XenobioX

Synergistic Support for Xenobiotic Detox | VA-133

VitaAiD® Professional Therapeutics

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 Detoxifcation

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 impact our health by disrupting the methylation process**, including **heavy metals** (eg. cadmium, lead, arsenic, nickel, methylmercury), **endocrine disrupters** (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.

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)	400 mcg
N-Acetyl-L-Cysteine	500 mg
D-Glucarate (from calcium d-glucarate)	350 mg
Betaine Anhydrous	150 mg

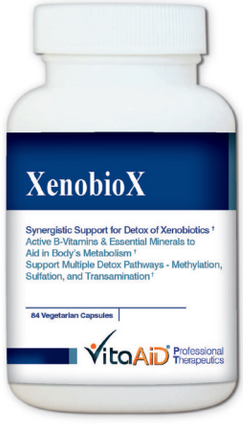
Other Ingredients: Silicon dioxide, L-Leucine, pullulan/ hypromellose (capsule)

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.

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.

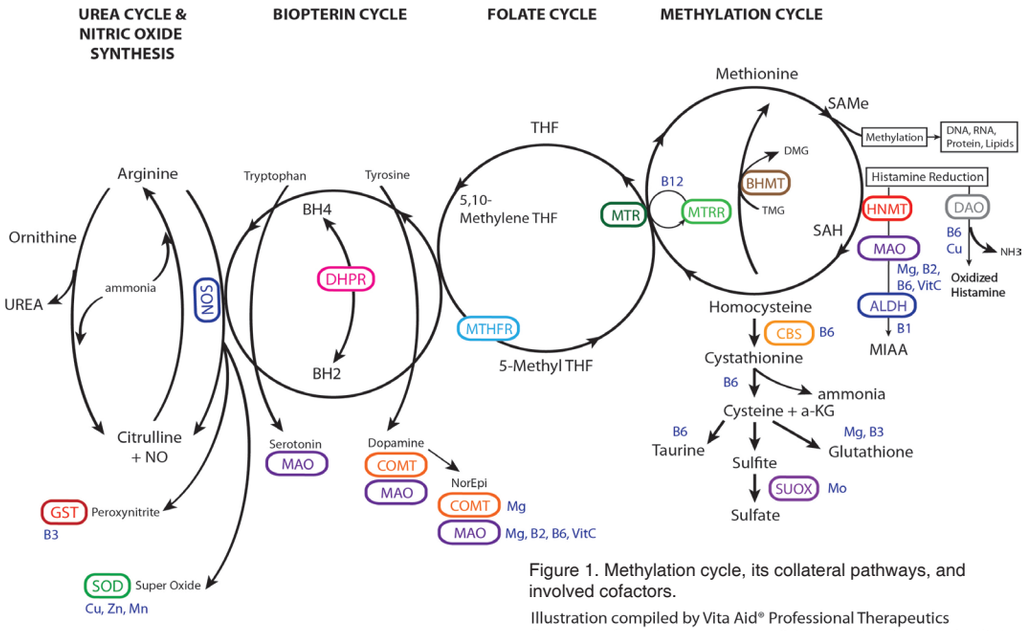


Figure 1. Methylation cycle, its collateral pathways, and involved cofactors.

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

D-Glucarate – Supports Glucuronidation & Protects against Tumorgenesis 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 O_2 to the less active H_2O_2, which is then neutralized by GSH via GPx.

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:

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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.

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