ULTAGENOMICS

DECODE YOUR DNA. GUIDE YOUR HEALTH.

Environmental Toxin Response

GENETICS REPORT

1	3	56
Report	Report Panels	Gene Variants

Example Client
Prepared for

September 19, 2026
Report Date

Our genetic test provides insights into how your unique genetic profile influences health areas. By understanding these genetic tendencies, you and your healthcare provider can make more informed lifestyle and wellness choices to support long-term health goals.

THESE GENE RESULTS MIGHT TELL YOU

- ✓ How your body may respond to certain nutrients
- ✓ Dietary patterns and environmental factors
- ✓ Potential predispositions in detoxification, metabolism, and physical performance
- ✓ Why some wellness strategies may work differently for you

THESE GENE RESULTS WILL NOT TELL YOU

- ✕ Any medical diagnoses
- ✕ Cancer diagnoses or clinical cancer risk assessments
- ✕ Predictions about future health conditions
- ✕ How lifestyle factors outside of genetics impact health outcomes

This report is intended for informational purposes only and does not constitute medical advice. Results reflect genetic predispositions, not guarantees of health outcomes. Always consult a qualified healthcare practitioner before making changes to your health regimen.

A typical or decreased result does not mean there is no risk, and an increased result does not mean a condition will develop. Environment, lifestyle and family history also play a role. Any result that mentions cancer reflects a genetic tendency only. It is not a clinical cancer risk assessment and does not replace recommended cancer screening.

Talk with a healthcare provider before starting any supplement, especially if pregnant or breastfeeding, taking prescription medications, or managing a medical condition. Do not start, stop or change any medication based on these results without first talking with the prescribing provider.

Statements about supplements in this report have not been evaluated by the Food and Drug Administration. These products are not intended to diagnose, treat, cure, or prevent any disease.

Research on how genes relate to health is still developing. Some recommendations are based on limited evidence and may be updated as new research becomes available.

Testing is performed by a CLIA-certified, CAP-accredited laboratory. Genotyping is highly accurate, but no laboratory test is completely error-free. Results that will change clinical decisions may warrant confirmatory testing.

OVERVIEW

Results at a Glance

This report covers 3 panels and 10 individual results. The summary below shows where the genetic profile indicates areas worth focusing on, areas to monitor, and areas where results are favorable. The Moderate results are where targeted support may have the most impact.

0

NEEDS ATTENTION

3

MODERATE

- Benzene Risk
- Arsenic Exposure Risk
- Fluoride Exposure Risk

7

OPTIMAL

- General Chemical Sensitivity
- Pesticide Risk
- BPA Risk
- Mold Metabolism (CYP1A2)
- Mold Severity Risk
- Mercury Exposure Risk
- Lead Exposure Risk

1 Environmental Sensitivity

27 variants analyzed

In plain terms: Environmental sensitivity refers to an individual's genetic predisposition to react or respond differently to various environmental factors and chemicals. This section explores genetic risks associated with general chemical sensitivity, pesticides, and bisphenol A (BPA).

General chemical sensitivity encompasses an individual's susceptibility to adverse effects from exposure to a wide range of environmental chemicals, such as cleaning products, fragrances, and pollutants. Genetic variations can influence the body's ability to metabolize and eliminate these chemicals, potentially leading to heightened sensitivity or increased risk of adverse reactions.

Pesticide risk refers to an individual's genetic susceptibility to the potential harmful effects of pesticide exposure. Being aware of genetic predispositions for pesticide risk can guide decisions about pesticide exposure and encourage the use of protective measures when necessary.

Bisphenol A (BPA) risk focuses on an individual's genetic susceptibility to the potential health effects of BPA, a chemical commonly found in plastics and other consumer products. Genetic variations can influence how efficiently the body metabolizes and eliminates BPA, which can affect risk of BPA-related health concerns.

Understanding genetic predispositions for these enviro-toxins can support choices that minimize exposure and promote overall well-being.

While genetics play a role in environmental sensitivity, other factors such as lifestyle choices, overall health, and individual circumstances also contribute to how an individual responds to environmental factors.

YOUR RESULTS

General Chemical Sensitivity



TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical general chemical sensitivity genetic results have a typical sensitivity to environmental toxins. Their genetic profile suggests their body respond to chemicals in a manner similar to the general population. It is important for individuals with typical general chemical sensitivity genetics to be aware of potential chemical sensitivities and take appropriate precautions when handling or being exposed to envirotoxins. Hypoallergenic and fragrance-free cleaning and beauty supplies are advisable to mitigate exposure.

YOUR RESULTS

Benzene Risk



INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased risk for benzene genetic results possess genetic variations that elevate their susceptibility to the harmful effects of benzene exposure beyond the typical population level. This heightened genetic predisposition underscores the importance of vigilance and proactive measures to reduce benzene exposure. Individuals with an increased risk should be particularly mindful of avoiding environments with high benzene levels, using protective measures when exposure is unavoidable, and seeking regular medical check-ups to monitor for any health issues associated with benzene.

RECOMMENDATIONS

SUPPLEMENT

N-Acetylcysteine (NAC)

600 to 1,200 mg daily

NAC provides cysteine for glutathione synthesis needed by glutathione-S-transferase enzymes to conjugate benzene oxide and hydroquinone, the reactive intermediates responsible for benzene-induced chromosomal damage.

SUPPLEMENT

Sulforaphane

20 to 40 mg daily

Sulforaphane activates Nrf2, upregulating Phase II glutathione-S-transferase and UDP-glucuronosyltransferase enzymes that conjugate benzene oxide for urinary excretion.

DIET

Cruciferous Vegetables

2 to 5 cups daily

Cruciferous vegetables provide glucoraphanin that converts to sulforaphane, activating the Nrf2-driven Phase II enzyme cascade responsible for detoxifying benzene metabolites through glutathione conjugation and glucuronidation.

DIET

Avoid Charred and Smoked Foods

Charring and smoking food at high temperatures generates benzene and polycyclic aromatic hydrocarbons at concentrations that contribute meaningfully to dietary benzene load on top of environmental exposure.

LIFESTYLE

Avoid Benzene Sources

Gasoline vapor, tobacco smoke, industrial solvents, and adhesives are the largest benzene exposure sources, with petroleum products and cigarette smoke the most common household exposures.

LIFESTYLE

Adequate Hydration

64+ oz daily

Water-soluble benzene metabolites including trans-trans-muconic acid and phenylmercapturic acid are eliminated through renal excretion following Phase II conjugation, and high fluid intake ensures continuous renal flushing of these conjugates.

FURTHER TESTING

Urine trans-trans-Muconic Acid (TTMA)

TTMA is the specific urinary metabolite of benzene ring-opening that confirms active benzene exposure and reflects current metabolic processing through the primary detoxification pathway.

FURTHER TESTING

Complete Blood Count (CBC) with Differential

Benzene is a myelotoxin that selectively damages bone marrow progenitor cells, manifesting as progressive reductions in neutrophil, platelet, and red blood cell counts before clinical pancytopenia develops.

YOUR RESULTS

Pesticide Risk



TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical pesticide risk genetic results have a typical risk of adverse effects related to pesticide exposure, including organophosphates. Their genetic profile suggests their body respond to pesticide exposure in a manner similar to the general population. It is crucial for individuals with typical pesticide risk genetics to be mindful of potential pesticide exposures and take appropriate precautions to minimize exposure. This may include using protective clothing, practicing safe handling and application of pesticides, and considering organic or pesticide-free alternatives whenever possible.

YOUR RESULTS

BPA Risk

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical bpa risk genetic results have a typical risk of adverse effects associated with exposure to bisphenol A (BPA). BPA is a chemical commonly found in plastics, food containers, and other consumer products. Their genetic profile suggests their body may respond to BPA exposure in a manner similar to the general population. It is important for individuals with typical bpa risk genetics to be aware of potential sources of BPA exposure and take necessary steps to minimize exposure, such as using BPA-free products, avoiding prolonged contact with plastics, and choosing alternative packaging options whenever possible.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
CYP2C9 A1075C	rs1057910	**AA**	**XPC G2815T**	rs2228001	**TG**
CYP2C9 C430T	rs1799853	**CC**	**SLCO1B1 T521C**	rs4149056	**TT**
GSTM1 (A>G)	rs366631	**AG**	**ANKK1 G2137A**	rs1800497	**GG**
GSTA1 A-135G	rs3957357	**GG**	**CYP1A1 T1384C**	rs1048943	**TT**
GSTP1 A313G	rs1695	**AA**	**CYP2D6 C506-1T**	rs3892097	**CC**
SOD2 C512+816A	rs2758331	**AC**	**PTGS2 C306G**	rs5277	**CG**
SOD2 A47G	rs4880	**AG**	**SULT1A1 C628T**	rs1042028	**TC**
CAT (C>T)	rs1001179	**CC**	**NAT2 G857A**	rs1799931	**GG**
CYP2E1 C-1055T	rs2031920	**CC**	**NAT2 G191A**	rs1801279	**GG**
CYP2E1 g.G3739C	rs3813867	**GG**	**NAT2 G590A**	rs1799930	**AA**
BCHE C1699T	rs1803274	**CC**	**NAT2 T341C**	rs1801280	**TT**
BCHE C-32T	rs1126680	**CC**	**CAT C1167T**	rs769217	**CC**
UGT2B15 A253C	rs1902023	**CC**	**HSPA1L G1478A**	rs2227956	**AA**
PON1 T575C	rs662	**TT**			

2 Mold Severity

9 variants analyzed

In plain terms: Mold severity refers to an individual's genetic predisposition for metabolizing mold toxins and their risk of experiencing adverse health effects from mold exposure. This section explores genetics for mold metabolism, specifically focusing on CYP1A2 gene variations, as well as symptom severity risk after exposure.

The CYP1A2 gene plays a crucial role in the metabolism of mold toxins. Genetic variations in this gene can impact how efficiently the body processes and eliminates mold toxins, which can influence susceptibility to mold-related health issues. Understanding genetic predispositions for mold metabolism can provide insights into the body's ability to handle mold exposure.

Mold severity risk assesses genetic susceptibility to experiencing adverse health effects from mold exposure, specifically ochratoxin A (OTA) and aflatoxin B (AFB1), which are predominantly food-based, but also environmental. Genetic variations can affect the body's response to mold toxins, potentially increasing risk of respiratory symptoms, allergies, or other health concerns when exposed to mold. Being aware of genetic predispositions for mold severity can assist in taking proactive measures to reduce mold exposure and creating a healthier living environment.

While genetics play a role in mold severity, other factors such as the extent and duration of mold exposure, overall health, and individual sensitivities also contribute to an individual's response.

Common sources of OTA mold include: cereals, beans, coffee, wine, beer, fresh and dried fruits, cocoa, nuts, spices, meats, milk, and infant formula. OTA also can be found in water-damaged houses and heating ducts.

Common sources of AFB1 mold include: Groundnuts, Tree nuts, Maize, Rice, Figs, Other dried foods, Spices, Crude vegetable oils, Cocoa beans

YOUR RESULTS

Mold Metabolism (CYP1A2)



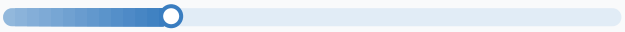
RAPID METABOLISM

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with rapid metabolism mold metabolism (cyp1a2) genetic results have a rapid metabolism of mold-related substances. Their genetic profile suggests their body may have an enhanced ability to metabolize and eliminate molds efficiently. This may provide a relatively better ability to handle mold exposure and reduce susceptibility to adverse health effects. However, it is still important for individuals with rapid metabolism mold metabolism (cyp1a2) genetics to be cautious of potential mold-related risks and take appropriate preventive measures to minimize exposure.

YOUR RESULTS

Mold Severity Risk



DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with decreased mold severity risk genetic results have a low severity risk of mold-related asthma and allergies. Their genetic profile suggests their body may have a decreased susceptibility to developing severe asthma or allergies in response to mold exposure. While they may still experience mild symptoms, they are less likely to experience severe respiratory reactions or exacerbations. However, it is still important for individuals with low mold severity risk genetics to be mindful of potential mold exposure, take appropriate precautions, and seek medical advice if symptoms worsen or persist.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
XPC G2815T	rs2228001	**TG**	**CYP1A2 T739G**	rs2069526	**TT**
GSTA1 A-135G	rs3957357	**GG**	**CYP1A2 G-3860A**	rs2069514	**GG**

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
GSTM1 (A>G)	rs366631	**AG**	**CYP1A2 C-163A**	rs762551	**AC**
CYP1A2 C1291T	rs28399424	**CC**	**SLCO1B1 T521C**	rs4149056	**TT**
CYP1A2 C-729T	rs12720461	**CC**			

3 Element and Heavy Metal Management

20 variants analyzed

In plain terms: Element and heavy metal management is a crucial aspect of maintaining optimal health and well-being. This section explores genetic predispositions for managing exposure to certain elements and heavy metals, including arsenic, mercury, lead, and fluoride. Understanding genetic risk factors can help make informed choices to minimize exposure and support the body's natural detoxification processes.

Arsenic exposure risk refers to the likelihood of being more susceptible to arsenic toxicity. Arsenic is a naturally occurring element that can be found in water, soil, and certain foods. High levels of arsenic exposure have been associated with various health concerns. Genetic variations can influence how efficiently the body metabolizes and eliminates arsenic, affecting overall sensitivity to its harmful effects.

Mercury exposure risk refers to the susceptibility to the negative effects of mercury toxicity. Mercury is a toxic heavy metal found in certain fish, dental amalgam fillings, and environmental pollutants. Genetic factors can impact how efficiently the body processes and eliminates mercury, influencing overall sensitivity to its harmful effects on the nervous system and other organs.

Lead exposure risk refers to the genetic predisposition for increased susceptibility to the toxic effects of lead exposure. Lead is a heavy metal commonly found in older homes, soil, and certain industrial settings. High levels of lead exposure can have detrimental effects on neurological development, cardiovascular health, and other organ systems.

Fluoride exposure risk refers to the genetic predisposition for increased sensitivity to the potential adverse effects of fluoride exposure. Fluoride is a mineral commonly found in water, toothpaste, and certain dental treatments. While fluoride is beneficial for dental health in appropriate doses, excessive exposure can lead to dental fluorosis and other health concerns.

Understanding genetic risk factors for these elements and heavy metals can help guide choices and minimize exposure. Strategies for managing exposure may include drinking filtered water, choosing low-mercury fish options, maintaining good oral hygiene practices, and reducing environmental exposures.

YOUR RESULTS

Arsenic Exposure Risk



INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased arsenic exposure risk genetic results have an increased risk of adverse effects related to arsenic exposure. Their genetic profile suggests their body may be less efficient at processing or eliminating arsenic, potentially increasing the risk of negative impacts. It is crucial for individuals with increased arsenic exposure risk genetics to be extra vigilant and take proactive steps to minimize exposure to arsenic. This may include ensuring access to safe drinking water, reducing consumption of foods known to have higher arsenic content, and being cautious of potential occupational or environmental sources of arsenic contamination.

RECOMMENDATIONS

SUPPLEMENT

Folate (Folic Acid or L-Methylfolate)

400 mcg daily

Supports arsenic methylation and excretion and may help lower blood arsenic. Adequate B12 should accompany it.

SUPPLEMENT

Selenium

55 to 100 mcg daily (diet first)

May modestly support arsenic handling; human data are limited.

DIET

Arsenic-Certified Water Filter

Reverse osmosis or arsenic-specific adsorptive filters cut inorganic arsenic in drinking water, the main exposure route in most regions; EPA and WHO guidance support reducing water arsenic.

DIET

Limit Rice and Rice Products

Rice accumulates inorganic arsenic; rinse and cook in extra water, and limit rice-based cereals and snacks.

LIFESTYLE

Avoid Tobacco Smoke and Treated-Wood Exposure

Tobacco and older pressure-treated wood or pesticide residues are additional arsenic sources and add to the toxic burden.

LIFESTYLE

Document Occupational and Regional Exposure Sources

Note well water, mining, smelting, agricultural or wood-treatment exposure so results can be interpreted and sources reduced.

FURTHER TESTING

Urinary Arsenic with Speciation

Separates toxic inorganic arsenic from harmless seafood-derived forms; avoid seafood for several days beforehand so total arsenic is not falsely elevated.

FURTHER TESTING

Drinking Water Arsenic Test

Tests private wells, which are not regulated, for arsenic; the most direct way to find and fix a source.

YOUR RESULTS

Fluoride Exposure Risk



INCREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with increased fluoride exposure risk genetic results have an increased risk of adverse effects associated with fluoride exposure. Their genetic profile suggests their body may be less efficient at processing or eliminating fluoride, potentially increasing the risk of adverse impacts on oral health or other potential concerns.

RECOMMENDATIONS

SUPPLEMENT

Iodine

150 to 300 mcg daily

Fluoride competes directly with iodine at the sodium iodide symporter on thyroid follicular cells, reducing iodine uptake and thyroid hormone synthesis in proportion to the fluoride-to-iodine ratio in circulation.

SUPPLEMENT

Magnesium (glycinate)

200 to 400 mg daily

Fluoride has high affinity for magnesium and forms intracellular magnesium fluoride complexes that precipitate and deplete free magnesium, inhibiting the hundreds of magnesium-dependent enzymes in energy metabolism, DNA repair, and protein synthesis.

DIET

Limit Strongly Brewed Black Tea

Up to 2 to 3 cups daily

Tea leaves concentrate fluoride, so very large amounts add up.

DIET

Reduce Black Tea Consumption

Black tea leaves accumulate fluoride from soil through their root system, resulting in concentrations of 1 to 6 mg fluoride per cup, which can substantially exceed the amount from drinking water when consumed in quantity.

LIFESTYLE

Use a Pea-Sized Amount of Toothpaste

Spit, don't swallow

Keeps the protective benefit while limiting swallowed fluoride.

LIFESTYLE

Check Local Water Fluoride

From the water utility's annual report

Shows how much fluoride comes from tap water.

FURTHER TESTING

Spot Urine Fluoride

Spot urine fluoride provides an accessible measure of recent fluoride exposure and renal elimination, with levels above 1.5 mg/L in non-fluoridated water areas indicating elevated dietary or environmental fluoride sources requiring identification.

FURTHER TESTING

Thyroid Function Panel (TSH, Free T4, Free T3)

Fluoride's competition with iodine at the sodium iodide symporter makes thyroid function monitoring the most clinically important follow-up for individuals with fluoride exposure risk and concurrent iodine transport variants.

YOUR RESULTS

Mercury Exposure Risk

DECREASED

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with decreased mercury exposure risk genetic results have a lower risk of adverse effects associated with mercury exposure. Their genetic profile suggests their body may be more efficient at processing and eliminating mercury, reducing the likelihood of negative impacts. However, it is still important to follow recommended guidelines for mercury exposure, such as avoiding or minimizing consumption of high-mercury fish and being cautious of other potential sources of mercury contamination, to maintain optimal health.

YOUR RESULTS

Lead Exposure Risk

TYPICAL

UNDERSTANDING WHAT THIS MAY MEAN

Individuals with typical lead exposure risk genetic results have a typical risk of adverse effects associated with lead exposure. Their genetic profile suggests a response to lead that is similar to the general population. It is important to be aware of potential sources of lead exposure, such as lead-based paint, contaminated soil, or certain occupational settings, and take appropriate measures to minimize exposure. This may include regular cleaning, proper hygiene practices, and following guidelines for lead-safe environments, especially for pregnant women and children.

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
GSTP1 C341T	rs1138272	**CC**	**GCLC g.G4891A**	rs17883901	**GG**
ANKK1 G2137A	rs1800497	**GG**	**GSTP1 A313G**	rs1695	**AA**
SOD2 T*1993G	rs10370	**TG**	**GSTM1 (A>G)**	rs366631	**AG**
CREB1 T262-2836C	rs6740584	**TT**	**MT4 G143A**	rs11643815	**GG**
CREB1 g.A2363G	rs2253206	**AA**	**CPOX4 T814G**	rs1131857	**TT**
AS3MT G529-56C	rs3740393	**GG**	**MTHFR G677A**	rs1801133	**GA**
AS3MT T860C	rs11191439	**TT**	**MTHFR T1298G**	rs1801131	**TT**

GENETIC SUMMARY

GENE	RS ID	YOUR GENOTYPE	GENE	RS ID	YOUR GENOTYPE
ALAD C264G	rs1800435	**CC**	**GSTO2 A424G**	rs156697	**GG**
VDR C1024+283T	rs1544410	**CC**	**GSTO1 g.G4494A**	rs2164624	**AA**
GCLM G-588A	rs41303970	**AG**	**GSTO1 C419A**	rs4925	**AA**

APPENDIX A

Supplement Recommendations

Based on your results

Supplements listed below are recommended for panels where at least one result was outside the typical range.

SUPPLEMENT	RATIONALE	PANEL
N-Acetylcysteine (NAC) 600 to 1,200 mg daily	NAC provides cysteine for glutathione synthesis needed by glutathione-S-transferase enzymes to conjugate benzene oxide and hydroquinone, the reactive intermediates responsible for benzene-induced chromosomal damage.	Environmental Sensitivity
Sulforaphane 20 to 40 mg daily	Sulforaphane activates Nrf2, upregulating Phase II glutathione-S-transferase and UDP-glucuronosyltransferase enzymes that conjugate benzene oxide for urinary excretion.	Environmental Sensitivity
Folate (Folic Acid or L-Methylfolate) 400 mcg daily	Supports arsenic methylation and excretion and may help lower blood arsenic. Adequate B12 should accompany it.	Element and Heavy Metal Management
Selenium 55 to 100 mcg daily (diet first)	May modestly support arsenic handling; human data are limited.	Element and Heavy Metal Management
Iodine 150 to 300 mcg daily	Fluoride competes directly with iodine at the sodium iodide symporter on thyroid follicular cells, reducing iodine uptake and thyroid hormone synthesis in proportion to the fluoride-to-iodine ratio in circulation.	Element and Heavy Metal Management
Magnesium (glycinate) 200 to 400 mg daily	Fluoride has high affinity for magnesium and forms intracellular magnesium fluoride complexes that precipitate and deplete free magnesium, inhibiting the hundreds of magnesium-dependent enzymes in energy metabolism, DNA repair, and protein synthesis.	Element and Heavy Metal Management

APPENDIX B

Further Testing Recommendations

Based on your results

Lab tests and assessments recommended for panels where at least one result was outside the typical range.

TEST / ASSESSMENT	CLINICAL RATIONALE	PANEL
Urine trans-trans-Muconic Acid (TTMA)	TTMA is the specific urinary metabolite of benzene ring-opening that confirms active benzene exposure and reflects current metabolic processing through the primary detoxification pathway.	Environmental Sensitivity
Complete Blood Count (CBC) with Differential	Benzene is a myelotoxin that selectively damages bone marrow progenitor cells, manifesting as progressive reductions in neutrophil, platelet, and red blood cell counts before clinical pancytopenia develops.	Environmental Sensitivity
Urinary Arsenic with Speciation	Separates toxic inorganic arsenic from harmless seafood-derived forms; avoid seafood for several days beforehand so total arsenic is not falsely elevated.	Element and Heavy Metal Management
Drinking Water Arsenic Test	Tests private wells, which are not regulated, for arsenic; the most direct way to find and fix a source.	Element and Heavy Metal Management
Spot Urine Fluoride	Spot urine fluoride provides an accessible measure of recent fluoride exposure and renal elimination, with levels above 1.5 mg/L in non-fluoridated water areas indicating elevated dietary or environmental fluoride sources requiring identification.	Element and Heavy Metal Management
Thyroid Function Panel (TSH, Free T4, Free T3)	Fluoride's competition with iodine at the sodium iodide symporter makes thyroid function monitoring the most clinically important follow-up for individuals with fluoride exposure risk and concurrent iodine transport variants.	Element and Heavy Metal Management