TOXDetect
PROFILE®

TOXDetect Profile® Interpretation Guide

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MOSAIC DIAGNOSTICS

Introduction

The information provided in this guide is intended solely for educational purposes and should not be construed as treatment recommendations. It is recommended that you consult with your healthcare provider for any necessary treatment.

The interpretation information included in this guide includes all of the metabolite/parent compounds assessed on MosaicDX TOXDetect Profile® test report.

Legend:

		example:
Metabolite **Parent**	⟶	1) Monoethylphthalate (MEP) **Diethylphthalates**

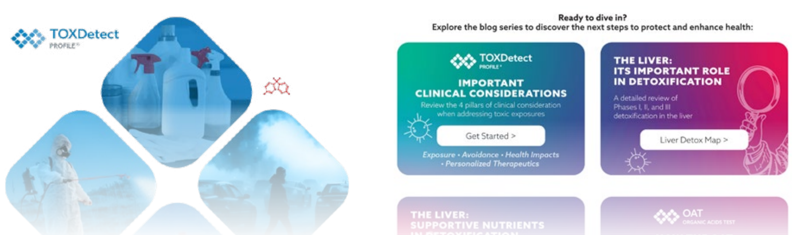
The TOXDetect Profile is a urinary assessment of exposure to environmental toxicants. The profile screens for exposure to common chemicals used to make plastics (phthalates, bisphenols), pesticides, and other industrial chemicals found in personal care products, cleaning products, food packaging, and flame retardants added to furniture and textiles.

The **TOXDetect Profile measures 27 metabolites**, providing insight into exposure to phthalates, bisphenols, volatile organic compounds, parabens, pesticides, glyphosate, and more.

The increased use of environmental toxicants has had several negative consequences for human health and the environment. Exposure to certain toxic chemicals has been linked to an increased risk of cancer, birth defects, neurological disorders, immune dysfunction, endocrine disruption and other health problems. Additionally, these chemical toxicants can pollute air, water, and soil, harming wildlife and ecosystems.

For more information on important clinical considerations when dealing with known or suspected exposure to environmental toxicants please see our **TOXDetect Blog Series**.

TOXDetect PROFILE®



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PHTHALATES

1) Monoethylphthalate (MEP)
Diethylphthalates

2) Monobutyl phthalate (MBP)
Di-n-butyl Phthalate (DBP)

3) Mono-2ethylhexyl phthalate (MEHP)
Di(2-ethylhexyl) Phthalate (DEHP)

4) Mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP)
Di(2-ethylhexyl) Phthalate (DEHP)

5) Monoisobutyl phthalate (MiBP)
Di(2-ethylhexyl) Phthalate (DEHP)

1-5) PHTHALATES are referred to as "the everywhere chemical" due to the fact they are used in hundreds of products, including toys, food packaging, shampoo, vinyl flooring, and more.

Enhanced insight into phthalate exposure is provided by measuring five phthalate metabolites. Phthalates are a series of widely used chemicals found in most products that have contact with plastics during production, packaging, or delivery. These plasticizers which make plastic more flexible, and durable are associated with a number of health problems including reproductive, neurological, respiratory, and increased risk of certain types of cancer.[1,2,3] Most significantly they are known as endocrine disruptors.[4] Exposure can occur through various routes including ingestion - phthalates can leach from food and beverage packaging materials, inhalation - phthalates can be released into the air from products such as vinyl flooring, shower curtains and air fresheners, dermal contact - phthalates can be absorbed through the skin from personal care products, as well as from vinyl gloves and vinyl flooring.[3] Phthalates are metabolized through various pathways, they are conjugated with glucuronic acid or sulfate in the liver, these conjugated metabolites are eliminated from the body through urine or feces.[5] Induced perspiration can be a useful method to facilitate the elimination of certain toxic phthalate compounds, including DEHP and MEHP.[6]

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BISPHENOLS

Bisphenol A (BPA)
Bisphenol A (BPA)

Bisphenol S (BPS)
Bisphenol S (BPS)

6) Bisphenol A (BPA) is a chemical produced in the manufacturing process of polycarbonate plastics and epoxy resins. Found in consumer products such as food and beverage containers, medical devices, shatterproof windows, toys, dental sealants, some metal food container liners, water supply pipes, and more, makes exposure quite common. Recently, BPA is often replaced in many of these products by bisphenol analogs such as bisphenol S (BPS) which has comparable toxicological effects. BPA is most often leached into food from food and beverage containers then ingested. Leaching from containers depends more on the temperature of the liquid or vessel than the age of the container. Other exposure sources of BPA can occur through dust, air, water, dermal absorption, and breastmilk.[7-8] BPA exposure has both developmental and reproductive effects.[8-9] Exposure to BPA has been linked to various cardiovascular and metabolic diseases such as hypertension, diabetes mellitus, obesity, and more. [10]

Neurotoxic effects of BPA including depression, anxiety, neurodevelopmental, and behavioral disorders are documented.[9] BPA can also modulate the immune response increasing susceptibility to autoimmune disease and infections.[13] BPA has been found to promote carcinogenesis through several mechanisms especially leading to an increased risk of hormone-dependent cancers. [9-10] Human metabolism of BPA involves both phase I and phase II reactions in the liver and then quickly clears via the urine especially via glucuronidation. Glucuronidation is primarily driven by uridine diphosphate glucuronosyltransferases (UGTs) in the liver and digestive tract. UGT hepatic activity is low in infancy and increases with age.[11]

7) Bisphenol S (BPS) is generated as a result of exposure to bisphenol S. Bisphenol S (BPS) is metabolized in the body through phase II metabolism, specifically glucuronidation and sulfation.[12] Bisphenols are synthetic compounds used in the production of plastics and resins, commonly found in various consumer products, including food and drink containers, water bottles, thermal receipt papers, dental sealants, toys, cosmetics, and the lining of canned goods.[13-14] Along with being a known endocrine disruptor, BPA has raised concerns due to potential health impacts related to reproductive and developmental effects, increased risk of obesity, diabetes, cardiovascular disease, and certain cancers. In response to concerns, many companies now produce "BPA-Free" products, however, some BPA alternatives like BPS have also raised concerns about potential similar effects.[15-16]

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VOCs - VOLATILE ORGANIC COMPOUNDS

2-3-4 Methylhippuric Acid (2,-3-,4-MHA)
Xylene

Phenylglyoxylic Acid (PGO)
Styrene/Ethylbenzene

N-Acetyl Phenyl Cysteine (NAP)
Benzene

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

N-Acetyl (Propyl) Cysteine (NAPR)
1-bromopropane

N-Acetyl (3,4-Dihydroxybutyl) Cysteine (NADB)
1,3 butadiene

2-Hydroxyethyl Mercapturic Acid (HEMA)
Ethylene Oxide, Vinyl Chloride

8) 2-3-4 METHYLHIPPURIC ACID (2,-3-,4-MHA) Is a metabolite generated as a result of exposure to xylene, an aromatic hydrocarbon widely used in industry and medical laboratories. It is used extensively as a solvent in the rubber, printing, and leather industries. It is also used as a thinner for paints, cleaning agents, and varnishes. Xylene is released primarily from industrial sources. One can also come in contact with xylene through automobile exhaust and a variety of consumer products such as cigarette smoke, paints, varnish, rust preventives, and shellac. Literature suggests that xylene exposure causes toxic effects on various systems of the body. Central nervous system toxicity may lead to headaches, irritability, depression, insomnia, agitation, extreme tiredness, tremors, impaired concentration, and damage to short-term memory. Longer term effects can damage the liver and kidneys.[17] Xylene is primarily eliminated through metabolism in the liver and subsequent excretion of 70-80% of metabolites in urine within 24 hours after exposure. Xylene is metabolized in the liver by side-chain (CH_3) dehydroxylation, finally forming the metabolite methylhippuric acid.[18]

9) PHENYLGLYOXYLIC ACID (PGO) Is a metabolite generated as a result of exposure to styrene/ethylbenzene widely used to make plastics and rubber, which are used to manufacture a variety of products, such as insulation, pipes, automobile parts, printing cartridges, food containers, and carpet backing. Exposure occurs through breathing indoor air that has styrene vapors from building materials, photocopiers, tobacco smoke, and other products. Styrene may also leach from polystyrene containers used for food products, especially when food is heated in these containers.[19] Short term exposure can cause CNS depression and skin and respiratory irritation. Long term exposure can damage the reproductive system and cause problems such as infertility and birth defects,[20-21] can cause neurological damage such as memory loss, difficulty concentrating, and can cause impaired motor function.[22] Exposure to PGO has been linked to an increased risk of leukemia and lymphoma.[23] In the liver, styrene is metabolized to styrene-7,8-

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oxide (SO) by cytochrome P-450 enzymes. SO can then be further metabolized to styrene glycol, mandelic acid, and phenylglyoxylic acid, which are excreted in the urine. Glutathione conjugation is also a significant pathway for detoxification.[24-25]

10) N-ACETYL PHENYL CYSTEINE (NAP) is a metabolite generated as a result of the exposure to benzene, an industrial solvent. Its use has been reduced due to toxicity and potential health hazards. Exposure has been associated with a range of acute and long-term adverse health effects and diseases, including cancer and hematological effects. Exposure can occur occupationally, in the general environment and in the home as a result of the ubiquitous use of benzene-containing petroleum products, including motor fuels and solvents. Active and passive exposure to tobacco smoke is also a significant source of exposure. Benzene exposure has been linked to respiratory, hepatic, cardiovascular, immune, nervous, and endocrine system dysfunction.[26-32] High exposure to benzene may cause nausea, vomiting, dizziness, poor coordination, central nervous system depression, and even death.[32,33] The metabolism of benzene is complex and involves multiple enzymatic pathways. Benzene is primarily metabolized in the liver by the cytochrome P450 enzyme system. It undergoes oxidation to form several metabolites. These metabolites can further undergo conjugation with glucuronic acid or sulfate to form more water-soluble compounds that can be excreted in urine.[35,36]

11) N-ACETYL (2-CYANOETHYL) CYSTEINE (NACE) Is a metabolite generated as a result of the exposure to acrylonitrile. Acrylonitrile exposure comes from the use of products containing acrylonitrile, such as acrylic fiber clothing or carpeting, acrylonitrile-based plastics, leaching into foods from plastic food containers, and cigarette smoke. Humans exposed to high levels via inhalation experience respiratory tract irritation, labored breathing, dizziness, cyanosis, limb weakness and convulsions.[37] Long-term exposure to acrylonitrile has been associated with subjective symptoms such as headache, fatigue, and general malaise.[38] Acrylonitrile is primarily metabolized by the liver, involving the conjugation with glutathione by glutathione transferases. This chemical reaction results in the formation of N-Acetyl (2-Cyanoethyl) Cysteine, which is excreted in the urine.[39-40] It is considered a probable human carcinogen, with evidence suggesting an association with lung cancer.[41-42]

12) N-ACETYL (PROPYL) CYSTEINE (NAPR) is a metabolite generated as a result of exposure to 1-bromopropane, a solvent in adhesives, dry cleaning, degreasing, and electronic and metal cleaning products. Low levels of NAPR can cause headaches, decreased sensation in the fingers and toes, and a drunk-like feeling. Long-term exposure can have lasting effects on the nervous system including weakness, incoordination, loss of feeling, inability to walk, and damage to nerves. Health impacts of 1-bromopropane exposure include neurotoxicity, reproductive toxicity, hematopoietic disorders, DNA damage, and respiratory toxicity. It can also cause symptoms such as headache, mucosal irritation, decreased sensation, paresthesia, and stumbling.[43-46]
In the metabolism of 1-bromopropane, conjugation reactions involving the attachment of a cysteine group result in the formation of metabolites like N-Acetyl (Propyl) Cysteine, aiding in its detoxification and elimination.[47] Supplementation with glutathione or NAC may accelerate elimination.

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13) N-ACETYL (3,4-DIHYDROXYBUTYL) CYSTEINE (NADB) is a metabolite generated as a result of exposure to 1,3 butadiene, a petrochemical used to produce synthetic rubber used for car and truck tires and is also an environmental toxicant found in car exhaust, combustion of fuels for warmth or energy production and cigarette smoke. It is associated with adverse health impacts, including cancer, and cardiovascular disease.[48-49] The International Agency for Research on Cancer (IARC) concluded that 1,3 butadiene is a human carcinogen. Exactly how humans metabolize 1,3 butadiene is unknown. The predominant route of exposure is inhalation, about half of inhaled 1,3 butadiene is broken down and exhaled. The remaining is broken down to its metabolites in the liver and excreted in the urine.[50]

14) 2-HYDROXYETHYL MERCAPTURIC ACID (HEMA) - is a metabolite generated as a result of exposure to ethylene oxide or vinyl chloride. Ethylene oxide is a man made substance widely used in the production of various chemicals such as plastics, textiles and antifreeze (ethylene glycol). Additionally, ethylene oxide is commonly used as a sterilizing agent for medical equipment. Inhalation is the most common route of exposure in occupational settings and via tobacco smoke. There is some evidence that exposure to ethylene oxide can cause a pregnant woman to lose a pregnancy. The International Agency for Research on Cancer (IARC) concluded that ethylene oxide is a known human carcinogen, exposure is linked to increased risk of leukemia and non-Hodgkin's lymphoma.[51-52] Ethylene oxide is then metabolized by epoxide hydrolase (EH) and glutathione S-transferase (GST) enzymes. These enzymes handle the breakdown and removal of ethylene oxide from the body.[53]

Vinyl chloride is colorless gas used primarily to manufacture polyvinyl chloride (PVC) widely used in numerous products such as pipes, wire and cable insulation, packaging materials, various construction materials and disposable medical products. Inhalation is the most common route of exposure primarily in occupational settings, also via smoke from cigars or cigarettes. Low level exposure is possible via contaminated drinking water. Individuals living near hazardous waste sites and landfills may be exposed to higher levels. Acute high level exposure can produce headaches, dizziness, drowsiness, and loss of consciousness. Long term exposure can result in hepatocellular changes and increased incidence of liver cancer. The International Agency for Research on Cancer (IARC) concluded that vinyl chloride is carcinogenic to humans.[54] Metabolism in humans is attributed to the P-450 monooxygenases in the liver. Intermediates are detoxified primarily via glutathione conjugation and excreted in the urine.[54]

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PARABENS

15) Methylparaben (MeP)
Methylparaben (MeP)

16) Ethylparaben (EtP)
Ethylparaben (EtP)

17) Propylparaben (PrP)
Propylparaben (PrP)

18) Butylparaben (BuP)
Butylparaben (BuP)

15-18) PARABENS are a class of synthetic chemicals commonly used as preservatives in cosmetics, personal care products, pharmaceuticals, and some food items. Some individuals may experience skin irritation or allergic reactions to parabens. Concerns about their impact on human health include the potential for endocrine disruption, links to breast cancer, and increased BMI.[55-58] Enhanced insight into paraben exposure is provided by measuring four types of parabens and a metabolite of exposure, p-hydroxybenzoic acid. Parabens are a class of synthetic chemicals commonly used as preservatives in cosmetics, personal care products, pharmaceuticals, and some food items. The two most commercially used parabens are methylparaben and propylparaben. They help prevent the growth of bacteria, yeast, and mold, thereby extending the shelf life of these products. Some individuals may experience skin irritation or allergic reactions to parabens. Concerns about their impact on human health include the potential for endocrine disruption, links to breast cancer, and increased BMI.[59-63] Parabens are metabolized in humans through hydrolysis to p-hydroxybenzoic acid, followed by conjugation and excretion in urine.[64-65]

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PESTICIDES

Atrazine mercapturate

Atrazine mercapturate

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

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

3-Phenoxybenzoic Acid (3-PBA)

Pyrethoids, Permethrin, Cypermethrin, Cyhalothrins, Fenpropathrin, Deltamethrin, Trihalomethrin

Diethylphosphate (DEP)

Organophosphates

19) Atrazine mercapturate Is a urinary metabolite of atrazine, a widely used triazine herbicide. Atrazine inhibits photosynthesis in plants and is used to kill broadleaf weeds especially in agriculture for crops such as corn and along roadways. Other uses of atrazine include sugarcane crops, golf course turf, and residential lawns. Predominant use is in the Midwest region of the United States.[66-67] Atrazine may migrate out of the soil via surface runoff to bodies of water such as rivers, lakes or groundwater where it does not significantly degrade. The half-life in surface water is over 200 days. Atrazine may also evaporate from the soil into the atmosphere.[68] Exposure to atrazine has been associated with primarily endocrine disruptive impacts. Interference with the hypothalamic-pituitary-gonadal axis affecting steroid synthesis, reproductive, and developmental processes are reported. Atrazine is considered neurotoxic and influences dopamine synthesis.[69] In addition, it has been associated with neurobehavioral changes causing anxiety and social deficits.[70-71] Exposure may lead to hepatotoxic effects.[72]

Some studies have found increased risk of lung, prostate, non-Hodgkin lymphoma, and kidney cancers associated with exposure.[73] Atrazine is primarily metabolized by humans in the liver, creating various toxic metabolites. The CYP1A2 and CYP3A4 enzymes are primarily responsible for phase I metabolism of atrazine in the liver followed by phase II biotransformation including glutathione conjugation. Atrazine is rapidly excreted from the body, usually within 24-48 hours of exposure, but may be distributed into adipose tissue where it can take longer to be excreted, and potentially remain toxic.[68,74]

20) 2,4-DICHLOROPHENOXYACETIC ACID (2,4-D) is the result of exposure to 2,4-Dichlorophenoxyacetic Acid (2,4-D) is one of the most widely used herbicides in the world. It is commonly used in agriculture and landscaping. Chronic exposure to lower levels of 2,4-D has been associated with potential health effects, including endocrine disruption, reproductive effects, developmental effects, and increased risk of non-Hodgkin lymphoma.[75-77] The specific enzymes and genes involved in the metabolism of 2,4-D in humans have not been extensively studied. In animals it is metabolized through processes like conjugation, forming glucuronide, sulfation, and other conjugations, which generate products that are excreted in urine.[78]

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21) 3-Phenoxybenzoic Acid (3-PBA) is a metabolite generated as a result of exposure to pyrethroids, one of the most commonly used pesticides in household and agricultural fields accounting for 30% of insecticide used worldwide. They are modeled after the natural insecticidal compounds found in chrysanthemum flowers, known as pyrethrins. They are widely used in agriculture, household insect control, and veterinary medicine. Pyrethroids work by targeting the nervous system of insects, causing hyperexcitation and paralysis. The most common potential impacts to health include neurobehavioral, neurodevelopmental, and endocrine disruption.[79-80] Exposure has also been associated with an increased risk of all-cause and cardiovascular disease mortality.[81-82] There is limited information on the metabolism of pyrethroids, their metabolism involves enzymes such as cytochrome P450 isoforms and carboxylesterases.[83]

22) DIETHYLPHOSPHATE (DEP) is a metabolite generated as a result of exposure to a number of organophosphate pesticides used widely in agriculture to control pests, as well as in residential settings to manage insects and rodents. The organophosphate pesticides work by inhibiting the activity of acetylcholinesterase, an enzyme essential for proper nerve function. Exposure to organophosphates has been associated with neurological deficits, neurodegenerative diseases, peripheral nerve effects, and neurodevelopmental issues. Additionally, long-term exposure has been linked to oxidative stress, psychological effects, and liver function abnormalities.[84-86] Organophosphates metabolize to dialkyl phosphate metabolites in humans through various enzymatic reactions. Cytochrome P450 (CYP) enzymes and paraoxonases (PONs) play a significant role in the formation of these metabolites.[87]

Organophosphate Pesticides that are converted to DEP

Chlorethoxyphos	Ethion
Chlorfenvinphos	Malathion
Chlorpyrifos- methyl	Parathion
Coumaphos	Phorate
Diazinon	Sulfotep
Diaxathion	TEPP
Disulfoton	Terbufos
Dimathoate	Triazophos

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OTHER

DIPHENYL PHOSPHATE (DPP)
Triphenyl Phosphate

N-ACETYL (CARBOMETHYL) CYSTEINE (NAE)
Acrylamide

PERCHLORATE (PERC)
Perchlorate

OXYBENZONE (OBZ)
Oxybenzone

23) DIPHENYL PHOSPHATE (DPP) is a metabolite generated as a result of exposure to triphenyl phosphate (TPHP), commonly used as a flame retardant in consumer products such as furniture, electronics, and textiles. It is also present in personal care products, such as nail polish and cosmetics and contact with these products can lead to dermal absorption. It can be released into the air from products or during manufacturing processes, causing exposure via inhalation. Another route of exposure is from food and beverages due to migration from packaging materials or contamination during food processing. Exposure to triphenyl phosphate can alter endocrine function and impacts reproduction. Altered thyroid function and decreased semen quality has been observed in humans.[88-91] TPHP is primarily metabolized by cytochrome P450 enzymes, specifically CYP1A2 and CYP2E1, in the liver. These enzymes catalyze the oxidation of TPHP, leading to the formation of its major metabolite, diphenyl phosphate (DPP).[92-93]

24) N-ACETYL (CARBOMETHYL) CYSTEINE (NAE) is a metabolite generated as a result of exposure to acrylamide, which is formed when starchy foods, such as potatoes, grains, and coffee beans, are cooked at high temperatures. Other potential sources of acrylamide exposure include cigarette smoke, as acrylamide is formed during the combustion of tobacco, and certain cosmetic products that may contain acrylamide as a contaminant.[94] Acrylamide has been linked to an increased risk of cancer, particularly in organs such as the kidneys, ovaries, and uterus. Additionally, acrylamide exposure has been associated with neurotoxicity, which can lead to cognitive and motor abnormalities. Other potential health effects include genotoxicity, reproductive toxicity, hepatotoxicity, immunotoxicity, and increased cardiovascular risk.[95-97] To decrease exposure, people can use lower cooking temperatures and opt for cooking methods that produce less acrylamide like boiling, steaming, and microwaving foods instead of baking, roasting, or frying.[98]

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25) PERCHLORATE (PERC) is a metabolite generated as a result of exposure to perchlorate, a chemical used in fireworks, road flares, explosives, and rocket fuel. Perchlorates are considered environmental contaminants due to their widespread use and persistence in the environment. Perchlorates can leach into groundwater from industrial facilities, military sites, or areas where perchlorate-containing products are used or disposed of improperly causing contamination of drinking water. Perchlorate can also enter the food supply through contaminated water used for irrigation or through food processing. Milk is also a source of perchlorate, the content in milk is related to the presence of perchlorate in feed. Certain crops such as leafy greens, vegetables, and fruit have been found to accumulate perchlorate. The main target organ for perchlorate is the thyroid gland. Perchlorate inhibits the thyroid's uptake of iodine. This interference can disrupt thyroid function and lead to health problems such as hypothyroidism (underactive thyroid) or other thyroid disorders. Pregnant women, infants, and children are particularly vulnerable to the effects of perchlorate exposure on thyroid function. Perchlorate does not appear to be modified in the body, either by degradation or covalent binding. [99-100]

26) OXYBENZONE (OBZ) is a chemical compound of the benzophenone class often used in sunscreen and other personal care products due to its ability to absorb ultraviolet light protecting the skin from UV radiation making exposure common.[101] Oxybenzone is often found in effluents of wastewater treatment plants and surface waters. Adverse impacts of oxybenzone on marine and aquatic ecosystems has caused it to be classified as an emerging environmental contaminant.[102] The chemical composition of oxybenzone allows it easily to pass through the skin and placenta barriers. The UV filter is a recognized endocrine disrupting chemical and has been found in both fetal and umbilical cord blood. Some recent findings have associated higher levels of oxybenzone in women's urine with an increased incidence of giving birth to neonates with Hirschsprung's Disease.[103] Some studies have indicated menstrual cycle abnormalities, increased incidence of endometriosis, and uterine fibroid formation associated with exposure. Oxybenzone shows estrogenic activity at commonly found concentrations in the human body.[104] Oxybenzone undergoes hydroxylation in phase I metabolism, and then further conjugation in phase II metabolism.[10] It is rapidly photo-oxidized after topical application producing a potent electrophile that reacts with antioxidants. This inactivation of important antioxidant systems causes concern for the harmful impact it may have on the epidermis.[106]

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GLYPHOSATE

GLYPHOSATE (GLY)

Glyphosate is a broad-spectrum herbicide used in over 750 different products, ranging from agriculture and forestry to home use. Glyphosate is the world's most widely produced herbicide and a key ingredient in products like Roundup™.[107] Glyphosate residues can be found in food and water, leading to exposure through consumption. Studies have shown that dietary intake is a significant source of glyphosate exposure, with higher levels detected in individuals consuming a conventional versus an organic diet.[108-110] Glyphosate can be found in indoor dust, which can lead to exposure through inhalation or ingestion of contaminated dust particles. This route of exposure is relevant for both urban and rural settings.[111-112]

Glyphosate has been classified as a "probable carcinogen" by the International Agency for Research on Cancer (IARC), particularly associated with non-Hodgkin lymphoma.[113] Glyphosate alters the gut microbiome by reducing microbial diversity and disrupting beneficial bacteria. Glyphosate affects microbes through interference with key pathways, including the shikimate pathway which is essential for the synthesis of aromatic amino acids in plants, fungi, and many bacteria. Exposure to glyphosate also impairs microbial functions essential for digestion, immunity, and health.[114] Evidence indicates glyphosate can have significant adverse effects on the brain and behavior, increasing the risk for serious neurological diseases such as Parkinson's disease and Alzheimer's disease.[115-118] Glyphosate exposure can lead to significant metabolic alterations, including disruptions in lipid metabolism and glucose homeostasis, higher urinary concentrations of glyphosate have been associated with an increased risk of T2DM.[119-120]

Glyphosate is rapidly metabolized in the body, with half-lives generally ranging from a few hours to about a day.[121-124] Due to its high solubility in water glyphosate is readily absorbed across epithelial tissues, including the intestine, liver, and kidney. [125] Higher urinary glyphosate levels are associated with increased biomarkers of liver dysfunction and renal injury.[126-127] The best way to reduce exposure to glyphosate is to eat organic foods. Multiple studies have demonstrated that an organic diet significantly reduces urinary glyphosate levels. Other ways to reduce exposure are avoiding living in areas where glyphosate is sprayed especially during spray season, genetically modified organism (GMO) foods, and animal products from which GMO foods were used to feed the animal.[128-131]

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