
بسم الله الرحمن الرحیم

Environmental toxins & contamination of foods

Hidden threats to digestive health

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"Man is what he eats"

-Ludwig Feuerbach, 1850

600 million

- people fall ill due to contaminated food annually (almost 1 in 10 people).
- 420 000 die every year, primarily in low- and middle-income countries.
- Children under 5 years: 40% of foodborne disease burden (125,000 deaths/year).
- Economic loss: ~$110 billion/year (productivity + medical costs).



https://www.who.int/news-room/fact-sheets/detail/food-safety

The GI Tract – First Line of Exposure

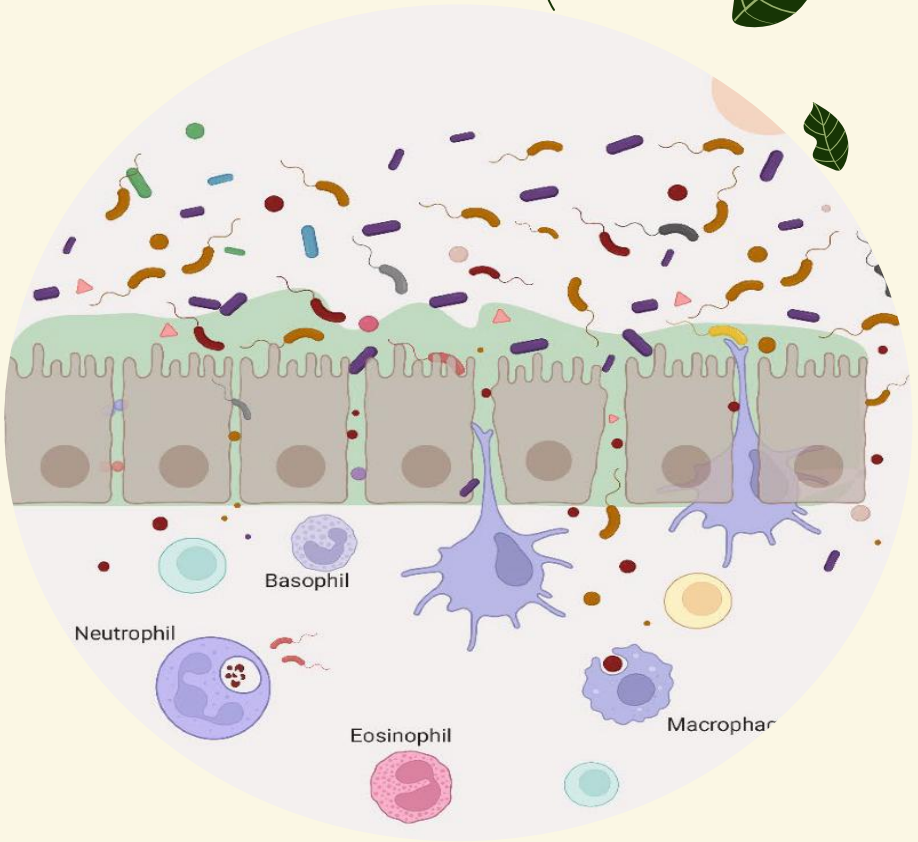
Ingestion is the most common route of entry for foodborne toxins, **80-90%** of toxin exposure occurs through diet (WHO, 2022).

- **Why?** Bioaccumulation in food chains (e.g., mercury and pesticides)
  - GI tract is uniquely vulnerable:
    - High exposure surface area
    - Rapid cellular turnover
    - Microbiota interactions

The GI Tract – First Line of Exposure

- Mechanisms of Damage:
  - Dysbiosis: Pesticides reduce microbial diversity (linked to IBD/IBS).
  - Barrier Disruption: Phthalates/ Bisphenol A (BPA) ↑ intestinal permeability ("leaky gut").
  - Direct Toxicity: Arsenic → oxidative stress → colorectal cancer (IARC Group 1).

Following chemical exposure (pesticides), a schematic representation is shown of a generic epithelial tissue presenting several inflammatory response outcomes after disruption of the barrier integrity due to pesticide exposure, such as **loss of mucus and villi**, increase of barrier permeability by **cellular junction rupture**, **leaking**, **dysbiosis**, and **inflammatory response**



Lima, C., Falcão, M. A. P., Rosa, J. G. S., Disner, G. R., & Lopes-Ferreira, M. (2022). Pesticides and Their Impairing Effects on Epithelial Barrier Integrity, Dysbiosis, Disruption of the AhR Signaling Pathway and Development of Immune-Mediated Inflammatory Diseases. *International Journal of Molecular Sciences*, *23*(20), 12402. https://doi.org/10.3390/ijms232012402

Types of Environmental Food Contaminants

Biological

Bacteria, viruses
parasites, prions

Chemical

Toxins, pesticides,
heavy metals,
industrial contaminants

Physical

Foreign bodies
microplastics



Biological Contaminants

Bacterial Pathogens

- **Salmonella:** eggs, poultry and other products of animal origin
- **Campylobacter:** raw milk, raw or undercooked poultry and drinking water
- **E. coli (EHEC):** unpasteurized milk, undercooked meat and contaminated fresh fruits and vegetables.
    - Symptoms: fever, headache, nausea, vomiting, abdominal pain and diarrhea, HUS (EHEC)

Bacterial Pathogens

- **Listeria monocytogenes**: Ready-to-eat meats, unpasteurized dairy products and can grow at refrigeration temperatures
  - Severe in pregnant women, neonates, elderly
  - Diarrhea, abdominal pain
- **Vibrio cholerae**: contaminated water, Raw vegetables and various types of raw or undercooked seafood
  - Symptoms may include abdominal pain, vomiting and profuse watery diarrhea

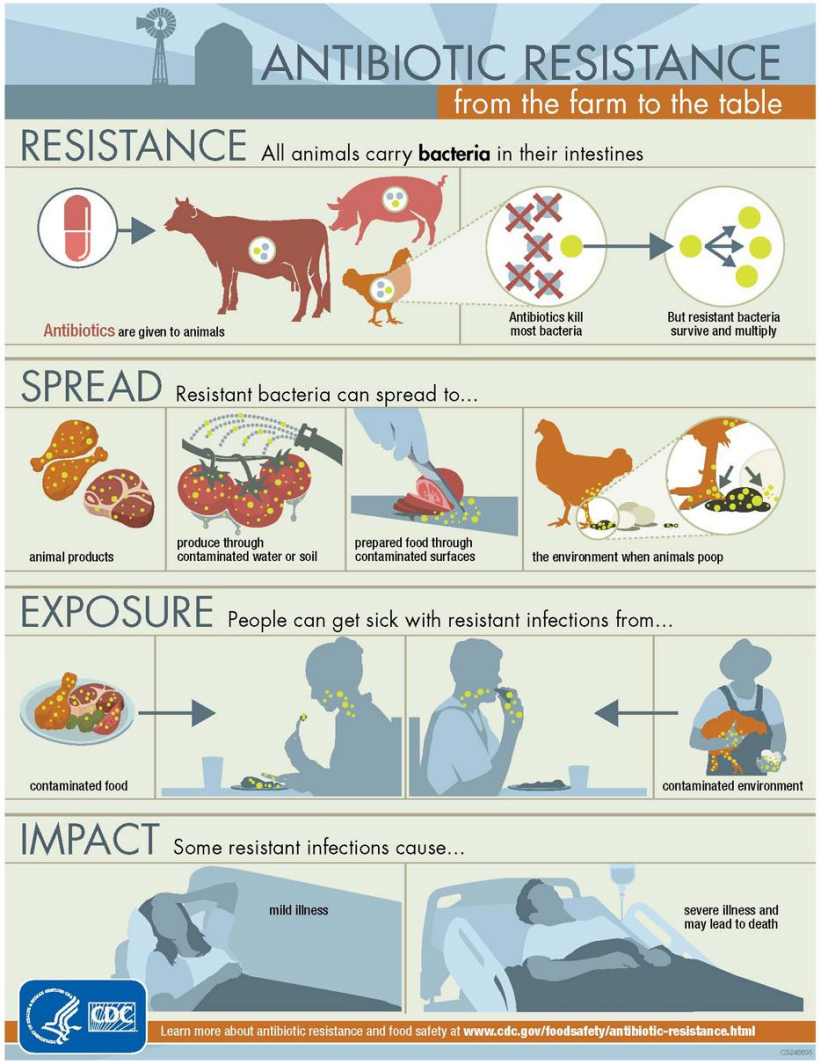
Bacterial toxins

Example: Clostridium perfringens enterotoxin, Bacillus cereus emetic toxin.

GI Impact: Acute food poisoning → vomiting, diarrhea, necrotizing enteritis.

Bacterial Resistance – A GI Challenge

- Overuse of antibiotics in agriculture → resistant GI pathogens.
- GI flora alteration → increased susceptibility.
- GI specialists must be vigilant for resistant enteric infections.



Viral Foodborne Threats

- **Norovirus**: Highly contagious, leading cause of outbreaks
- **Hepatitis A:** Undercooked seafood, contaminated produce
    - Long incubation; liver dysfunction

Parasitic Contaminants

Taenia spp., Echinococcus, Ascaris, Giardia, Entamoeba histolytica

- Soil-contaminated crops, undercooked meat
- May lead to malabsorption, chronic diarrhea, liver cysts

Mycotoxins

Fungal contamination in crops (corn, peanuts, coffee, grains) due to poor storage (humidity/heat).

- **Aflatoxins (Aspergillus):**

  Mechanism: Metabolized by liver → DNA adducts → hepatocellular carcinoma (IARC Group 1 carcinogen).

- **Ochratoxins (Aspergillus, Penicillium):**

Mechanism: Nephrotoxic + intestinal barrier disruption → potential role in IBD

Nazareth TM, Soriano Pérez E, Luz C, Meca G, Quiles JM. Comprehensive Review of Aflatoxin and Ochratoxin A Dynamics: Emergence, Toxicological Impact, and Advanced Control Strategies. Foods. 2024 Jun 18;13(12):1920. doi: 10.3390/foods13121920. PMID: 38928866; PMCID: PMC11203095.

Aflatoxin and GI Health

Fungal Species: Aspergillus flavus & A. parasiticus (produce aflatoxin B1, the most toxic variant).

High-Risk Conditions: Warm (>25°C), humid (>70% RH) storage of crops

Commonly Contaminated Foods:

- Grains: Corn, rice
- Nuts: Peanuts, pistachios, almonds (high-fat content promotes fungal growth).
- Spices: Chili, black pepper
- Dairy: Indirect exposure via aflatoxin M1 (cows fed contaminated feed).

Crops prone to contaminations



Corn



Peanut



Pistachio



Coconut



Rice

Aflatoxin and GI Health

Mechanisms of Toxicity

- **Liver Toxicity & Carcinogenesis:**

Hepatocellular carcinoma (HCC) (IARC Group 1 carcinogen).

Synergy with HBV: AFB1 + HBV ↑ HCC risk 30-fold (HBV impairs aflatoxin detoxification).

- **GI Tract Impact:**

Gut Microbiota Disruption: ↓ Lactobacillus, ↑ Enterobacteriaceae → dysbiosis

Mucosal Inflammation: ↑ Intestinal permeability ("leaky gut") → potential IBD exacerbation.

Liu Y, Wu F. Global burden of aflatoxin-induced hepatocellular carcinoma: a risk assessment. Environ Health Perspect. 2010 Jun;118(6):818-24. doi: 10.1289/ehp.0901388. Epub 2010 Feb 19. PMID: 20172840; PMCID: PMC2898859.

Prions – Rare but Deadly

Bovine Spongiform Encephalopathy (BSE) → Variant Creutzfeldt-Jakob disease (vCJD)

- Consumption of infected brain/spinal tissues
- Resistant to standard sterilization
- Neurodegenerative, no cure

Naturally Occurring Toxins

Marine biotoxins: Shellfish, fish

- Neurotoxicity, diarrhea

Mushroom toxins: Wild foraged species

- Liver failure, death



Chemical Contaminants

Pesticides

- **Organophosphates (e.g., chlorpyrifos, malathion)**

Use: Widely sprayed on fruits, vegetables, and grains.

Persistence: Short half-life (days-weeks), but frequent reapplication → chronic exposure.

- **Organochlorines (e.g., dichlorodiphenyltrichloroethane (DDT), lindane)**

Legacy Issue: Banned in many countries but persist in soil/fatty foods (dairy, meat).

- **Neonicotinoids (e.g., imidacloprid)**

Absorbed by plants → residues in pollen, fruits, and drinking water.

- **Glyphosate**

Pesticides & Mechanisms of GI Toxicity

- **Mucosal Inflammation**

  Oxidative Stress: Pesticides ↑ ROS → damage gut epithelial tight junctions → leaky gut.

  Immune Activation: Organophosphates trigger NF-κB pathway → pro-inflammatory cytokines
- **Microbiome Disruption**

  ↓ Beneficial bacteria (e.g., Lactobacillus, Bifidobacterium).

  ↑ Pathobionts (e.g., E. coli, Enterococcus) → dysbiosis
- **Neurotoxicity & Gut-Brain Axis**

  Neonicotinoids disrupt acetylcholine signaling → altered gut motility (IBS-like symptoms).

Chronic Low-Dose Exposure to pesticides & GI Diseases

- **Irritable Bowel Syndrome (IBS)**

Farmers/agricultural workers show higher IBS prevalence - OR: 1.26 (1).

- **Food Intolerances**

Pesticide-induced barrier dysfunction → ↑ antigen penetration → immune reactions.

Dysbiosis & IBD Risk

Dieldrin & DDT exposure linked to ulcerative colitis in cohort studies (dieldrin: HR=1.59), DDT: HR=1.38) (2).

Glyphosate depletes Faecalibacterium prausnitzii (anti-inflammatory commensal).

1. Rueda-Ruzafa L, Roman P, Cardona D, Requena M, Ropero-Padilla C, Alarcón R. Environmental pesticide exposure and the risk of irritable bowel syndrome: A case-control study. Environ Toxicol Pharmacol. 2023 Mar;98:104076. doi: 10.1016/j.etap.2023.104076. Epub 2023 Feb 7. PMID: 36754128. 2. Chen D, Parks CG, Hofmann JN, Beane Freeman LE, Sandler DP. Pesticide use and inflammatory bowel disease in licensed pesticide applicators and spouses in the Agricultural Health Study. Environ Res. 2024 May 15;249:118464. doi: 10.1016/j.envres.2024.118464. Epub 2024 Feb 12. PMID: 38354883; PMCID: PMC11065595.

Heavy Metals in Food

Cadmium: Rice, leafy vegetables, shellfish

- Mechanism: Accumulates in enterocytes → oxidative stress + malabsorption

Arsenic: rice, water

- Mechanism: ↑ ROS → colorectal cancer (IARC Group 1).

Lead: Cereals, vegetables (soil contamination)

- Neurotoxicity, anemia

Mercury: Fish (especially large predatory fish e.g. Tuna, swordfish)

- CNS toxicity, fetal damage

Heavy Metals in Food

A. Arsenic (Inorganic As^{3+}/As^{5+})

- GI Mucosal Damage:

Disrupts tight junctions → ↑ intestinal permeability

Induces apoptosis in enterocytes → chronic diarrhea

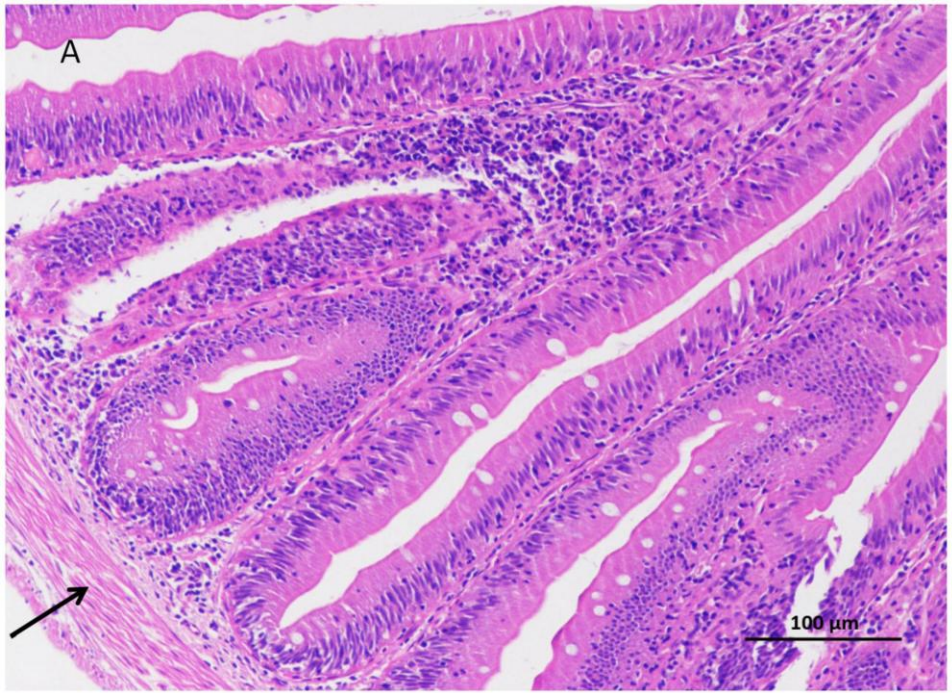
- Carcinogenesis:

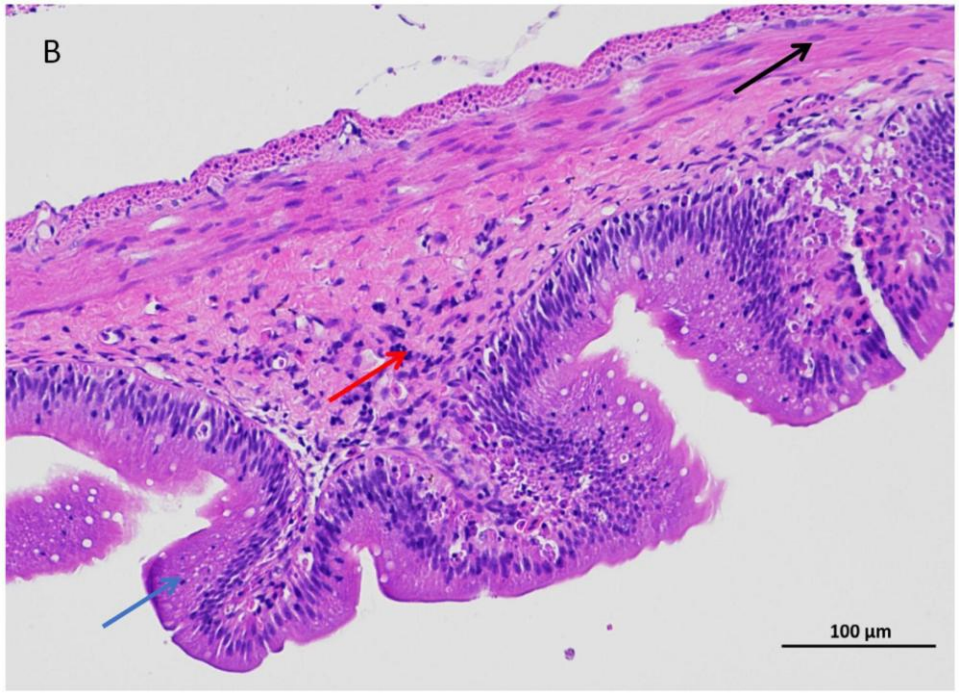
IARC Group 1 carcinogen (skin, bladder, colorectal cancer).

Methylation in liver → DNA damage + ROS generation.

Iran-Specific Concerns: Arsenic in Well Water

Clinical Signs: Hyperkeratosis, chronic diarrhea, ↑ HCC risk (synergy with HBV).





Arsenic caused distal intestinal histopathological changes (200 × magnification). (A) Control group, (B) arsenic (As)-treated group. Blue arrows: shortened intestinal villi, right black arrow: thinner muscular layer, and red arrows: inflammatory cell infiltration.

Shi, X., Xu, W., Che, X., Cui, J., Shang, X., Teng, X., & Jia, Z. (2023). Effect of arsenic stress on the intestinal structural integrity and intestinal flora abundance of Cyprinus carpio. Frontiers in Microbiology, 14.

Heavy Metals in Food

B. Lead (Pb^{2+})

- GI Dysmotility: Inhibits calcium channels in enteric nerves → constipation/colic.
- Neurotoxicity: Competes with zinc/iron → cognitive impairment (even low-dose).

C. Cadmium (Cd^{2+})

- Pancreatic & Colorectal Cancer:
    - Accumulates in pancreatic acinar cells → oxidative stress → malignancy.
    - Epigenetic effects: Hypermethylation of tumor suppressor genes.

D. Mercury (Methyl-Hg)

- Gut-Brain Axis Disruption: Binds to sulfhydryl groups in neurons → peripheral neuropathy.

Industrial Pollutants- Persistent Organic Pollutants (POPs)

- **Dioxins**: meat/dairy fat

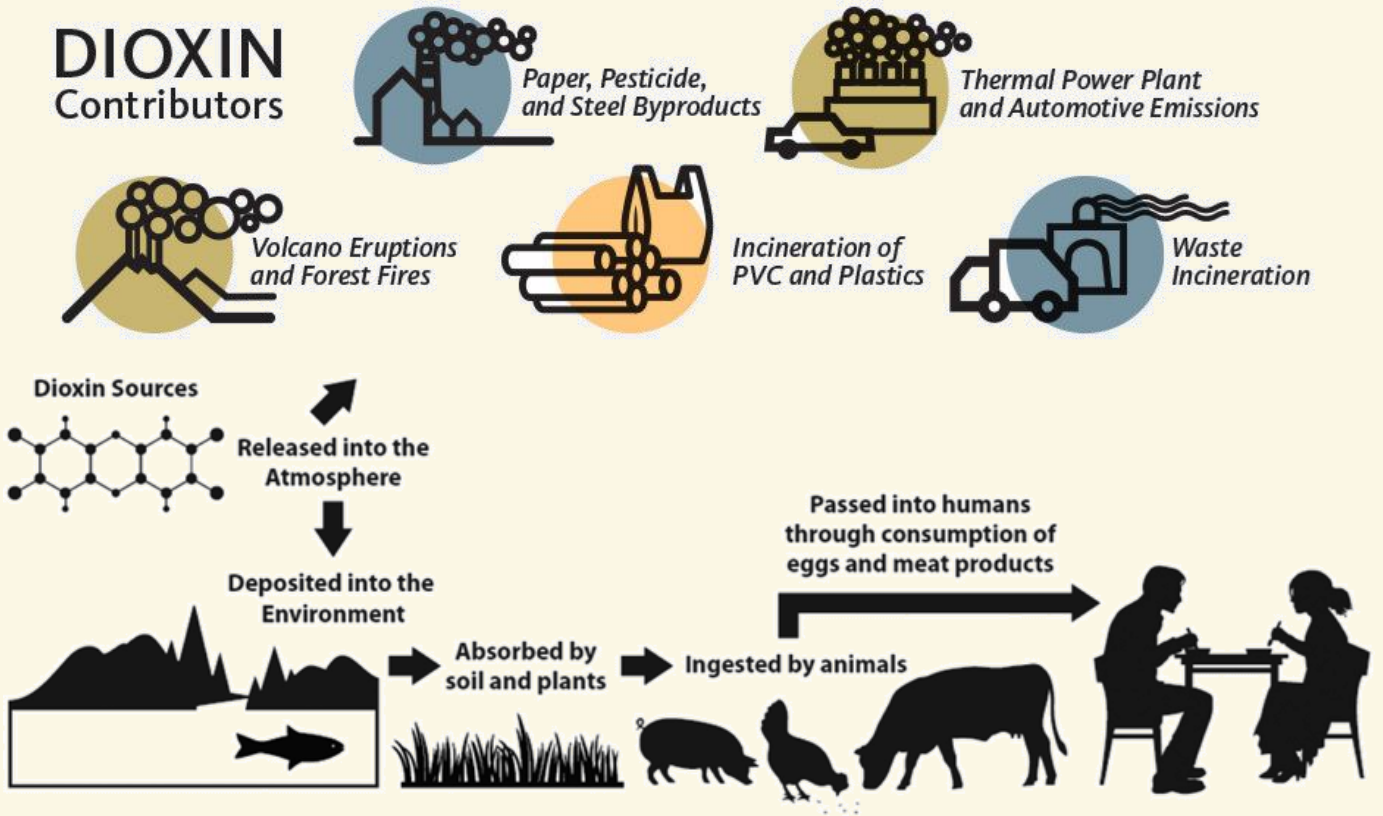
Mechanism: Aryl hydrocarbon receptor (AhR) activation → immune dysregulation → IBD exacerbation.

- **Polychlorinated biphenyls (PCBs)**: farm-raised fish

Endocrine disruption → altered gut motility.

- Both: carcinogenic, endocrine disruption, immune suppression, Long half-life in body fat

Industrial Pollutants- Persistent Organic Pollutants (POPs)



5 Atmospheric pathways of dioxins' entry in the food web: emission, deposition, bioaccumulation/uptake, ingestion by animal and ingestion by humans.

Kurwadkar, Sudarshan. (2020). Dioxin. 10.1201/9781315170961-1.

Plasticizers

Toxin	Common Sources	Key Properties
Bisphenol A (BPA)	Canned food linings, plastic containers	Estrogen mimic, leaches when heated
Phthalates	Fast food packaging, vinyl gloves	Binds fats, disrupts androgen signaling
Perfluoroalkyls (PFAS)	Grease-proof food wrappers, popcorn bags	"Forever chemicals" (bioaccumulative)





plasticizers





Plasticizers - Mechanisms of GI Toxicity

A. Gut Barrier Disruption

BPA: Downregulates occludin/claudin-5 → ↑ intestinal permeability

Phthalates (DEHP): Activates PPAR-γ → alters tight junction proteins.

B. Dysbiosis

PFAS: Reduces Bifidobacterium → ↑ Proteobacteria (linked to IBD)

BPA: Promotes obesogenic microbiota (↑ Firmicutes: Bacteroidetes ratio).

C. Endocrine Disruption

All compounds: Bind to estrogen/androgen receptors → aberrant gut hormone signaling (e.g., GLP-1).



physical Contaminants

Physical contaminants

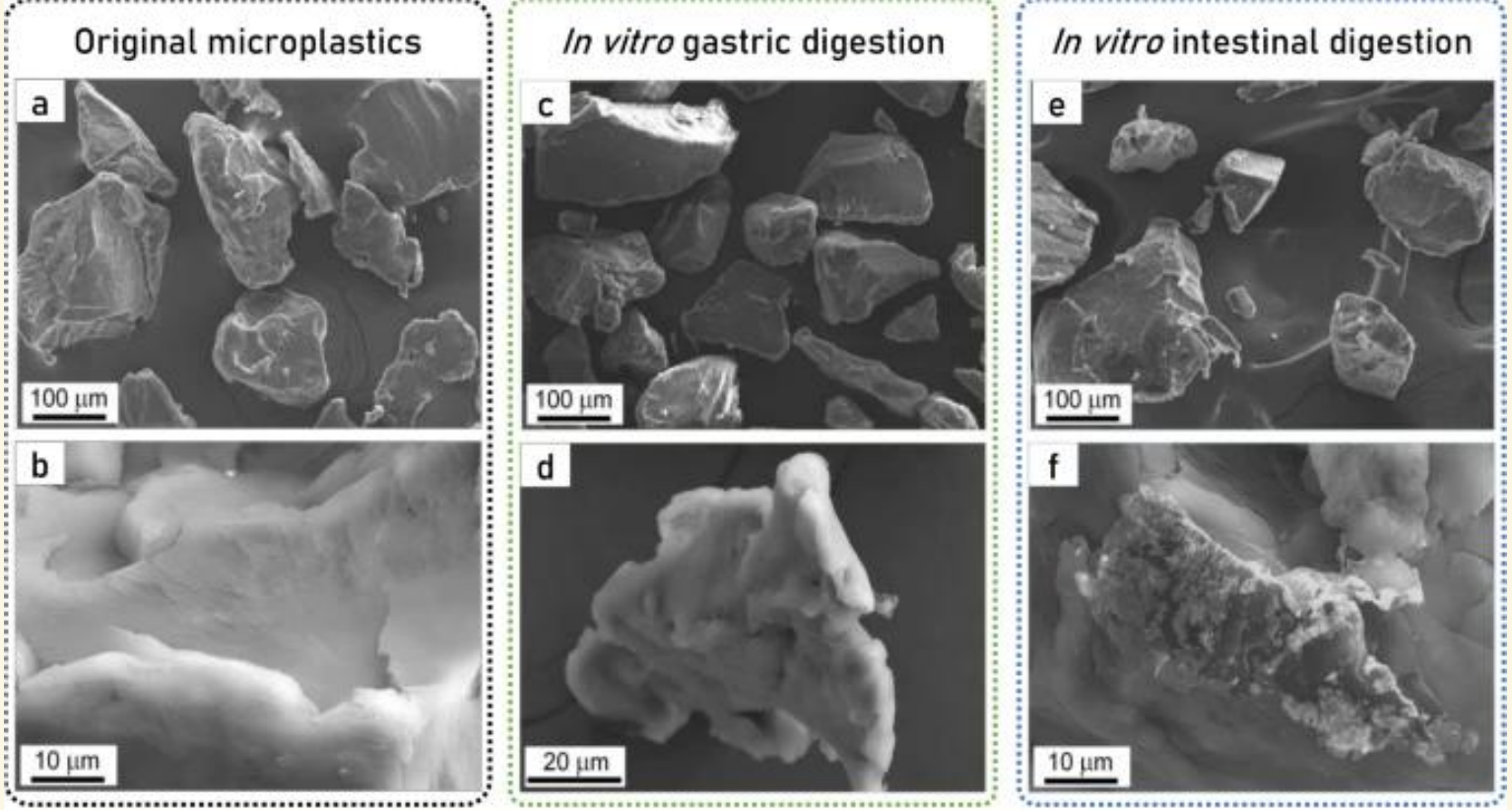
A. Microplastics (<5mm): Bottled water, seafood, food packaging.

- GI Impact: Mucosal irritation → chronic inflammation.
- Carriers of pathogens/toxins (e.g., adsorbed heavy metals).

B. Nanoparticles (TiO_2, SiO_2): Food additives (E171, anti-caking agents).

- Mechanism: ↑ Intestinal permeability → immune activation

Source	Contamination Level	Key Findings
Bottled Water	Up to 325 particles/L	Higher than tap water (polypropylene, PET)
Seafood	Mussels, oysters, fish guts	~0.5–10 MPs/gram (fibers, fragments)
Table Salt	Sea salt > rock salt > lake salt	Up to 1,800 MPs/kg (global avg.)
Honey/Sugar	Airborne deposition	~660 MPs/kg in commercial honey



Micrographs by field emission scanning electron microscope (FESEM) of polyethylene terephthalate microplastics: (a,b) original PET microplastics (MPs), (c) and (d) PET MPs after in vitro gastric digestion, (e,f) PET MPs after in vitro gastrointestinal digestion

Tamargo, A., Molinero, N., Reinosa, J.J. et al. PET microplastics affect human gut microbiota communities during simulated gastrointestinal digestion, first evidence of plausible polymer biodegradation during human digestion. Sci Rep 12, 528 (2022). https://doi.org/10.1038/s41598-021-04489-w

Mechanisms of GI Toxicity

A. Physical Damage & Epithelial Stress

<10 µm particles translocate across intestinal epithelium → systemic circulation

Larger fragments cause abrasion → mucosal microtears.

B. Immune Activation & Inflammation

Macrophage Recruitment: MPs mistaken for pathogens → TLR4/NF-κB activation → pro-inflammatory cytokines (IL-6, TNF-α).

C. Dysbiosis

Microbiome Shifts: ↓ Akkermansia muciniphila (mucus protector) → ↑ Escherichia (pro-inflammatory).

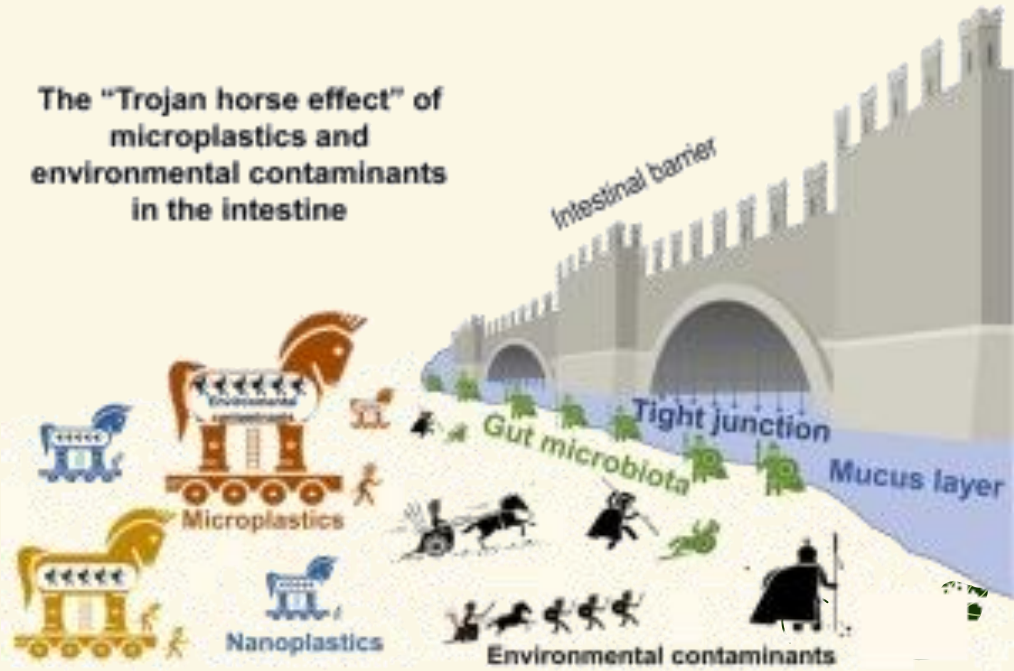
Fecal MP concentration in IBD patients (41.8 items/g dm) was significantly higher than that in healthy people (28.0 items/g dm).

Yan Z, Liu Y, Zhang T, Zhang F, Ren H, Zhang Y. Analysis of Microplastics in Human Feces Reveals a Correlation between Fecal Microplastics and Inflammatory Bowel Disease Status. Environ Sci Technol. 2022 Jan 4;56(1):414-421. doi: 10.1021/acs.est.1c03924. Epub 2021 Dec 22. PMID: 34935363.

Synergy with Other Toxins

- **"Trojan Horse" Effect**:

  MPs carry **pathogens (e.g., Vibrio)**, **pesticides**, or **plasticizers** → compounded toxicity.



L. Hildebrandt, F.L. Nack, T. Zimmermann, D. Pröfrock, Microplastics as a Trojan horse for trace metals, Jurnal of Hazardous Materials Letters, Volume 2, 2021,100035, ISSN 2666-9110,

Other Physical Contaminants in Food

- **Examples:**

Glass shards, metal fragments, bone chips, stones, plastic bits, jewelry, hair, insects

- **Mechanisms of damage:**

Mechanical injury to teeth, oral mucosa, esophagus, or bowel obstruction (especially in children or elderly), Perforation in rare cases (e.g. sharp bones or metal), Emotional distress and food refusal in vulnerable patients

- **Gastrointestinal Impact:** Oral trauma, esophagitis, gastric mucosal injury Foreign body ingestion → possible endoscopic or surgical removal

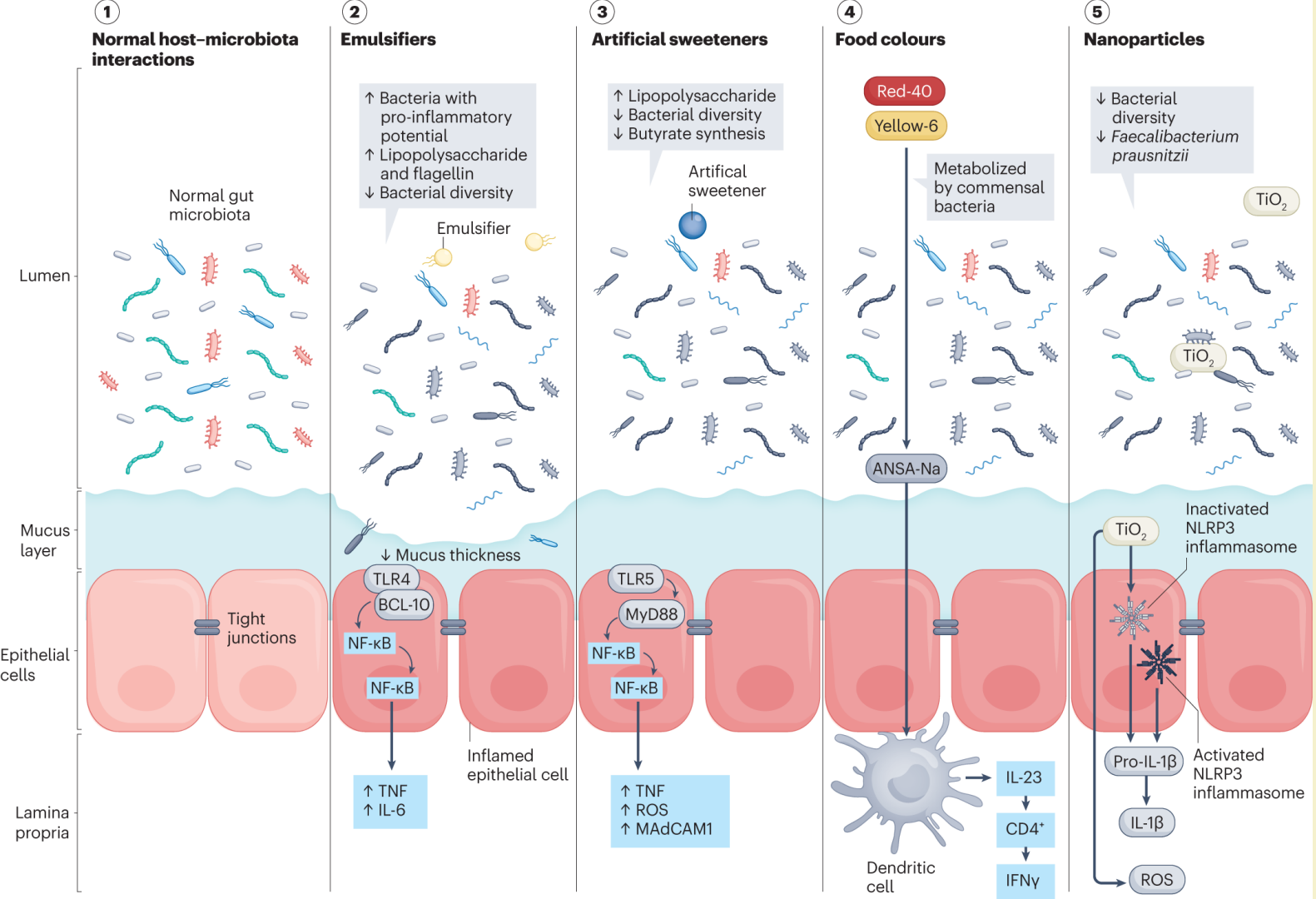
Other Chemical Hazards

Cyanogenic glycosides: Cassava, almonds

Radioactive nucleotides: From environmental fallout

Allergens and additives: Immune and inflammatory GI responses

Additive	Found In	Key Concerns
Emulsifiers (CMC, polysorbate-80)	Ice cream, bread, sauces	Gut barrier disruption, dysbiosis
Artificial Sweeteners (saccharin, sucralose)	Diet sodas, "sugar-free" products	Microbiome shifts, glucose intolerance
Preservatives (sulfites, benzoates)	Wine, dried fruit, processed meats	Oxidative stress, mast cell activation



Different effects of emulsifiers, sweeteners, colors and nanoparticles on the microbiome, mucosal barrier and inflammation in the gut.

Whelan, K., Bancil, A.S., Lindsay, J.O. *et al.* Ultra-processed foods and food additives in gut health and disease. *Nat Rev Gastroenterol Hepatol* **21**, 406–427 (2024). https://doi.org/10.1038/s41575-024-00893-5

Key Carcinogens & Their Target Cancers

Toxin	Primary Food Source	Associated Cancer	Mechanism
Aflatoxin B1	Moldy nuts/grains (peanuts, corn)	Hepatocellular Carcinoma (HCC)	DNA adducts (p53 mutation at codon 249)
N-Nitroso Compounds (NOCs)	Processed meats (bacon, hot dogs)	Colorectal Cancer (CRC)	Alkylating agents → DNA damage
Arsenic (Inorganic)	Rice, contaminated water	Stomach, Bladder, HCC	Oxidative stress + impaired DNA repair
Acrylamide	Fried/grilled starches (bread, potatoes)	Esophageal, CRC	Epoxide formation → mutagenesis

Key Epidemiological Data

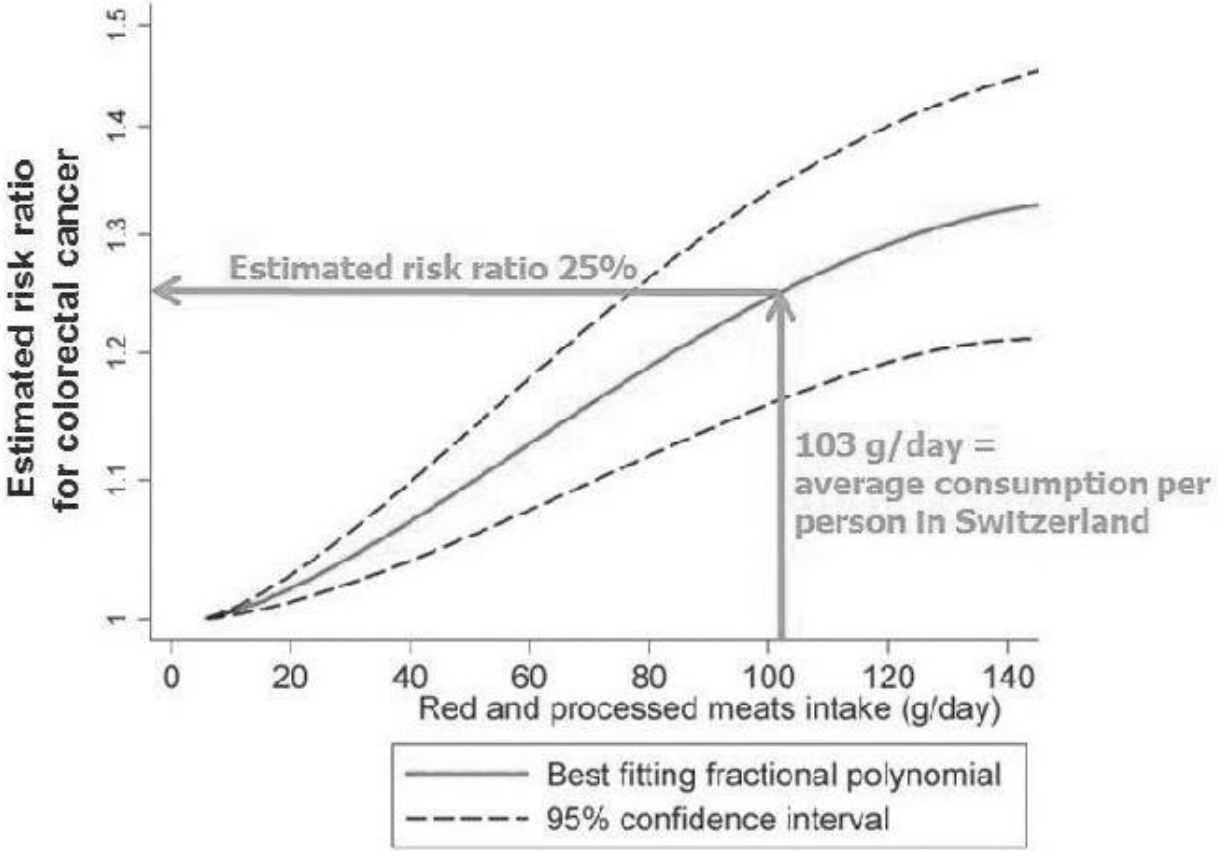
A. Processed Meats and CRC Risk

- **50g/day processed meat ↑ CRC risk by 18%** (OR: 1.18, 95% CI: 1.10–1.28) (1)
- *Mechanism*: Heme iron + nitrites → NOCs in colon.

B. Aflatoxin and HCC (HBV Synergy)

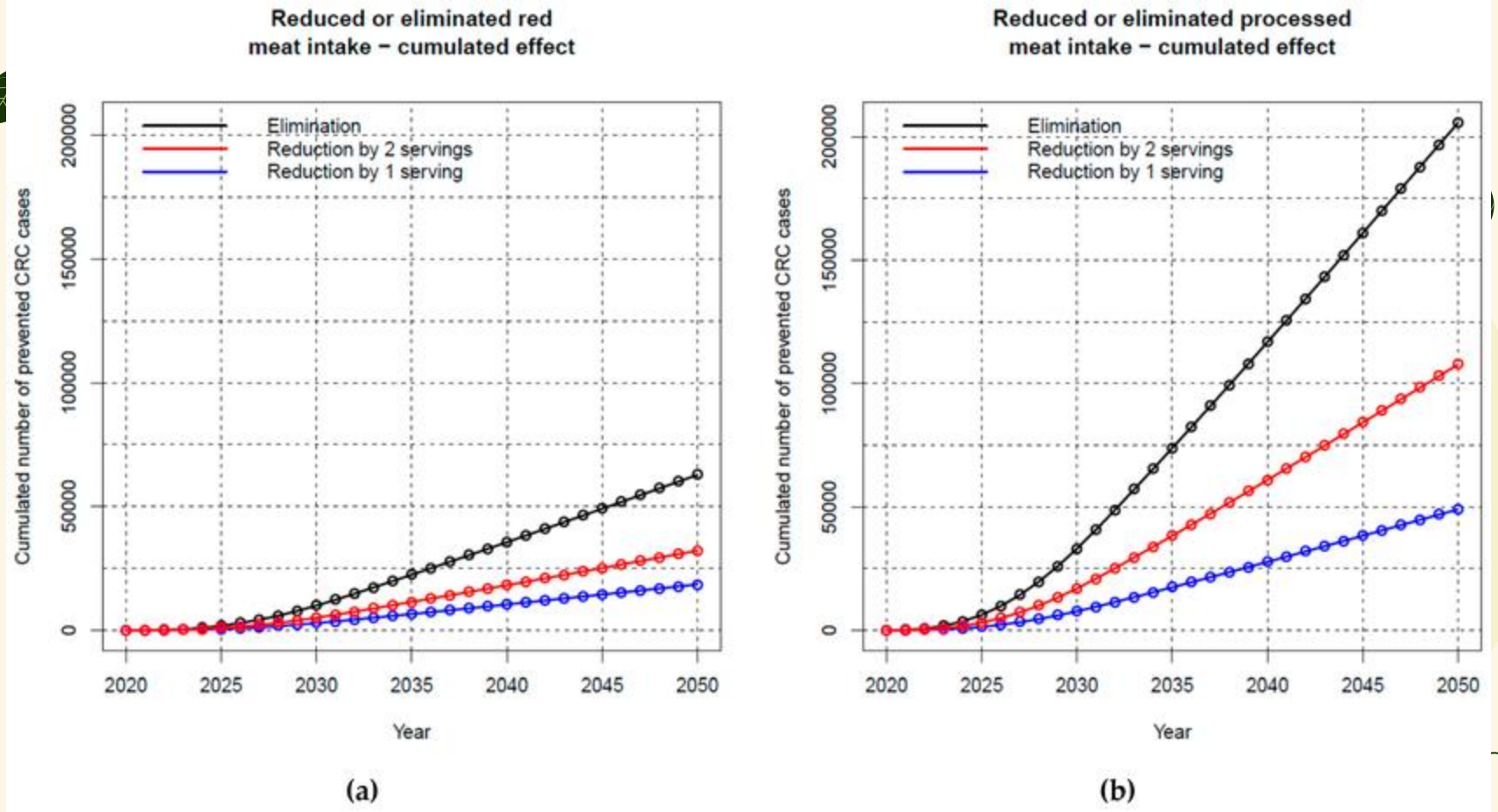
- **HBV + Aflatoxin exposure**: **OR = 35.5** (vs. HBV alone OR = 15.2) (2).

1. Aykan NF. Red Meat and Colorectal Cancer. Oncol Rev. 2015 Dec 28;9(1):288. doi: 10.4081/oncol.2015.288. PMID: 26779313; PMCID: PMC4698595. 2. Liu Y, Chang CC, Marsh GM, Wu F. Population attributable risk of aflatoxin-related liver cancer: systematic review and meta-analysis. Eur J Cancer. 2012 Sep;48(14):2125-36. doi: 10.1016/j.ejca.2012.02.009. Epub 2012 Mar 8. PMID: 22405700; PMCID: PMC3374897.



Relative risk of colorectal cancer with an increasing consumption of red meat (processed and unprocessed). The arrows indicate the estimated risk ratio with the average consumption according to the Sixth Swiss Nutrition Report

Battaglia, Evelyne & Baumer, Beatrice & Conrad, Beatrice & Darioli, Roger & Schmid, Alexandra & Keller, Ulrich. (2015). Aspects sanitaires de la consommation de viande. Forum Médical Suisse – Swiss Medical Forum. 15. 10.4414/fms.2015.02296.



Cumulative prevented colorectal cancer cases over time in case of a reduction in (a) red and (b) processed meat consumption (men and women combined). Abbreviations: CRC, colorectal cancer.

Niedermaier, Tobias & Gredner, Thomas & Hoffmeister, Michael & Mons, Ute & Brenner, Hermann. (2023). Impact of Reducing Intake of Red and Processed Meat on Colorectal Cancer Incidence in Germany 2020 to 2050-A Simulation Study. Nutrients. 15. 10.3390/nu15041020.

Clinical Implications

- Screening High-Risk Groups:
  - **HCC**: HBV patients in aflatoxin-endemic regions (Africa/Asia).
  - **CRC**: Processed meat consumers with pks positive E. coli stool PCR.
- Dietary Mitigation:
  - **Vitamin C/E**: Blocks nitrosamine formation in stomach.
  - **Chlorophyllin**: Binds aflatoxin in gut (reduced urinary AFB1-DNA adducts by 55%).

P.A. Egner, J. Wang, Y. Zhu, B. Zhang, Y. Wu, Q. Zhang, G. Qian, S. Kuang, S.J. Gange, L.P. Jacobson, K.J. Helzlsouer, G.S. Bailey, J.D. Groopman, & T.W. Kensler, Chlorophyllin intervention reduces aflatoxin–DNA adducts in individuals at high risk for liver cancer, Proc. Natl. Acad. Sci. U.S.A. 98 (25) 14601-14606, https://doi.org/10.1073/pnas.251536898 (2001).



Clinical Implications for GI Practice

Recognizing and Managing Foodborne Toxin Exposure

Recognizing Toxin-Related Symptoms

A. Non-Specific Presentations

- "Functional" GI Symptoms:
  - Bloating, diarrhea, constipation (mimicking IBS) → Could indicate pesticide or emulsifier exposure.
  - Fatigue, brain fog → Potential heavy metal (Pb, As) or mold (mycotoxin) toxicity.

B. Red Flags for Chronic Exposure

- IBD Flares:
  - New/worsening symptoms despite therapy? Consider dietary emulsifiers (CMC, polysorbate-80) or processed foods, pesticide/plasticizer exposure.
- NAFLD Progression:
  - Unexplained ALT/AST spikes? Check for aflatoxin (nuts, grains), PFAS (fast food packaging), heavy metals.

Key Elements of Patient History

A. Dietary Habits

- High-Risk Foods:
  - Daily rice consumption → arsenic.
  - Processed meats → N-nitroso compounds (CRC risk).
  - Non-organic strawberries/spinach → pesticide residues.
- Food Storage:
  - Moldy grains/nuts → aflatoxins.

B. Occupational/Environmental Risks

- Farmers: Pesticides (organophosphates).
- Textile/Plastic Workers: Phthalates, BPA.
- Living Near Industrial Sites: Heavy metals (Pb, Cd).

Role of Diet in Prevention & Management

A. For IBD Patients

- Eliminate Emulsifiers: Trial removing processed foods (may reduce flares).
- Fiber-Rich Diet: Butyrate producers (oats, legumes) counteract dysbiosis.

B. For NAFLD/IBS

- Organic Diet: ↓ Pesticide exposure → improved microbiota diversity.
- Heavy Metal Detox Foods: Cilantro (chelates Pb), selenium (antagonizes As).

C. For All Patients

- Safe Food Prep: Wash/peel produce, avoid plastic microwaving.
- Water Filtration: Reverse osmosis for arsenic/PFAS.



Patient Education: Practical Steps to Reduce Exposure

A. Food Handling & Storage

- Wash Produce Thoroughly:
  - Use baking soda soak (reduces pesticide residues by 60-90%)
  - Peel non-organic fruits/vegetables (removes surface toxins).
- Avoid Plastic Containers:
  - Never microwave plastic (heat → BPA/phthalate leaching).
  - Replace with glass/ceramic for storage.

B. Dietary Diversity & Fiber

- Rotate Grains: Alternate rice (arsenic risk) with quinoa, millet, or buckwheat.
- Prioritize Fiber: 30g/day from veggies, legumes → binds toxins (e.g., pectin reduces heavy metal absorption).

C. Organic & Local Foods

- "Dirty Dozen" Rule: Buy organic for thin-skinned produce (strawberries, spinach, apples).
- Local Markets: Reduces preservatives/packaging toxins in transported foods.

2024 GUIDE



TO PESTICIDES IN PRODUCE

DIRTY DOZEN

1. Strawberries
2. Spinach
3. Grapes
4. Peaches
5. Pears
6. Nectarines
7. Apples
8. Bell & Hot Peppers
9. Cherries
10. Blueberries
11. Green Beans
12. Kale, Collard & Mustard Greens

CLEAN 15

1. Avocados
2. Sweet corn
3. Pineapple
4. Onions
5. Papaya
6. Sweet peas (frozen)
7. Asparagus
8. Honeydew melon
9. Kiwi
10. Cabbage
11. Watermelon
12. Mushrooms
13. Mangoes
14. Sweet potatoes
15. Carrots

Based on the EWG's 2024 Shopper's Guide to Pesticides in Produce™

EWG's 2024 Dirty Dozen: The Environmental Working Group (EWG) publishes an annual "Dirty Dozen" list highlighting fruits and vegetables with the highest levels of pesticide residues.



Gut-Protective Nutrition Strategies

A. Probiotics & Fermented Foods

- Targeted Strains:
  - Lactobacillus rhamnosus (binds aflatoxins) (Front Microbiol, 2022).
  - Saccharomyces boulardii (restores barrier after pesticide exposure).

Traditional Options: Yogurt, kefir, kimchi → boost microbial diversity.

B. Plant-Rich, Antioxidant Diet

- Counteract Oxidative Stress:
  - Cruciferous veggies e.g. broccoli, cauliflower, cabbage (sulforaphane detoxifies heavy metals).
  - Turmeric/black pepper (curcumin chelates arsenic).
- Healthy Fats:
  - Omega-3s (wild salmon, flaxseeds) reduce PFAS-induced inflammation.



Take home messages

1. The gut is the first target of foodborne toxins — both microbial and chemical and physical.
2. Chronic exposure can drive IBD, dysbiosis, liver disease, and GI cancers.
3. The microbiome mediates many toxic effects through inflammation and barrier disruption.
4. Underrated threats include mycotoxins, heavy metals, BPA, and POPs.
5. Food safety is clinical — not just public health; exposures belong in patient history.
6. Prevention starts with awareness — in clinics, kitchens, and policy.
7. GI specialists must lead in connecting gut health to environmental food risks.

The Gut Remembers What We Forget

Start small. Think big. Dig deeper.

We monitor symptoms. We measure labs. But we often overlook what starts it all: *exposure*.

As gastroenterologists, we are not just healers of the gut, *we are its guardians.*





Thanks!

Do you have any questions?
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