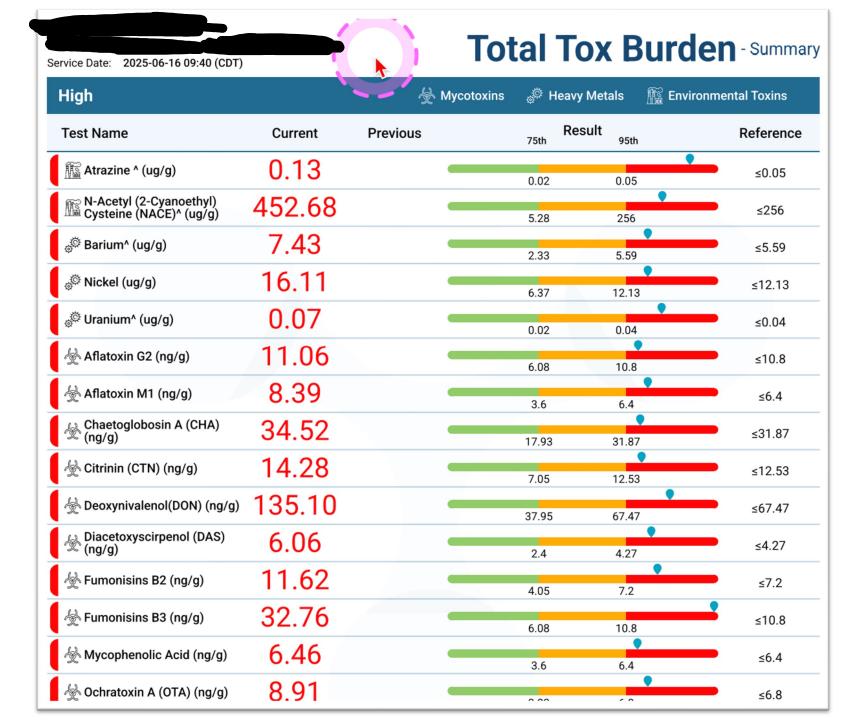


87 Total Environmental Burdens

Total Tox Burden Get Started At Home Test Available The Total Tox Burden Test is a comprehensive urine-based panel that measures the levels of mycotoxins, heavy metals, and environmental chemicals in the body. Utilizing advanced liquid chromatography-mass spectrometry (LC-MS/MS) technology, this test provides detailed insights into toxin accumulation and exposure, helping guide personalized detoxification strategies and reduce the health risks associated with environmental and chemical toxins. What We Measure 20 Heavy Metals 3 29 Mycotoxins 3 38 Environmental Chemicals

https://vibrant-wellness.com/tests/toxins/total-tox-burden?utm_source=chatgpt.com



June 16th 2025

55 Female



C-Reactive Protein, Cardiac

Test	Current Resul	t and Flag	Previous Re	sult and Date	Units	Reference Interval
▲ C-Reactive Protein, Cardiac 01	10.42	High	18.13	06/13/2025	mg/L	0.00-3.00
	Relative Risk for Future Cardiovascular Event					
				Low	<1.00	
				Average	1.00 - 3.00	
				High	>3.00	

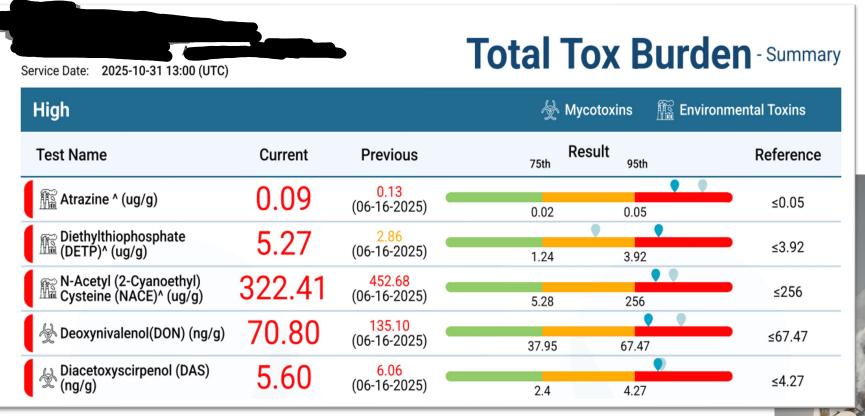
CBC With Differential/Platelet

Test	Current Resul	t and Flag	Previous Res	ult and Date	Units	Reference Interval
▼ WBC 01	2.6	Low	2.4	06/13/2025	x10E3/uL	3.4-10.8

Hemoglobin A1c

Test	Current Result and Flag	Previous Result and Date		Units	Reference Interval
Hemoglobin A1c ⁰¹	5.5	5.8	06/13/2025	%	4.8-5.6

October 10th 2025







BACKGROUND

Aflatoxin G2 is a minor mycotoxin produced by the fungi species, Aspergillus nomiae, and Aspergillus flavus.

ASSOCIATED RISK

Aflatoxin G2 binds to DNA and can lead to DNA alterations. It is primarily implicated in hepatic diseases.

POSSIBLE SOURCES

Contaminated plant (such as peanuts, maize, or rice) and animal products (such as meat or dairy), Inhaling dust (generated during the handling and processing of contaminated crops and feeds such as cottonseed).

DETOX SUGGESTIONS

To mitigate aflatoxin G2 effects, it is important to include a diet rich in antioxidants, stay hydrated, and consider liver-supporting supplements like milk thistle. Prevention through food safety practices is key, as there is no direct method to detoxify aflatoxin from the body.



BACKGROUND

Aflatoxin M1 is a metabolite of aflatoxin B1, which is produced by molds such as Aspergillus flavus and Aspergillus parasiticus. Aflatoxin M1 is formed when animals, particularly dairy cows, consume feed contaminated with aflatoxin B1, and it is excreted in their milk.

ASSOCIATED RISK

Aflatoxin M1 has been regarded as a human carcinogen. It can cause liver damage, immune suppression, internal haemorrhaging, muscle tremors, and impact gain and efficiency.

POSSIBLE SOURCES

Contaminated milk. Aflatoxin M1 is mainly found in the milk of cattle fed with contaminated aflatoxin feed. Consumption of such animal products exposes humans to Aflatoxin M1.

DETOX SUGGESTIONS

To mitigate aflatoxin M1 effects, it is important to include a diet rich in antioxidants, stay hydrated, and consider liver-supporting supplements like milk thistle. Prevention through food safety practices is key, as there is no direct method to detoxify aflatoxin from the body.



BACKGROUND

Chaetoglobosin A (CHA) is a mycotoxin produced by molds like Chaetomium globosum. It is associated with cytotoxic effects and is found in various agricultural products, including grains and cereals.

ASSOCIATED RISK

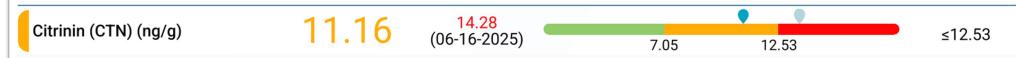
Exposure to CHA poses significant health risks, primarily due to its cytotoxic properties. CHA has been linked to cellular damage and toxicity, potentially affecting multiple organ systems upon exposure.

POSSIBLE SOURCES

Exposure to CHA can occur through ingestion of contaminated grains and cereals, as well as through inhalation of mold spores in contaminated environments.

DETOX SUGGESTIONS

Detoxification of mycotoxins including, CHA involves glutathione conjugation and glucuronidation. Nutrients like N-acetyl cysteine (NAC), vitamins C and E, selenium, and calcium D-glucarate aid in neutralizing and eliminating the toxin. Silybins from milk thistle enhance liver function, promoting glutathione conjugation via upregulation of glutathione S-transferase (GST)



BACKGROUND

Citrinin (CTN) is a polyketide mycotoxin, a secondary metabolite produced by certain fungi species, primarily Aspergillus, Penicillium, and Monascus. It is commonly found in a variety of foods including rice, wheat, flour, barley, maize, rye, oats, peanuts, and fruits. Citrinin tends to occur post-harvest, particularly in stored grains.

ASSOCIATED RISK

Citrinin poses significant health risks upon ingestion, particularly affecting the kidneys. Its toxic effects include renal degeneration, leading to weight loss. Chronic exposure to citrinin has been linked to nephrotoxicity, which can result in renal dysfunction and damage.

POSSIBLE SOURCES

The presence of citrinin is widespread in agricultural products, especially grains and cereals, making it a concern globally. It is often found in stored grains, where fungal growth can occur under certain conditions.

DETOX SUGGESTIONS

Detoxification of mycotoxins including citrinin involves various mechanisms. Glutathione conjugation and glucuronidation are essential processes for neutralizing and eliminating the toxin. Nutrients such as N-acetyl cysteine (NAC), vitamins C and E, selenium, and calcium D-glucarate play crucial roles in aiding detoxification pathways. Additionally, compounds like silybins from milk thistle can enhance liver function, thereby promoting glutathione conjugation through the upregulation of glutathione S-transferase (GST), which aids in the detoxification process.





11.62 (06-16-2025)



≤7.2

BACKGROUND

Fumonisins B2 is a mycotoxin produced by the fungi, Aspergillus niger, Fusarium fujikuro Fusarium moniliforme, Fusarium proliferatum, and Fusarium nygamai.

ASSOCIATED RISK

Fumonisin B2 can lead to a variety of toxic effects such as autophagy, apoptosis, neurotoxicity, immunotoxicity, reproductive toxicity, tissue and organ toxicity, and carcinogenicity [9]. Leukopenia, sepsis, bone marrow suppression, hemosiderosis, and multiple haemorrhages can be caused due to Fumonisin B2 intoxication

POSSIBLE SOURCES

Fumonisin B-infected maize.

DETOX SUGGESTIONS

Detoxification of mycotoxins including, Fumonisin B2 involves glutathione conjugation and glucuronidation. Nutrients like N-acetyl cysteine (NAC), vitamins C and E, selenium, and calcium D-glucarate aid in neutralizing and eliminating the toxin. Silybins from milk thistle enhance liver function, promoting glutathione conjugation via upregulation of glutathione S-transferase (GST).

Test Name	Current	Previous	Resul	t 95th	Reference
Ochratoxin A (OTA) (ng/g)	4.13	8.91 (06-16-2025)	3.83	6.8	≤6.8

BACKGROUND

Ochratoxin is a mycotoxin produced by various fungal species such as Aspergillus ochraceus, Aspergillus carbonarius, Aspergillus niger and Penicillium verrucosum.

ASSOCIATED RISK

Ochratoxin A has been recognised as a renal toxin owing to its ability to induce nephrotoxicity and renal tumors. It displays a long elimination half-life and stimulates the major inflammatory cytokines released. Ochratoxin A is efficiently absorbed from the gastrointestinal tract into the small intestine where it seen to effectively interrupt the intestinal barrier functions.

POSSIBLE SOURCES

Contaminated Barley, oats, rye, wheat, coffee beans, pork.

DETOX SUGGESTIONS

Detoxification of ochratoxin involves the use of activated charcoal (AC) to bind and neutralize the toxin in the gastrointestinal tract. To minimize the risk of nutrient depletion, AC should be taken separately from essential nutrients. Concurrent use of an oral multimineral formula or IV nutrient therapy can help replenish any lost nutrients during detoxification.



9.65

32.76 (06-16-2025)



≤10.8

BACKGROUND

Fumonisins B3 is a mycotoxin produced by the fungi, Fusarium moniliforme, Fusarium proliferatum, and Fusarium nygamai.

ASSOCIATED RISK

Fumonisin B3 can lead to a variety of toxic effects such as autophagy, apoptosis, neurotoxicity, immunotoxicity, reproductive toxicity, tissue and organ toxicity, and carcinogenicity [9]. Leukopenia, sepsis, bone marrow suppression, hemosiderosis, and multiple haemorrhages can be caused due to Fumonisin B3 intoxication.

POSSIBLE SOURCES

Fumonisin B-infected maize.

DETOX SUGGESTIONS

Detoxification of mycotoxins including, Fumonisin B3 involves glutathione conjugation and glucuronidation. Nutrients like N-acetyl cysteine (NAC), vitamins C and E, selenium, and calcium D-glucarate aid in neutralizing and eliminating the toxin. Silybins from milk thistle enhance liver function, promoting glutathione conjugation via upregulation of glutathione S-transferase (GST).



BACKGROUND

Gliotoxin is a mycotoxin produced by the fungi, Aspergillus fumigatus, Eurotium chevalieri, Gliocladium fimbriatum, and several Trichoderma and Penicillium species.

ASSOCIATED RISK

Gliotoxin can promote immunosuppression by inhibiting or interfering with the activation of transcription factors that are involved in T-cell activation. Gliotoxin can penetrate and impair the integrity of the human blood-brain barrier which can have severe neurological implications. Gliotoxin can have adverse effects on the kidney and liver too.

POSSIBLE SOURCES

Indoors, in buildings with water damage or in damp properties with water leaks and poor ventilation, Spores produced by gliotoxin-producing molds.

DETOX SUGGESTIONS

Detoxification of mycotoxins including, Gliotoxin involves glutathione conjugation and glucuronidation. Nutrients like N-acetyl cysteine (NAC), vitamins C and E, selenium, and calcium D-glucarate aid in neutralizing and eliminating the toxin. Silybins from milk thistle enhance liver function, promoting glutathione conjugation via upregulation of glutathione S-transferase (GST).

Mycophenolic Acid (ng/g)

4.50

6.46 (06-16-2025)



≤6.4

BACKGROUND

Mycophenolic acid is a mycotoxin produced by multiple species of Penicillium and other molds such as Aspergillus spp. From a pharmacological standpoint, it is used as an immunosuppressive drug in transplantation immunology.

ASSOCIATED RISK

Exposure to mycophenolic acid can lead to recurrent infections caused by the dysfunction of the individual's immune system. This can be mediated either through the local inhibition of innate immunity of the cilia cells lining the mucosal epithelium, or via systemic inhibition involving acquired immunity. Prolonged exposure to mycophenolic acid can give rise to further dysregulation of immune checkpoints leading to uncontrolled cell proliferation or cancer.

POSSIBLE SOURCES

Mycophenolic acid-producing molds in moisture-damaged buildings.

DETOX SUGGESTIONS

Detoxification of mycotoxins including, mycophenolic acid involves glutathione conjugation and glucuronidation. Nutrients like N-acetyl cysteine (NAC), vitamins C and E, selenium, and calcium D-glucarate aid in neutralizing and eliminating the toxin. Silybins from milk thistle enhance liver function, promoting glutathione conjugation via upregulation of glutathione S-transferase (GST).

Trichothecenes				
Test Name	Current	Previous	Result 75th 95th	Reference
Deoxynivalenol(DON) (ng/g)	70.80	135.10 (06-16-2025)	37.95 67.47	≤67.47

BACKGROUND

Deoxynivalenol (DON) is a class of mycotoxins primarily produced by Fusarium fungi. It is one of the most abundant and important trichothecenes in food and feed and is a significant contaminant due to its frequent occurrence in toxicologically relevant concentrations worldwide. It infects oats, barley, corn, wheat, and rice in the field or in storage.

ASSOCIATED RISK

DON affects human health, causing acute temporary nausea, vomiting, diarrhea, abdominal pain, headache, dizziness, and fever. Fusarium toxin contamination cannot be avoided completely since toxin production depends strongly on environmental conditions such as temperature and humidity. As a result, human exposure to this toxin poses a permanent health risk. Toxic effects of DON include immune suppression, cytotoxic, skin necrosis, hemorrhage, anemia, granulocytopenia, oral epithelial lesions, hematopoietic, alimentary toxic aleukia (ATA), hypotension, and coagulopathy. Intestinal toxicity of DON is attributed to mitochondrial dysfunction (caused due to reactive oxygen species) as a critical factor.

POSSIBLE SOURCES

DON plant-based food like grains or animal proteins like kidney, liver, eggs, dairy products, beer, bread, breakfast cereals, sorghum, buckwheat, noodles, and malt.

DETOX SUGGESTIONS

Activated charcoal (AC) can adsorb deoxynivalenol, aiding in its elimination from the body. To prevent potential nutrient depletion, AC should be taken separately from food, medication, or supplements. Additionally, supporting liver function with silybins from milk thistle and providing antioxidant support with vitamins C and E can assist in detoxification.



POSSIBLE SOURCES

Drinking groundwater, contaminated food, injections, and waste sites.

ASSOCIATED RISK

Barium dissolves in the stomach and can result in symptoms like hypokalemia, diarrhea, nausea, vomiting, heart rhythm abnormalities, muscle cramps, and kidney disorders. Other symptoms include increased/decreased blood pressure and numbness around the face.

DETOX SUGGESTIONS

Barium is primarily eliminated from the body through conversion into the nontoxic barium sulfate in the gastrointestinal tract. This process can be facilitated by oral sulfate salts, such as sodium or magnesium sulfate, which decrease absorption. In severe cases, hemodialysis may be necessary to rapidly increase barium clearance, especially when supportive measures like intravenous potassium supplementation are ineffective.



POSSIBLE SOURCES

Contaminated food and water, dermal exposures, and inhalation.

ASSOCIATED RISK

Ingestion of uranium may lead to kidney problems. As a result, the kidneys are the most impacted organ system by uranium exposure, both chronic and acute. Uranium may also impact DNA and cause chromosomal abnormalities. The main manifestation of uranium exposure is the cellular depletion of antioxidants which increases oxidative stress. Altered genomic stability and increased oxidative stress are hallmarks of aging. As a result, uranium intoxication may disrupt many biological processes which could lead to the risk of accelerated aging and developing age-associated conditions.

DETOX SUGGESTIONS

To detoxify uranium from the body, maintain adequate hydration to facilitate urinary excretion and avoid exposure to uranium sources. Chelation therapy is not typically recommended for uranium detoxification due to limited effectiveness and potential risks.



POSSIBLE SOURCES

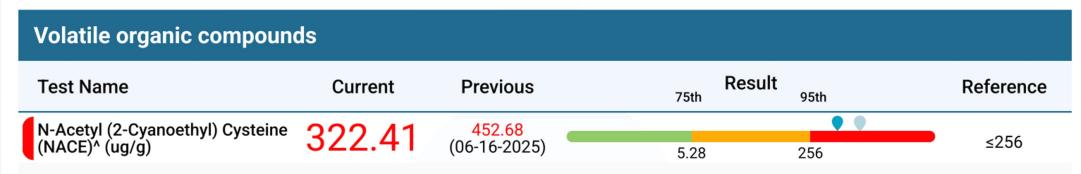
Contaminated food, jewelry, cosmetics, keys, cell phones, paper clips, electrical equipment, alloy, orthodontic braces, eyeglass frames, and clothing fasteners.

ASSOCIATED RISK

Nickel toxicity poses a significant risk, leading to allergies, cardiovascular and kidney diseases, lung fibrosis, nasal and lung cancer, along with symptoms such as low blood pressure, muscle tremors, nausea, vomiting, haemorrhages, heart attacks, oral and/or intestinal cancer, and kidney dysfunction.

DETOX SUGGESTIONS

Chelation therapy utilizing agents such as EDTA (ethylenediaminetetraacetic acid) or DMSA (dimercaptosuccinic acid) facilitates the removal of nickel from the body by binding to the metal ions and aiding in their excretion via urine or feces. These chelating agents work by forming stable complexes with nickel, thereby reducing its toxicity. Additionally, antioxidants like vitamin C play a crucial role in mitigating oxidative stress induced by nickel exposure, supporting overall detoxification processes.



BACKGROUND

Acrylonitrile is a colourless liquid with a pungent odour. It is used in the production of acrylic fibres, resins, and rubber.

ASSOCIATED RISK

Exposure to acrylonitrile can lead to headaches, nausea, dizziness, fatigue, and chest pains. Furthermore, individuals exposed to elevated levels of airborne acrylonitrile, particularly in occupational settings, are at a higher risk of experiencing damage to their lungs, liver, and central nervous system.

POSSIBLE SOURCES

Smoking tobacco and cigarettes are another potential exposure. Use of any of these products could lead to exposure to acrylonitrile.

DETOX SUGGESTIONS

To facilitate the detoxification of N-Acetyl (2-Cyanoethyl) Cysteine (NACE) from the body, hydration is vital to promote urinary excretion. Additionally, supporting liver function through a nutrient-rich diet and antioxidant intake aids in metabolizing and eliminating NACE efficiently.