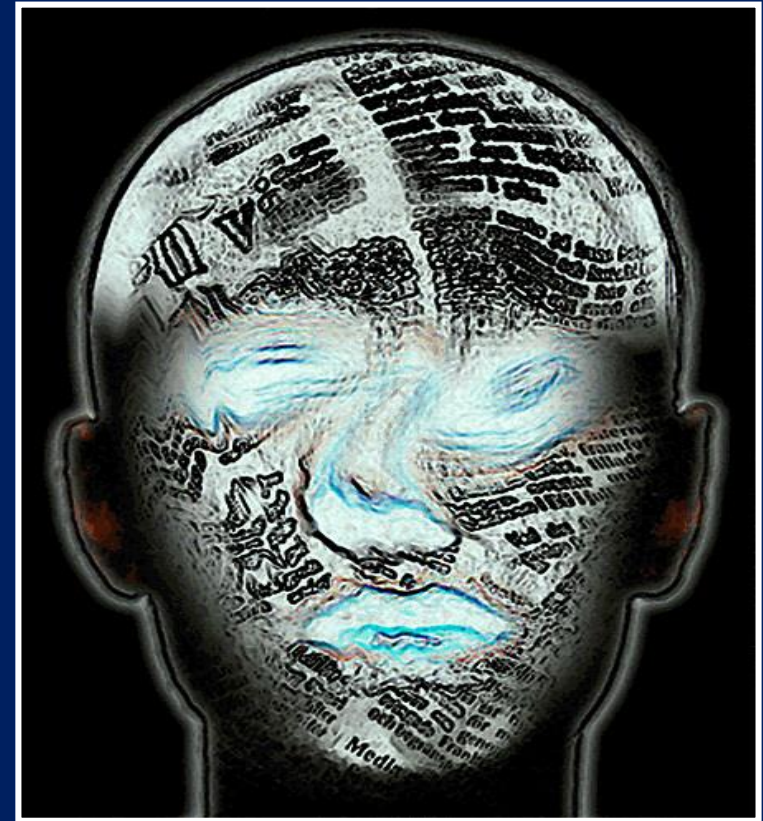
- ✓ **Vulnerable and susceptible populations:** the very young, the very old, women of reproductive age group, workers, populations with existing medical conditions, etc.






# 4 CURRENT GAPS: Risk Characterization

- Multiple exposures and potential adverse interactions
- Multiple and critical health endpoints
- Latency period (fetal basis of adult disease) and multigenerational effects



# TAKE HOME POINTS

- The knowledge on the adverse effects of plastics on human health is not comprehensive and conclusive and at this time may be difficult to assess. However, it is prudent that precautionary principle should be advocated because of their potential irreversible effects.
- Because of the intrinsic toxic characteristics of plastics (endogenous chemicals, adsorbed chemicals, microbes), it is important to strengthen the exposure pathways by documenting internal and external exposures.
- Risk analysis frameworks to evaluate hazards and risks to exposures to plastics should be undertaken and supported.



The point is this:  
whoever sows sparingly  
will also reap sparingly,  
and whoever sows bountifully  
will also reap bountifully.  
2 Corinthians 9:6

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**Thank you for your kind attention.**