

Total Toxins Summary

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

| High (>95th percentile) | | | | | | Mycotoxins |
|--------------------------|----------------|-----------------|----------------|-----------------|-------------|------------|
| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE | |
| Aflatoxin B1 (AFB1) | 5055.98 | | 0 3.9 6.93 | | ≤6.93 ng/g | |
| Aflatoxin B2 (AFB2) | 3357.87 | | 0 4.58 8.13 | | ≤8.13 ng/g | |
| Aflatoxin G1 | 5839.93 | | 0 3.68 6.53 | | ≤6.53 ng/g | |
| Aflatoxin G2 | 2423.47 | | 0 6.08 10.8 | | ≤10.8 ng/g | |
| Aflatoxin M1 | 5557.57 | | 0 3.6 6.4 | | ≤6.4 ng/g | |
| Chaetoglobosin A (CHA) | 3717.08 | | 0 17.9 31.8 | | ≤31.87 ng/g | |
| Citrinin (CTN) | 3961.83 | | 0 7.05 12.5 | | ≤12.53 ng/g | |
| Deoxynivalenol(DON) | 783.53 | | 0 37.9 67.4 | | ≤67.47 ng/g | |
| Diacetoxyscirpenol (DAS) | 3157.32 | | 0 2.4 4.27 | | ≤4.27 ng/g | |
| Dihydrocitrinone | 4543.72 | | 0 9.3 16.5 | | ≤16.53 ng/g | |
| Enniatin B1(ENN B1) | 948.01 | | 0 0.13 0.22 | | ≤0.22 ng/g | |
| Fumonisin B1 | 3182.72 | | 0 3.45 6.13 | | ≤6.13 ng/g | |
| Fumonisin B2 | 3296.01 | | 0 4.05 7.2 | | ≤7.2 ng/g | |

| SPECIMEN INFORMATION | | |
|--------------------------------------|--------|---------|
| Provoking Status: unavailable | Agent: | Dosage: |

Results are creatinine corrected to account for urine dilution variations. Reference intervals are based upon NHANES(cdc.gov/nhanes) data if available, and are representative of a large population cohort under non-provoked conditions. Chelation (provocation) agents can increase urinary excretion of metals/elements.

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High (>95th percentile)

Mycotoxins

| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
|------------------------|----------------|-----------------|----------------|-----------------|--------------|
| Fumonisin B3 | 2784.37 | | 0 6.08 10.8 | | ≤10.8 ng/g |
| Gliotoxin | 1261.74 | | 0 116 207 | | ≤207.87 ng/g |
| Mycophenolic Acid | 1512.39 | | 0 3.6 6.4 | | ≤6.4 ng/g |
| Nivalenol (NIV) | 5029.32 | | 0 1.8 3.2 | | ≤3.2 ng/g |
| Ochratoxin A (OTA) | 4595.35 | | 0 3.83 6.8 | | ≤6.8 ng/g |
| Patulin | 5109 | | 0 6.53 11.6 | | ≤11.6 ng/g |
| Roridin A | 4812.43 | | 0 4.28 7.6 | | ≤7.6 ng/g |
| Roridin E | 5126.69 | | 0 0.75 1.33 | | ≤1.33 ng/g |
| Roridin L2 | 4157.39 | | 0 3.83 6.8 | | ≤6.8 ng/g |
| Satratoxin G | 5195.86 | | 0 0.1 0.18 | | ≤0.18 ng/g |
| Satratoxin H | 3682.05 | | 0 0.1 0.18 | | ≤0.18 ng/g |
| Sterigmatocystin (STC) | 3118.99 | | 0 0.3 0.53 | | ≤0.53 ng/g |
| T-2 Toxin | 883.29 | | 0 0.1 0.18 | | ≤0.18 ng/g |

SPECIMEN INFORMATION

Provoking Status: **unavailable**

Agent:

Dosage:

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| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

| High (>95th percentile) | | | | Mycotoxins | | Heavy Metals |
|-------------------------|----------------|-----------------|----------------|-----------------|--|--------------|
| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | | REFERENCE |
| Verrucarin A | 4223.33 | | 0 0.75 1.33 | | | ≤1.33 ng/g |
| Verrucarin J | 3392.25 | | 0 5.18 9.2 | | | ≤9.2 ng/g |
| Zearalenone (ZEN) | 3768.04 | | 0 0.38 0.67 | | | ≤0.67 ng/g |
| Aluminum | 118.98 | | 0 17.8 45.1 | | | ≤45.15 ug/g |
| Antimony* | 6.31 | | 0 0.07 0.16 | | | ≤0.16 ug/g |
| Arsenic* | 269.74 | | 0 11.9 52 | | | ≤52 ug/g |
| Barium* | 12.24 | | 0 2.33 5.59 | | | ≤5.59 ug/g |
| Beryllium* | 14.45 | | 0 0.2 0.76 | | | ≤0.76 ug/g |
| Bismuth | 24.54 | | 0 0.58 2.53 | | | ≤2.53 ug/g |
| Cadmium* | 34.59 | | 0 0.29 0.8 | | | ≤0.8 ug/g |
| Cesium* | 13.27 | | 0 6.37 10.3 | | | ≤10.3 ug/g |
| Gadolinium | 6.11 | | 0 0.17 0.45 | | | ≤0.45 ug/g |
| Lead* | 114.54 | | 0 0.52 1.16 | | | ≤1.16 ug/g |

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Total Toxins Summary

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

| High (>95th percentile) | | Heavy Metals | | Environmental Toxins | |
|---|----------------|-----------------|----------------|----------------------|-------------|
| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
| Mercury* | 81.01 | | 0 0.57 1.61 | | ≤1.61 ug/g |
| Nickel | 20.54 | | 0 6.37 12.1 | | ≤12.13 ug/g |
| Palladium | 19.62 | | 0 0.15 0.2 | | ≤0.2 ug/g |
| Platinum* | 9.59 | | 0 0.1 0.9 | | ≤0.9 ug/g |
| Tellurium | 21.57 | | 0 0.42 0.89 | | ≤0.89 ug/g |
| Thallium* | 362.42 | | 0 0.24 0.43 | | ≤0.43 ug/g |
| Thorium | 20.4 | | 0 0.02 0.07 | | ≤0.07 ug/g |
| Tin* | 6.94 | | 0 1 3.72 | | ≤3.72 ug/g |
| Tungsten* | 5.08 | | 0 0.12 0.33 | | ≤0.33 ug/g |
| Uranium* | 12.26 | | 0 0.02 0.04 | | ≤0.04 ug/g |
| 2-Hydroxyethyl Mercapturic Acid (HEMA)* | 1064.2 | TNP | 0 1.7 4.75 | | ≤4.75 ug/g |
| 2-Methylhippuric Acid (2MHA)* | 6831.22 | <0.01 | 0 77.9 248 | | ≤248 ug/g |
| 2,2-bis(4-Chlorophenyl) acetic acid (DDA) | 7064.74 | | 0 7.9 19 | | ≤19 ug/g |

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| SPECIMEN INFORMATION | | |
|--------------------------------------|--------|---------|
| Provoking Status: unavailable | Agent: | Dosage: |

Results are creatinine corrected to account for urine dilution variations. Reference intervals are based upon NHANES(cdc.gov/nhanes) data if available, and are representative of a large population cohort under non-provoked conditions. Chelation (provocation) agents can increase urinary excretion of metals/elements.

Total Toxins Summary

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
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High (>95th percentile)

Environmental Toxins

| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
|---|----------------|-----------------|----------------|-----------------|--------------|
| 2,4-Dichlorophenoxyacetic Acid (2,4-D)* | 2465.39 | 1 | 0 0.5 1.55 | | ≤1.55 ug/g |
| 3-Methylhippuric Acid (3MHA) | 2936.05 | >10000 | 0 64.8 612 | | ≤612.83 ug/g |
| 3-Phenoxybenzoic Acid (3PBA)* | 3120.15 | >10000 | 0 1.01 5.44 | | ≤5.44 ug/g |
| 4-Methylhippuric Acid (4MHA) | 5652.4 | TNP | 0 65.5 752 | | ≤752.72 ug/g |
| 4-Nonylphenol | 6120.36 | <0.01 | 0 0.42 2.06 | | ≤2.06 ug/g |
| Atrazine * | 4737.24 | 5 | 0 0.02 0.05 | | ≤0.05 ug/g |
| Atrazine mercapturate* | 5305.49 | 6 | 0 0.02 0.05 | | ≤0.05 ug/g |
| Bisphenol A (BPA)* | 8513.69 | 7 | 0 2.12 5.09 | | ≤5.09 ug/g |
| Butylparaben* | 2232.85 | 8 | 0 0.25 4.39 | | ≤4.39 ug/g |
| Diethyl phosphate (DEP)* | 5623.27 | | 0 3.2 15.7 | | ≤15.7 ug/g |
| Diethyldithiophosphate (DEDTP)* | 5236.6 | | 0 0.17 0.3 | | ≤0.3 ug/g |
| Diethylthiophosphate (DETP)* | 1189.7 | | 0 1.24 3.92 | | ≤3.92 ug/g |
| Dimethyl phosphate (DMP)* | 1785.73 | | 0 9.1 33.6 | | ≤33.6 ug/g |

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SPECIMEN INFORMATION

Provoking Status: **unavailable**

Agent:

Dosage:

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High (>95th percentile)

Environmental Toxins

| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
|--|----------------|-----------------|----------------|-----------------|------------|
| Dimethyldithiophosphate (DMDTP)* | 2651.53 | | 0 0.67 6.12 | | ≤6.12 ug/g |
| Dimethylthiophosphate (DMTP)* | 7775.27 | | 0 5.91 33.7 | | ≤33.7 ug/g |
| Diphenyl Phosphate (DPP) | 5704.99 | | 0 1.1 3.7 | | ≤3.7 ug/g |
| Ethylparaben * | 4321.25 | | 0 5.41 99.3 | | ≤99.3 ug/g |
| Glyphosate | 580.15 | | 0 1.65 7.6 | | ≤7.6 ug/g |
| Methylparaben* | 8827.17 | | 0 180 653 | | ≤653 ug/g |
| Mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP)* | 8576.21 | | 0 14.1 37.7 | | ≤37.7 ug/g |
| Mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP)* | 6140.3 | | 0 8.99 23.4 | | ≤23.4 ug/g |
| Mono-2-ethylhexyl phthalate (MEHP)* | 1978.65 | | 0 2.73 8.47 | | ≤8.47 ug/g |
| Mono-ethyl phthalate (MEtP)* | 595.58 | | 0 94.2 541 | | ≤541 ug/g |
| N-Acetyl (2-Cyanoethyl) Cysteine (NACE)* | 1445.39 | | 0 5.28 256 | | ≤256 ug/g |
| N-Acetyl (2-Hydroxypropyl) Cysteine (NAHP)* | 5090.41 | | 0 101 403 | | ≤403 ug/g |
| N-Acetyl (3,4-Dihydroxybutyl) Cysteine* | 9073.19 | | 0 374 583 | | ≤583 ug/g |

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|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

| High (>95th percentile) | | | | Environmental Toxins | PFAS |
|--|----------------|-----------------|----------------|----------------------|-------------|
| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
| N-Acetyl (Propyl) Cysteine (NAPR)* | 3102.95 | | 0 11.3 46.1 | | ≤46.1 ug/g |
| N-acetyl phenyl cysteine (NAP)* | 1802.48 | | 0 1.29 3.03 | | ≤3.03 ug/g |
| N-acetyl-S-(2-carbamoyl-ethyl)-cysteine* | 6958.71 | | 0 82 199 | | ≤199 ug/g |
| Perchlorate (PERC)* | 1550.31 | | 0 4.89 10.7 | | ≤10.7 ug/g |
| Phenyl glyoxylic Acid (PGO)* | 4737.71 | | 0 285 518 | | ≤518 ug/g |
| Propylparaben* | 6174.69 | | 0 36.7 222 | | ≤222 ug/g |
| Tiglylglycine (TG) | 4460.57 | | 0 0.09 3.24 | | ≤3.24 ug/g |
| Triclosan (TCS)* | 1841.07 | | 0 29.9 358 | | ≤358 ug/g |
| GenX/HPFO-DA | 17.194 | | 0 1.04 6.68 | | ≤6.689 ug/g |
| 9-chlorohexadecafluoro-3-oxanonane-1-sulfonate | 19.058 | | 0 0.47 2.75 | | ≤2.75 ug/g |
| Dodecafluoro-3H-4,8-dioxanoate (NaDONA) | 14.197 | | 0 0.37 1.91 | | ≤1.916 ug/g |
| Perfluoro-[1,2-13C2] octanoic acid (M2PFOA) | 20.185 | | 0 0.45 2.05 | | ≤2.054 ug/g |
| Perfluoro-1-[1,2,3,4-13C4] octanesulfonic acid | 26.652 | | 0 0.64 2.68 | | ≤2.68 ug/g |

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| SPECIMEN INFORMATION | | |
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|-----------|------------|--------|---------------|--------------|------------------------|
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High (>95th percentile)

PFAS

| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
|--|----------------|-----------------|----------------|-----------------|-------------|
| Perfluoro-1-heptane sulfonic acid (PFHpS) | 28.225 | | 0 0.62 3.78 | | ≤3.783 ug/g |
| Perfluoro-n-[1,2-13C2] decanoic acid (MPFDA) | 7.915 | | 0 0.94 2.90 | | ≤2.907 ug/g |
| Perfluoro-n-[1,2-13C2] hexanoic acid | 16.595 | | 0 0.09 0.32 | | ≤0.325 ug/g |
| Perfluorobutanoic acid (PFBA) | 3.873 | | 0 0.06 0.11 | | ≤0.113 ug/g |
| Perfluorodecanoic acid (PFDeA) | 17.114 | | 0 0.69 2.39 | | ≤2.399 ug/g |
| Perfluorododecanoic acid (PFDoA) | 2.097 | | 0 0.54 1.76 | | ≤1.769 ug/g |
| Perfluoroheptanoic acid (PFHpA) | 24.455 | | 0 0.10 0.14 | | ≤0.142 ug/g |
| Perfluorohexane Sulfonic Acid (PFHxS) | 10.097 | | 0 0.11 1.68 | | ≤1.681 ug/g |
| Perfluorohexanoic acid (PFHxA) | 0.163 | | 0 0.01 0.15 | | ≤0.156 ug/g |
| Perfluorononanoic acid (PFNA) | 27.688 | | 0 0.65 1.31 | | ≤1.31 ug/g |
| Perfluorooctane sulfonic acid (PFOS) | 13.948 | | 0 0.65 3.21 | | ≤3.215 ug/g |
| Perfluorooctanoic acid (PFOA) | 8.895 | | 0 0.56 2.20 | | ≤2.205 ug/g |
| Perfluoropentanoic acid (PFPeA) | 16.696 | | 0 0.19 0.73 | | ≤0.731 ug/g |

SPECIMEN INFORMATION

Provoking Status: unavailable

Agent:

Dosage:

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High (>95th percentile)

PFAS

| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
|--------------------------------------|----------------|-----------------|----------------|-----------------|-------------|
| Perfluorotetradecanoic acid (PFTeDA) | 13.069 | | 0 1.47 4.91 | | ≤4.912 ug/g |
| Perfluoroundecanoic acid (PFUnA) | 17.938 | | 0 0.69 1.26 | | ≤1.267 ug/g |

Moderate (75th-95th percentile)

PFAS

| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
|------------------------------------|----------------|-----------------|----------------|-----------------|------------|
| Perfluorotridecanoic acid (PFTrDA) | 3.313 | | 0 1.26 3.96 | | ≤3.96 ug/g |

Urine Creatinine

| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
|------------------|----------------|-----------------|----------------|-----------------|-----------------|
| Urine Creatinine | 9.71 | | 0 0.24 2.16 | | 0.25-2.16 mg/mL |

COMMENTS

Urine Creatinine

Urine tests that measure ratio of analytes by creatine concentration will not be altered by urine volume, hydration status, or time of testing. When using creatinine concentration to measure urine analytes, the only interference with the test is if the person's creatinine levels are very high (which may be seen in kidney disease, diabetes, or competitive body builder athletes), or when creatinine levels are very low (which may be seen in people with muscle wasting or sarcopenia who have lost their lean muscle mass stores). High urine creatinine may cause falsely lower urine analyte results. Low urine creatinine may cause falsely higher urine analyte results. This does not invalidate the findings; rather, critical analysis should be used to correlate results with clinical history and symptomatology for intervention decision-making.

SAMPLE

SPECIMEN INFORMATION

Provoking Status: **unavailable**

Agent:

Dosage:

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PATIENT

NAME: TEST TEST
DATE OF BIRTH: 1991-11-11 GENDER: Female
TELEPHONE: 11234567890 AGE: 31

ACCESSION ID: 2306270585
SPECIMEN COLLECTED: 2023-06-27 10:00 (PST)
SPECIMEN RECEIVED: 2023-06-27 14:47 (PST)
FINAL REPORT DATE: 2023-06-27 15:15 (PST)
GENERATION DATE: 2023-06-27 15:25 (PST)

FASTING:

PROVIDER:

PRACTICE NAME: Vibrant IT3 Practice
PROVIDER NAME: Vibrant IT3333
PHLEBOTOMIST: No Matched Result

TELEPHONE: +15555555555
FAX #: 650-331-7393
ADDRESS: Apple Park Way, Millcreek, UT

Vibrant Wellness is pleased to present to you, 'Mycotoxins panel', to help you make healthy lifestyle, dietary and treatment choices in consultation with your healthcare provider. It is intended to be used as a tool to encourage a general state of health and well-being.

The Vibrant Mycotoxins Panel is a test to identify and quantify the level of a large set of mycotoxins from both food and environmental molds present in your urine. The results are provided in 3 tables subgrouping the mycotoxins into Aflatoxins, Trichothecenes and Other Mycotoxins.

The report begins with the summary page which lists only the mycotoxins whose levels are >95th percentile (Red) and 75th-95th percentile (Yellow) of reference range, normalized to Urine creatinine levels. Additionally, the previous value is also indicated for your referral (if available). Following this section is the complete list of the mycotoxins and their absolute levels normalized to Creatinine in a quantile format along with the reference ranges. These levels are shown with three shades of color – Green, Yellow and Red. Reference ranges were determined using urine samples from 1000 apparently healthy individuals. The result in green corresponds to 0 to 75th percentile, the result in yellow corresponds to 75th to 95th percentile and the result in red corresponds to greater than 95th percentile of reference range. All content provided in the report are purely for informational purposes only and should not be considered medical advice. Any changes based on the information should be made in consultation with your healthcare provider.

The Vibrant Wellness platform provides tools for you to track and analyze your general wellness profile. Testing for the Mycotoxins panel is performed by Vibrant America, a CLIA certified lab CLIA#:05D2078809. Vibrant Wellness provides and makes available this report and any related services pursuant to the Terms of Use Agreement (the "Terms") on its website at www.vibrant-wellness.com. By accessing, browsing, or otherwise using the report or website or any services, you acknowledge that you have read, understood, and agree to be bound by these terms. If you do not agree to accept these terms, you shall not access, browse, or use the report or website. The statements in this report have not been evaluated by the Food and Drug Administration and are only meant to be lifestyle choices for potential risk mitigation. Please consult your physician/dietitian for medication, treatment, or lifestyle management. This product is not intended to diagnose, treat, or cure any disease.

Pediatric ranges have not been established for this test. It is important that you discuss any modifications to your diet, exercise, and nutritional supplementation with your physician before making any changes.

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| Aflatoxin B1 (AFB1) | 5055.98 | | 0 3.9 6.93 | | ≤6.93 ng/g |
| Aflatoxin B2 (AFB2) | 3357.87 | | 0 4.58 8.13 | | ≤8.13 ng/g |
| Aflatoxin G1 | 5839.93 | | 0 3.68 6.53 | | ≤6.53 ng/g |
| Aflatoxin G2 | 2423.47 | | 0 6.08 10.8 | | ≤10.8 ng/g |
| Aflatoxin M1 | 5557.57 | | 0 3.6 6.4 | | ≤6.4 ng/g |
| Chaetoglobosin A (CHA) | 3717.08 | | 0 17.9 31.8 | | ≤31.87 ng/g |
| Citrinin (CTN) | 3961.83 | | 0 7.05 12.5 | | ≤12.53 ng/g |
| Deoxynivalenol(DON) | 783.53 | | 0 37.9 67.4 | | ≤67.47 ng/g |
| Diacetoxyscirpenol (DAS) | 3157.32 | | 0 2.4 4.27 | | ≤4.27 ng/g |
| Dihydrocitrinone | 4543.72 | | 0 9.3 16.5 | | ≤16.53 ng/g |
| Enniatin B1(ENN B1) | 948.01 | | 0 0.13 0.22 | | ≤0.22 ng/g |
| Fumonisin B1 | 3182.72 | | 0 3.45 6.13 | | ≤6.13 ng/g |
| Fumonisin B2 | 3296.01 | | 0 4.05 7.2 | | ≤7.2 ng/g |
| Fumonisin B3 | 2784.37 | | 0 6.08 10.8 | | ≤10.8 ng/g |
| Gliotoxin | 1261.74 | | 0 116 207 | | ≤207.87 ng/g |

Results are creatinine corrected to account for urine dilution variations.

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|------------------------|----------------|-----------------|--|-----------------|------------|
| Mycophenolic Acid | 1512.39 | |  | | ≤6.4 ng/g |
| Nivalenol (NIV) | 5029.32 | |  | | ≤3.2 ng/g |
| Ochratoxin A (OTA) | 4595.35 | |  | | ≤6.8 ng/g |
| Patulin | 5109 | |  | | ≤11.6 ng/g |
| Roridin A | 4812.43 | |  | | ≤7.6 ng/g |
| Roridin E | 5126.69 | |  | | ≤1.33 ng/g |
| Roridin L2 | 4157.39 | |  | | ≤6.8 ng/g |
| Satratoxin G | 5195.86 | |  | | ≤0.18 ng/g |
| Satratoxin H | 3682.05 | |  | | ≤0.18 ng/g |
| Sterigmatocystin (STC) | 3118.99 | |  | | ≤0.53 ng/g |
| T-2 Toxin | 883.29 | |  | | ≤0.18 ng/g |
| Verrucarin A | 4223.33 | |  | | ≤1.33 ng/g |
| Verrucarin J | 3392.25 | |  | | ≤9.2 ng/g |
| Zearalenone (ZEN) | 3768.04 | |  | | ≤0.67 ng/g |

Results are creatinine corrected to account for urine dilution variations.

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Urine Creatinine

| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
|------------------|----------------|-----------------|--|-----------------|-----------------|
| Urine Creatinine | 9.71 | |  | | 0.25-2.16 mg/mL |

COMMENTS

Urine Creatinine

Urine tests that measure ratio of analytes by creatine concentration will not be altered by urine volume, hydration status, or time of testing. When using creatinine concentration to measure urine analytes, the only interference with the test is if the person's creatinine levels are very high (which may be seen in kidney disease, diabetes, or competitive body builder athletes), or when creatinine levels are very low (which may be seen in people with muscle wasting or sarcopenia who have lost their lean muscle mass stores). High urine creatinine may cause falsely lower urine analyte results. Low urine creatinine may cause falsely higher urine analyte results. This does not invalidate the findings; rather, critical analysis should be used to correlate results with clinical history and symptomatology for intervention decision-making.

SAMPLE

Results are creatinine corrected to account for urine dilution variations.

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Aflatoxin

| TEST NAME | PERCENTILE | | REFERENCE | TEST NAME | PERCENTILE | | REFERENCE |
|---------------------|------------|---------|------------|---------------------|------------|---------|------------|
| | 75th | 95th | | | 75th | 95th | |
| Aflatoxin B1 (AFB1) | | 5055.98 | ≤6.93 ng/g | Aflatoxin B2 (AFB2) | | 3357.87 | ≤8.13 ng/g |
| Aflatoxin G1 | | 5839.93 | ≤6.53 ng/g | Aflatoxin G2 | | 2423.47 | ≤10.8 ng/g |
| Aflatoxin M1 | | 5557.57 | ≤6.4 ng/g | | | | |

COMMENTS

Aflatoxin B1 (AFB1)

Aflatoxin B1 (AFB1) is a mycotoxin produced by several strains of the fungus *Aspergillus flavus*. It is found in foods, specifically cereals such as corn and rice, tree nuts, oilseeds (peanut, cottonseed, etc.) and spices, notably those grown in tropical and sub-tropical regions. There is substantial evidence that aflatoxins cause hepatic carcinoma and AFB1, the most toxic aflatoxin, is classified as carcinogenic (Group 1) by the International Agency for Research on Cancer (IARC). Aflatoxins such as AFB1 can cause additive effects in individuals affected by hepatitis B. Wasting and weight loss, stunted growth and development in children, liver cirrhosis and aflatoxicosis are other conditions associated with aflatoxin ingestion.³

Aflatoxin B2 (AFB2)

Aflatoxin B2 (AFB2) is a mycotoxin produced by several *Aspergillus* spp. and found in contaminated foods or hay exposed to water or humid conditions. Exposure routes are primarily ingestion or inhalation. Ingestion can either occur directly from food such as grains, tree nuts, and oilseeds or can also occur from ingestion of milk or meat from animals fed contaminated feed. Toxicity of aflatoxins can be categorized as follows, in descending order of known toxic effects: aflatoxin B1, aflatoxin G1, aflatoxin B2, and aflatoxin G2. Animal studies have indicated that AFB2 has hepatotoxic, teratogenic, and carcinogenic effects.⁴

Aflatoxin G1

Aflatoxin G1 (AFG1) is a mycotoxin produced by the mold *Aspergillus parasiticus*. In the aflatoxin family, the most clinical research to date has been on aflatoxin B1 (AFB1). Nonetheless, aflatoxin G1 demonstrates cytotoxic and carcinogenic effects in numerous organ systems, such as hepatic, renal and lung tissues.¹ Per the available research, AFB1 is stronger than AFG1 regarding hepatic carcinogenicity but AFG1 induces a higher incidence of kidney tumors than AFB1. In comparing genotoxicity of different aflatoxins, studies indicate aflatoxin B1 is the most toxic aflatoxin, followed in descending order by aflatoxin G1, aflatoxin B2, and aflatoxin G2.²

Aflatoxin G2

Aflatoxin G2 (AFG2) is a mycotoxin primarily produced by the toxic fungi *Aspergillus parasiticus*, but also can be produced by related *Aspergillus* strains. The overall toxicity of aflatoxin G2 by itself has not been well characterized in the literature, however G2 appears to demonstrate the least genotoxic effects of studied aflatoxins.² It has been indicated that G2, while potentially less toxic, often occurs in combination with other aflatoxins in food sources.

Aflatoxin M1

Aflatoxin M1 (AFM1) is a mycotoxin produced primarily by *Aspergillus flavus* and *Aspergillus parasiticus*. Aflatoxin M1 (AFM1) is the monohydroxylated derivative of Aflatoxin B1 (AFB1). AFM1 is mainly found in the milk of cattle fed with contaminated Aflatoxin feed. Aflatoxin M1 can cause liver damage, poor production, immune suppression, internal hemorrhaging, muscle tremors, and impact gain and efficiency. Mortality rates may also increase due to the presence of these mycotoxins. AFM1 is highly resistant to processing technologies such as pasteurization, ultra-high temperature heating (UHT), and other processing methods, hence it is found in dairy products. AFM1 is found in lactating mothers too. Moreover, AFM1 is regarded as a human carcinogen.

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Other

| TEST NAME | PERCENTILE | | REFERENCE | TEST NAME | PERCENTILE | | REFERENCE |
|------------------------|------------|---------|-------------|------------------------|------------|---------|--------------|
| | 75th | 95th | | | 75th | 95th | |
| Chaetoglobosin A (CHA) | | 3717.08 | ≤31.87 ng/g | Citrinin (CTN) | | 3961.83 | ≤12.53 ng/g |
| Dihydrocitrinone | | 4543.72 | ≤16.53 ng/g | Enniatin B1 (ENN B1) | | 948.01 | ≤0.22 ng/g |
| Fumonisin B1 | | 3182.72 | ≤6.13 ng/g | Fumonisin B2 | | 3296.01 | ≤7.2 ng/g |
| Fumonisin B3 | | 2784.37 | ≤10.8 ng/g | Gliotoxin | | 1261.74 | ≤207.87 ng/g |
| Mycophenolic Acid | | 1512.39 | ≤6.4 ng/g | Ochratoxin A (OTA) | | 4595.35 | ≤6.8 ng/g |
| Patulin | | 5109 | ≤11.6 ng/g | Sterigmatocystin (STC) | | 3118.99 | ≤0.53 ng/g |
| Zearalenone (ZEN) | | 3768.04 | ≤0.67 ng/g | | | | |

COMMENTS

Chaetoglobosin A (CHA)

Chaetoglobosin A (CHA) is one of the chaetoglobosin mycotoxins produced from the fungus *Chaetomium globosum*. *Chaetomium globosum* is the third most common indoor fungal contaminant of damp buildings. It has been isolated from several places such as soil, dung, feeds, foods, textiles, plywood, carpet, wallpaper and wet walls. Inhalation of airborne spores and CHA from this fungus is known to produce respiratory as well as systemic infection in human beings. Case reports of infections include mild to severe illness, from sinusitis, onychomycosis, and cutaneous infections to disseminated cerebral disease, pneumonia and keratitis.¹⁹ Relatively low levels of CHA have been shown to be lethal to various tissue culture cell lines and it is thought that CHA is highly toxic, even at minimal doses.

Citrinin (CTN)

Citrinin (CTN) is a mycotoxin produced by several fungal strains in the *Penicillium*, *Aspergillus* and *Monascus* genera. It is generally formed in stored grains such as rice, wheat, oats, etc., but can also be found in other crops such as peanuts, olives, apples and cheese. Red yeast rice can be contaminated by citrinin, and studies are mixed regarding prevalence of a high presence in dietary supplements targeted for cholesterol management.¹² The kidney is the predominant organ of CTN related toxicity which is thought to be linked to oxidative stress and mitochondrial dysfunction. CTN is usually found in foods together with another nephrotoxic mycotoxin, ochratoxin A, which generally is thought to have stronger nephrotoxic effects. Other reports effects from CTN include liver and bone-marrow toxicity.¹³

Dihydrocitrinone

Dihydrocitrinone (DHC) is a metabolite of Citrinin (CTN), which is a mycotoxin produced by several fungal strains in the *Penicillium*, *Aspergillus* and *Monascus* genera and found in stored grains and other food products. Citrinin has nephrotoxic and genotoxic effects, and often occurs in combination with ochratoxin A. Studies show that dihydrocitrinone (DHC) has a significantly reduced cytotoxic and genotoxic potential as compared to CTN. Thus, DHC is considered a detoxification related step of CIT metabolism.¹⁸

Enniatin B1 (ENN B1)

Enniatin B1 (ENN B1) is a mycotoxin produced by *Fusarium* spp. fungi. It occurs in grains such as rice and corn, as well as fish, fruits, nuts, coffee and cocoa products. Pre-clinical research indicates that ENN B1 has cytotoxic effects related to oxidative stress, cell cycle disruption, mitochondrial modifications, and apoptosis. Genotoxic effects and adverse effects from acute exposures have not been found in pre-clinical studies. Additive cytotoxicity, when ENN B1 is in the presence of other mycotoxins, has been demonstrated.⁹

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Other

COMMENTS

Fumonisin B1

Fumonisin B1 (FB1) is a mycotoxin produced by species of the Fusarium family of fungi, such as *F. verticillioides* and *F. proliferatum*. FB1 is present in contaminated corn, cereal grains, and other agricultural products, as well as water damaged buildings. Many different fumonisins have so far been reported, however, FB1 is the most abundant fumonisin, accounting for 70% of the total fumonisins naturally occurring in infected food and feed samples. Fumonisin contaminated diets in children have shown negative impacts on growth, and in pregnant women, have been associated with neural tube defects in the developing fetus. Several studies show FB1 may have a contributing role in human esophageal and hepatic carcinogenesis. The International Agency for Research on Cancer (IARC) characterized FB1 as a group 2B possible carcinogen for humans.¹⁰ Toxic effects result from oxidative stress, apoptosis, necrosis and cell-cycle alterations.¹¹

Fumonisin B2

Fumonisin B2 (FB2) is a mycotoxin produced by *Fusarium* spp. such as *Fusarium verticillioides* and *F. proliferatum*. Many different fumonisins are reported, however, toxicology studies have mainly focused on fumonisin B1, with detection of concurrent fumonisins B2 and B3 growth noted. In terms of quantity of fumonisins detected, fumonisin B1 (FB1) accounts for the largest concentration of fumonisin mycotoxin, however fumonisin B2 (FB2) is also frequently found in a lesser amount in cereals, corn, and other agricultural products. More recently it was found the *Aspergillus niger* also produces FB2 in grape products, coffee bean, dried figs and beer. While less clinically relevant data is available for FB2, pre-clinical evidence suggests similar mechanisms of toxicity to FB1, such as oxidative stress, cytotoxicity, and disturbance of sphingolipid metabolism. The overall ranking of severity of toxicity of fumonisins is in descending order, FB1, FB2, followed by FB3.¹¹

Fumonisin B3

Fumonisin B3 (FB3) is a mycotoxin produced by *Fusarium* spp. such as *Fusarium verticillioides* and *F. proliferatum*. Many different fumonisins are reported, however, toxicology studies have mainly focused on fumonisin B1, with detection of co-occurring fumonisins B2 and B3 in food products observed. While less clinically relevant data is available for fumonisin B3, pre-clinical evidence suggests toxicity is less than fumonisins B1 and B2.¹¹

Gliotoxin

Gliotoxin (GT) is the main mycotoxin produced by *Aspergillus fumigatus* but can also be produced by *Gliocadium fimbriatum* and species of *Trichoderma* and *Penicillium*. Gliotoxin is produced from mold growth in compost, decaying leaf piles, and water-damaged buildings. Exposure from inhalation of spores and particulates has a wide range of immune system and health effects. Gliotoxin causes immunosuppression via NF-kB inhibition and interference with transcription factors involved with T-cell activation. Furthermore, gliotoxin is linked to invasive pulmonary aspergillosis, disruption of the blood-brain barrier, and many non-specific symptoms such as headache, malaise, skin hypersensitivity, eye irritation, olfactory disruption, and respiratory arrest.^{15,16}

Mycophenolic Acid

Mycophenolic Acid (MPA) is a mycotoxin produced by a number of *Penicillium* species. *Penicillium brevicompactum*, a species able to produce mycophenolic acid (MPA), has also been frequently identified in indoor environments that can be found on building materials, in dust, and in air samples. MPA is a known immunosuppressant. The main mechanism of action of MPA is the specific inhibition of inosine monophosphate dehydrogenase, which is highly expressed in T- and B-lymphocytes. Enzyme inhibition blocks lymphocyte and monocyte proliferation and leads to immunosuppression.¹⁷ MPA has been formulated into an immunosuppressant prescription drug, mycophenolate mofetil, and has been used since the 1990's to prevent organ transplant rejection. Side effects include gastrointestinal toxicity and carcinogenesis, among others.

Ochratoxin A (OTA)

Ochratoxin A (OTA), a renal toxin, is produced majorly by *Aspergillus* and *Penicillium* fungal species. Ochratoxin A has been found in barley, oats, rye, wheat, coffee beans, and other plant products, with barley having a particularly high likelihood of contamination. It is also frequently found in pork intended for human consumption. OTA is absorbed in the small intestine and distributed via the blood, to mainly the kidneys, where higher concentrations are found. OTA toxicity is linked to renal conditions such as Balkan endemic nephropathy and chronic interstitial nephropathy. It is also a potential urothelial carcinogen via oxidative stress and direct genotoxic mechanisms. It has been theorized that increases in carcinogenicity and genotoxicity occur during co-exposure with citrinin (CIT), fumonisin (FB). OTA has a long elimination half-life and is linked to intestinal barrier disruption and stimulation of inflammatory cytokines.⁵

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Other

COMMENTS

Patulin

Patulin is a mycotoxin produced from several species of mold such as *Aspergillus*, *Byssoschlamys* and *Penicillium*. Patulin is mainly obtained through ingestion of mycotoxins in fruit such as apples and apple-derived products such as juice, cider puree, etc., that have been colonized by blue mold. Gastrointestinal disturbances such as nausea and vomiting have been reported in humans exposed to elevated patulin levels, however chronic exposure can have neurotoxic, immunosuppressive, and teratogenic properties. Limits on patulin levels in commercial agriculture products have been established by many countries.¹⁴

Sterigmatocystin (STC)

Sterigmatocystin (STC) is a mycotoxin produced by several *Aspergillus* species. STC can be present in a wide variety of crops such as grains, corn, nuts, cheese, coffee, etc., however rice and oats are affected the most.⁶ STC is an intermediate of the aflatoxin biosynthetic pathway. While its biological activity is like that of aflatoxins, studies show that the carcinogenic activity of STC is weaker than that of aflatoxin B1. Animal studies show hepatic necrosis and nephrotoxicity with acute exposure, and in-vitro studies show genotoxic effects. The clinical effects of STC in humans is relatively unclear therefore, this mycotoxin is not currently regulated in food production.⁷

Zearalenone (ZEN)

Zearalenone (ZEN) is a mycotoxin produced by fungi of the genus *Fusarium* and commonly found in maize, beans, grains and animal feeds. Zearalenone has been found to have immunotoxic, hepatotoxic and estrogenic effects. The estrogenic effects occur due to similarity of the ZEN structure to estrogen structures, where ZEN can bind to both alpha and beta estrogen receptors and disrupt endocrine function. Various estrogenic effects have been noted in animals and humans including fertility disorders, premature puberty, breast enlargement, and feminization traits in males.

Trichothecenes

| TEST NAME | PERCENTILE | | REFERENCE | TEST NAME | PERCENTILE | | REFERENCE |
|---------------------|------------|---------|-------------|--------------------------|------------|---------|------------|
| | 75th | 95th | | | 75th | 95th | |
| Deoxynivalenol(DON) | | 783.53 | ≤67.47 ng/g | Diacetoxyscirpenol (DAS) | | 3157.32 | ≤4.27 ng/g |
| Nivalenol (NIV) | | 5029.32 | ≤3.2 ng/g | Roridin A | | 4812.43 | ≤7.6 ng/g |
| Roridin E | | 5126.69 | ≤1.33 ng/g | Roridin L2 | | 4157.39 | ≤6.8 ng/g |
| Satratoxin G | | 5195.86 | ≤0.18 ng/g | Satratoxin H | | 3682.05 | ≤0.18 ng/g |
| T-2 Toxin | | 883.29 | ≤0.18 ng/g | Verrucarin A | | 4223.33 | ≤1.33 ng/g |
| Verrucarin J | | 3392.25 | ≤9.2 ng/g | | | | |

COMMENTS

Deoxynivalenol(DON)

Deoxynivalenol (DON) is a type B trichothecene primarily produced by the *Fusarium* species of fungi. It is one of the most common mycotoxin contaminants found in wheat, corn, barley and other cereal based grains. It is also found in animal feed as well as grain-based products such as beer, noodles and soy sauce.²¹ DON can cause health effects at chronic low doses as well as acute effects at high or toxic doses. Most common symptoms are related to intestinal effects such as inflammation and intestinal barrier disruption. Intestinal toxicity of DON is attributed to mitochondrial dysfunction (caused due to reactive oxygen species) as a critical factor. Higher dose exposure can cause emesis, weight loss, immune suppression, as well as hematologic, skin, cardiovascular, nervous system and endocrine issues.²¹

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Trichothecenes

COMMENTS

Diacetoxyscirpenol (DAS)

Diacetoxyscirpenol (DAS), also known as anguidine, is a type A trichothecene mycotoxin primarily produced by *Fusarium* fungi. DAS has mainly been reported in numerous grains (principally wheat, sorghum, maize, barley, and oats), but can also present in potato products, soybeans, and coffee. DAS is commonly found to co-occur with other *Fusarium* mycotoxins such as type A and B trichothecenes and zearalenone.²⁴ Trichothecenes, in general, are classified as gastrointestinal toxins, dermatotoxins, immunotoxins, hematotoxins, and gene toxins. DAS, along with T-2 and HT-2, are considered among the most toxic in the trichothecene group. DAS are recognized as having multiple inhibitory effects on eukaryote cells, including inhibition of protein, DNA, and RNA synthesis; inhibition of mitochondrial function; effects on cell division; and membrane effects. The toxic effects of DAS in humans and animals are similar and include vomiting, diarrhea, hypotension, and myelosuppression.²⁵

Nivalenol (NIV)

Nivalenol (NIV) is a Type B trichothecene mycotoxin produced by several *Fusarium* spp. It is commonly found in crops such as wheat, barley and corn and persists in foods despite food processing. Structurally, NIV is similar to DON, and often found alongside DON in foods, however, the oxidative stress and toxicity of NIV is greater than that of DON.²² Nivalenol is thought to signal a series of cellular processes that result in inflammation and apoptosis in fast growing cells with resultant immunosuppression, gastrointestinal toxicity and genotoxicity. There are associations in the literature with esophageal and gastric carcinomas, as well as Kashin-Beck disease.²³

Roridin A

Roridin A is a macrocyclic trichothecene produced from *Stachybotrys chartarum* and it has been found in water damaged buildings as well as on mold contaminated grain and straw crops. Humans can be exposed to *Stachybotrys* through dermal contact, ingestion and inhalation. Macrocyclic trichothecenes are the most cell-toxic trichothecenes currently known. Pre-clinical studies show roridin A has the potential to cause apoptosis of olfactory neurons with resultant atrophy of the olfactory epithelium and olfactory bulbs after inhalation exposure. Pathology is potentiated by the simultaneous exposure to lipopolysaccharide, which is also released in water contaminated buildings. ³³

Roridin E

Roridin E is a well-known macrocyclic trichothecene mycotoxin produced by various species of fungi such as *Fusarium*, *Myrothecium*, *Trichoderma*, *Trichothecium*, *Cephalosporium*, *Verticimonosporium*, and *Stachybotrys*. Trichothecenes are produced on many different grains like wheat, oats, or maize, primarily by *Fusarium* species. However, some molds that produce trichothecene mycotoxins, such as *Stachybotrys chartarum*, can grow in damp indoor environments and may contribute to health problems among building occupants. Trichothecenes are considered extremely toxic and have been used as biological warfare agents. Trichothecenes have multiorgan effects, including anorexia and weight loss; growth retardation; nervous disorders; cardiovascular alterations; immunodepression; hemostatic derangements; skin toxicity; decreased reproductive capacity; bone marrow damage; and alimentary toxic aleukia.

Roridin L2

Roridin L2, a fungal metabolite, is a common trichothecene produced by *Stachybotrys chartarum* and a biosynthetic precursor of Satratoxin G. Both are present in water damaged homes. It is theorized that structural differences between roridin L2 and satratoxin G account for differing levels of toxicity, with the former having a less toxic impact due to potential inability to diffuse through the cell membrane.³⁴

Satratoxin G

Satratoxin G is a macrocyclic trichothecene mycotoxin produced by *Stachybotrys chartarum*, commonly called black mold. Humans can be exposed to *Stachybotrys* through dermal contact, ingestion and inhalation. Macrocyclic trichothecenes are the most cell-toxic trichothecenes currently known. Satratoxins within this category are known to inhibit protein biosynthesis and induce cell death in neuronal cell lines.²⁸ Known adverse health effects of satratoxin G through inhalation include neurotoxicity and inflammation within the nose and brain,²⁹ as well pulmonary effects.

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Trichothecenes

COMMENTS

Satratoxin H

Satratoxin H is a macrocyclic trichothecene mycotoxin produced by *Stachybotrys chartarum*, commonly known as black mold, as well as the mushroom *Podostroma cornudamae*. Satratoxins within this category are known to inhibit protein biosynthesis and induce cell death in neuronal cell lines.³⁰ Satratoxin H is one of the causative agents associated with "sick building syndrome" and results from water damage to cellulose-rich building materials such as gypsum or wallpaper. It has also been found in foods on corn, barley and oat samples. Respiratory, neurological, ocular, hematological, and dermatological symptoms have been attributed to *Stachybotrys* mycotoxin exposure, including idiopathic pulmonary hemorrhage in infants.³¹

T-2 Toxin

T-2 toxin is a Type A trichothecene produced by species of *Fusarium* and it has the highest toxicity of secondary metabolites produced in this genus.²⁶ Humans have exposure to T-2 from improperly stored grains where *Fusarium* can develop. Systemic illness can occur from either GI, inhalational or transdermal contact. T-2 toxins can cross the blood-brain barrier and accumulate in the central nervous system (CNS). Oral ingestion of T-2 toxins has led to alimentary toxic aleukia, amongst other severe systemic effects. The mechanism involved in T-2 toxicity is an inhibitory effect on protein synthesis, generally mediated via oxidative stress-mediated DNA damage and apoptosis. Due to its extreme stability and toxicity, T-2 has been categorized as a biological weapon.²⁷

Verrucarin A

Verrucarin A is a Type D, macrocyclic trichothecene mycotoxin which is produced by *Stachybotrys*, *Fusarium*, and *Myrothecium* species. It can occur from mold growth on water damaged buildings or naturally in crops used for human and animal consumption. While verrucarin A is a known toxic compound, it has been studied for selective anti-cancer effects. Known cytotoxic effects include inhibition of protein and DNA and RNA synthesis, interference with mitochondrial function, as well as effects on cell division and on cell membranes.²⁰

Verrucarin J

Verrucarin J is a trichothecene produced by *Stachybotrys chartarum*. They can grow in damp indoor environments and may contribute to health problems among building occupants. Verrucarin J molecules are small enough to be airborne and easily inhaled. Inhalation is the most dangerous form of exposure, but with Verrucarin J being lipophilic, mycotoxins can easily cross cell membranes, which means they can be absorbed through the mouth and even the skin. Verrucarin J can inhibit protein synthesis as well as DNA and RNA damage in human cells.

Risk and Limitations

This test has been developed and its performance characteristics determined by Vibrant America LLC., a CLIA certified lab. These assays have not been cleared or approved by the U.S. Food and Drug Administration.

Mycotoxins do not demonstrate absolute positive and negative predictive values for mold related illnesses. Its clinical utility has not been fully established. Clinical history and current symptoms of the individual must be considered by the healthcare provider prior to any interventions. Test results should be used as one component of a physician's clinical assessment. Quantification of mycotoxins in urine is not FDA-recognized diagnostic indicator of mold exposure.

Mycotoxins testing is performed at Vibrant America, a CLIA certified laboratory. Vibrant America has effective procedures in place to protect against technical and operational problems. However, such problems may still occur. Examples include failure to obtain the result for a specific mycotoxin due to circumstances beyond Vibrant's control. Vibrant may re-test a sample to obtain these results but upon re-testing the results may still not be obtained. As with all medical laboratory testing, there is a small chance that the laboratory could report incorrect results. A tested individual may wish to pursue further testing to verify any results.

The information in this report is intended for educational purposes only. While every attempt has been made to provide current and accurate information, neither the author nor the publisher can be held accountable for any errors or omissions.

Vibrant Wellness makes no claims as to the diagnostic or therapeutic use of its tests or other informational materials. Vibrant Wellness reports and other information do not constitute medical advice and are not a substitute for professional medical advice. Please consult your healthcare provider for questions regarding test results, or before beginning any course of medication, supplementation, or dietary changes. Users should not disregard, or delay in obtaining, medical advice for any medical condition they may have, and should seek the assistance of their health care professionals for any such conditions.

SAMPLE

Heavy Metals

PATIENT

NAME: TEST TEST
DATE OF BIRTH: 1991-11-11 GENDER: Female
TELEPHONE: 11234567890 AGE: 31

ACCESSION ID: 2306270585
SPECIMEN COLLECTED: 2023-06-27 10:00 (PST)
SPECIMEN RECEIVED: 2023-06-27 14:47 (PST)
FINAL REPORT DATE: 2023-06-27 15:15 (PST)
GENERATION DATE: 2023-06-27 15:25 (PST)

FASTING:

PROVIDER:

PRACTICE NAME: Vibrant IT3 Practice
PROVIDER NAME: Vibrant IT3333
PHLEBOTOMIST: No Matched Result

TELEPHONE: +15555555555
FAX #: 650-331-7393
ADDRESS: Apple Park Way, Millcreek, UT

Vibrant Wellness is pleased to present to you, 'Heavy Metals panel', to help you make healthy lifestyle, dietary and treatment choices in consultation with your healthcare provider. It is intended to be used as a tool to encourage a general state of health and well-being.

The Heavy Metals is a test to measure levels of Heavy Metals Toxins in your urine that you might be exposed to.

Reference ranges are established based on NHANES study where applicable. Other reference ranges are established based on 1000 apparently healthy urine samples.

The report begins with the summary page which lists only the heavy metal toxins whose levels are >95th percentile (Red) and 75th-95th percentile (Yellow) of reference range, normalized to Urine creatinine levels. Additionally, the previous value is also indicated for your referral (if available). Following this section is the complete list of the heavy metal toxins and their absolute levels normalized to Creatinine in a quantile format along with the reference ranges. These levels are shown with three shades of color – Green, Yellow and Red. The result in green corresponds to 0 to 75th percentile, the result in yellow corresponds to 75th to 95th percentile and the result in red corresponds to greater than 95th percentile of reference range. All content provided in the report are purely for informational purposes only and should not be considered medical advice. Any changes based on the information should be made in consultation with your healthcare provider.

The Vibrant Wellness platform provides tools for you to track and analyze your general wellness profile. Testing for the Heavy Metals panel is performed by Vibrant America, a CLIA certified lab CLIA#:05D2078809. Vibrant Wellness provides and makes available this report and any related services pursuant to the Terms of Use Agreement (the "Terms") on its website at www.vibrant-wellness.com. By accessing, browsing, or otherwise using the report or website or any services, you acknowledge that you have read, understood, and agree to be bound by these terms. If you do not agree to accept these terms, you shall not access, browse, or use the report or website. The statements in this report have not been evaluated by the Food and Drug Administration and are only meant to be lifestyle choices for potential risk mitigation. Please consult your physician/dietitian for medication, treatment, or lifestyle management. This product is not intended to diagnose, treat, or cure any disease.

Pediatric ranges have not been established for this test. It is important that you discuss any modifications to your diet, exercise, and nutritional supplementation with your physician before making any changes.

Heavy Metals Summary

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

High (>95th percentile)

| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
|------------|----------------|-----------------|----------------|-----------------|-------------|
| Aluminum | 118.98 | | 0 17.8 45.1 | | ≤45.15 ug/g |
| Antimony* | 6.31 | | 0 0.07 0.16 | | ≤0.16 ug/g |
| Arsenic* | 269.74 | | 0 11.9 52 | | ≤52 ug/g |
| Barium* | 12.24 | | 0 2.33 5.59 | | ≤5.59 ug/g |
| Beryllium* | 14.45 | | 0 0.2 0.76 | | ≤0.76 ug/g |
| Bismuth | 24.54 | | 0 0.58 2.53 | | ≤2.53 ug/g |
| Cadmium* | 34.59 | | 0 0.29 0.8 | | ≤0.8 ug/g |
| Cesium* | 13.27 | | 0 6.37 10.3 | | ≤10.3 ug/g |
| Gadolinium | 6.11 | | 0 0.17 0.45 | | ≤0.45 ug/g |
| Lead* | 114.54 | | 0 0.52 1.16 | | ≤1.16 ug/g |
| Mercury* | 81.01 | | 0 0.57 1.61 | | ≤1.61 ug/g |
| Nickel | 20.54 | | 0 6.37 12.1 | | ≤12.13 ug/g |
| Palladium | 19.62 | | 0 0.15 0.2 | | ≤0.2 ug/g |

* Indicates NHANES population data reference ranges.

SPECIMEN INFORMATION

Provoking Status: **unavailable**

Agent:

Dosage:

Results are creatinine corrected to account for urine dilution variations. Reference intervals are based upon NHANES(cdc.gov/nhanes) data if available, and are representative of a large population cohort under non-provoked conditions. Chelation (provocation) agents can increase urinary excretion of metals/elements.

Heavy Metals Summary

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

High (>95th percentile)

| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
|-----------|----------------|-----------------|----------------|-----------------|------------|
| Platinum* | 9.59 | | 0 0.1 0.9 | | ≤0.9 ug/g |
| Tellurium | 21.57 | | 0 0.42 0.89 | | ≤0.89 ug/g |
| Thallium* | 362.42 | | 0 0.24 0.43 | | ≤0.43 ug/g |
| Thorium | 20.4 | | 0 0.02 0.07 | | ≤0.07 ug/g |
| Tin* | 6.94 | | 0 1 3.72 | | ≤3.72 ug/g |
| Tungsten* | 5.08 | | 0 0.12 0.33 | | ≤0.33 ug/g |
| Uranium* | 12.26 | | 0 0.02 0.04 | | ≤0.04 ug/g |

* Indicates NHANES population data reference ranges.

Urine Creatinine

| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
|------------------|----------------|-----------------|----------------|-----------------|-----------------|
| Urine Creatinine | 9.71 | | 0 0.24 2.16 | | 0.25-2.16 mg/mL |

COMMENTS

Urine Creatinine

Urine tests that measure ratio of analytes by creatine concentration will not be altered by urine volume, hydration status, or time of testing. When using creatinine concentration to measure urine analytes, the only interference with the test is if the person's creatinine levels are very high (which may be seen in kidney disease, diabetes, or competitive body builder athletes), or when creatinine levels are very low (which may be seen in people with muscle wasting or sarcopenia who have lost their lean muscle mass stores). High urine creatinine may cause falsely lower urine analyte results. Low urine creatinine may cause falsely higher urine analyte results. This does not invalidate the findings; rather, critical analysis should be used to correlate results with clinical history and symptomatology for intervention decision-making.

SPECIMEN INFORMATION

Provoking Status: **unavailable**

Agent:

Dosage:

Results are creatinine corrected to account for urine dilution variations. Reference intervals are based upon NHANES(cdc.gov/nhanes) data if available, and are representative of a large population cohort under non-provoked conditions. Chelation (provocation) agents can increase urinary excretion of metals/elements.

Heavy Metals

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Heavy Metals * Indicates NHANES population data reference ranges.

| TEST NAME | PERCENTILE | | REFERENCE | TEST NAME | PERCENTILE | | REFERENCE |
|------------|------------|--------|-------------|-----------|------------|--------|-------------|
| | 75th | 95th | | | 75th | 95th | |
| Aluminum | | 118.98 | ≤45.15 ug/g | Antimony* | | 6.31 | ≤0.16 ug/g |
| Arsenic* | | 269.74 | ≤52 ug/g | Barium* | | 12.24 | ≤5.59 ug/g |
| Beryllium* | | 14.45 | ≤0.76 ug/g | Bismuth | | 24.54 | ≤2.53 ug/g |
| Cadmium* | | 34.59 | ≤0.8 ug/g | Cesium* | | 13.27 | ≤10.3 ug/g |
| Gadolinium | | 6.11 | ≤0.45 ug/g | Lead* | | 114.54 | ≤1.16 ug/g |
| Mercury* | | 81.01 | ≤1.61 ug/g | Nickel | | 20.54 | ≤12.13 ug/g |
| Palladium | | 19.62 | ≤0.2 ug/g | Platinum* | | 9.59 | ≤0.9 ug/g |
| Tellurium | | 21.57 | ≤0.89 ug/g | Thallium* | | 362.42 | ≤0.43 ug/g |
| Thorium | | 20.4 | ≤0.07 ug/g | Tin* | | 6.94 | ≤3.72 ug/g |
| Tungsten* | | 5.08 | ≤0.33 ug/g | Uranium* | | 12.26 | ≤0.04 ug/g |

COMMENTS

Aluminum

Aluminum (atomic number 13) is the most widely distributed metal in the environment and has many consumer applications—including pots, pans, beverage cans, foil, antacids, antiperspirants, cosmetics, and food additives (e.g., baking powder, coloring agents, and anticaking agents). Therefore, aluminum intoxications may occur frequently. Exposures to aluminum may extensively occur in occupations associated with mining and processing of ore, scrap metal recycling, welding, etc. Humans living in environments contaminated by industrial wastes may also be exposed to high levels of aluminum. Intake of aluminum can occur by inhalation of aerosols or particles, ingestion of food, water, medicaments, skin contact, vaccination, dialysis, and infusions. The mechanisms of aluminum toxicity include changes in cell membrane permeability, inhibition of enzyme activity, protein denaturation/transformation, and disruption of iron homeostasis leading to iron overload-induced lipid peroxidation and increased reactive oxygen species. Aluminum poisoning can affect blood content, musculoskeletal system, kidney, liver, respiratory and nervous system. Early symptoms of aluminum toxicity include flatulence, headaches, colic, dryness of the skin and mucous membranes, and tendencies for colds. Later symptoms may include paralytic muscular conditions, loss of memory, and mental confusion.

Antimony

Antimony (atomic number 51) is a silvery-white metal found in the earth's crust. Its main applications are industrial, including semiconductors, batteries, castings, bearings, pewter, and flame-retardant materials. Antimony is also used to treat the parasitic diseases schistosomiasis and leishmaniasis. The mechanism of antimony toxicity is enzyme inhibition—especially enzymes involved in cellular respiration and carbohydrate/protein metabolism—due to the binding of sulfhydryl groups. Occupational exposure to antimony occurs mainly in workers involved in industries producing antimony and antimony trioxide, metal mining, smelting and refining, coal-fired power plants, refuse incineration, or those working in indoor firing ranges. Inhalation of aerosols and oral intake of foods contaminated with antimony are the major routes of antimony exposure. However, antimony can be absorbed by dermal routes as well. Antimony intoxication may cause respiratory irritation, pneumoconiosis, antimony spots on the skin, and gastrointestinal conditions. Major clinical manifestations associated with antimony toxicity include eye and lung irritation, skin irritation, stomach pain, diarrhea, vomiting, and stomach ulcers.

Heavy Metals

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Heavy Metals

COMMENTS

Arsenic

Arsenic (atomic number 33) is a naturally occurring element distributed throughout the earth's crust and in groundwater. At lower levels, it is also found in the air and in food products. Ingestion and inhalation are the most common routes of exposure to arsenic. However, dermal exposure may lead to illness. Arsenic-contaminated water—used for drinking, food preparation, and irrigation of food crops—poses the greatest threat to public health. According to the American Cancer Society, the foods with the highest levels of arsenic are seafood, rice (including rice cereal), mushrooms, and poultry. Because tobacco plants can take up arsenic naturally present in the soil, people who smoke may have higher levels. The mechanisms of arsenic toxicity include inactivating enzymes involved in cellular energy pathways, DNA synthesis, and DNA repair. Acute exposure to arsenic can lead to gastroenteritis followed by hypotension. Chronic exposure can lead to the risk of developing skin lesions, cardiovascular diseases, diabetes, affected cognitive abilities, and cancer.

Barium

Barium (atomic number 56) is an alkaline earth metal that is typically found in food and groundwater. It is extensively used in electronic tubes, rodenticides, colorants in paint, and X-ray contrast medium. Exposure to barium may occur through drinking groundwater, as well as through skin contact, from ingesting it accidentally with polluted material/food, and from direct injection via X-ray contrast medium. The mechanism of toxicity for barium includes interfering with the potassium channels and, in the GI tract, stimulating acid and histamine secretion and peristalsis. Major symptoms associated with barium intoxication include hypokalemia, diarrhea, nausea, vomiting, heart rhythm abnormalities, muscle cramps, and kidney disorders.

Beryllium

Beryllium (atomic number 4) is a silvery-white metal that is lighter than aluminum but stronger than steel. Its strength-to-weight ratio, reflectivity, transparency to X-rays, thermal stability and conductivity, and high melting point makes it an essential material in the defense, nuclear, aerospace, medical, information technology, and telecommunications industries. Humans are exposed to it via airborne particles of beryllium metal, alloys, oxides, and ceramics—including golf clubs, bicycles, dental appliances, automotive parts, computer parts, and microwaves. Beryllium particles are inhaled into the lungs and the upper respiratory tract. Additionally, hand-to-mouth exposures and skin contact with ultra fine particles may also occur. The mechanism of beryllium toxicity is not well understood. Mostly likely, beryllium combines with certain proteins, releasing toxic substances. Acute beryllium toxicity can cause pneumonitis, cough, chest pain, dyspnea, and pneumonia. Chronically, it results in sarcoid-like granulomata mainly in the lungs and is occasionally subcutaneous. It is a skin irritant and can also cause conjunctivitis, rhinitis, and pharyngitis. Additionally, some individuals exposed to beryllium develop sensitization and are at risk of developing chronic beryllium disease (CBD) which is characterized by abnormally exaggerated immune responses.

Bismuth

Bismuth (atomic number 83) is a high-density, silvery, pink-tinged metal that is so brittle it is usually mixed with other metals to make it useful. Its alloys with cadmium or tin have low melting points and are used in electrical fuses, fire detectors and extinguishers, and solders. It is also a byproduct of iron ore manufacturing. Bismuth is a commonly used supplemental product for the treatment of symptoms associated with gastric ulcers, excess abdominal gas, and diarrhea. Intoxication with bismuth is rare. However, bismuth exposure can occur via inhalation of soldering fumes when working near lead-free pipes (which contain bismuth as a lead substitute) or by the consumption of water containing traces of bismuth or from the intake of over-the-counter medicines that contain bismuth (chewable or liquid). Small doses of bismuth may cause mild gastrointestinal discomforts such as nausea or epigastric discomfort. On the other hand, chronic ingestion of bismuth may lead to symptoms of nausea, vomiting, encephalopathy (confusion, disorientation, possibly seizures), acute neurological symptoms such as ataxia, confusion, short term memory impairment, dysarthria, myoclonus (involuntary muscle jerk), and paresthesia (burning or prickling sensation in the hands, arms, legs, or feet, etc.). Renal and hepatic failure may occur with high levels of toxicity. In chronic bismuth poisoning, individuals may also have a blue-black gum line and lichen planus-like skin rashes.

Cadmium

Cadmium (atomic number 48) is a natural element found in tiny amounts in air, water, soil, and food. It is used in batteries, alloys for electroplating (auto industries), the production of pigments, and as stabilizers for polyvinyl plastic. Exposure to cadmium occurs primarily occurs via ingestion of foods grown in contaminated soil or by the inhalation of cigarette smoke. According to the Agency for Toxic Substances and Disease Registry, dermal absorption of cadmium is negligible. Cadmium toxicity generates reactive oxygen species, interferes with DNA repair, and binds the mitochondria affecting cell proliferation, differentiation, and apoptosis. Symptoms of cadmium toxicity include anemia, liver disease, vomiting, diarrhea, kidney disease, and impaired bone density. Long-term exposure to cadmium may lead to cancer and organ system toxicity such as skeletal, urinary, reproductive, cardiovascular, central and peripheral nervous, and respiratory systems.

Heavy Metals

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
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Heavy Metals

COMMENTS

Cesium

Cesium (atomic number 55) is a naturally occurring element found combined with other elements in rocks, soil, and dust in low amounts. It is used to make atomic clocks, optical glass, and vacuum tubes. Nuclear explosions or the breakdown of uranium in fuel elements can produce radioactive forms of cesium. Exposure to stable or radioactive cesium occurs from ingesting contaminated food or drinking water or breathing contaminated air. In muscle cells, cesium competes with, and displaces, potassium. Such exposures may cause nausea, vomiting, diarrhea, bleeding, fatigue, muscle weakness, and palpitations. In severe conditions, it can cause cardiac arrhythmia, coma, and even death. Cesium can cause DNA damage which can affect genomic stability. Altered in genomic stability is a hallmark of aging. Thus, cesium toxicity may accelerate aging owing to its contribution to genomic instability.

Gadolinium

Gadolinium (atomic number 64) is a rare earth metal. It is typically used in microwave technology, color TV tubes, synthetic gemstones, compact discs, and computer memory. It is also used as a contrast dye for MRI testing, wherein it is injected into the bloodstream and it gets stored in the blood vessels and in abnormal tissue, thus enabling the easy detection of problems found in the body. As a result, the most common route of gadolinium exposure is via the injection of the contrast dye used with an MRI. The symptoms of gadolinium toxicity can present shortly after an MRI and can present as aching, burning, tingling, tight skin, lesions, hyperpigmentation, muscle twitching, worsening vision, tinnitus, swallowing, and voice problems, hair loss, edema, and balance problems. Limited clinical data suggests the potential mechanisms of gadolinium toxicity include expression and release of cytokines involved in tissue fibrosis, blockage of calcium-dependent enzymes, elevation of reactive oxygen species, and apoptosis.

Lead

Lead (atomic number 82) is the most predominant toxic heavy element in the environment. It is soft, malleable, and ductile with poor conductivity and resistance to corrosion. Lead continues to accumulate in the environment due to its abundant distribution across the globe, non-biodegradable nature, and ongoing use. Exposure to lead and its compounds occurs mostly in lead-related occupations with various sources like leaded gasoline, industrial processes such as smelting of lead and its combustion, pottery, boat building, lead-based painting, lead-containing pipes, battery recycling, grids, pigments, printing of books, and so on. Additionally, traditional herbal medicines, ayurvedics, and cosmetics, can be high in lead. Lead is a highly poisonous metal affecting almost every organ in the body. It attacks the brain and central nervous system, which may cause coma, convulsions, and even death. Lead toxicity can particularly affect children's brain development, resulting in reduced intelligence quotient (IQ), affected behavioral and cognitive functions. Lastly, lead exposure may also cause anemia, hypertension, renal impairment, immunotoxicity, and toxicity to the reproductive organs. Lead is seen to influence protein stability by altering rates of protein synthesis and degradation, and protein folding. It is believed that alterations in brain proteins could be the cause of lead-induced neurotoxicity. Dysregulated protein stability is a hallmark of aging. Thus, lead intoxication may disrupt many biological processes which could lead to the risk of accelerated aging and developing age-associated conditions owing to its contributions to altered protein stability.

Mercury

The metal mercury (atomic number 80) has multiple uses as an alloy. It is used in making thermometers. It is also used as a filling in dentistry along with silver. Mercury is found in high levels in the atmosphere surrounding coal-burning plants, incinerators, and other types of industry. It is also found in significant levels in large fish such as tuna, swordfish, king mackerel, grouper, marlin, bluefish, shark, orange roughy, and tilefish. Mercury can be breathed in through polluted air, absorbed through the oral cavity from amalgam fillings, injected into veins from mercury-containing vaccines, and absorbed through the digestive system from contaminated food, drugs, and supplements. All forms of mercury can affect the nervous system. The mechanisms of mercury toxicity includes binding sulfhydryl, phosphoryl, carboxyl, amide and amine groups of proteins (including enzymes), rendering them inactive. Acute exposure to high levels of metallic mercury can result in nausea, vomiting, lung damage, diarrhea, increased blood pressure, skin rash, and eye irritation. Long-term effects can give rise to the brain and/or kidney damage, damage to a developing fetus, changes in vision, tremors, hearing, memory problems, and irritability.

Nickel

Nickel (atomic number 28) is extensively distributed in the environment, air, water, and soil. Nickel is used to make jewelry, coins, batteries, spark plugs, catalysts, stainless steel (including cooking and eating utensils), machinery parts, nickel alloys, industrial plumbing, and electroplating. Lung inhalation is the major route of exposure for nickel-induced toxicity. However, food is the major source of nickel exposure. The Agency for Toxic Substances and Disease Registry (ATSDR) estimates that average intake for adults is 100 to 300 micrograms per day. It may also be absorbed through the skin. The mechanisms of nickel toxicity include depletion of glutathione levels and bonding to the sulfhydryl groups of proteins. Contact with nickel may cause a variety of side effects on human health, such as contact dermatitis (nickel allergy), cardiovascular and kidney diseases, lung fibrosis, and lung and nasal cancer. The symptoms accompanied with its intoxication include low blood pressure, malaise, muscle tremor, tetany and paralysis, nausea, vomiting, hemorrhages, heart attack, oral and/or intestinal cancer, and kidney dysfunction.

Heavy Metals

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Heavy Metals

COMMENTS

Palladium

Palladium (atomic number 46) is a naturally occurring metal whose use more than doubled in the last part of the 20th century. It is extensively used in dental appliances, electrical appliances, mobile phones, laptop computers, and jewelry—and as chemical catalysts used to make pharmaceuticals. Humans are mainly exposed to palladium via ingestion of contaminated water and food or by inhalation. However, dermal exposure to palladium may also cause skin and eye irritation; nickel and palladium share the same periodic table group, so people with known nickel allergy may be more susceptible. Recent studies suggest the mechanism of palladium toxicity include mitochondrial membrane potential collapse and depletion of cellular glutathione levels. Exposure to palladium can cause acute toxicity or hypersensitivity with respiratory symptoms, urticaria, and, less frequently, contact dermatitis.

Platinum

The metal platinum (atomic number 78) has many applications. It is used in jewelry, surgical tools, dental appliances, laboratory utensils, electrical resistance wires, and electrical contact points. Platinum exposure is most likely to occur by breathing in contaminated air (exhaust from leaded gasoline), absorption through the skin by working with platinum-containing jewelry, or via ingestion of platinum-containing medicines used for chemotherapy. Platinum exposure may also occur through contaminated food—the highest levels occurring in eggs, meat, and grain products. Platinum as a metal is not very dangerous but platinum salts can cause several deleterious health effects, including hearing damage, allergic reactions to the skin and mucosa, damage to organs such as intestines, kidneys, and bone marrow, and even cancer. Additionally, platinum compounds are seen to cause DNA damage which can further affect genomic stability. Alterations in genomic stability have been implicated in aging. Thus, platinum toxicity may accelerate aging owing to its contribution to genomic instability which is a hallmark of aging.

Tellurium

Tellurium (atomic number 52) is one of the rarest elements on earth. Tellurium toxicity is rare and mostly occupationally exposed workers are affected. Tellurium is used in solar cells, rewritable CDs and DVDs, as a catalyst in oil refining, to vulcanize rubber, to tint glass or ceramics, and improves the machinability of alloys. The main exposure source of tellurium is contaminated water and plant life. Foods with the highest tellurium levels are meat, dairy products, and cereals. After tellurium exposure, the major symptoms include loss of appetite, dryness of the mouth, suppression of sweating, a metallic taste in the mouth, and most notable, a sharp garlic odor of the breath, sweat and urine. Severe tellurium inhalation poisoning results in irritation of the respiratory tract, respiratory depression, and circulatory collapse.

Thallium

Thallium (atomic number 81) is a soft, bluish-white metal naturally occurring in the earth's crust. It is most commonly used in the semiconductor industry and, in rare cases, glass manufacturing. Thallium exposure can come from food, water, and air. Produce grown in contaminated soil and contaminated groundwater are the most common routes of thallium exposure in humans. According to uptake studies, Brassicaceous plants have the highest levels. Thallium is present in cigarette smoke, and smokers have approximately twice as much thallium in their bodies as those who do not smoke. The initial symptoms of thallium poisoning may include fever, gastrointestinal problems, delirium, convulsions, and coma. Acute toxicity may subside to be replaced by a gradual development of mild gastrointestinal disturbances, polyneuritis, encephalopathy, tachycardia, skin eruptions, stomatitis, atrophic changes of the skin, nail changes (Mee's lines), and skin hyperesthesia (mainly in the soles of the feet and the tibia). Additionally, degenerative changes in the heart, liver, and kidney, subarachnoid hemorrhage, bone marrow depression, psychotic behavior with hallucinations, and dementia may also occur. Thallium can disrupt protein bonds and may even cause DNA damage. These aspects are considered to be the hallmarks of aging which result in reduced longevity. Thus, thallium intoxication may accelerate aging owing to its contribution to genomic and proteomic instability.

Thorium

Thorium (atomic number 90) is a naturally occurring radioactive element present in the air, water, soil, and rocks. It is found in trace amounts in most animals. Thorium is used to make welding rods, fire brick, camera and telescope lenses, gas lantern mantles, and in the ceramics industry (glazes). It is also incorporated into metals used in the aerospace industry and nuclear reactions. Until the 1950s, thorium dioxide was used as a radiology contrast agent. Thorium is currently being used as a novel alpha-therapy for the treatment of resistant tumors. Thorium is a known human carcinogen. It can enter the body through the respiratory, gastrointestinal, and dermatological systems. Occupational thorium exposure can occur to those individuals working near radioactive waste disposal sites, and/or uranium, thorium, tin, phosphate mining, and gas mantle production industries. Symptoms and side effects of thorium toxicity are most likely to manifest in the hematological, hepatic, and respiratory systems, as well as possible cancers. The most common symptoms of thorium toxicity are respiratory distress and pneumonia, pulmonary hypertension, and fibrosis. Individuals who breathe thorium dust may develop lung disease. Studies have also shown that individuals exposed to thorium may have an increased risk of bone cancer because thorium may be stored in bone.

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Heavy Metals

COMMENTS

Tin

Tin (atomic number 50) is a soft, white, silvery metal and naturally occurring element. Major uses of tin include cans and containers, electrical, construction, and transportation. Tin can also be combined with carbon to form organotin compounds used to make plastics (including plastic pipes), food packages, paints, wood preservatives, rodenticides and pesticides. Human exposure to tin may occur by inhalation, ingestion, or dermal absorption. Occupational exposure to tin may be significant in some industrial environments. Tin-lined cans used to package food are the most important contributor to dietary tin intake/intoxication. Tin salts are poorly absorbed and rapidly excreted in the feces which is why they have low toxicity. The small amounts of tin that stays in the body gets deposited in bone, liver, kidneys and lymph nodes. Effects of tin are growth retardation, changes in enzyme activities, anemia, interactions with the absorption and excretion of calcium, copper, iron and zinc, and morphological changes in liver and kidney. Therefore, if a large amount of canned food is eaten daily over a long period, disturbances of gastric acid secretion (along with GI symptoms including nausea, vomiting, and diarrhea) and a reduction in iron absorption or heme metabolism may occur.

Tungsten

Tungsten (atomic number 74) is a naturally occurring element that is typically found in the solid form in rocks and minerals. It is used in light bulb filaments, as part of X-ray tubes, as a catalyst to speed up chemical reactions, as a component of steel in high-speed tools, in turbine blades, in darts, and in golf club components. Tungsten has the highest melting point of all metals and maintains tensile strength even at very high temperatures. Replacing lead and depleted uranium, heavy metal tungsten alloys are increasingly used in military applications such as helicopter rotors, kinetic energy penetrators for defeating heavy armor, guided missiles, and fragmentation warheads. Tungsten intoxications are relatively rare. However, breathing contaminated air, drinking contaminated water, skin contact with compounds that contain tungsten, or eating food that contains tungsten are the most common ways tungsten toxicity occurs. The symptoms associated with tungsten toxicity may include breathing problems, nausea, seizures, rapid onset of clouded consciousness which may lead to coma and encephalopathy, renal conditions, and hypocalcemia. Limited evidence from animal studies suggest tungsten exposure is carcinogenic, but this may be contributed to or modified by the presence of other heavy metals like nickel and cobalt in tungsten alloys.

Uranium

Uranium (atomic number 92) is a naturally occurring radioactive element found on earth found in nearly all rocks and soils. It is used as fuel for nuclear power plants and the nuclear reactors that run naval ships and submarines. It can also be used in nuclear weapons. Depleted uranium is used in military applications, including as a shield to protect against ionizing radiation, as armor in military vehicles, in munitions to help them penetrate enemy armored vehicles, and as a counterbalance on helicopter rotors. Uranium can be ingested through the lungs, and gastrointestinal (GI) tract, and can be absorbed through the skin. Uranium can stick to plant roots so unwashed root vegetables are a primary source of uranium in the diet. However, Brazil nuts are also found to have high levels. The majority of uranium that is inhaled through the lungs or ingested through the GI tract is not absorbed and leaves the body through the feces. However, water-soluble sources of uranium being ingested may lead to kidney problems. As a result, the kidneys are the most impacted organ system by uranium exposure, both chronic and acute. The primary mechanism of uranium toxicity is direct damage to DNA from alpha particle interactions. Therefore, uranium may also cause chromosomal abnormalities. The main manifestation of uranium exposure is cellular depletion of antioxidants and the formation of reactive oxygen species (ROS), as well as increased 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.

Risk and Limitations

This test has been developed and its performance characteristics determined by Vibrant America LLC., a CLIA certified lab. These assays have not been cleared or approved by the U.S. Food and Drug Administration.

Heavy Metals Toxins panel does not demonstrate absolute positive and negative predictive values for any condition. Its clinical utility has not been fully established. Clinical history and current symptoms of the individual must be considered by the healthcare provider prior to any interventions. Test results should be used as one component of a physician's clinical assessment.

Heavy Metals Panel testing is performed at Vibrant America, a CLIA certified laboratory. Vibrant America has effective procedures in place to protect against technical and operational problems. However, such problems may still occur. Examples include failure to obtain the result for a specific toxin due to circumstances beyond Vibrant's control. Vibrant may re-test a sample to obtain these results but upon re-testing the results may still not be obtained. As with all medical laboratory testing, there is a small chance that the laboratory could report incorrect results. A tested individual may wish to pursue further testing to verify any results.

The information in this report is intended for educational purposes only. While every attempt has been made to provide current and accurate information, neither the author nor the publisher can be held accountable for any errors or omissions.

Vibrant Wellness makes no claims as to the diagnostic or therapeutic use of its tests or other informational materials. Vibrant Wellness reports and other information do not constitute medical advice and are not a substitute for professional medical advice. Please consult your healthcare practitioner for questions regarding test results, or before beginning any course of medication, supplementation, or dietary changes. Users should not disregard, or delay in obtaining, medical advice for any medical condition they may have, and should seek the assistance of their health care professionals for any such conditions.

SAMPLE

Environmental Toxins

PATIENT

NAME: TEST TEST
DATE OF BIRTH: 1991-11-11 GENDER: Female
TELEPHONE: 11234567890 AGE: 31

ACCESSION ID: 2306270585
SPECIMEN COLLECTED: 2023-06-27 10:00 (PST)
SPECIMEN RECEIVED: 2023-06-27 14:47 (PST)
FINAL REPORT DATE: 2023-06-27 15:15 (PST)
GENERATION DATE: 2023-06-27 15:25 (PST)

FASTING:

PROVIDER:

PRACTICE NAME: Vibrant IT3 Practice
PROVIDER NAME: Vibrant IT3333
PHLEBOTOMIST: No Matched Result

TELEPHONE: +15555555555
FAX #: 650-331-7393
ADDRESS: Apple Park Way, Millcreek, UT

Vibrant Wellness is pleased to present to you, 'Environmental Toxins Panel', to help you make healthy lifestyle, dietary and treatment choices in consultation with your healthcare provider. It is intended to be used as a tool to encourage a general state of health and well-being.

The Vibrant Environmental Toxins Panel is a test to measure levels of Environmental Toxins in your urine. The panel is sub-grouped into Pesticides, Pthalates, Parabens, Acrylic, Alkyl phenols and Volatile Organic Compounds.

Reference ranges are established based on NHANES study where applicable. Other reference ranges are established based on 1000 apparently healthy urine samples.

The report begins with the summary page which lists only the environmental toxins whose levels are >95th percentile (Red) and 75th-95th percentile (Yellow) of reference range, normalized to Urine creatinine levels. Additionally, the previous value is also indicated for your referral (if available). Following this section is the complete list of the environmental toxins and their absolute levels normalized to Creatinine in a quantile format along with the reference ranges. These levels are shown with three shades of color – Green, Yellow and Red. The result in green corresponds to 0 to 75th percentile, the result in yellow corresponds to 75th to 95th percentile and the result in red corresponds to greater than 95th percentile of reference range. All content provided in the report are purely for informational purposes only and should not be considered medical advice. Any changes based on the information should made in consultation with your healthcare provider.

The Vibrant Wellness platform provides tools for you to track and analyze your general wellness profile. Testing for the Environmental Toxins panel is performed by Vibrant America, a CLIA certified lab CLIA#:05D2078809. Vibrant Wellness provides and makes available this report and any related services pursuant to the Terms of Use Agreement (the "Terms") on its website at www.vibrant-wellness.com. By accessing, browsing, or otherwise using the report or website or any services, you acknowledge that you have read, understood, and agree to be bound by these terms. If you do not agree to accept these terms, you shall not access, browse, or use the report or website. The statements in this report have not been evaluated by the Food and Drug Administration and are only meant to be lifestyle choices for potential risk mitigation. Please consult your physician/dietitian for medication, treatment, or lifestyle management. This product is not intended to diagnose, treat, or cure any disease.

Pediatric ranges have not been established for this test. It is important that you discuss any modifications to your diet, exercise, and nutritional supplementation with your physician before making any changes.

Environmental Toxins Summary

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

High (>95th percentile)

| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
|---|----------------|-----------------|----------------|-----------------|--------------|
| 2-Hydroxyethyl Mercapturic Acid (HEMA)* | 1064.2 | TNP | 0 1.7 4.75 | | ≤4.75 ug/g |
| 2-Methylhippuric Acid (2MHA)* | 6831.22 | <0.01 | 0 77.9 248 | | ≤248 ug/g |
| 2,2-bis(4-Chlorophenyl) acetic acid (DDA) | 7064.74 | | 0 7.9 19 | | ≤19 ug/g |
| 2,4-Dichlorophenoxyacetic Acid (2,4-D)* | 2465.39 | 1 | 0 0.5 1.55 | | ≤1.55 ug/g |
| 3-Methylhippuric Acid (3MHA) | 2936.05 | >10000 | 0 64.8 612 | | ≤612.83 ug/g |
| 3-Phenoxybenzoic Acid (3PBA)* | 3120.15 | >10000 | 0 1.01 5.44 | | ≤5.44 ug/g |
| 4-Methylhippuric Acid (4MHA) | 5652.4 | TNP | 0 65.5 752 | | ≤752.72 ug/g |
| 4-Nonylphenol | 6120.36 | <0.01 | 0 0.42 2.06 | | ≤2.06 ug/g |
| Atrazine * | 4737.24 | 5 | 0 0.02 0.05 | | ≤0.05 ug/g |
| Atrazine mercapturate* | 5305.49 | 6 | 0 0.02 0.05 | | ≤0.05 ug/g |
| Bisphenol A (BPA)* | 8513.69 | 7 | 0 2.12 5.09 | | ≤5.09 ug/g |
| Butylparaben* | 2232.85 | 8 | 0 0.25 4.39 | | ≤4.39 ug/g |
| Diethyl phosphate (DEP)* | 5623.27 | | 0 3.2 15.7 | | ≤15.7 ug/g |
| Diethyldithiophosphate (DEDTP)* | 5236.6 | | 0 0.17 0.3 | | ≤0.3 ug/g |
| Diethylthiophosphate (DETP)* | 1189.7 | | 0 1.24 3.92 | | ≤3.92 ug/g |

* Indicates NHANES population data reference ranges.

Results are creatinine corrected to account for urine dilution variations. Reference intervals are based upon NHANES(cdc.gov/nhanes) data if available, and are representative of a large population cohort under non-provoked conditions.

Environmental Toxins Summary

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

High (>95th percentile)

| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
|--|----------------|-----------------|----------------|-----------------|------------|
| Dimethyl phosphate (DMP)* | 1785.73 | | 0 9.1 33.6 | | ≤33.6 ug/g |
| Dimethyldithiophosphate (DMDTP)* | 2651.53 | | 0 0.67 6.12 | | ≤6.12 ug/g |
| Dimethylthiophosphate (DMTP)* | 7775.27 | | 0 5.91 33.7 | | ≤33.7 ug/g |
| Diphenyl Phosphate (DPP) | 5704.99 | | 0 1.1 3.7 | | ≤3.7 ug/g |
| Ethylparaben * | 4321.25 | | 0 5.41 99.3 | | ≤99.3 ug/g |
| Glyphosate | 580.15 | | 0 1.65 7.6 | | ≤7.6 ug/g |
| Methylparaben* | 8827.17 | | 0 180 653 | | ≤653 ug/g |
| Mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP)* | 8576.21 | | 0 14.1 37.7 | | ≤37.7 ug/g |
| Mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP)* | 6140.3 | | 0 8.99 23.4 | | ≤23.4 ug/g |
| Mono-2-ethylhexyl phthalate (MEHP)* | 1978.65 | | 0 2.73 8.47 | | ≤8.47 ug/g |
| Mono-ethyl phthalate (MEtP)* | 595.58 | | 0 94.2 541 | | ≤541 ug/g |
| N-Acetyl (2-Cyanoethyl) Cysteine (NACE)* | 1445.39 | | 0 5.28 256 | | ≤256 ug/g |
| N-Acetyl (2-Hydroxypropyl) Cysteine (NAHP)* | 5090.41 | | 0 101 403 | | ≤403 ug/g |
| N-Acetyl (3,4-Dihydroxybutyl) Cysteine* | 9073.19 | | 0 374 583 | | ≤583 ug/g |
| N-Acetyl (Propyl) Cysteine (NAPR)* | 3102.95 | | 0 11.3 46.1 | | ≤46.1 ug/g |

* Indicates NHANES population data reference ranges.

Results are creatinine corrected to account for urine dilution variations. Reference intervals are based upon NHANES(cdc.gov/nhanes) data if available, and are representative of a large population cohort under non-provoked conditions.

Environmental Toxins Summary

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

High (>95th percentile)

| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
|---|----------------|-----------------|----------------|-----------------|------------|
| N-acetyl phenyl cysteine (NAP)* | 1802.48 | | 0 1.29 3.03 | | ≤3.03 ug/g |
| N-acetyl-S-(2-carbamoylethyl)-cysteine* | 6958.71 | | 0 82 199 | | ≤199 ug/g |
| Perchlorate (PERC)* | 1550.31 | | 0 4.89 10.7 | | ≤10.7 ug/g |
| Phenyl glyoxylic Acid (PGO)* | 4737.71 | | 0 285 518 | | ≤518 ug/g |
| Propylparaben* | 6174.69 | | 0 36.7 222 | | ≤222 ug/g |
| Tiglylglycine (TG) | 4460.57 | | 0 0.09 3.24 | | ≤3.24 ug/g |
| Triclosan (TCS)* | 1841.07 | | 0 29.9 358 | | ≤358 ug/g |

* Indicates NHANES population data reference ranges.

Urine Creatinine

| TEST NAME | CURRENT RESULT | PREVIOUS RESULT | CURRENT RESULT | PREVIOUS RESULT | REFERENCE |
|------------------|----------------|-----------------|----------------|-----------------|-----------------|
| Urine Creatinine | 9.71 | | 0 0.24 2.16 | | 0.25-2.16 mg/mL |

COMMENTS

Urine Creatinine

Urine tests that measure ratio of analytes by creatine concentration will not be altered by urine volume, hydration status, or time of testing. When using creatinine concentration to measure urine analytes, the only interference with the test is if the person's creatinine levels are very high (which may be seen in kidney disease, diabetes, or competitive body builder athletes), or when creatinine levels are very low (which may be seen in people with muscle wasting or sarcopenia who have lost their lean muscle mass stores). High urine creatinine may cause falsely lower urine analyte results. Low urine creatinine may cause falsely higher urine analyte results. This does not invalidate the findings; rather, critical analysis should be used to correlate results with clinical history and symptomatology for intervention decision-making.

Results are creatinine corrected to account for urine dilution variations. Reference intervals are based upon NHANES(cdc.gov/nhanes) data if available, and are representative of a large population cohort under non-provoked conditions.

Environmental Toxins

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Environmental phenols * Indicates NHANES population data reference ranges.

| TEST NAME | PERCENTILE | | REFERENCE | TEST NAME | PERCENTILE | | REFERENCE |
|------------------|------------|---------|------------|--------------------|------------|---------|------------|
| | 75th | 95th | | | 75th | 95th | |
| 4-Nonylphenol | | 6120.36 | ≤2.06 ug/g | Bisphenol A (BPA)* | | 8513.69 | ≤5.09 ug/g |
| Triclosan (TCS)* | | 1841.07 | ≤358 ug/g | | | | |

COMMENTS

4-Nonylphenol

4-Nonylphenols are used in manufacturing antioxidants, lubricating oil additives, laundry and dish detergents, emulsifiers, and solubilizers. These compounds are also precursors used to produce paints, pesticides, cosmetics, and plastics. Nonylphenol persists in aquatic environments and is moderately bioaccumulative. It is not readily biodegradable, and it can take months or longer to degrade in surface waters, soils, and sediments. It has a potential role as an endocrine disruptor and xenoestrogen due to its ability to act with estrogen-like activity. Nonylphenol exposure has also been associated with breast cancer.

Bisphenol A (BPA)

BPA is one of the highest volume of chemicals produced worldwide. It is a starting material for the synthesis of plastics. BPA-based plastic is clear and tough, and is made into plastic bottles including water bottles, sports equipment, CDs, and DVDs. Epoxy resins containing BPA are used to line water pipes, as coatings on the inside of many food and beverage cans and in making thermal paper such as that used in sales receipts. Exposure to Bisphenol A cause fertility problems, male impotence, heart disease and other conditions. BPA is a xenoestrogen, exhibiting estrogen-mimicking, hormone-like properties that raise concern about its suitability in some consumer products and food containers.

Triclosan (TCS)

Triclosan (TCS) is an antibacterial and antifungal agent present in some consumer products, including toothpaste, soaps, detergents, toys, and surgical cleaning treatments. Humans are exposed to triclosan through skin absorption when washing hands or in the shower, brushing teeth, using mouthwash, or doing dishes, and through ingestion when swallowed. Additional exposure is possible through ingesting plants grown in soil treated with sewage sludge or eating fish exposed to it. Triclosan has been associated with a higher risk of food allergies. Triclosan has also been found to be a weak endocrine disruptor. Prenatal triclosan exposure was associated with increased cord testosterone levels in the infants.

Herbicides * Indicates NHANES population data reference ranges.

| TEST NAME | PERCENTILE | | REFERENCE | TEST NAME | PERCENTILE | | REFERENCE |
|---|------------|---------|------------|------------|------------|---------|------------|
| | 75th | 95th | | | 75th | 95th | |
| 2,4-Dichlorophenoxyacetic Acid (2,4-D)* | | 2465.39 | ≤1.55 ug/g | Atrazine * | | 4737.24 | ≤0.05 ug/g |
| Atrazine mercapturate* | | 5305.49 | ≤0.05 ug/g | Glyphosate | | 580.15 | ≤7.6 ug/g |

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Herbicides

COMMENTS

2,4-Dichlorophenoxyacetic Acid (2,4-D)

2,4-Dichlorophenoxyacetic Acid (2,4-D) is a systemic herbicide which selectively kills most broad-leaved weeds. People can be exposed to herbicides by breathing them in or by skin contact from their residential use or living near application sites, or by eating contaminated food and drinking contaminated water. Oral ingestion is associated with neuritis, weakness, nausea, abdominal pain, headache, dizziness, peripheral neuropathy, stupor, seizures, brain damage, and impaired reflexes. 2,4-D is a known endocrine disruptor and can block hormone distribution and cause glandular breakdown. It is linked to immune system damage, birth defects, and reproductive issues, possibly due to its frequent contamination with dioxins. Men who work with 2,4-Dichlorophenoxyacetic acid (2,4-D) are at risk of having abnormally shaped sperm, which can lead to fertility issues. It has also been classified as a possible carcinogen.

Atrazine

Atrazine is a widely used herbicide that prevents pre and post-emergence broadleaf weeds in crops like maize (corn) and sugarcane, as well as on turf like golf courses and residential lawns. It used to be the most commonly detected pesticide contaminating drinking water, and studies suggest it is an endocrine disruptor, an agent that can alter the natural hormonal system. The implications for children's health are related to effects during pregnancy and during sexual development.

Atrazine mercapturate

Atrazine mercapturate is a metabolite of atrazine, which is one of the most widely used herbicides to prevent pre and postemergence broadleaf weeds in crops such as maize (corn) and sugarcane and on turf, such as golf courses and residential lawns. It used to be the most commonly detected pesticide contaminating drinking water and studies suggest it is an endocrine disruptor, an agent that can alter the natural hormonal system. The implications for children's health are related to effects during pregnancy and during sexual development.

Glyphosate

Glyphosate is the most used herbicide worldwide, and its residues can be found in food, drinking-water, crops, animal feed, groundwater, rain, and air. Residues have also been found in the urine of 60–80% of the general population in the United States. Potential health harms linked to glyphosate-based herbicides include microbiome disruption, increased risk of celiac disease, endocrine disruption, reproduction and fertility effects, cardiovascular disorder, central nervous system dysfunction, learning impairment, anxiety, depression, and renal disease. In 2015 the IARC, the specialized cancer agency of the WHO, classified glyphosate as a Group 2A carcinogen. Non-Hodgkin lymphoma has been significantly associated with occupational exposure to glyphosate in the literature. Multiple myeloma has also been associated with glyphosate exposure. Mechanisms shown in pre-clinical research for carcinogenicity and other health harms include increased production of reactive oxygen species (ROS), DNA adduct formation, mutagenic effects, and chromosomal damage.

Mitochondrial Marker

| TEST NAME | PERCENTILE | | REFERENCE | TEST NAME | PERCENTILE | | REFERENCE |
|--------------------|------------|---------|------------|-----------|------------|------|-----------|
| | 75th | 95th | | | 75th | 95th | |
| Tiglylglycine (TG) | | 4460.57 | ≤3.24 ug/g | | | | |

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Mitochondrial Marker

COMMENTS

Tiglylglycine (TG)

Tiglylglycine (TG) is a marker used to assess for mitochondrial dysfunction and/or genetic disorders. When tiglylglycine levels are elevated, it can indicate disorders of the respiratory chain, mitochondrial dysfunction, or genetic causes. Toxic chemical exposure may be one of the most common causes of mitochondrial dysfunction. Other triggers include infections, inflammation, and nutritional deficiencies. Mitochondrial dysfunction has been linked with aging, diabetes, autism, chronic fatigue syndrome, Parkinson's, and Alzheimer's syndromes.

Other Markers * Indicates NHANES population data reference ranges.

| TEST NAME | PERCENTILE | | REFERENCE | TEST NAME | PERCENTILE | | REFERENCE |
|--------------------------|------------|---------|------------|--|------------|---------|-----------|
| | 75th | 95th | | | 75th | 95th | |
| Diphenyl Phosphate (DPP) | | 5704.99 | ≤3.7 ug/g | N-acetyl-S-(2-carbamoyl-ethyl)-cysteine* | | 6958.71 | ≤199 ug/g |
| Perchlorate (PERC)* | | 1550.31 | ≤10.7 ug/g | | | | |

COMMENTS

Diphenyl Phosphate (DPP)

Diphenyl phosphate (DPP) is an aryl phosphate ester (APE) used as an industrial catalyst and chemical additive and is the primary metabolite of flame retardant APEs. DPP is used in the manufacture of phosphoric acid diesters such as triphenyl phosphate, trixylenyl phosphate, isodecyl diphenyl phosphate, cresyl diphenyl phosphate and isopropylphenyl diphenyl phosphate. It is widely used as a protective agent for hydroxyl group in organic synthesis. It finds application as an additive for paints and coatings. DPP impacts cardiac development. DPP has the potential to impair mitochondrial function as well as induce renal toxicity, hepatotoxicity, and hemotoxicity.

N-acetyl-S-(2-carbamoyl-ethyl)-cysteine

N-acetyl-S-(2-carbamoyl-ethyl)-cysteine (NAE) is a metabolite of acrylamide. Acrylamide can polymerize to form polyacrylamide. These chemicals are used in many industrial processes such as plastics, food packaging, cosmetics, dyes, and the treatment of drinking water. High-temperature processed foods (e.g., potato chips and French fries) and cigarette smoking are the two major sources of exposure. This is because asparagine, an important amino acid for central nervous system function, can produce acrylamide when cooked at a high temperature in the presence of sugars. Foods rich in asparagine include asparagus, potatoes, legumes, nuts, seeds, beef, eggs, and fish, and are potential sources of exposure to acrylamide. Frequent intake of acrylamide has been linked with an increased risk of neurological damage and even cancer.

Perchlorate (PERC)

Perchlorate (PERC) is a naturally occurring and man-made anion and is a powerful oxidizing agent. It is mainly used for propellants in rocket fuel as well as control static electricity in food packaging. Perchlorate may be accumulated into vegetables through water cycle and other parts in the environment. Perchlorate contamination in food and water has been found to be harmful for human health. Perchlorate is classified as a possible carcinogen. It may disrupt the thyroid's ability to produce hormones. It has also been linked with lung toxicity and aplastic anemia.

Parabens * Indicates NHANES population data reference ranges.

| TEST NAME | PERCENTILE | | REFERENCE | TEST NAME | PERCENTILE | | REFERENCE |
|----------------|------------|---------|------------|----------------|------------|---------|------------|
| | 75th | 95th | | | 75th | 95th | |
| Butylparaben* | | 2232.85 | ≤4.39 ug/g | Ethylparaben * | | 4321.25 | ≤99.3 ug/g |
| Methylparaben* | | 8827.17 | ≤653 ug/g | Propylparaben* | | 6174.69 | ≤222 ug/g |

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Parabens

COMMENTS

Butylparaben

Butylparaben belongs to the paraben family and is one of the most common antimicrobial preservatives in cosmetics such as such as makeup, moisturizers, hair-care products, and shaving creams. It is also used in medication suspensions, and as a flavoring additive in food. When exposed to high levels of butylparaben via inhalation, irritation to the respiratory tract results; symptoms include coughing and shortness of breath. Ingestion of large doses of butylparaben may cause irritation to the gastrointestinal (GI) tract. Butylparaben is an endocrine disruptor.

Ethylparaben

Ethylparaben is produced naturally and found in several fruits and insects, where it acts as an antimicrobial agent. It is also can be used as feed preservatives and antiseptic for bacteria. Ethylparaben is mainly used as antiseptics in cosmetics, food and medicine. Although parabens are generally considered safe when used in low percentages, a study claimed to have found a link between parabens and breast cancer. Ethylparaben is readily absorbed from the gastrointestinal tract or through the skin. It is hydrolyzed to p-hydroxybenzoic acid and rapidly excreted in urine without accumulating in the body. Populations exposed to large amounts of Ethylparaben may have a high burden of estrogenicity-related disease and endocrine disruption.

Methylparaben

Methylparaben belongs to the paraben family and is an anti-fungal agent often used in a variety of cosmetics and personal-care products. It is also used as a food preservative. Methylparaben is generally recognized as safe for food and cosmetic antibacterial preservation. Methylparaben is readily absorbed from the gastrointestinal tract or through the skin. Studies indicate that methylparaben applied on the skin may react with UVB (Ultraviolet type B), leading to increased skin aging and DNA damage. Methylparaben was responsible for disrupting estrogenic and androgenic receptors too.

Propylparaben

Propylparaben belongs to the paraben family and is often used in water-based cosmetics, such as creams, lotions, shampoos, and bath products. It is also used as a food additive and has also been shown to have anti-fungal and anti-microbial properties. Propylparaben is generally recognized as safe for food and cosmetic antibacterial preservation. Although parabens are generally considered safe when used in low percentages, a study claimed to have found a link between parabens and breast cancer. Parabens are potential endocrine disruptors due to their ability to mimic estrogen.

Pesticides * Indicates NHANES population data reference ranges.

| TEST NAME | PERCENTILE | | REFERENCE | TEST NAME | PERCENTILE | | REFERENCE |
|---|------------|---------|------------|---------------------------------|------------|---------|------------|
| | 75th | 95th | | | 75th | 95th | |
| 2,2-bis(4-Chlorophenyl) acetic acid (DDA) | | 7064.74 | ≤19 ug/g | 3-Phenoxybenzoic Acid (3PBA)* | | 3120.15 | ≤5.44 ug/g |
| Diethyl phosphate (DEP)* | | 5623.27 | ≤15.7 ug/g | Diethyldithiophosphate (DEDTP)* | | 5236.6 | ≤0.3 ug/g |
| Diethylthiophosphate (DETP)* | | 1189.7 | ≤3.92 ug/g | Dimethyl phosphate (DMP)* | | 1785.73 | ≤33.6 ug/g |
| Dimethyldithiophosphate (DMDTP)* | | 2651.53 | ≤6.12 ug/g | Dimethylthiophosphate (DMTP)* | | 7775.27 | ≤33.7 ug/g |

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Pesticides

COMMENTS

2,2-bis(4-Chlorophenyl) acetic acid (DDA)

DDT metabolism in humans yields 2,2-bis (4-chlorophenyl) acetic acid (DDA) as the principal urinary metabolite and potential exposure biomarker. DDT is a persistent organic pollutant that is readily adsorbed to soils and sediments, which can act both as sinks and as long-term sources of exposure. DDT was a commonly used pesticide for insect control. DDT was used to control malaria and typhus. DDT is an endocrine disruptor and indicates possible disruption in semen quality, menstruation, gestational length, and duration of lactation. Chronic exposure to DDT will build up in areas of the body with high lipid content and can affect reproductive capabilities and the embryo or fetus. It is considered likely to be a human carcinogen, especially for breast cancer. DDE is a metabolite of DDT and is excreted as DDA in the urine.

3-Phenoxybenzoic Acid (3PBA)

3-phenoxybenzoic acid (3PBA) is the result of exposure to pyrethroid insecticides (pyrethrins). Pyrethrins are the collective name for a group of pesticidal compounds derived from pyrethrum flowers in the genus Chrysanthemum. Pyrethroids may affect neurological development, disrupt hormones, induce cancer, and suppress the immune system. Inhaling high levels of pyrethrins or pyrethroids may bring about asthmatic breathing, sneezing, nasal stuffiness, headache, nausea, incoordination, tremors, convulsions, facial flushing and swelling, and burning and itching sensation. Pyrethroids are axonic poisons that work by keeping the sodium channels open in the neuronal membranes.

Diethyl phosphate (DEP)

Diethyl phosphate, (DEP) indicates exposure to an organophosphate insecticide. Organophosphates function by inhibiting the action of cholinesterase enzymes in nerve cells. They can be absorbed through the lungs or skins or by eating them on contaminated food. Even at low levels, organophosphates may be hazardous to the nerves system, especially for fetuses and young children. Repeated or prolonged exposure may induce impaired memory and concentration, disorientation, severe depressions, irritability, confusion, headache, speech difficulties, delayed reaction times, nightmares, sleepwalking, drowsiness, or insomnia. An influenza-like condition with headache, nausea, weakness, loss of appetite, and malaise.

Diethyldithiophosphate (DEDTP)

Diethyldithiophosphate (DEDTP) is a metabolite of organophosphates, which are one of the most common causes of poisoning worldwide and are frequently intentionally used as pesticides. They can enter the body through the lungs or skin, or by eating contaminated food. Even at low levels, organophosphates may be hazardous to the nervous system, especially for foetuses and young children. Repeated or prolonged exposure may induce impaired memory and concentration, disorientation, severe depression, irritability, confusion, headache, speech difficulties, delayed reaction times, nightmares, sleepwalking, drowsiness, or insomnia. Organophosphates function by inhibiting the action of cholinesterase enzymes in nerve cells. An influenza-like condition with headache, nausea, weakness, loss of appetite, and malaise.

Diethylthiophosphate (DETP)

Diethylthiophosphate (DETP) is a metabolite of organophosphates, which are one of the most common causes of poisoning worldwide and are frequently intentionally used as pesticides. They can enter the body through the lungs or skin, or by eating contaminated food. Even at low levels, organophosphates may be hazardous to the nervous system, especially for foetuses and young children. Repeated or prolonged exposure may induce impaired memory and concentration, disorientation, severe depression, irritability, confusion, headache, speech difficulties, delayed reaction times, nightmares, sleepwalking, drowsiness, or insomnia. Organophosphates function by inhibiting the action of cholinesterase enzymes in nerve cells. An influenza-like condition with headache, nausea, weakness, loss of appetite, and malaise.

Dimethyl phosphate (DMP)

Dimethyl phosphate (DMP) indicates exposure to an organophosphate insecticide. Organophosphates function by inhibiting the action of cholinesterase enzymes in nerve cells. They can enter the body through the lungs or skin, or by eating contaminated food. Even at low levels, organophosphates may be hazardous to the nervous system, especially for foetuses and young children. Repeated or prolonged exposure may induce impaired memory and concentration, disorientation, severe depression, irritability, confusion, headache, speech difficulties, delayed reaction times, nightmares, sleepwalking, drowsiness, or insomnia, an influenza-like condition with headache, nausea, weakness, loss of appetite, and malaise.

Environmental Toxins

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Pesticides

COMMENTS

Dimethyldithiophosphate (DMDTP)

Dimethyldithiophosphate (DMDTP) is a metabolite of organophosphates, which are one of the most common causes of poisoning worldwide and are frequently intentionally used as pesticides. They can enter the body through the lungs or skin, or by eating contaminated food. Even at low levels, organophosphates may be hazardous to the nervous system, especially for foetuses and young children. Repeated or prolonged exposure may induce impaired memory and concentration, disorientation, severe depression, irritability, confusion, headache, speech difficulties, delayed reaction times, nightmares, sleepwalking, drowsiness, or insomnia. Organophosphates function by inhibiting the action of cholinesterase enzymes in nerve cells. An influenza-like condition with headache, nausea, weakness, loss of appetite, and malaise.

Dimethylthiophosphate (DMTP)

Dimethylthiophosphate (DMTP) is a metabolite of organophosphates, which are one of the most common causes of poisoning worldwide and are frequently intentionally used as pesticides. They can enter the body through the lungs or skin, or by eating contaminated food. Even at low levels, organophosphates may be hazardous to the nervous system, especially for foetuses and young children. Repeated or prolonged exposure may induce impaired memory and concentration, disorientation, severe depression, irritability, confusion, headache, speech difficulties, delayed reaction times, nightmares, sleepwalking, drowsiness, or insomnia. Organophosphates function by inhibiting the action of cholinesterase enzymes in nerve cells. An influenza-like condition with headache, nausea, weakness, loss of appetite, and malaise. Organophosphates and their metabolite, DMTP, generate oxidative stress, which in turn induces genomic instability through DNA damage. Alterations in genomic stability have been implicated in aging. Thus, DMTP may accelerate ageing owing to its contribution to genomic instability, which is a hallmark of aging.

Phthalates * Indicates NHANES population data reference ranges.

| TEST NAME | PERCENTILE | | REFERENCE | TEST NAME | PERCENTILE | | REFERENCE |
|--|------------|---------|------------|--|------------|--------|------------|
| | 75th | 95th | | | 75th | 95th | |
| Mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP)* | | 8576.21 | ≤37.7 ug/g | Mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP)* | | 6140.3 | ≤23.4 ug/g |
| Mono-2-ethylhexyl phthalate (MEHP)* | | 1978.65 | ≤8.47 ug/g | Mono-ethyl phthalate (MEtP)* | | 595.58 | ≤541 ug/g |

COMMENTS

Mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP)

Mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP) is a metabolite of mono(2-ethylhexyl) phthalate (MEHP), which belongs to the most common environmental toxin phthalates. Phthalates, often known as plasticizers, are a group of chemicals used to make plastics more flexible and harder to break. They are widely used in cosmetics, adhesives, detergents, lubricating oils, automotive plastics, and plastic clothes. People are exposed to phthalates by eating or drinking contaminated foods but also by breathing in air that contains phthalate vapours or dust. Inhaling phthalates can irritate the nose and throat, causing coughing and wheezing, headaches, dizziness, and nausea. MEHHP measured from the blood of pregnant women has been significantly associated with the decreased penis width, shorter anogenital distance, and the incomplete descent of testes of their newborn sons. Phthalates have been classified as endocrine disruptors which may cause reproductive damage, depressed leukocyte function, and even cancer. Phthalate exposure has also been associated with diabetes and insulin resistance, breast cancer, obesity, metabolic disorders, and immune disorders. Phthalate exposure and adverse child neurodevelopment, including autistic behaviours and lower cognitive and motor development, have also been reported.

Mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP)

Mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP) is a metabolite of mono(2-ethylhexyl) phthalate (MEHP), which belongs to the most common environmental toxin phthalates. Phthalates, often known as plasticizers, are a group of chemicals used to make plastics more flexible and harder to break. They are widely used in cosmetics, adhesives, detergents, lubricating oils, automotive plastics, and plastic clothes. People are exposed to phthalates by eating or drinking contaminated foods but also by breathing in air that contains phthalate vapours or dust. Inhaling phthalates can irritate the nose and throat, causing coughing and wheezing, headaches, dizziness, and nausea. MEHHP measured from the blood of pregnant women has been significantly associated with the decreased penis width, shorter anogenital distance, and the incomplete descent of testes of their newborn sons. Phthalates have been classified as endocrine disruptors which may cause reproductive damage, depressed leukocyte function, and even cancer. Phthalate exposure has also been associated with diabetes and insulin resistance, breast cancer, obesity, metabolic disorders, and immune disorders. Phthalate exposure and adverse child neurodevelopment, including autistic behaviours and lower cognitive and motor development, have also been reported.

Environmental Toxins

| LAST NAME | FIRST NAME | GENDER | DATE OF BIRTH | ACCESSION ID | DATE OF SERVICE |
|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Phthalates

COMMENTS

Mono-2-ethylhexyl phthalate (MEHP)

The active metabolite of di-(2-ethylhexyl) phthalate (DEHP), the most common environmental toxin phthalates, is di-(2-ethylhexyl) phthalate (MEHP). Phthalates, often known as plasticizers, are a group of chemicals used to make plastics more flexible and harder to break. They are widely used in cosmetics, adhesives, detergents, lubricating oils, automotive plastics, and plastic clothes. People are exposed to phthalates by eating or drinking contaminated foods but also by breathing in air that contains phthalate vapours or dust. Inhaling phthalates can irritate the nose and throat, causing coughing and wheezing, headaches, dizziness, and nausea. DEHP measured from the blood of pregnant women has been significantly associated with the decreased penis width, shorter anogenital distance, and the incomplete descent of testes of their newborn sons. Phthalates have been classified as endocrine disruptors which may cause reproductive damage, depressed leukocyte function, and even cancer. Phthalate exposure has also been associated with diabetes and insulin resistance, breast cancer, obesity, metabolic disorders, and immune disorders. Phthalate exposure and adverse child neurodevelopment, including autistic behaviours and lower cognitive and motor development, have also been reported.

Mono-ethyl phthalate (MEtP)

Mono-ethyl Phthalate (MEtP) is a metabolite of diethyl phthalate, which belongs to the most common environmental toxin group, phthalates. Phthalates, often known as plasticizers, are a group of chemicals used to make plastics more flexible and harder to break. They are widely used in cosmetics, adhesives, detergents, lubricating oils, automotive plastics, and plastic clothes. People are exposed to phthalates by eating or drinking contaminated foods but also by breathing in air that contains phthalate vapours or dust. Inhaling phthalates can irritate the nose and throat, causing coughing and wheezing, headaches, dizziness, and nausea. Phthalates have been classified as endocrine disruptors which may cause reproductive damage, depressed leukocyte function, and even cancer. Phthalate exposure has also been associated with diabetes and insulin resistance, breast cancer, obesity, metabolic disorders, and immune disorders. Phthalate exposure and adverse child neurodevelopment, including autistic behaviours and lower cognitive and motor development, have also been reported.

Volatile organic compounds * Indicates NHANES population data reference ranges.

| TEST NAME | PERCENTILE | | REFERENCE | TEST NAME | PERCENTILE | | REFERENCE |
|---|------------|---------|--------------|--|------------|---------|---------------|
| | 75th | 95th | | | 75th | 95th | |
| 2-Hydroxyethyl Mercapturic Acid (HEMA)* | | 1064.2 | ≤4.75 ug/g | 2-Hydroxyisobutyric Acid (2HIB) | 384.44 | | ≤1215.72 ug/g |
| 2-Methylhippuric Acid (2MHA)* | | 6831.22 | ≤248 ug/g | 3-Methylhippuric Acid (3MHA) | | 2936.05 | ≤612.83 ug/g |
| 4-Methylhippuric Acid (4MHA) | | 5652.4 | ≤752.72 ug/g | N-Acetyl (2-Cyanoethyl) Cysteine (NACE)* | | 1445.39 | ≤256 ug/g |
| N-Acetyl (2-Hydroxypropyl) Cysteine (NAHP)* | | 5090.41 | ≤403 ug/g | N-Acetyl (3,4-Dihydroxybutyl) Cysteine* | | 9073.19 | ≤583 ug/g |
| N-Acetyl (Propyl) Cysteine (NAPR)* | | 3102.95 | ≤46.1 ug/g | N-acetyl phenyl cysteine (NAP)* | | 1802.48 | ≤3.03 ug/g |
| Phenyl glyoxylic Acid (PGO)* | | 4737.71 | ≤518 ug/g | | | | |

COMMENTS

2-Hydroxyethyl Mercapturic Acid (HEMA)

2-Hydroxyethyl Mercapturic Acid (HEMA) is a metabolite of ethylene oxide and/or vinyl chloride. Ethylene oxide is widely used in the production of agrochemicals detergents, pharmaceuticals, and cosmetics. Ethylene oxide is also the raw material to make sterilant in rubber, plastics, and electronics. Chronic exposure to ethylene oxide has been determined to be mutagenic to humans. Ethylene oxide is toxic by inhalation and may cause acute poisoning, accompanied by a variety of symptoms including headache, nausea, and vomiting. Studies of humans exposed to ethylene oxide show an increased incidence of breast cancer and leukemia. Exposure to vinyl chloride has been associated with increased incidence of autism. Exposure may come from the use of plastic containers for cooking, reheating, eating or drinking (especially warm or hot) food or beverages. High concentrations of vinyl chloride may cause central nervous system depression, nausea, headache, dizziness, liver damage and liver cancer, degenerative bone changes, thrombocytopenia, enlargement of the spleen, autism and even death.

Environmental Toxins

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|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Volatile organic compounds

COMMENTS

2-Methylhippuric Acid (2MHA)

2-Methylhippuric acid (2MHA) is a metabolite of the isomers of xylene. In addition to being a solvent, xylenes are also used in perfumes, detergents, pesticides, and fuel. The main effect of inhaling xylene vapour is depression of the central nervous system (CNS), with symptoms such as headaches, dizziness, nausea, and vomiting. Long-term exposure may lead to irritability, depression, insomnia, agitation, extreme tiredness, tremors, hearing loss, impaired concentration, and short-term memory loss. A condition called chronic solvent-induced encephalopathy, commonly known as "organic solvent syndrome," has been associated with xylene exposure. Exposure to xylene is known to result in immunologic, respiratory, carcinogenic, reproductive, neurologic, and cardiovascular effects.

3-Methylhippuric Acid (3MHA)

3-Methylhippuric acid (3MHA) is a metabolite of the isomers of xylene. In addition to being a solvent, xylenes are also used in perfumes, detergents, pesticides, and fuel. The main effect of inhaling xylene vapour is depression of the central nervous system (CNS), with symptoms such as headache, dizziness, nausea, and vomiting. Long-term exposure may lead to irritability, depression, insomnia, agitation, extreme tiredness, tremors, hearing loss, impaired concentration, and short-term memory loss. A condition called chronic solvent-induced encephalopathy, commonly known as "organic solvent syndrome," has been associated with xylene exposure. Exposure to xylene is known to result in immunologic, respiratory, carcinogenic, reproductive, neurologic, and cardiovascular effects.

4-Methylhippuric Acid (4MHA)

4-Methylhippuric acid (4MHA) is a metabolite of the isomers of xylene. In addition to being a solvent, xylenes are also used in perfumes, detergents, pesticides, and fuel. The main effect of inhaling xylene vapour is depression of the central nervous system (CNS), with symptoms such as headache, dizziness, nausea, and vomiting. Long-term exposure may lead to irritability, depression, insomnia, agitation, extreme tiredness, tremors, hearing loss, impaired concentration, and short-term memory loss. A condition called chronic solvent-induced encephalopathy, commonly known as "organic solvent syndrome," has been associated with xylene exposure. Exposure to xylene is known to result in immunologic, respiratory, carcinogenic, reproductive, neurologic, and cardiovascular effects.

N-Acetyl (2-Cyanoethyl) Cysteine (NACE)

N-acetyl (2-cyanoethyl) cysteine (NACE) is a result of exposure to acrylonitrile and NACE is the major metabolite. Acrylonitrile is a colourless liquid with a pungent odor. It is used in the production of acrylic fibers, resins, and rubber. Use of any of these products could lead to exposure to acrylonitrile. Smoking tobacco and cigarettes is another potential exposure. Exposure to acrylonitrile can lead to headaches, nausea, dizziness, fatigue, and chest pains. The European Union has classified acrylonitrile as a carcinogen. Workers exposed to high levels of airborne acrylonitrile are diagnosed more frequently with damage to their lungs, liver, and central nervous system.

N-Acetyl (2,Hydroxypropyl) Cysteine (NAHP)

N-Acetyl (2, hydroxypropyl) Cysteine (NAHP) is a metabolite of propylene oxide, which is majorly used to produce polyurethane plastics and fumigant. These materials are used in polyester resins for the textile and construction industries as well as for lubricants, surfactants, and oil demulsifiers. Propylene oxide has been classified as a possible human carcinogen. Frequent exposure may lead to an increased risk of cancer.

N-Acetyl (3,4-Dihydroxybutyl) Cysteine

N-Acetyl (3,4-Dihydroxybutyl) Cysteine (NADB) is a metabolite of 1,3-butadiene, which is important industrially as a monomer in the production of synthetic rubber. Individuals that come into contact with rubber, such as car tires, could absorb up to 1,3 butadiene through the skin. Although butadiene breaks down quickly in the atmosphere, it is nevertheless found in the ambient air in urban and suburban areas as a consequence of its constant emission from motor vehicles. Butadiene has been listed as a carcinogen, and long-term exposure is associated with cardiovascular disease, leukemia, and other cancers.

Environmental Toxins

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|-----------|------------|--------|---------------|--------------|------------------------|
| TEST | TEST | Female | 1991-11-11 | 2306270585 | 2023-06-27 10:00 (PST) |

Volatile organic compounds

COMMENTS

N-Acetyl (Propyl) Cysteine (NAPR)

N-Acetyl (Propyl) Cysteine (NAPR) is a metabolite of 1-bromopropane (nPB), which is used as a liquid or gaseous solvent with a characteristic hydrocarbon odor. It is widely used for metal cleaning, foam gluing, and dry cleaning. nPB has been classified as a human carcinogen. Occupation exposure to nPB in higher concentrations has resulted in significant injuries. Reported symptoms of overexposure affect the nervous system and include confusion, slurred speech, dizziness, paresthesias, and difficulty walking; unusual fatigue and headaches; development of arthralgias; visual disturbances (difficulty focusing); and muscle twitching. Symptoms may persist for over a year.

N-acetyl phenyl cysteine (NAP)

Benzene synthesizes N-acetyl phenyl cysteine (NAP) as a metabolite. Environmentally, benzene is an important solvent. The major sources of benzene exposure are tobacco smoke, automobile service stations, exhaust from motor vehicles, and industrial emissions. Ingestion and dermal absorption of benzene can also result from contact with contaminated water. Benzene may cause drowsiness, dizziness, rapid or irregular heartbeat, and headaches in people who breathe it in. Long term exposure to benzene may increase the risk of cancer, aplastic anemia, bone marrow failure, acute leukemia, and cardiovascular disease. Benzene is extremely toxic and targets the liver, kidney, lung, heart, and brain as well as causing DNA strand breaks and chromosomal damage. Benzene and its metabolites induce oxidative stress, which causes genomic instability through DNA damage. Changes to genomic stability have been linked to aging. Thus, NAP toxicity may accelerate aging owing to its contribution to genomic instability, which is a hallmark of aging.

Phenyl glyoxylic Acid (PGO)

Phenyl glyoxylic Acid (PGO) is a metabolite of styrene. Styrene is used in the manufacturing of plastics, in building materials, and is found in car exhaust fumes. Polystyrene and its copolymers are widely used as food-packaging materials. Styrene is a known carcinogen, especially in the case of eye contact. Long-term exposure to styrene may cause central nervous system and kidney effects, headaches, depression, fatigue, hearing loss, balance and concentration problems, and even cancer.

Environmental Toxins



Risk and Limitations

This test has been developed and its performance characteristics determined by Vibrant America LLC., a CLIA certified lab. These assays have not been cleared or approved by the U.S. Food and Drug Administration.

Environmental Toxins panel does not demonstrate absolute positive and negative predictive values for any condition. Its clinical utility has not been fully established. Clinical history and current symptoms of the individual must be considered by the healthcare provider prior to any interventions. Test results should be used as one component of a physician's clinical assessment.

Environmental Toxins Panel testing is performed at Vibrant America, a CLIA certified laboratory. Vibrant America has effective procedures in place to protect against technical and operational problems. However, such problems may still occur. Examples include failure to obtain the result for a specific toxin due to circumstances beyond Vibrant's control. Vibrant may re-test a sample to obtain these results but upon re-testing the results may still not be obtained. As with all medical laboratory testing, there is a small chance that the laboratory could report incorrect results. A tested individual may wish to pursue further testing to verify any results.

The information in this report is intended for educational purposes only. While every attempt has been made to provide current and accurate information, neither the author nor the publisher can be held accountable for any errors or omissions.

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SAMPLE