Casual Friday Series

Forever Chemicals and the Fires They Start Pt. II

A Biogenetix Clinical Presentation

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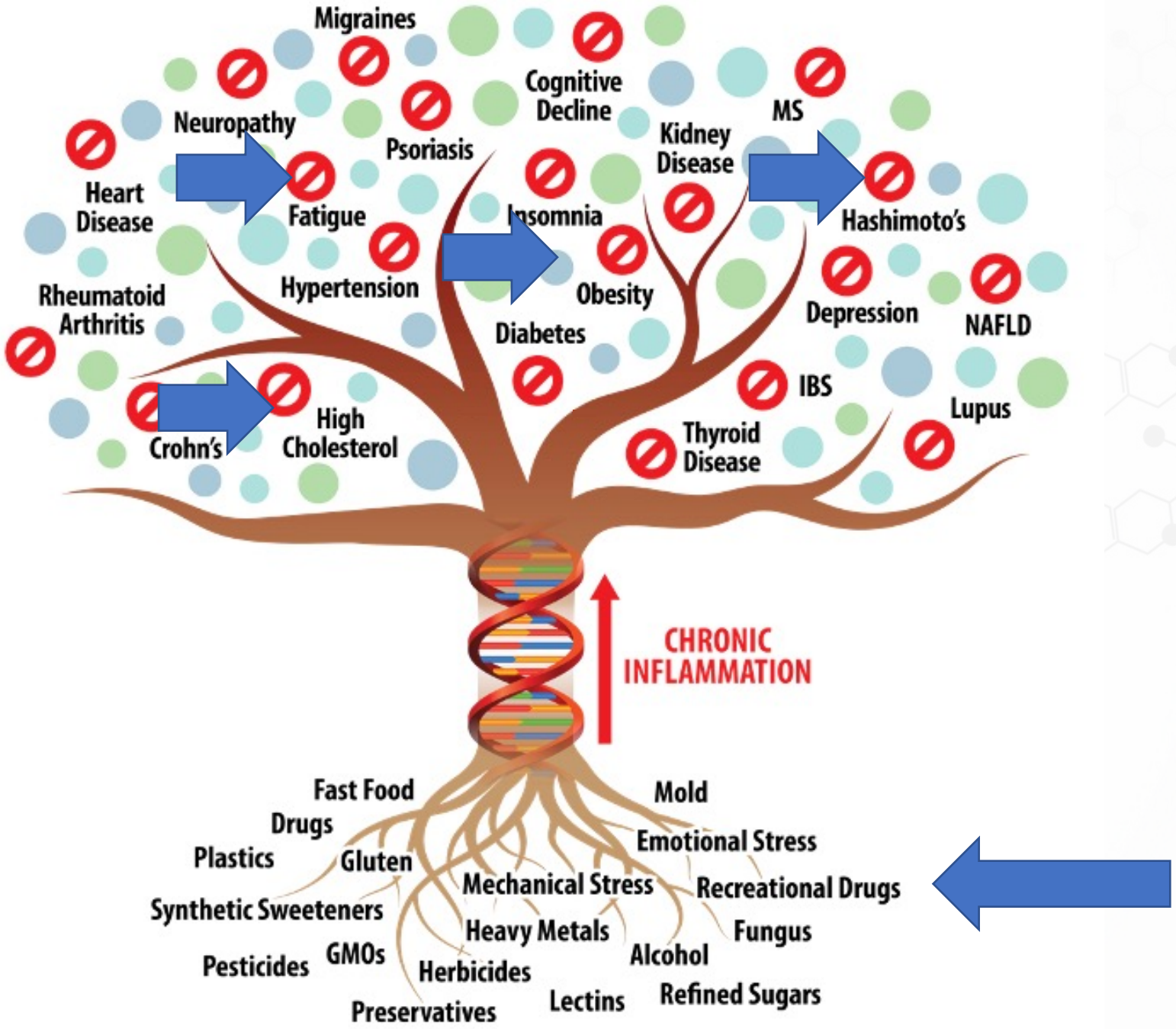

Migraines
Neuropathy
Psoriasis
Cognitive Decline
MS
Kidney Disease
Heart Disease
Fatigue
Insomnia
Hashimoto's
Hypertension
Obesity
Depression
NAFLD
Rheumatoid Arthritis
Diabetes
IBS
Lupus
Crohn's
High Cholesterol
Thyroid Disease
CHRONIC INFLAMMATION
Fast Food
Mold
Drugs
Emotional Stress
Plastics
Gluten
Mechanical Stress
Recreational Drugs
Synthetic Sweeteners
Heavy Metals
Fungus
Pesticides
GMOs
Alcohol
Herbicides
Lectins
Refined Sugars
Preservatives

Centers for Disease Control and Prevention
CDC 24/7: Saving Lives, Protecting People™

Per- and Polyfluorinated Substances (PFAS) Factsheet

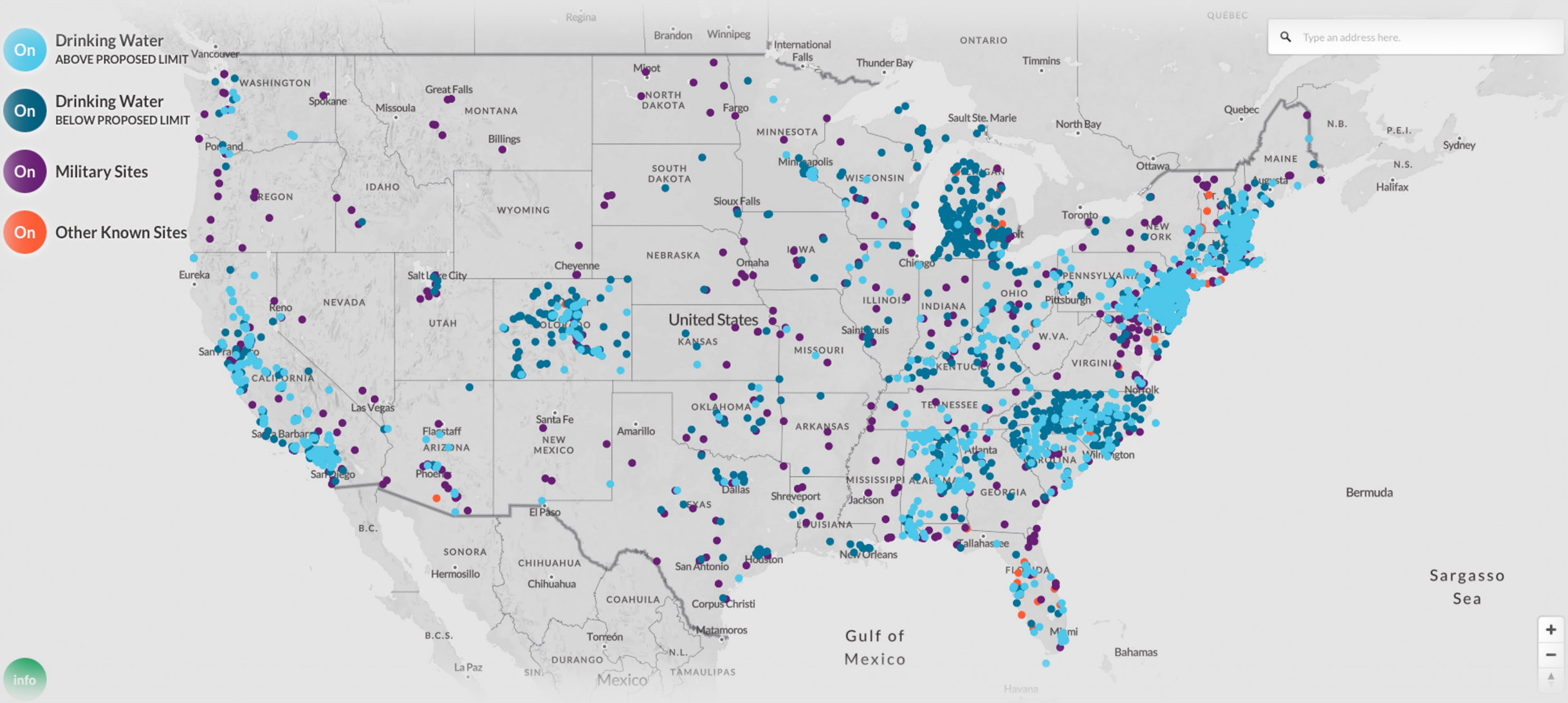
Print

The per-and polyfluoroalkyl substances (PFAS) are a group of chemicals used to make fluoropolymer coatings and products that resist heat, oil, stains, grease, and water. Fluoropolymer coatings can be in a variety of products. These include clothing, furniture, adhesives, food packaging, heat-resistant non-stick cooking surfaces, and the insulation of electrical wire. Many PFAS, including perfluorooctane sulfonic acid (PFOS) and perfluorooctanoic acid (PFOA), are a concern because they:

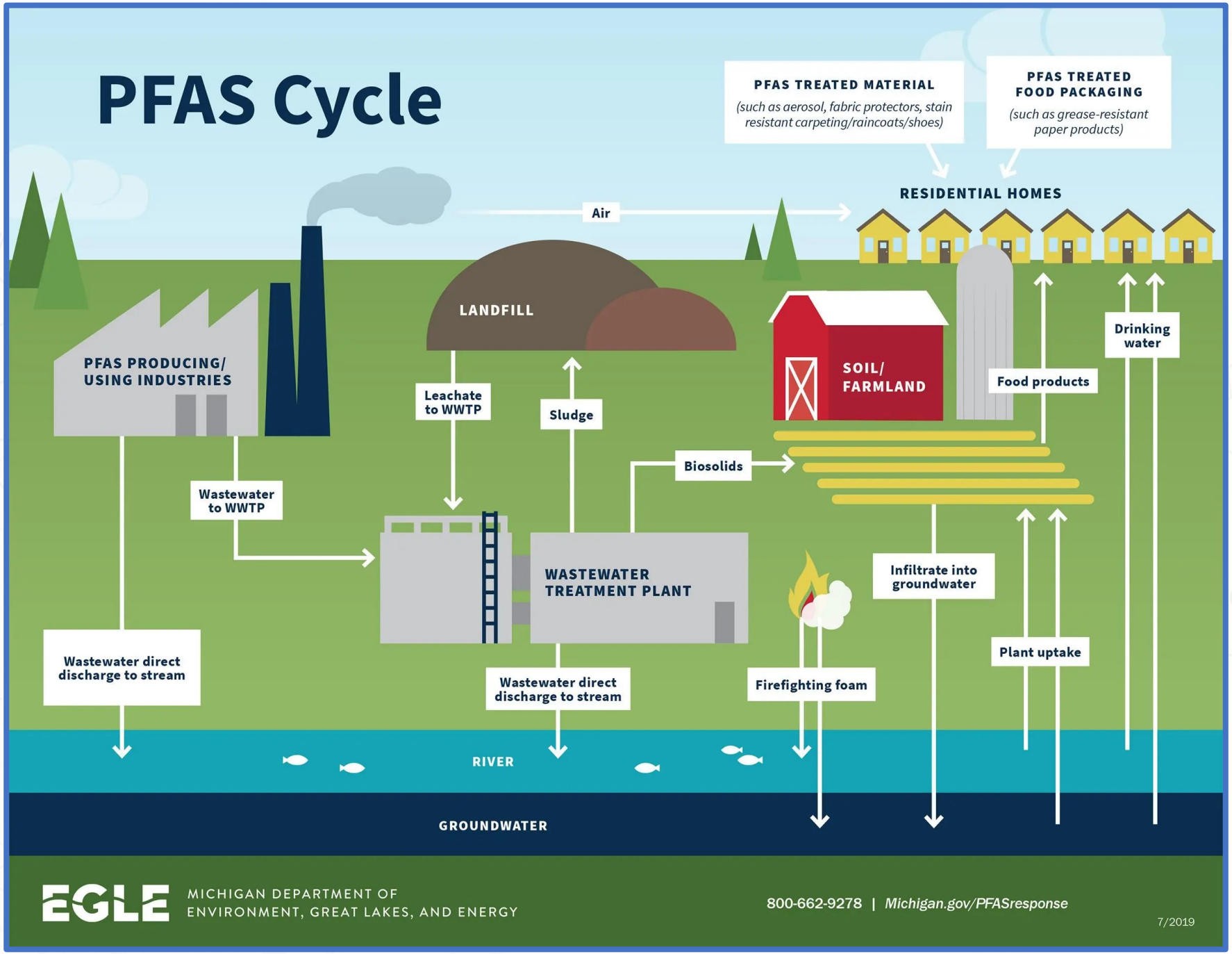
- do not break down in the environment,
- can move through soils and contaminate drinking water sources,
- build up (bioaccumulate) in fish and wildlife.

PFAS are found in rivers and lakes and in many types of animals on land and in the water.


PFAS
Domestic &
Imported Sources
WATER-RESISTANT CLOTHING
FIREFIGHTING FOAMS
PAINT
PERSONAL CARE PRODUCTS
COSMETICS
NONSTICK COOKWARE
CARPET
FAST FOOD PACKAGING
PESTICIDES
STAIN-PROOF FURNITURE
MICROWAVE POPCORN BAGS
STAIN-RESISTANT PRODUCTS



https://www.ewg.org/interactive-maps/pfas_contamination/map/


PFAS Cycle
PFAS TREATED MATERIAL
(such as aerosol, fabric protectors, stain resistant carpeting/raincoats/shoes)
PFAS TREATED FOOD PACKAGING
(such as grease-resistant paper products)
RESIDENTIAL HOMES
Air
LANDFILL
PFAS PRODUCING/ USING INDUSTRIES
SOIL/ FARMLAND
Drinking water
Food products
Leachate to WWTP
Sludge
Biosolids
Wastewater to WWTP
WASTEWATER TREATMENT PLANT
Infiltrate into groundwater
Plant uptake
Wastewater direct discharge to stream
Wastewater direct discharge to stream
Firefighting foam
RIVER
GROUNDWATER
EGLE
MICHIGAN DEPARTMENT OF ENVIRONMENT, GREAT LAKES, AND ENERGY
800-662-9278 | Michigan.gov/PFASresponse
7/2019

Not All In-Home Drinking Water Filters Completely Remove Toxic PFAS

Research by Duke and NC State scientists finds most filters are only partially effective at removing PFAS. A few, if not properly maintained, can even make the situation worse.

- Reverse osmosis filters and two-stage filters reduced PFAS levels, including GenX, by 94% or more in water, though the small number of two-stage filters tested necessitates further testing to determine why they performed so well.
- Activated-carbon filters removed 73% of PFAS contaminants, on average, but results varied greatly. In some cases, the chemicals were completely removed; in other cases they were not reduced at all. Researchers saw no clear trends between removal efficiency and filter brand, age or source water chemical levels. Changing out filters regularly is probably a very good idea, nonetheless, researchers said.
- The PFAS-removal efficiency of whole-house systems using activated carbon filters varied widely. In four of the six systems tested, PFSA and PFCA levels actually increased after filtration. Because the systems remove disinfectants used in city water treatment, they can also leave home pipes susceptible to bacterial growth.

https://pubs.acs.org/doi/10.1021/acs.estlett.0c00004

https://nicholas.duke.edu/news/not-all-home-drinking-water-filters-completely-remove-toxic-pfas

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Association between environmental chemicals co-exposure and peripheral blood immune-inflammatory indicators

Yong Liu, [1,2] Zhihui Zhang, [3] Dongran Han, [2] Yiding Zhao, [1] Xiaoning Yan,[✉1,*] and Shengnan Cui[✉4,5,*]

People seldom come into contact with a single chemical substance in the real environment, but are generally affected by a variety of chemical substances, and there are interactions and non-linear interrelations among them. Therefore, it is necessary to explore the impact of mixed exposure on human health based on the two new methods of WQSR and BKMR. When WQSR is used to study the co-exposure effect of chemical mixtures, this method can only evaluate the co-exposure effect of chemicals in a single effect direction, but it has limitations in evaluating the interaction between chemicals and the double effect direction (19). BKMR is a supervised method for evaluating exposure mixtures, which characterizes the co-exposure effects under different effect directions by non-parametric methods (especially kernel functions). The method can also evaluate potential interactions and non-linear relationships. However, BKMR has limitations when estimating involves simultaneous exposure to high and low levels of chemicals (21). This study integrates various statistical methods to evaluate the effects of chemical mixture co-exposures on the immune-inflammation indicators, demonstrating that mixed chemical exposures (PFAHs, PAHs, Metallic and non-metallic elements) can indeed affect the immune-inflammation indicators, which in turn affects health.

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In this study, we found different trends in the influence of different chemicals on immune-inflammatory indicators. Therefore, in the case of co-exposure to multiple chemicals, the final influence trend of immune-inflammatory indicators may be influenced by exposure measurement of different chemicals, and the higher exposure measurement has a higher weight on the overall trend. In this study, we found that PFDE, PFUA, As and Hg in the comprehensive exposure chemicals had a high negative weight on the immune inflammation spectrum, while P01, P02, P04, and Cd had a high positive weight on the immune inflammation spectrum.

PFASs belong to long-chain perfluoroalkyl carboxylic acids (PFDE and PFUA, etc.) with significant persistence and bioaccumulation potential (26), which have been widely used in food packaging, household cleaning products, furniture, interior decoration, textiles, cosmetics, medical equipment, and other fields (27–29). Polycyclic aromatic hydrocarbons (PAHs) are a group of chemicals formed during the incomplete combustion of coal, oil, gas, and garbage including vehicle exhaust, bitumen, coal tar, wildfires, agricultural burning, etc., (13, 30). Arsenic (As) is a toxic metal that is widely distributed in the environment and is present in soil, food, and water, leading to unavoidable human exposure to arsenic. Cadmium is mainly released from nickel-cadmium batteries, coatings and coatings, plastic stabilizers, fossil fuel combustion, phosphate fertilizer and garbage incineration. Mercury pollution mainly comes from coal burning, non-ferrous metal production and cement production (31).

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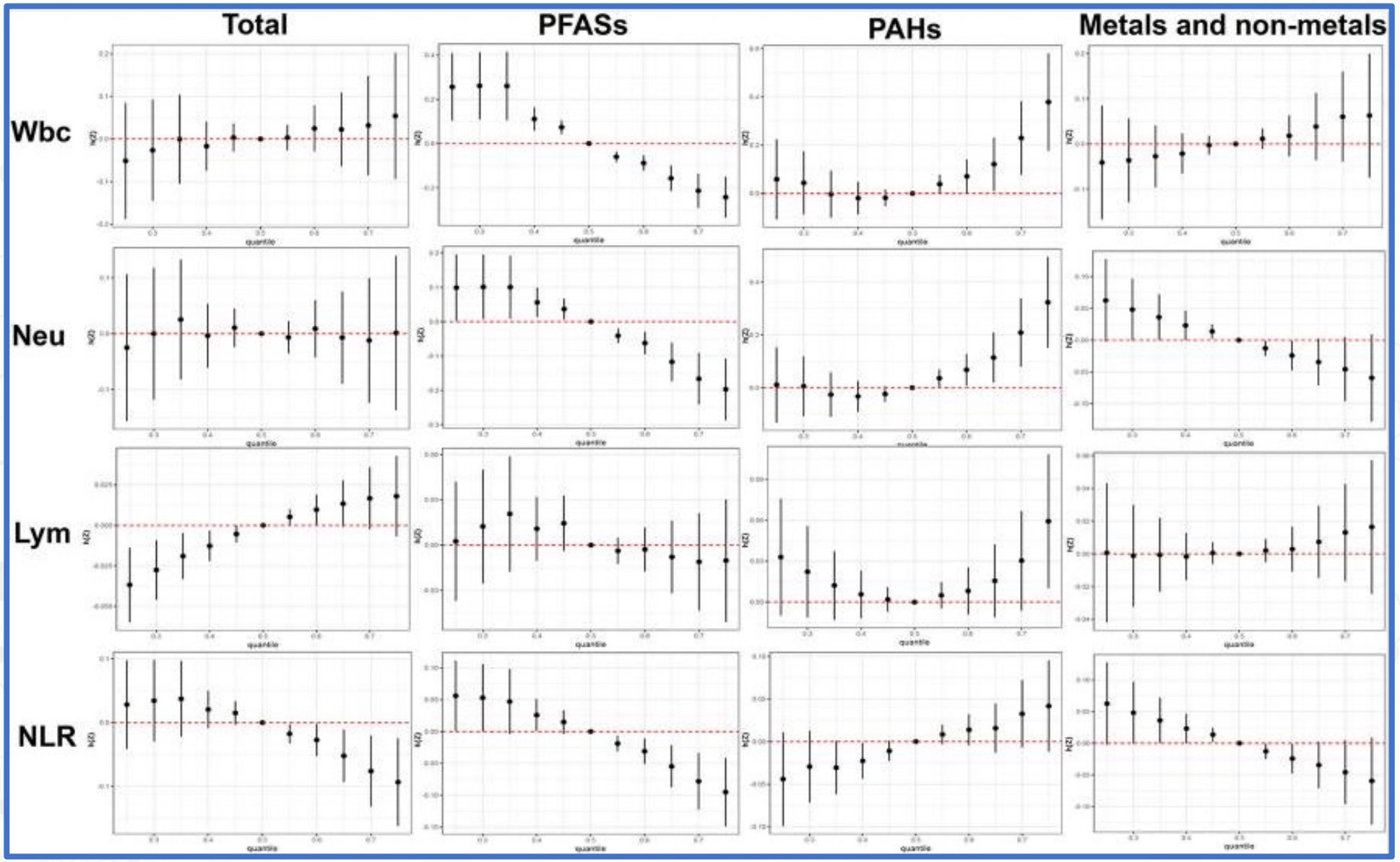
Long-term abnormal action of immune-inflammatory cells in the body can lead to IID (immune-inflammatory disease) (32). One study noted an increased risk of myocardial infarction (MI) with IIDs, 69% increased risk of RA, 41% increased risk of psoriatic arthritis, and increased cardiovascular risk was also observed in patients with psoriasis (33, 34). A recent study showed that the presence of one IID increased the risk of patients developing additional IIDs by 5–62%, and developing any two secondary IIDs increases the risk by 3–75% (35).

This study found that when exposed to all chemicals (PFAHs, PAHs, Metallic and non-metallic elements), Wbc and Lym showed an increasing trend, NLR showed a decreasing trend, while Neu showed no obvious monotonic trend. Since neutrophils and lymphocytes are the main components of white blood cells, NLR reflects the balance between the two aspects of the immune system: acute and chronic inflammation (indicated by neutrophil counts) and adaptive immunity (lymphocyte counts) (6, 36). Therefore, lymphocyte immunity is mainly activated under comprehensive chemical exposure. Lymphocytes are composed of T lymphocytes, B lymphocytes and NK cells, among which T lymphocytes are involved in cellular immunity and are also considered to be major drivers of many inflammatory and autoimmune diseases (37).


Total
PFASs
PAHs
Metals and non-metals
Wbc
Neu
Lym
NLR
quantile

CBC With Differential/Platelet

Test	Current Result and Flag	Previous Result and Date		Units	Reference Interval
WBC [01]	5.8	5.5	06/21/2023	x10E3/uL	3.4-10.8
RBC [01]	4.21	4.40	06/21/2023	x10E6/uL	3.77-5.28
Hemoglobin [01]	12.7	13.2	06/21/2023	g/dL	11.1-15.9
Hematocrit [01]	39.1	40.8	06/21/2023	%	34.0-46.6
MCV [01]	93	93	06/21/2023	fL	79-97
MCH [01]	30.2	30.0	06/21/2023	pg	26.6-33.0
MCHC [01]	32.5	32.4	06/21/2023	g/dL	31.5-35.7
RDW [01]	12.8	12.3	06/21/2023	%	11.7-15.4
Platelets [01]	246	268	06/21/2023	x10E3/uL	150-450
Neutrophils [01]	43	49	06/21/2023	%	Not Estab.
Lymphs [01]	47	38	06/21/2023	%	Not Estab.
Monocytes [01]	5	6	06/21/2023	%	Not Estab.
Eos [01]	3	5	06/21/2023	%	Not Estab.
Basos [01]	2	2	06/21/2023	%	Not Estab.
Neutrophils (Absolute) [01]	2.5	2.7	06/21/2023	x10E3/uL	1.4-7.0
Lymphs (Absolute) [01]	2.7	2.1	06/21/2023	x10E3/uL	0.7-3.1
Monocytes(Absolute) [01]	0.3	0.3	06/21/2023	x10E3/uL	0.1-0.9
Eos (Absolute) [01]	0.2	0.3	06/21/2023	x10E3/uL	0.0-0.4
Baso (Absolute) [01]	0.1	0.1	06/21/2023	x10E3/uL	0.0-0.2
Immature Granulocytes [01]	0	0	06/21/2023	%	Not Estab.
Immature Grans (Abs) [01]	0.0	0.0	06/21/2023	x10E3/uL	0.0-0.1

CBC With Differential/Platelet

Test	Current Result and Flag	Previous Result and Date		Units	Reference Interval
WBC [01]	5.9	5.5	07/13/2023	x10E3/uL	3.4-10.8
RBC [01]	4.86	4.78	07/13/2023	x10E6/uL	4.14-5.80
Hemoglobin [01]	14.6	14.5	07/13/2023	g/dL	13.0-17.7
Hematocrit [01]	43.7	43.0	07/13/2023	%	37.5-51.0
MCV [01]	90	90	07/13/2023	fL	79-97
MCH [01]	30.0	30.3	07/13/2023	pg	26.6-33.0
MCHC [01]	33.4	33.7	07/13/2023	g/dL	31.5-35.7
RDW [01]	12.5	12.6	07/13/2023	%	11.6-15.4
Platelets [01]	258	253	07/13/2023	x10E3/uL	150-450
Neutrophils [01]	45	54	07/13/2023	%	Not Estab.
Lymphs [01]	46	38	07/13/2023	%	Not Estab.
Monocytes [01]	7	7	07/13/2023	%	Not Estab.
Eos [01]	1	0	07/13/2023	%	Not Estab.
Basos [01]	1	1	07/13/2023	%	Not Estab.
Neutrophils (Absolute) [01]	2.8	2.9	07/13/2023	x10E3/uL	1.4-7.0
Lymphs (Absolute) [01]	2.7	2.1	07/13/2023	x10E3/uL	0.7-3.1
Monocytes(Absolute) [01]	0.4	0.4	07/13/2023	x10E3/uL	0.1-0.9
Eos (Absolute) [01]	0.0	0.0	07/13/2023	x10E3/uL	0.0-0.4
Baso (Absolute) [01]	0.0	0.0	07/13/2023	x10E3/uL	0.0-0.2
Immature Granulocytes [01]	0	0	07/13/2023	%	Not Estab.
Immature Grans (Abs) [01]	0.0	0.0	07/13/2023	x10E3/uL	0.0-0.1

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Inflammation is a complex process in which various cells, such as B lymphocytes, T lymphocytes, epithelial cells, etc.), pro-inflammatory cytokines [such as IL-1β, IL-6 and tumor necrosis factor α (TNFα)], transcription factor nuclear factor κB (NF-κB) and signal transduction and transcriptional activator 3 (STAT3) play important roles in inflammatory responses. Among them, IL-6 is a pleiotropic cytokine that is a major player in chronic inflammation (which is closely associated with chronic inflammatory diseases, autoimmune diseases, and cancer) and cytokine storm (such as the cytokine storm of COVID-19) (41–45). The synergistic action of NF-κB and STAT3 can induce super activation of NF-κB and subsequently produce a variety of inflammatory factors (46). Since IL-6 is a target of NF-κB, both NF-κB and STAT3 are activated in non-immune cells, triggering a positive feedback loop for NF-κB activation via the IL-6 STAT3 axis. This positive feedback loop is called the IL-6 amplifier (IL-6 Amp), which can be further enhanced because activated IL-6 Amp can enhance chemokine production and recruit lymphocytes in lesions, including Th17 cells (46–51). Inflammatory bowel disease, including chronic inflammatory disease, autoimmune diseases and cancer) is a non-immune cells and immune cells through complex interactions between IL - 6 Amp induction, confirmed the model between inflammation and cancer is a process of continuous, rather than by tissue specificity immune tolerance and the destruction of the cancer-causing mutations cause (52). In conclusion, exposure to mixed chemicals (PFASs, PAHs, and Metals), as long-term pro-inflammatory factors in human beings, induces chronic inflammation in human body by triggering IL-6 Amp, which not only affects human health, but also continuously increases medical expenditure (53, 54).

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Neurotransmission Targets of Per- and Polyfluoroalkyl Substance Neurotoxicity: Mechanisms and Potential Implications for Adverse Neurological Outcomes

Per- and polyfluoroalkyl substances (PFAS) are a group of persistent environmental pollutants that are ubiquitously found in the environment and virtually in all living organisms, including humans. PFAS cross the blood–brain barrier and accumulate in the brain. Thus, PFAS are a likely risk for neurotoxicity. Studies that measured PFAS levels in the brains of humans, polar bears, and rats have demonstrated that some areas of the brain accumulate greater amounts of PFAS. Moreover, in humans, there is evidence that PFAS exposure is associated with attention-deficit/hyperactivity disorder (ADHD) in children and an increased cause of death from Parkinson's disease and Alzheimer's disease in elderly populations. Given possible links to neurological disease, critical analyses of possible mechanisms of neurotoxic action are necessary to advance the field. This paper critically reviews studies that investigated potential mechanistic causes for neurotoxicity including (1) a change in neurotransmitter levels, (2) dysfunction of synaptic calcium homeostasis, and (3) alteration of synaptic and neuronal protein expression and function. We found growing evidence that PFAS exposure causes neurotoxicity through the disruption of neurotransmission, particularly the dopamine and glutamate systems, which are implicated in age-related psychiatric illnesses and neurodegenerative diseases. Evaluated research has shown there are highly reproduced increased glutamate levels in the hippocampus and catecholamine levels in the hypothalamus and decreased dopamine in the whole brain after PFAS exposure. There are significant gaps in the literature relative to the assessment of the nigrostriatal system (striatum and ventral midbrain) among other regions associated with PFAS-associated neurologic dysfunction observed in humans. In conclusion, evidence suggests that PFAS may be neurotoxic and associated with chronic and age-related psychiatric illnesses and neurodegenerative diseases. Thus, it is imperative that future mechanistic studies assess the impact of PFAS and PFAS mixtures on the mechanism of neurotransmission and the consequential functional effects.

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Mounting data suggest PFAS are likely neurotoxic and may modulate neurological function (exemplified by an outstanding recent review by Cao and Ng in 2021,[55] broadly addressing neurotoxicity). PFAS accumulate in the greatest amounts in the blood, liver, lung, and kidney.[69] Reports of neurotoxicity are present at relatively low amounts accumulating in the brain.[23,35,66,67] However, recent experiments quantifying PFAS in cerebrospinal fluid (CSF) and post-mortem human brains suggest these earlier studies may be underestimating the concentrations that PFAS accumulate in the brain.[63] When a single dose of PFOS was administered, it rapidly accumulated in the brain of rats within hours of administration of a single dose.[35] PFAS have been demonstrated to accumulate in the brains of many species including adult post-mortem humans, [65–67] human fetuses,[70] polar bears,[8,9,44] mice,[36] rats,[23,35,61,63] and northern leopard frogs.[6] Considering the heterogeneity of the brain structure, studies have additionally investigated the region-specific accumulation of PFAS, which could potentially target which brain functions are at greatest risk of dysfunction and disease.

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The human brain regions with the highest total PFAS levels were the hypothalamus with 206.9 ng/g and the caudate nucleus with 109.2 ng/g, followed by the cerebellum (54.4 ng/g) and lenticular nucleus (45.3 ng/g).[65] Interestingly, the polar bear samples collected in 2006 also had the largest total PFAS in the hypothalamus (60.1 ng/g), followed by the pons/medulla (48.6 ng/g) and thalamus (41.2 ng/g), whereas the 2011–2012 polar bear samples had the highest PFAS levels in the brain stem (35.7 ng/g) and occipital lobe (31.3 ng/g).[9,44] In the rats that were only exposed to high levels of PFOS, the 1 mg/kg/d exposure had the highest accumulation in the brain stem (363 ng/g) and the rest of the brain (striatum and thalamus with 396 ng/g) and the 10 mg/kg/d dose had the highest levels in the hypothalamus (15 706 ng/g) and hippocampus (8966 ng/g).[23]

The cross-species variability, variability in composition and levels of the exposed PFAS mixtures, and differences in distribution and elimination of PFAS from these brain regions may explain the variability in the brain region PFAS levels. Importantly, the regions with the highest long-term accumulation of PFAS may not necessarily be at the highest risk of neurotoxicity due to the many region-specific neuron and glia populations and unique connectivity to other regions of the brain.[45] Therefore, to understand the mechanism of how PFAS are inducing neurotoxicity and the impact on human disease, we must look at the functional targets within cells that are most susceptible to adverse effects from PFAS exposure, which includes neurotransmission.

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Rodent models exposed to PFAS before weaning and near and after sexual maturity had increases in dopamine and serotonin in the whole brain,[36] hippocampus,[30,34] and hypothalamus.[28,33] Interestingly, male mice developmentally exposed to PFAS mixtures, frogs developmentally exposed to PFOS or PFOA, and female juvenile Atlantic cod exposed to a PFAS mixture resulted in decreases in whole brain dopamine levels.[24–26] Long et al. detected decreases in dopamine levels in the dorsal striatum and increases in hippocampal glutamate levels of male and female mice subchronically exposed to PFAS for 3 months during adulthood.[27] These studies suggest potential deficits in dopaminergic neurotransmission and glutamatergic excitotoxity from longer PFAS exposures.[27]

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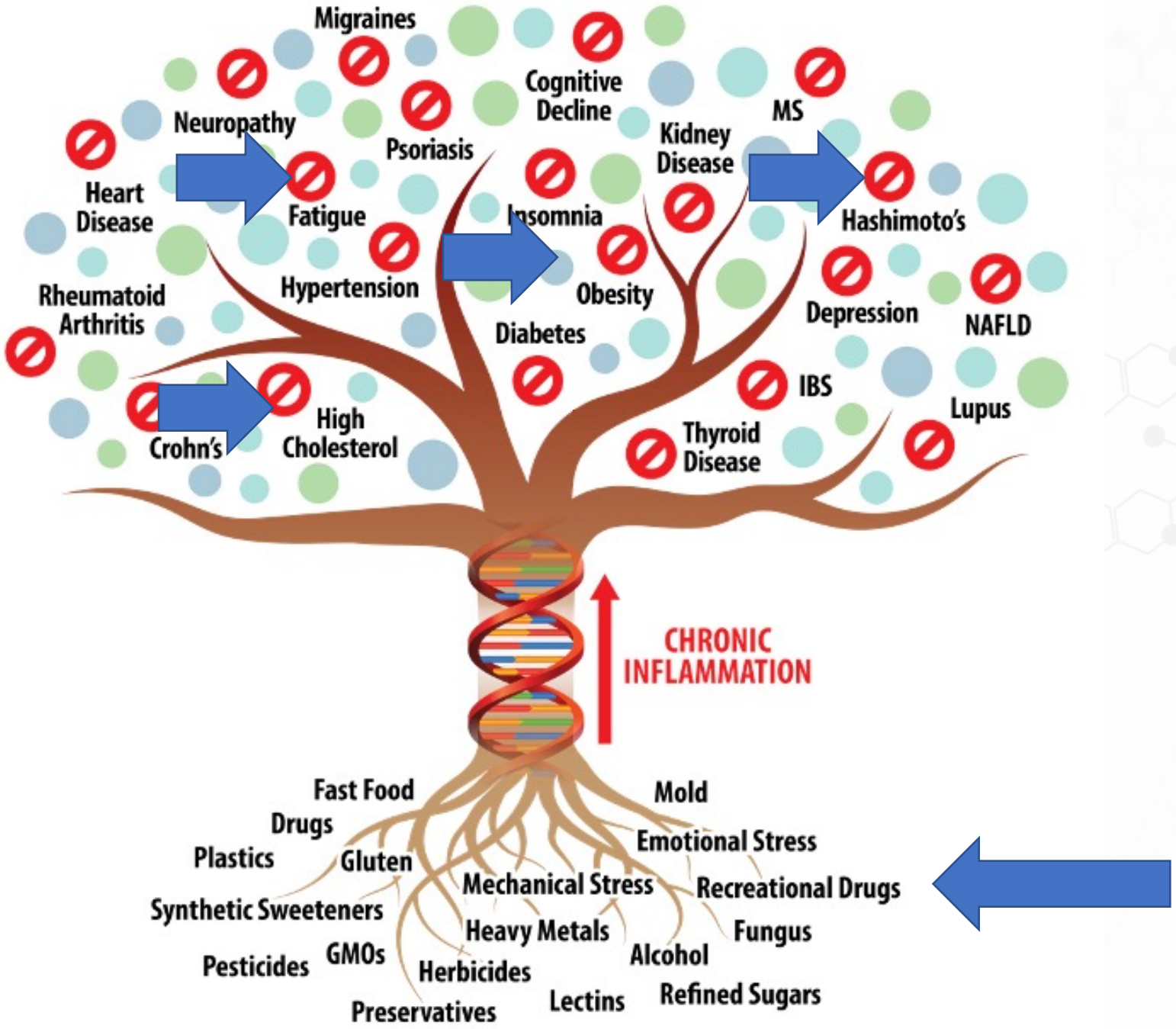
3.1.3. GABA. There is some evidence in animal and cell models that the PFAS, PFOS and PFOA, may alter GABAergic neurotransmission. Adult male mice lactationally exposed to PFOS were determined to have elevated levels of extracellular glutamate and GABA and no changes in extracellular glycine in the dorsal hippocampus.[30] Another developmental PFOS study in rats conducted metabolomic analysis of the cortex and found elevated levels of multiple inhibitory neurotransmitters including taurine, GABA, and glycine.[32] Furthermore, in human induced pluripotent stem cell (hiPSC)-derived neurons, Tukker et al. demonstrated PFOS and PFOA acted as noncompetitive $GABA_A$ receptor antagonists.[85] Taken together, it can be hypothesized that PFAS inhibit GABA synaptic neurotransmission by competitively inhibiting the $GABA_A$ receptor and increased GABA excretion (and other inhibitory neurotransmitters) may be a brain region-dependent compensatory mechanism.


Migraines
Neuropathy
Psoriasis
Cognitive Decline
MS
Kidney Disease
Heart Disease
Fatigue
Insomnia
Hashimoto's
Hypertension
Obesity
Depression
NAFLD
Rheumatoid Arthritis
Diabetes
IBS
Lupus
Crohn's
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