

Key Features:

Supports Multiple Detox Mechanisms:

- Active B-vitamins & essential minerals - support **Phases 1 & 2 detox** and prevent the overload of toxic/reactive metabolites
- **D-glucarate** – supports glucuronidation & reduces xenobiotic metabolites from being recycled
- **NAC & Selenium** - quench reactive oxygen species
- **Support the collateral metabolic pathways** of the methylation cycle and reduce the symptoms caused by the metabolic overload from methylation enhancement, such as anxiety, sulfite sensitivity, histamine intolerance & allergic reactions, insomnia & fatigue, GI Upset, and memory decline.

Description:

Xenobiotics are a group of chemical substances that are NOT naturally produced or expected to be present within our body. They may be grouped as drugs, pollutants, heavy metals, food additives, and herbicides/pesticides.

Xenobiotics affect our health profoundly as they can impact many biochemical pathways in our body, such as **disrupting the endocrine system, inhibiting important rate-limiting enzymes, generating free radicals, and damaging genetic materials**.

Though our body packs various detoxification mechanisms, chronic exposure to xenobiotics can attenuate our detox ability as many vital nutrients are depleted and genetic materials are damaged.

XenobioX is a synergistic formulation to provide support for xenobiotic detoxification. By providing the active B-vitamins, essential minerals and amino acids, it helps to enhance the detoxification pathways (ie. hydroxylation, methylation, sulfation, deamination/transamination, glutathione-conjugation, glucuronidation), as well as nourish the **collateral pathways of the methylation cycle** (ie. sulfite, COMT/MAO, histamine, SOD/GST).

Phase 1 & Phase 2 Detoxification

Phase 1 detoxification is the process of making fat-soluble toxins more water-soluble so that the toxins can be metabolized and excreted. Biochemical reactions involved may include oxidation, reduction, hydrolysis, and hydroxylation. Common cofactors involved in Phase 1 are B2 & B3 (via FAD/NAD), copper, magnesium, and iron.

Phase 2 detoxification is responsible for the active & toxic metabolites produced from Phase 1. It works via various types of conjugating reactions, such as methylation, sulfation, glucuronidation, and glutathione-conjugation.

Methylation – The Primary Target

Methylation is the most important reaction of all as it is involved in **DNA turnover, neurotransmitter synthesis and reduction, detoxification, and tissue regeneration**. Many xenobiotics

Quantity: 84 Vegetarian Capsules

Ingredients (per 2 capsules):

Vitamin B1 (from thiamine HCl).....	30 mg
Vitamin B2 (from riboflavin-5'-phosphate).....	20 mg
Niacinamide.....	50 mg
Vitamin B5 (from calcium d-pantothenate).....	50 mg
Vitamin B6 (from calcium pyridoxal-5'-phosphate).....	40 mg
5-MTHF (from calcium 5-methylfolate).....	800 mcg
Vitamin B12 (methylcobalamin).....	600 mcg
Zinc (from zinc bisglycinate).....	20 mg
Molybdenum (from molybdenum glycinate).....	200 mcg
Selenium (from selenium bisglycinate).....	200 mcg
N-Acetyl-L-Cysteine.....	500 mg
D-Glucarate (from calcium d-glucarate).....	350 mg
Betaine Anhydrous.....	150 mg

Other Ingredients: Silicon dioxide, L-Leucine, pullulan/hypromellose (capsule)

Suggested Use: Adults - Take 2 capsules with food, 1-2 times a day, or as directed by your health care practitioner. Take a few hours before or after taking other medications.

impact our health by disrupting the methylation process, including **heavy metals** (eg. cadmium, lead, arsenic, nickel, methylmercury), **endocrine disruptors** (eg. BPA, dioxin, diethylstilbestrol), and **air pollutants** (eg. benzene).¹ Dysfunction in methylation can, therefore, result in a wide array of symptoms and conditions.

The most direct outcome of methylation dysfunction is **hyperhomocysteinemia**, which is an independent risk factor for cardiovascular diseases.² Other conditions associated are ADHD, Alzheimer's disease, cancers (eg. breast, prostate, colon, and brain), anxiety, depression, schizophrenia, etc.

Support Methylation & Its Collateral Pathways

Cofactors Required: B1, B2, B3, B6, B9 (5-MTHF), B12, Vit C, Cu, Fe, Mg, Mn, Mo, Zn

MTHFR (5,10-methylene-tetrahydrofolate reductase) is the rate-limiting enzyme in the folate cycle that catalyzes the reduction of 5,10-methylene tetrahydrofolate to **5-methyl-tetrahydrofolate (5-MTHF)**, the major form of folate in plasma.

Supplying 5-MTHF with other methyl-donors can directly drive both the methylation and folate cycles and improve methylation efficiency.

However, it is just as important to support the **collateral pathways**



associated with methylation to ensure a positive outcome.
[Figure 1]

The reason is that when the folate and methylation cycles are enhanced by the methyl-donors, it **creates additional metabolites for the collateral pathways to process** and can potentially deplete the vitamin and mineral cofactors involved.

The collateral pathways are **sulfite, histamine, COMT, MAO, GST (glutathione S-transferase), and SOD (superoxide dismutase)**.

The most common adverse reactions from unsupported collateral pathways may include agitation and anxiety, sulfite sensitivity, insomnia, allergic

reactions, GI upset, fatigue, and memory decline.

D-Glucarate – Supports Glucuronidation & Protects against Tumorigenesis Caused by Xenobiotics

Glucuronidation is another important reaction in the body to conjugate xenobiotic metabolites – especially the endocrine disruptors – for excretion through bile. It requires uridine diphosphate glucuronic acid and **vitamin B3** as its cofactors.

However, there are species of bacteria in our gut that produce an enzyme called **beta-glucuronidase**. This enzyme is able to **deconjugate the hormone metabolites and toxins** in the gut by **cleaving off the glucuronate group**, and consequently, **allow the reactivated metabolites and toxins to damage the gut linings and/or re-enter the circulation**. In fact, studies have shown a **positive correlation between levels of beta-glucuronidase activity in the gut and risk of colon and lung cancer**.^{3,4} **D-glucarate** is a nutrient commonly found in fruits and vegetables. It has demonstrated the ability to **inhibit beta-glucuronidase** and **may provide cancer protective effects against xenobiotics**.^{3,4,5}

Total Antioxidant Capacity

SOD & GPx - The Dynamic Duo

Superoxide dismutase (SOD) and glutathione peroxidase (GPx) work hand-in-hand to help quench reactive oxygen species (ROS).

Zinc is one of the most important cofactors of **Superoxide Dismutase (SOD)** in the **mitochondria and cytoplasm**. SOD works by converting radicalized O₂ to the less active H₂O₂, which is then neutralized by GSH via GPx.

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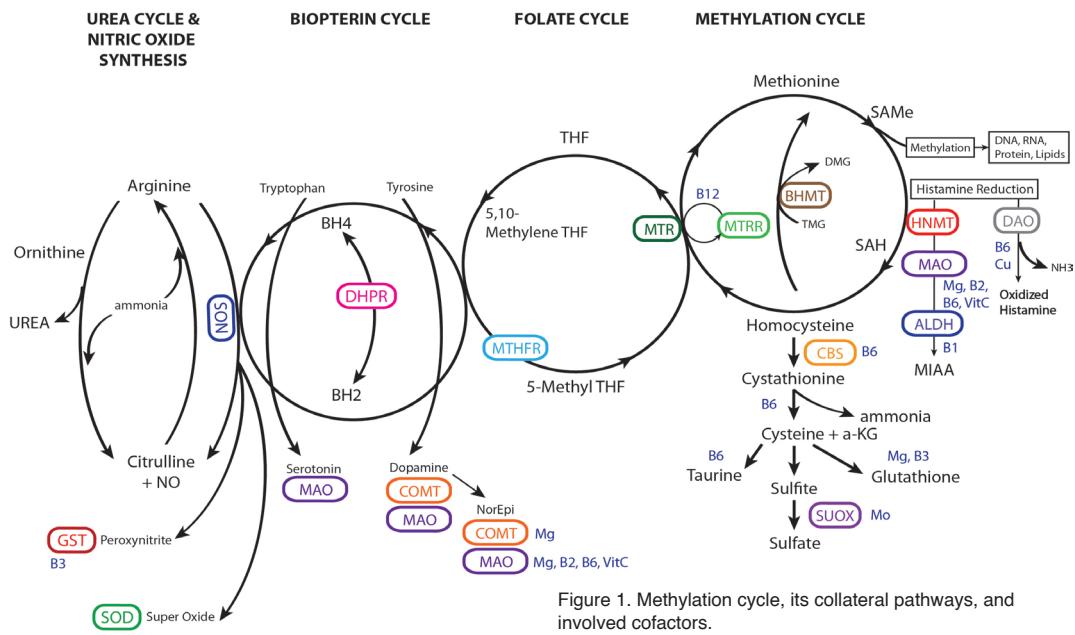


Figure 1. Methylation cycle, its collateral pathways, and involved cofactors.

Illustration compiled by Vita Aid® Professional Therapeutics
 Based on information provided by © Neurological Research Institute

Selenium serves its antioxidant purpose through being the **cofactor for Glutathione Peroxidase (GPx)**, an antioxidant enzyme that quenches ROS and reactive nitrogen species (RNS) at the expense of reduced glutathione (GSH).

Generation & Regeneration of GSH

N-acetyl cysteine (NAC) is one of the major precursors to GSH - the body's most important molecule to neutralize free radicals, conjugate chemicals and heavy metals, and protect against carcinogenesis.

Vitamin B3 (via NAD) is the coenzyme of **Glutathione S-Transferase (GST)**. GST catalyzes the reduction of oxidized glutathione (GS-SG ==> 2x GSH) – **restoring glutathione to its active form**.

Reference:

1. Baccarelli A, Bollati V. Epigenetics and environmental chemicals. *Curr Opin Pediatr* (2009); 21(2):243-251.
2. Brosnan JT, Jacobs RL, Stead LM, Brosnan ME. Methylation demand: a key determinant of homocysteine metabolism. *Acta Biochim Pol.* (2004); 51(2): 405-13.
3. Kim DH, Jin YH. Intestinal bacterial beta-glucuronidase activity of patients with colon cancer. *Arch Pharm Res.* (2001); 24(6): 564-7.
4. Hanausek M, Walaszek Z, Slaga TJ. Detoxifying cancer causing agents to prevent cancer. *Integrative Cancer Therapies* (2003); 2(2):139-144.
5. Walaszek Z, Hanausek M, Narog M, Raich PC, Slaga TJ. Mechanisms of lung cancer chemoprevention by d-glucarate. *Chest* (2004); 125: 149S-150S.