Casual Friday Series

Toxicity and Fibromyalgia

A Biogenetix Clinical Presentation

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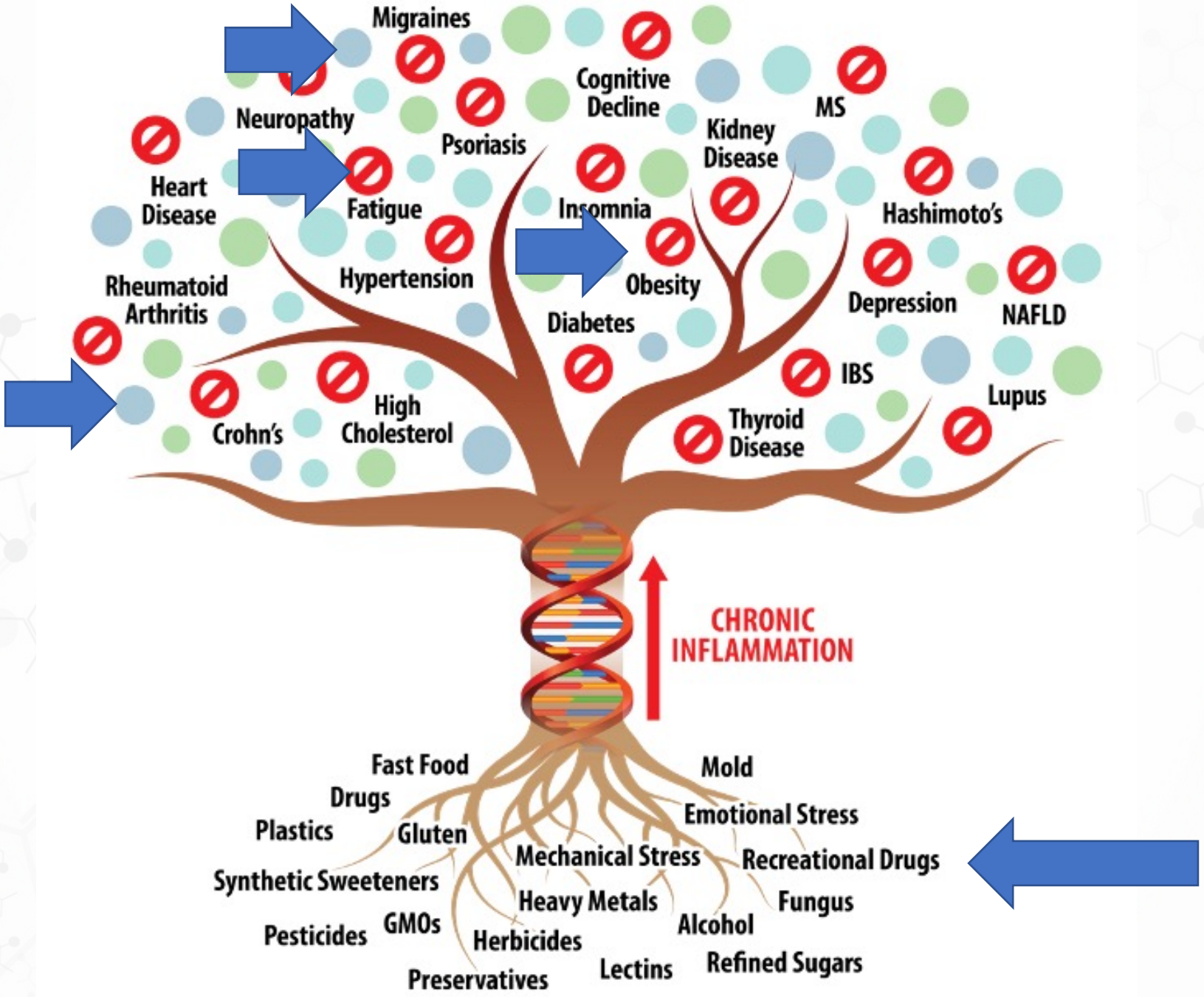
Disclaimer

- *Information in this presentation is not intended to diagnose, treat, reverse, cure, or prevent any disease. While this presentation is based on medical literature, findings, and text, The following statements have not been evaluated by the FDA.*
- *The information provided in this presentation is for your consideration only as a practicing health care provider. Ultimately you are responsible for exercising professional judgment in the care of your own patients.*


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

Next Gen Diagnostic Strategies:

- Headaches
- Neuralgia
- Neuropathy
- DJD/DDD
- Diffuse mm px syndromes
- Fibromyalgia
- RA
- Etc.

Diagnosis

In the past, doctors would check 18 specific points on a person's body to see how many of them were painful when pressed firmly. Newer guidelines from the American College of Rheumatology don't require a tender point exam.

Instead, the main factor needed for a fibromyalgia diagnosis is widespread pain throughout your body for at least three months.

To meet the criteria, you must have pain in at least four of these five areas:

- **Left upper region,** including shoulder, arm or jaw
- **Right upper region,** including shoulder, arm or jaw
- **Left lower region,** including hip, buttock or leg
- **Right lower region,** including hip, buttock or leg
- **Axial region,** which includes neck, back, chest or abdomen

https://www.mayoclinic.org/diseases-conditions/fibromyalgia/diagnosis-treatment/drc-20354785#:~:text=Newer%20guidelines%20from%20the%20American,for%20at%20least%20three%20months.

Treatment

In general, treatments for fibromyalgia include both medication and self-care strategies. The emphasis is on minimizing symptoms and improving general health. No one treatment works for all symptoms, but trying a variety of treatment strategies can have a cumulative effect.

Medications

Medications can help reduce the pain of fibromyalgia and improve sleep. Common choices include:

- **Pain relievers.** Over-the-counter pain relievers such as acetaminophen (Tylenol, others), ibuprofen (Advil, Motrin IB, others) or naproxen sodium (Aleve, others) may be helpful. Opioid medications are not recommended, because they can lead to significant side effects and dependence and will worsen the pain over time.
- **Antidepressants.** Duloxetine (Cymbalta) and milnacipran (Savella) may help ease the pain and fatigue associated with fibromyalgia. Your doctor may prescribe amitriptyline or the muscle relaxant cyclobenzaprine to help promote sleep.
- **Anti-seizure drugs.** Medications designed to treat epilepsy are often useful in reducing certain types of pain. Gabapentin (Neurontin) is sometimes helpful in reducing fibromyalgia symptoms, while pregabalin (Lyrica) was the first drug approved by the Food and Drug Administration to treat fibromyalgia.

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Therapies

A variety of different therapies can help reduce the effect that fibromyalgia has on your body and your life. Examples include:

- **Physical therapy.** A physical therapist can teach you exercises that will improve your strength, flexibility and stamina. Water-based exercises might be particularly helpful.
- **Occupational therapy.** An occupational therapist can help you make adjustments to your work area or the way you perform certain tasks that will cause less stress on your body.
- **Counseling.** Talking with a counselor can help strengthen your belief in your abilities and teach you strategies for dealing with stressful situations.

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Toxicol Rep. 2021; 8: 1538–1557.
Published online 2021 Jul 31. doi: 10.1016/j.toxrep.2021.07.014
PMCID: PMC8365328
PMID: 34430217

Endocrine disruptors also function as nervous disruptors and can be renamed endocrine and nervous disruptors (ENDs)

Gilles-Eric Seralini* and Gerald Jungers

Endocrine disruption (ED) and endocrine disruptors (EDs) emerged as scientific concepts in 1995, after numerous chemical pollutants were found to be responsible for reproductive dysfunction. The World Health Organization established in the United Nations Environment Programme a list of materials, plasticizers, pesticides, and various pollutants synthesized from petrochemistry that impact not only reproduction, but also hormonal functions, directly or indirectly. Cells communicate via either chemical or electrical signals transmitted within the endocrine or nervous systems. To investigate whether hormone disruptors may also interfere directly or indirectly with the development or functioning of the nervous system through either a neuroendocrine or a more general mechanism, we examined the scientific literature to ascertain the effects of EDs on the nervous system, specifically in the categories of neurotoxicity, cognition, and behaviour. To date, we demonstrated that all of the 177 EDs identified internationally by WHO are known to have an impact on the nervous system. Furthermore, the precise mechanisms underlying this neurodisruption have also been established. It was previously believed that EDs primarily function via the thyroid. However, this study presents substantial evidence that approximately 80 % of EDs operate via other mechanisms. It thus outlines a novel concept: EDs are also neurodisruptors (NDs) and can be collectively termed endocrine and nervous disruptors (ENDs). Most of ENDs are derived from petroleum residues, and their various mechanisms of action are similar to those of “spam” in electronic communications technologies. Therefore, ENDs can be considered as an instance of spam in a biological context.

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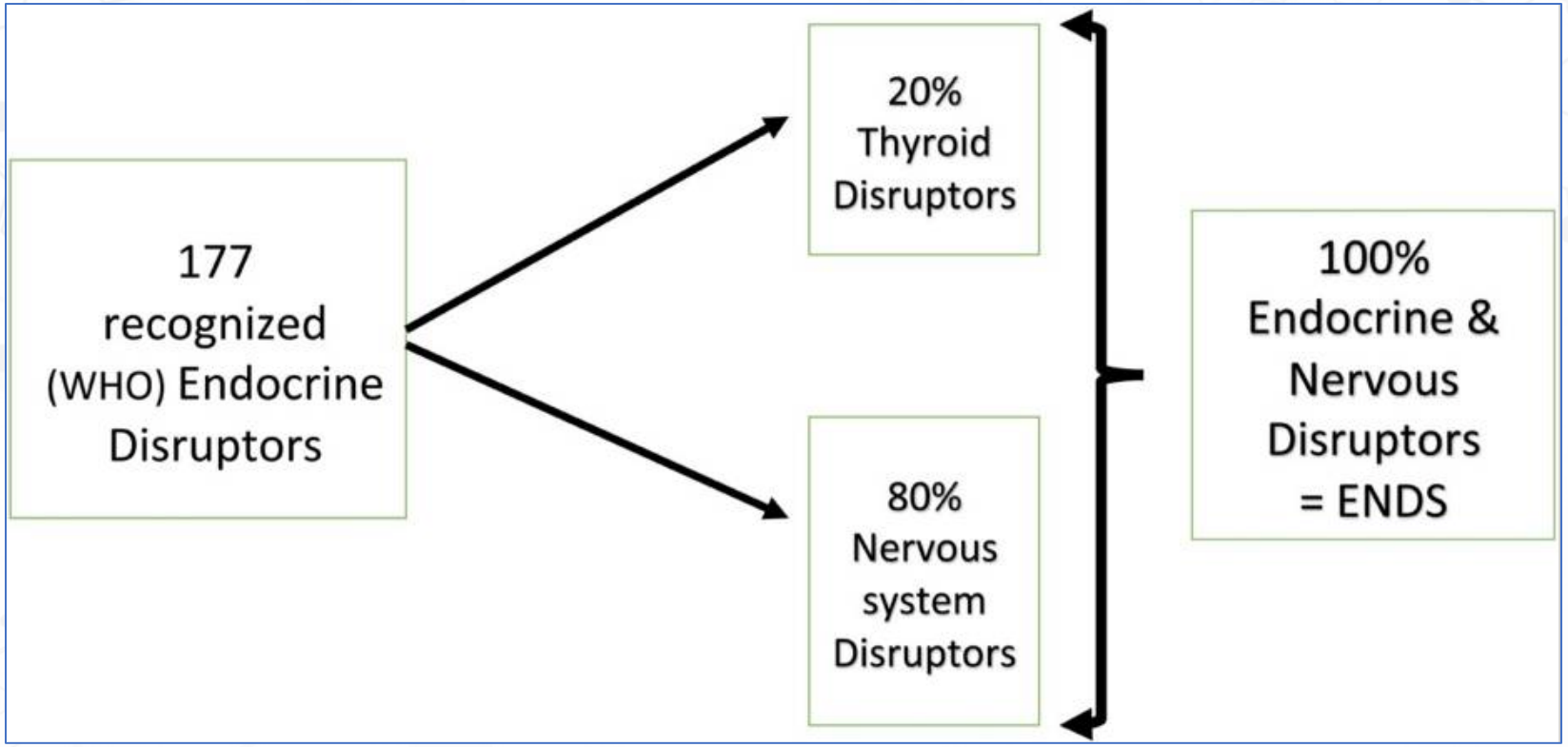

Front Neurol. 2023; 14: 940378.
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Association between urinary phthalate levels and chronic pain in US adults, 1999–2004: A nationally representative survey

Guoping Jin, [1] Yaoyao Nie, [2] Jiayao Fan, [3] Ye Yang, [2] Dingwan Chen, [2] Yingjun Li, [2] and Li Ju[✉4,*]

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Phthalates (PAEs), esters of 1,2-benzene dicarboxylic acid, are synthetic organic chemicals commonly used in industrial or consumer applications as plasticizers. PAEs are easily chipped off products and become contaminants in the workplace, indoor air, or food (6–8). Subsequently, people are daily exposed to them by inhalation, oral intake, or skin contact. In a cross-sectional survey, most people had detectable PAEs metabolites in urine, including di-(2-ethylhexyl)-phthalate (DEHP), diethyl phthalate (DEP), and di-n-butyl phthalate (DBP) (9). With this constant exposure, various human adverse health outcomes occur. Studies showed PAEs exposure led to reproductive toxicity, developmental toxicity, and cardiovascular disorders (10–12). Exposure to PAEs could also affect the pathogenesis of endometriosis (13). Our previous studies showed an association between PAEs with sarcopenia and osteoarthritis (14, 15). However, there is much less information about the association between PAEs and chronic pain. Usually, chronic pain is a common concomitant symptom of another severe disease, such as coronary heart disease, endometriosis, and osteoarthritis, as mentioned above. More than that, chronic pain has been recognized as an independent condition with its own taxonomy and definitions (16). Thus, the question of whether there is direct pain after PAEs exposure.

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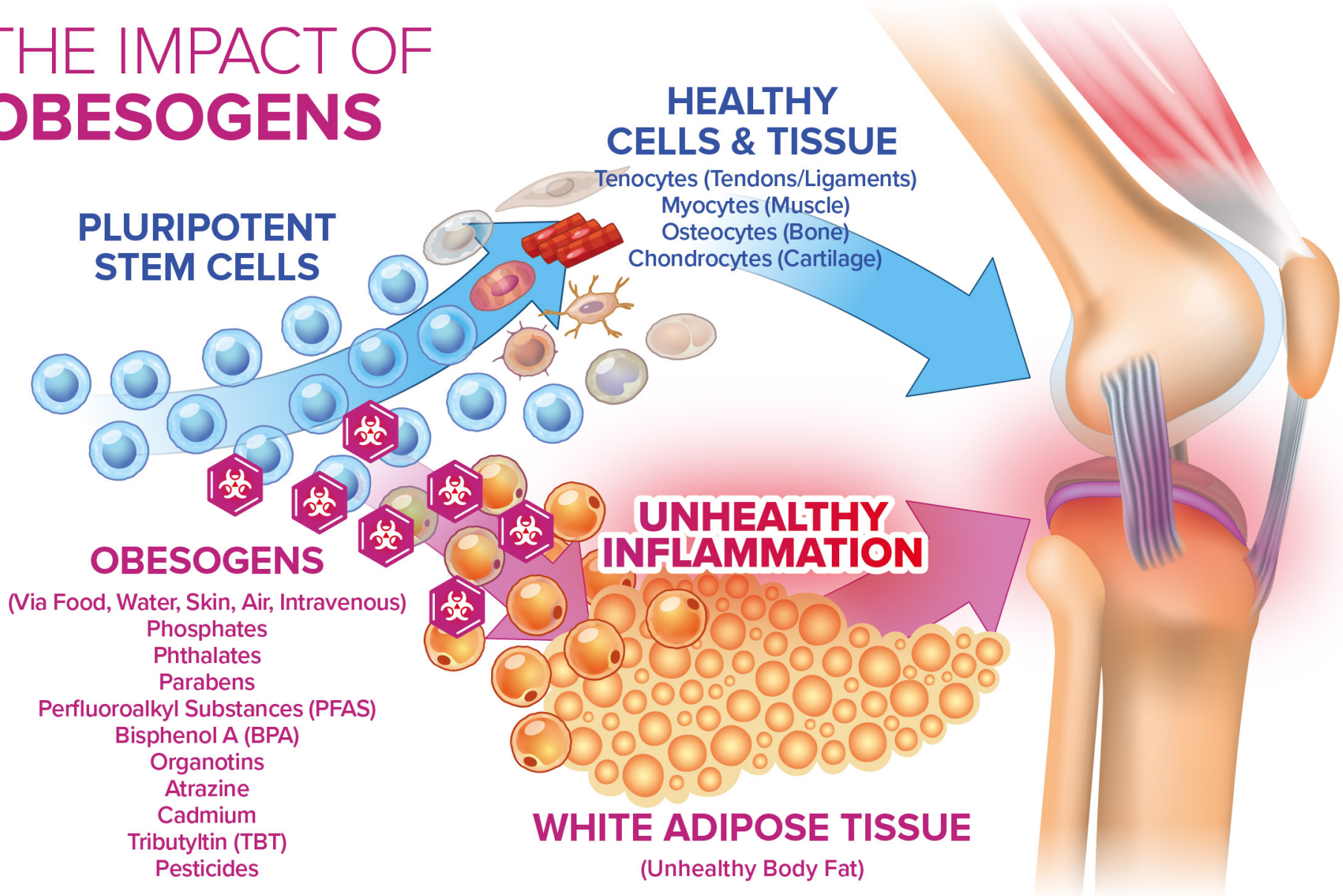
A total of 4,196 participants were considered in our final analysis. Chronic pain prevalence reached 52.19% (n = 2,138) among the participants, with women accounting for a large proportion (57.75% vs. 42.25%). After multivariable logistic regression analysis, a higher prevalence of chronic pain was observed among participants in the third tertile of mono-(2-ethyl)-hexyl phthalate (MEHP) (OR = 1.23, 95% CI = 1.02–1.48, P = 0.034) and mono-benzyl phthalate (MBzP) (OR = 1.28, 95% CI = 1.04–1.58, P = 0.022) in our adjusted model. The logtransformed concentration of MBzP also showed a significant association with chronic pain prevalence (OR = 1.09, 95% CI = 1.01–1.18, P = 0.036) in the adjusted model. In further analysis, the positive correlations of urinary phthalate metabolites with chronic pain remained robust when stratified by gender and chronic pain site.

THE IMPACT OF **OBESOGENS**



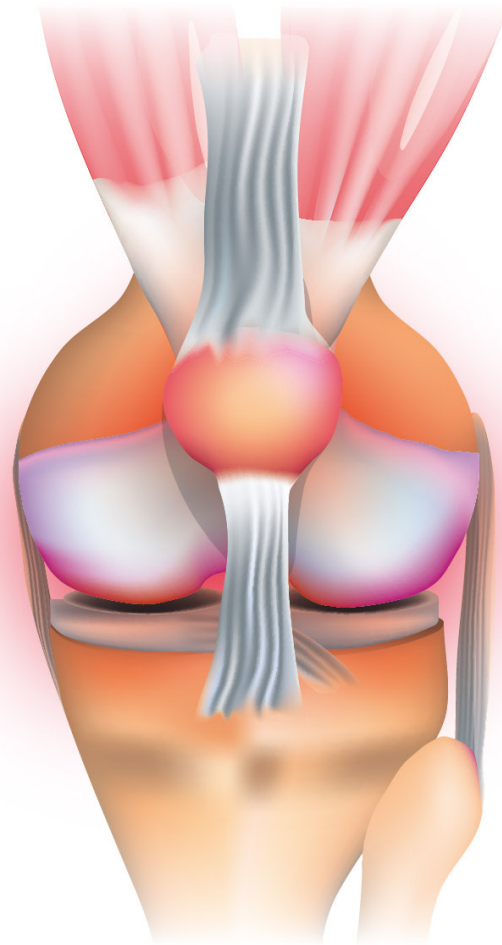
High (>95th percentile)

Mycotoxins | Heavy Metals | Environmental Toxins

TEST NAME	CURRENT RESULT	PREVIOUS RESULT	CURRENT RESULT / PREVIOUS RESULT	REFERENCE
Aflatoxin G1	17.99		0 – 3.68 – 6.53	≤6.53 ng/g
Fumonisins B1	9.03		0 – 3.45 – 6.13	≤6.13 ng/g
Fumonisins B2	17.23		0 – 4.05 – 7.2	≤7.2 ng/g
Ochratoxin A (OTA)	22.89		0 – 3.83 – 6.8	≤6.8 ng/g
Patulin	34.1		0 – 6.53 – 11.6	≤11.6 ng/g
Satratoxin H	0.35		0 – 0.1 – 0.18	≤0.18 ng/g
Barium*	12.04		0 – 2.33 – 5.59	≤5.59 ug/g
Tungsten*	0.84		0 – 0.12 – 0.33	≤0.33 ug/g
2-Hydroxyethyl Mercapturic Acid (HEMA)*	18.72		0 – 1.7 – 4.75	≤4.75 ug/g
2-Hydroxyisobutyric Acid (2HIB)	1585.91		0 – 795 – 1215	≤1215.72 ug/g
2,2-bis(4-Chlorophenyl) acetic acid (DDA)	24.07		0 – 7.9 – 19	≤19 ug/g
Bisphenol A (BPA)*	17.47		0 – 2.12 – 5.09	≤5.09 ug/g
Mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP)*	74.88		0 – 8.99 – 23.4	≤23.4 ug/g

* Indicates NHANES population data reference ranges.

THE **ADIPOKINE** SPIRAL



1
Obesogens Drive
Stem Cells into Adipocytes

2
Adiposity Increases

3
Increased
Adipokine Activity

4
Triggering
Increased
Unhealthy Inflammation

5
Ongoing
Tissue Damage

High (>95th percentile)

Mycotoxins | Environmental Toxins

TEST NAME	CURRENT RESULT	PREVIOUS RESULT	CURRENT RESULT / PREVIOUS RESULT	REFERENCE
Ochratoxin A (OTA)	15.35		0 — 3.83 — 6.8	≤6.8 ng/g
Glyphosate	24.1		0 — 1.65 — 7.6	≤7.6 ug/g
Mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP)*	55.96		0 — 8.99 — 23.4	≤23.4 ug/g

* Indicates NHANES population data reference ranges.

Moderate (75th-95th percentile)

Mycotoxins | Heavy Metals | Environmental Toxins

TEST NAME	CURRENT RESULT	PREVIOUS RESULT	CURRENT RESULT / PREVIOUS RESULT	REFERENCE
Aflatoxin G2	6.41		0 — 6.08 — 10.8	≤10.8 ng/g
Aflatoxin M1	5.63		0 — 3.6 — 6.4	≤6.4 ng/g
Diacetoxyscirpenol (DAS)	2.51		0 — 2.4 — 4.27	≤4.27 ng/g
Enniatin B1(ENN B1)	0.18		0 — 0.13 — 0.22	≤0.22 ng/g
Gliotoxin	179.07		0 — 116 — 207	≤207.87 ng/g

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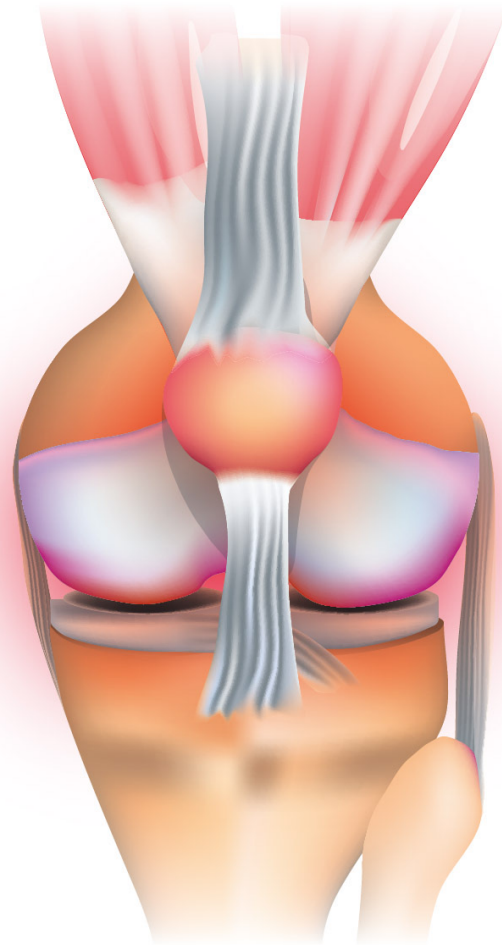

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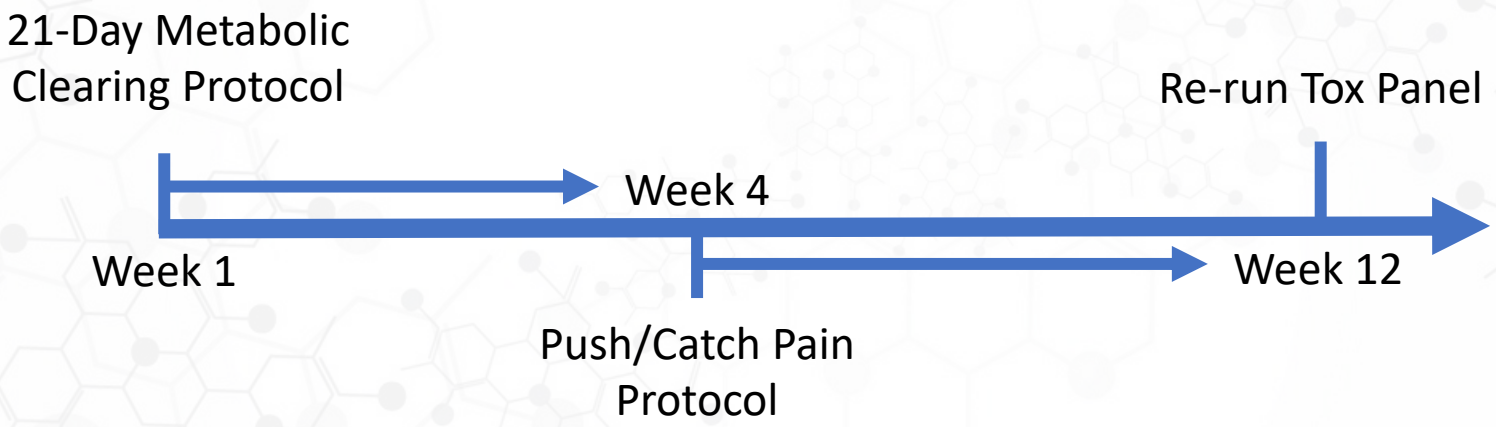
Bolt-On Treatment Strategy



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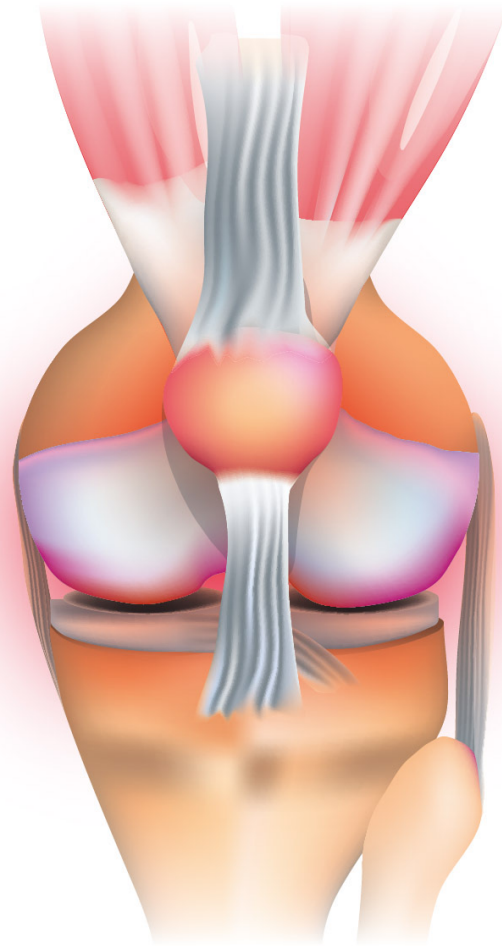


1 Obesogens Drive Stem Cells into Adipocytes

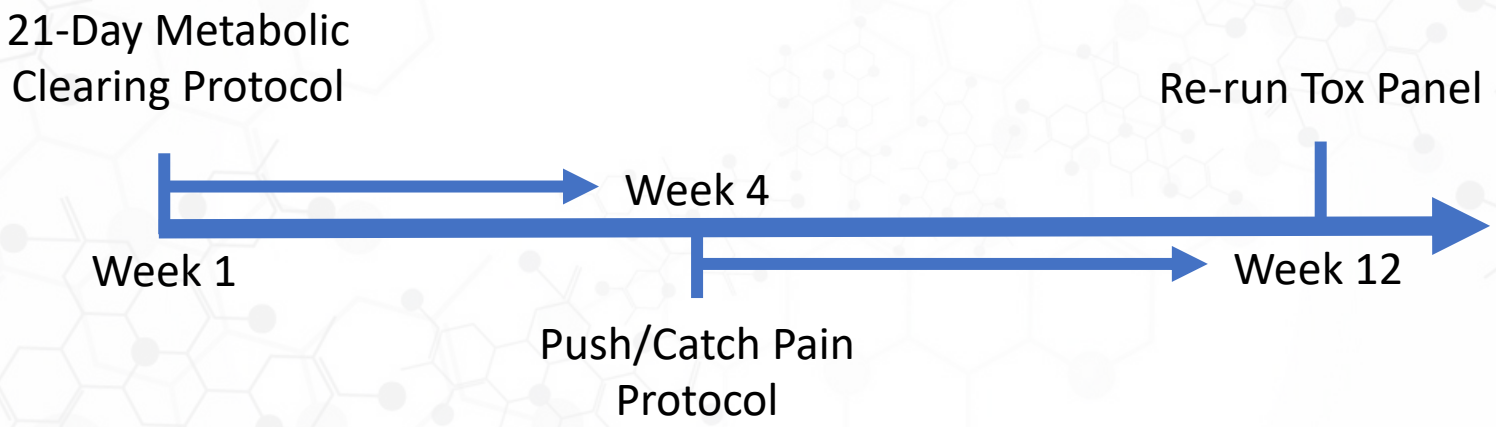
2 Adiposity Increases

3 Increased Adipokine Activity

4 Triggering Increased Unhealthy Inflammation

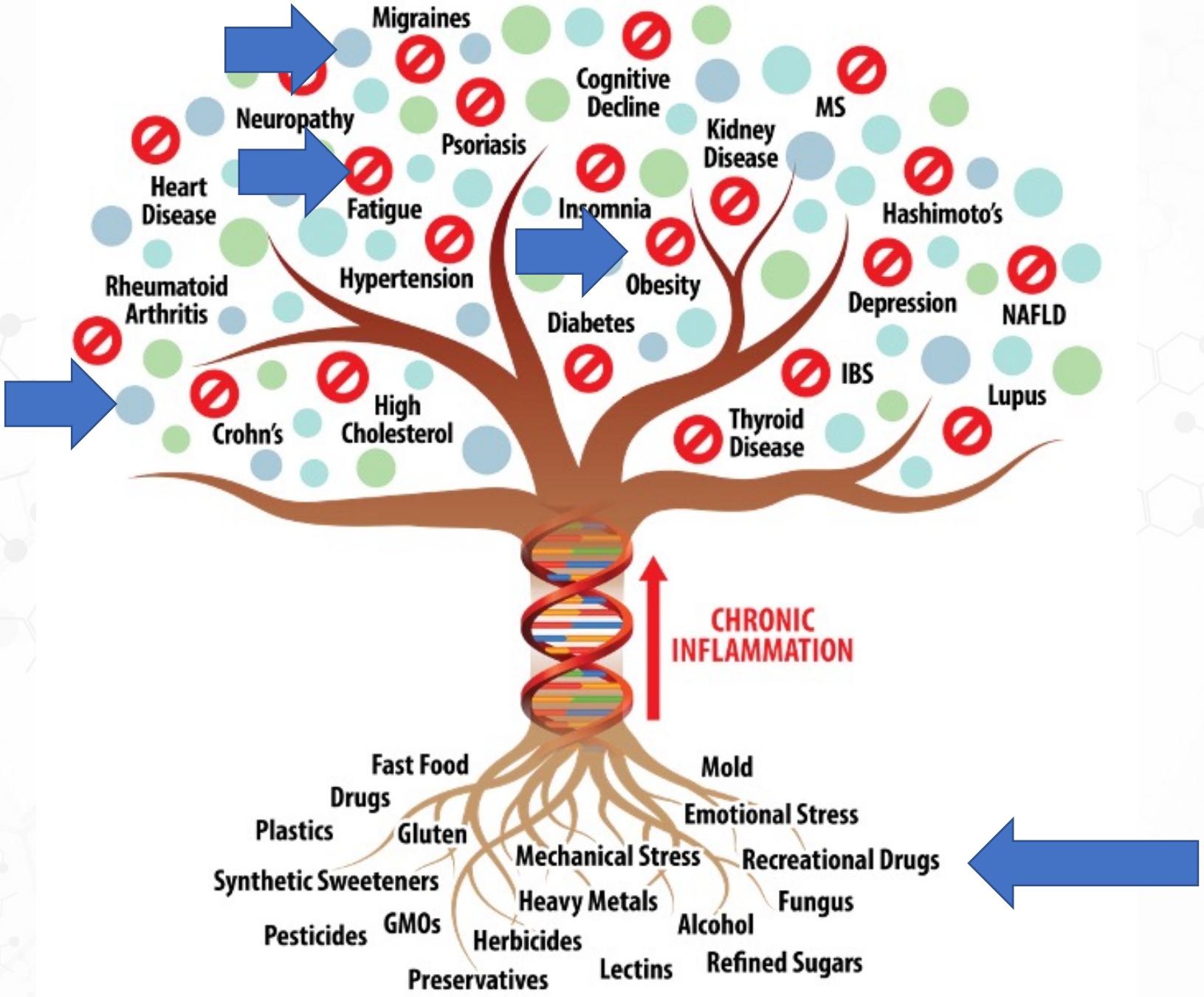
5 Ongoing Tissue Damage

Bolt-On Treatment Strategy



Step 1: 21-Day Metabolic Clearing Kit

Step 2: Push/Catch Pain Protocol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