2018 TRADITIONAL FOOD CONTAMINANT AND FOOD SAFETY REPORT

By:
Candace E. Kraft
Environmental Biologist

Owen Holly Maroney
Community Dietitian

Great Lakes Indian Fish & Wildlife Commission
Planning and Development Division
P.O. Box 9, 72682 Maple Street – Odanah, WI 54861
715-682-6619
www.glifwc.org

Version 1.0

Table of Contents

Background

Great Lakes Indian Fish & Wildlife Commission (GLIFWC) is an organization consisting of 11 sovereign tribal governments in Minnesota, Wisconsin, and Upper Michigan. The 11 Ojibwe[1] (Anishinaabe) member tribes include: Fond du Lac, Mille Lacs, St. Croix, Lac Courte Oreilles, Red Cliff, Bad River, Lac du Flambeau, Sokaogon-Mole Lake, Lac Vieux Desert, Keweenaw Bay, and Bay Mills reservations.

Figure 1: GLIFWC Member Tribe Locations and Ceded Territory Map.

All 11 tribal governments are signatories to treaties retaining off-reservation treaty rights to hunt, fish, and gather. GLIFWC assists its member tribes in implementing off-reservation treaty seasons and in the protection of treaty rights and natural resources. GLIFWC provides natural resource management expertise, conservation enforcement, legal and policy analysis, and public information services. GLIFWC exercises powers delegated by its member tribes.

[1] There are multiple terms used to refer to Ojibwe people. The Ojibwe people often call themselves Anishinaabe, which in Anishinaabemowin (the language of the Ojibwe people) meaning original people. An anglicized term for Ojibwe commonly used interchangeably is Chippewa. (GLIFWC 2016).

Incorporating Traditional Ecological Knowledge (TEK) into natural resource management plans, projects, and rules and regulations, upholds GLIFWC's Strategic Plan to infuse Anishinaabe culture and values into all of its endeavors. Although TEK has many definitions, GLIFWC recognizes it as a subset of wealth of Anishinaabe *gikendaasowin*, or traditional knowledge, based on a deep, enduring connection to the environment through observations shared throughout generations.

For Anishinaabe people, there are inseparable spiritual connections to animals, fish, and plants that carry on traditional lifeways centered on the land, air, and water that sustain treaty resources. Culturally, treaty resources embody both physical resources and spiritual resources. Accessing, utilizing, and sharing treaty resources helps tribal members to connect, practice, and sustain spiritual beliefs and lifeways from generation to generation.

Federal courts reaffirmed treaty rights for GLIFWC member tribes, which includes the right to the utilization and retail sale of treaty harvested materials, according to various stipulations beginning in 1987[2]. Tribal leaders expressed community members' desire and challenges to integrating traditional foods into tribally-operated, federal nutrition programs and to provide these foods for retail sale within their communities. On April 8th, 2015, tribal leaders and delegates, along with federal and state agency representatives, began a series of meetings with the Wisconsin Department of Agriculture, Trade and Consumer Protection (WI DATCP) to discuss these issues along with regulatory concerns.

In response to these meetings, on March 28, 2017, GLIFWC's Board of Commissioners directed GLIFWC to apply for grant funding to facilitate integration of traditional foods into tribally operated federal nutrition programs and to provide these foods for retail sale. The following September, GLIFWC successfully received grant funds from the Administration for Native Americans[3] for a 3-year "GLIFWC Chippewa Ceded Territory Traditional Food Regulatory System Project".

This project will provide the necessary scientific and legal foundation for GLIFWC member tribal communities to establish a Traditional Food Regulatory System capable of expanding the utilization of traditional foods in their communities. Project staff will provide trainings which will enable tribal communities to implement a food regulatory system with trained tribal members.

[2] Lac Courte Oreilles Band of Indians v. Wisconsin (1987) and Mille Lacs v. Minnesota (1999)
[3] Within the Administration for Children & Families, underneath the U.S. Department of Health & Human Services.

Introduction

This Traditional Food Contaminant and Food Safety Report is provided as part of the "GLIFWC Chippewa Ceded Territories Traditional Food Regulatory System Project" and provides a scientific foundation for policy development.

This report is a culmination of a thorough review of relevant scientific literature involving contamination of a biological, chemical, or physical nature in 14 traditional foods of the Anishinaabe people of the Great Lakes region (See Figure 1 on page 1).

Ten of the traditional foods were selected by tribal members via a Traditional Food Interest Survey administered during the winter of 2017-2018 (an excerpt of the survey can be seen in Appendix 1). The survey was made available for tribal leadership, tribal members of GLIFWC member tribes, tribally operated nutrition programs, and tribal food service employees and management. The survey was made available online, through tribal newsletters, over the phone, and by postal mail. The survey divided traditional foods into classifications, as seen in Table 1 on page 4, and asked which food and how frequently respondents would like to have access to these foods. Additionally, within each of these questions, respondents had the opportunity to write in answers or additional comments. A total of 326 surveys were completed, with seventy-seven percent of respondents being tribal members from GLIFWC member tribes. An additional 11% work as a food handler or administratively at a food establishment that serves tribal members (e.g. school on or near reservations, Head Start, casino, etc.).

Survey results were tabulated and analyzed to create an initial "Interest List" to include the food from each section with the highest interest rating. Once generated, the list was then presented to GLIFWC's Board of Commissioners, Voigt Intertribal Task Force, Lakes Committee, and GLIFWC's Advisory and Guidance Input Group of Elders (GAAGIGE) for additional input from tribal leadership, and elders from participating member tribes. Four recommendations were suggested by GAAGIGE: wild leeks/ramps, wild beach pea, hazelnuts, and morel mushrooms. Recommendations were added to the list to complete a list of 14 Identified Traditional Foods (as seen below).

Table 1: Categorization of Identified Traditional foods

IDENTIFIED TRADITIONAL FOODS LIST

Food	***Classification***
White-Tailed Deer	Large Game
Rabbit/Hare	Small Game
Duck	Migratory Birds
Turkey	Upland Game Birds
Whitefish	Great Lakes Fish
Walleye	Inland Fish
Fresh Berries	Fruit
Wild Leeks/Ramps	Bulb Vegetable
Wild Beach Pea	Legume
Hazelnut	Tree nut
Morel Mushroom	Fungi
Wild Rice	Grain
Berry Jams/Jellies	Value-Added
Maple Syrup	Value-Added
Animal Fat	Value-Added
Venison Jerky	Value-Added

This report outlines current scientific literature with the unknown risks outlined in the companion document. In addition to the scientific literature, relevant TEK has been incorporated, when available, to better understand the what risks are known and what remains unknown.

TABLE 2: THIS MATRIX OUTLINES BIOLOGICAL, CHEMICAL, AND PHYSICAL HAZARDS ASSOCIATED WITH IDENTIFIED TRADITIONAL FOODS.

		HAZARDS				
		BIOLOGICAL				
Common Name	**Scientific Name**	**Pathogen**	**Disease / Virus**	**Parasite**	**Prion (CWD)**	**Allergen**
Large/Small Game						
White-tailed Deer	*Odocoileus virginiatus*	X	X	X	X	
Snowshoe Hare	*Lepus americanus*	X	X	X		
Cottontail Rabbit	*Sylvilagus floridanus*	X	X	X		
Birds						
Ducks/divers						
Scaup/Bluebill	*Aythya affinis; A. marila*	X	X	X		
Ring-necked Duck	*Aythya collaris*	X	X	X		
Duck/dabblers						
Blue-winged Teal	*Anas carolinensis*	X	X	X		
Green-winged Teal	*Anas discors*	X	X	X		
Mallard	*Anas platyrhynchos*	X	X	X		
Wood Duck	*Aix sponsa*	X	X	X		
Upland						
Wild Turkey	*Meleagris gallopavo*	X	X			
Fish						
Walleye	*Sander vitreus*	X		X		X
Whitefish	*Coregonus clupeaformis*	X		X		X
Plants/ Fungi						
Wild Strawberry	*Fragaria vesca*	X				
Wild Raspberry	*Rubus idaeus*	X				
Wild Blueberry	*Vaccinium angustifolium*	X				
Wild Blackberry	*Rubus allegheniensis*	X				
Highbush Cranberry	*Viburnum opulus*	X				
Wintergreen	*Gaultheria procumbens*	X				
Elderberry	*Sambucus canadensis*	X				
Wild Ramps/Leeks	*Allium tricoccum*	X				
Hazelnuts	*Corylus americana*	X				X
Beach Pea	*Lathyrus japonicus*	X				
Wild Rice	*Zizania palustris*	X				
Morel Mushroom	*Morchella esculenta*	X				
Value Added						
Maple Syrup	*Acer saccharum*	X				
Jams/Jellies	Various species	X				
Venison Jerky	*Odocoileus virginiatus*	X	X	X	X	X
Animal Fat (duck)	*Anas spp.*	X				
		Pathogen	**Disease / Virus**	**Parasite**	**Prion (CWD)**	**Allergen**

TABLE 2: THIS MATRIX OUTLINES BIOLOGICAL, CHEMICAL, AND PHYSICAL HAZARDS ASSOCIATED WITH IDENTIFIED TRADITIONAL FOODS. (CONTINUED FROM PREVIOUS PAGE)

		HAZARDS				
		CHEMICAL			PHYSICAL	
Common Name	**Scientific Name**	**Heavy Metal**	**Chemical / Pesticide**	**Natural Toxin**	**Bullet Fragment**	**Shot Pellet**
Large/Small Game						
White-tailed Deer	*Odocoileus virginiatus*	X	X		X	
Snowshoe Hare	*Lepus americanus*	X	X		X	X
Cottontail Rabbit	*Sylvilagus floridanus*	X	X		X	X
Birds						
Ducks/divers						
Scaup/Bluebill	*Aythya affinis; A. marila*	X	X			X
Ring-necked Duck	*Aythya collaris*	X	X			X
Duck/dabblers						
Blue-winged Teal	*Anas carolinensis*		X			X
Green-winged Teal	*Anas discors*		X			X
Mallard	*Anas platyrhynchos*		X			X
Wood Duck	*Aix sponsa*		X			X
Upland						
Wild Turkey	*Meleagris gallopavo*	X				X
Fish						
Walleye	*Sander vitreus*		X			
Whitefish	*Coregonus clupeaformis*		X			
Plants						
Wild Strawberry	*Fragaria vesca*		X			
Wild Raspberry	*Rubus idaeus*	X				
Wild Blueberry	*Vaccinium angustifolium*	X	X			
Wild Blackberry	*Rubus allegheniensis*	X				
Highbush Cranberry	*Viburnum opulus*					
Wintergreen	*Gaultheria procumbens*					
Elderberry	*Sambucus canadensis*			X		
Wild Ramps/Leeks	*Allium tricoccum*					
Hazelnuts	*Corylus americana*					
Beach Pea	*Lathyrus japonicus*	X		X		
Wild Rice	*Zizania palustris*	X				
Morel Mushroom	*Morchella esculentoides*	X				
Value Added						
Maple Syrup	*Acer saccharum*	X				
Jams/Jellies	Various species					
Venison Jerky	*Odocoileus virginiatus*	X			X	
Animal Fat (duck)	*Anas spp.*	X	X	X		
		Heavy Metals	**Chemical / Pesticide**	**Natural Toxin**	**Bullet Fragment**	**Shot Pellet**

1. Biological Hazards

Biological hazards are biological substances, including bacteria, viruses, parasites, and prions, that cause harm or illness. These are more commonly referred to as pathogens. This report focuses on pathogens that cause disease or illness in humans. Pathogens can be found in a wide variety of environments and sources. The following sections outlines both environmental and food pathogens, and zoonotic pathogens.

TABLE 3: AN OVERVIEW OF BIOLOGICAL HAZARDS IN IDENTIFIED ANISHINAABE TRADITIONAL FOODS.

Biological Hazard Overview	Deer	Rabbit/ Hare	Duck	Turkey	Whitefish	Walleye	Berries	Wild Ramps	Beach Pea	Hazelnut	Morel Mushroom	Wild Rice	Maple Syrup	Berry Jam/Jelly	Animal Fats	Venison Jerky
Bacteria																
E. coli	X	X	X	X	X	X	X	X			X					
Salmonella	X	X	X	X	X	X	X	X								X
Listeria	X	X	X	X	X	X										
Shigella			X		X	X	X	X								
C. jejuni			X	X	X	X	X	X								
Y. enterocolitica	X	X	X		X	X	X	X								
F. tularensis		X	X	X	X	X	X	X								
C. botulism	Foods stored without oxygen (canned, vacuum packed, etc.)															
	X	X	X	X	X	X		X			X				X	
C. perfringens	Foods cooked and held at between 41°- 139°F for extended periods of time.															
	X	X	X	X	X	X										
B. ceruse	Cooked foods which are improperly cooled and/or improperly reheated.															
				X	X	X						X				
S. aureus	Smoked foods and dehydrated foods.															
	X	X			X	X										X
Parasite																
T. gondii	X															
Cryptosporidium			X	X	X	X	X	X								
Giardia			X	X	X	X	X	X								
Nematodes					X	X										
Diphyllobothrium			X	X	X	X										
E. multilocularis							X	X								
Trematodes	X				X	X										
Virus																
West Nile Virus				X												
Norovirus Hepatitis A	Human source. Harvesters in contact with contaminated water and ready-to-eat foods which come into contact with an infected food handler are at risk.															
Prion	X															

Environmental, Zoonotic[4], and Food Pathogens

In aquatic environments, pathogens can live in the water, sediment, on and within aquatic animals. In terrestrial[5] environments, pathogens can live in soils, on plants, on and within terrestrial animals. These pathogens can cause illness to humans but may not cause illness in the animal carrying the pathogen. The following are bacterial, viral, and parasitic pathogens that may potentially be present in the traditional foods that are the subject of this study at the time of harvest (see Table 3 on page 7).

Table 4: Habitats Associated with biological hazards in identified traditional foods. (*RTE = Ready-to-eat foods are foods which do not need an additional processing step before consumption. Examples include: deli meat, venison jerky, and jam.)

SOURCE HABITATS OF BIOLOGICAL HAZARDS			
	AQUATIC	**TERRESTRIAL**	**OTHER**
BACTERIA			
Escherichia coli	X	X	–
Salmonella spp.	X	X	–
Listeria monocytogenes	X	X	–
Shigella spp.	X	–	HUMANS
Campylobacter jejuni	X	X	–
Yersinia enterocolitica	X	X	–
Francisella tularensis	–	X	–
Clostridium botulinum	–	X	RTE* FOOD
Clostridium perfringens	–	X	RTE* FOOD
Bacillus cereus	–	X	RTE* FOOD
Staphylococcus aureus	–	–	HUMANS
PARASITE			
Toxoplasmosis gondii	X	X	–
Cryptosporidium spp.	X	–	–
Giardia lamblia	X	–	–
Nematodes	X	X	–
Diphyllobothrium latum	X	–	–
Echinococcus multilocularis	–	X	–
Trematodes	X	X	–
VIRUS			
West Nile Virus	–	X	–
Norovirus	X	–	HUMANS
Hepatitis A	X	–	HUMANS
PRION			
Chronic Wasting Disease	–	X	–

[4] Zoonotic refers to pathogens which can transfer to humans.
[5] Terrestrial refers to land-based.

Special Note About Water Events

Many pathogens in aquatic habitats are due to contaminated matter from animals and humans. Water can be contaminated by fecal matter when animals or humans desiccate in or near water sources, or when agricultural run-off or sewage overflows are carried into water sources. Events that can cause increase pathogen contamination in water:

- Large snowmelts
- Heavy rains
- Flooding

During these times, water runs off the landscape into lakes, ponds, and streams carrying with it fecal matter, chemical pollutants, and debris. A study conducted near the coast of North Carolina, from 1996 to 2000, found that the bacterial count after hurricanes, which often cause flooding, always increased. The average bacterial count prior to storms were less than 100 colony- forming units (CFU) per 100 ml whereas after storms, the bacterial count ranged from 131 to 16,900 cfu/ 100 ml (Mallin 2002).

Runoff from heavy rains also causes bacteria to rise. According to the Wisconsin Stormwater Manual, fecal bacteria counts are 20 to 40 times higher in urban areas then the health standard for recreational water. These high levels of bacteria are considered typical for runoff from both small and large cities in Wisconsin (Prey n.d., 6).

Additionally, runoff from agricultural areas using manure or housing animals can flow into streams which lead to rivers and into lakes. Water bodies with pathogenic contamination do not always show signs such as algae blooms or odors. Often information about bacterial or other pathogen counts can be found at a local natural resource office or health agency.

A. Bacterial Pathogens

E. coli Brief		
Habitat: Aquatic and Terrestrial		
Sources:		
• Animal Feces (including intestines) • Animal Hides	• Fecal Contaminated Water • Cross-contamination	
Primary Carriers:	*At Risk from Contaminated Water/Soil:*	
• Deer/Venison	• Fish • Duck • Berries	• Wild Ramps • Morel Mushrooms • Beach Peas

Escherichia coli is a large group of bacteria which is naturally found in the environment and animal intestines, including humans. Many strains, or types, of *E. coli* are harmless to humans, however, some strains are pathogenic, causing illness in humans. The pathogenic stains related to food are a type of *E. coli* that are capable of forming toxins within human intestines. In the United States food supply, the strain of *E. coli* that is of greatest concern is shiga toxin- producing *E. coli* (STEC), the most commonly identified being the O157:H7 subclass (CDC 2018).

The main source of pathogenic *E. coli* in terrestrial habitats are ruminant[6] animals, including deer, and their feces, however, it can be found in bird and human feces (CDC 2018c). *E. coli* often enters aquatic habitats through fecal contaminated flood water or storm run-off, agricultural run-off, or sewage overflow (CDC 2015). Fresh produce can be contaminated by untreated water or fecal matter from animals. Common food sources of STEC are undercooked ground beef and contaminated produce (NRAEF 2012). The typical route of exposure is through consuming food or water that has been contaminated with the bacteria, and through cross-contamination[7] and contact with animal or people (CDC 2018).

After consumption of the bacteria, STEC produces toxins in the intestines which cause illness. The Center for Disease Control and Prevention (CDC) estimates each year STEC causes 265,000 illness, 3,600 hospitalizations, and 30 deaths in the United States (CDC 2016) . Consuming only a small amount of STEC can make someone sick.

[6] Ruminant animals that can be found in North America include deer, cows, sheep, and goats.
[7] Cross contamination, or cross contact, occurs when a food comes into contact with a surface (e.g. cutting boards, utensils, food handler gloves, etc.) or other food which has pathogenic microorganisms or other contaminants on it. The contaminants from the surface can transfer on to the food and contaminate the food. This is likely to happen when raw foods come in contact with foods that are ready-to-eat or share the same food contact surface such as a cutting board or knife (NRAEF 2012).

Anyone can become ill from STEC, however, children under the age of five, people with weakened immune systems, and the elderly are at an elevated risk of developing STEC related complications. For all populations, the onset of symptoms can occur within eight hours or up to 10 days after exposure, however, symptoms most often appear three to four days after exposure, which can last for six hours or up to 10 days. These symptoms include: severe abdominal cramps, diarrhea, and vomiting (CDC 2018; DHHS 2011). Approximately about 5-10% of individuals with a STEC infection will develop a complication known as hemolytic uremic syndrome, which can lead to kidney failure. With timely medical treatment most patients will recover within a few weeks, however, some patients may suffer permanent damage or death (CDC 2018).

E. coli can survive with or without oxygen, relatively acidic or basic environments, and at wide temperature ranges (DHHS 2011). *E. coli* can also be found on the hide, fur, or feathers of animals (CDC 2018). Harvesters interested in white-tailed deer, plants eaten raw, and waterfowl or fish that may be in fecal contaminated water are at a higher risk of exposure to *E. coli* bacteria.

***Salmonella* Brief**	
Habitat: Aquatic and Terrestrial	
Sources:	
• Animal Feces (including intestines) • Animal Hides • Bird Feces	• Fecal Contaminated Water • Cross-contamination
Primary Carriers:	*At Risk from Contaminated Water/Soil:*
• Deer/Venison (including jerky) • Rabbit/Hare • Duck • Turkey	• Fish • Berries • Wild Ramps

Salmonella spp. is group of bacteria that is responsible for a food related illness known as nontyphoidal salmonellosis or salmonellosis. *Salmonella* is widely dispersed in nature, living in the intestines of animals, especially birds and reptiles, in terrestrial habitats. *Salmonella* is often found in aquatic habitats that have been contaminated by sewage from flood water or storm run-off, agricultural run-off, and/or sewage overflows (Bibi et al. 2015). Exposure to *salmonella* can occur through: contact with animals carrying the bacteria, fecal contaminated water, person-to-person contact, and cross-contamination. *Salmonella* enters the body through consumption of a contaminated substance (Abraham 2012). In the United States, outbreaks salmonellosis are more often associated with consumption of the bacteria with meat and poultry

products, however, outbreaks related to produce have increased, which are often due to contaminated water or fecal contamination (Hanning 2009).

According to the CDC, about 1.2 million people become ill from salmonellosis, a type of infection *salmonella*, which causes approximately 450 deaths annually in the United States. Of the 1.2 million cases of salmonellosis, approximately one million cases are believed to come from food sources (CDC 2018).

Salmonella is very infectious bacteria, needing only a small amount to infect a person. Anyone can become infected however, infants, persons with weakened immune systems, and the elderly, are at risk for severe illness and death. Typical onset of symptoms is six to 72 hours after exposure and lasts for four to seven days. Symptoms include: diarrhea, abdominal cramps, and fever (Abraham 2012).

Salmonella can survive in acidic conditions, does not need access to oxygen, and can survive dehydration. Harvesters interested in fowl, plants eaten raw, and fish from fecal contaminated water are at the most risk of exposure to S*almonella* bacteria.

***Listeria* Brief**	
Habitat: Aquatic and Terrestrial	
Sources:	
• Animal Feces (including intestines) • Bird Feces • Contaminated Soil	• Fecal Contaminated Water • Cross-contamination
Primary Carriers:	*At Risk from Contaminated Water/Soil:*

• Deer/Venison
• Rabbit/Hare
• Duck
• Turkey
	• Fish
	• Foods typically eaten raw:
	  o Berries
	  o Wild Leeks/Ramps

Listeria monocytogenes, more commonly referred to as listeria, is a bacteria found in contaminated water, soil, poultry, and cattle. *L. monocytoneges* is especially suited to survive in soil, without oxygen, is salt tolerant salt, and continues to grow[8] robustly in refrigerator temperatures (38°F-40°F) unlike most foodborne bacteria. Additionally, *L. monocytoneges* is known to easily spread from contaminated processing equipment that is not properly cleaned and sanitized, such as meat slicers. In aquatic habitats, *L. monocytoneges* can be found in contaminated water and fish intestines. In terrestrial habitats, *L. monocytoneges* can be found in soil, poultry or ruminant intestines, and on

[8] Bacterial growth refers to population size of the bacteria. In suitable growing conditions, bacteria can multiply or produce millions of bacterial cells within hours. The size of the bacterial cell does not change, only the number of that specific bacteria changes.

plants in contact with fecal contaminated water or feces (Food Safety Working Group n.d.; DHHS 2011) .

Consuming *L. monocytoneges* can cause two different infections that affect different populations. The most dangerous infection is listeriosis, which typically infects pregnant women, young children, elderly populations, and persons with weakened or suppressed immune systems (CDC 2018). While the number of listeriosis cases are typically low, approximately 1,600 cases reported annually, the mortality rate is higher than other foodborne illnesses (FDA 2017).

Listeriosis symptoms include headache, stiff neck, confusion, loss of balance, and convulsions, fever, and muscle aches. It can cause meningitis[9] and septicemia. [10] For pregnant women, listeriosis can lead to miscarriage, stillbirth, premature delivery, or life threatening infections (CDC 2018). The onset of symptoms can occur within three to 70 days of ingesting contaminated foods, and can last several days to several weeks. (Food Safety Working Group n.d.).

Healthy populations do not typically become ill with listeriosis, however, may become ill with listerial gastroenteritis, a relatively rare but mild illness characterized by fever and diarrhea. Typically the onset of symptoms takes 24 hours and lasts for about two days (Ooi and Lorber 2005).

L. monocytoneges can be found on wild birds, wild game, and on food growing in contaminated environments. Harvesters interested in fowl, game animals, plants eaten raw, or fish that may be in fecal contaminated water are at the most risk of exposure to *L. monocytoneges* bacteria.

[9] Meningitis is the swelling or inflammation of the protective tissue covering the brain and spinal cord. Many agents can cause meningitis including bacteria, viruses, fungi, parasites, head injury, chronic illnesses, and more. Medical attention is required immediately (Centers for Disease Control and Prevention 2017b).

[10] Septicemia is also known as blood poisoning and occurs when pathogenic bacteria from other parts of the body enter the bloodstream. This is a very serious illness requiring immediate medical attention (Encyclopaedia Britannica 2018).

Shigella Brief	
Habitat: Aquatic	
Sources:	
• Human feces	• Fecal Contaminated Water • Cross-contamination
Primary Carriers:	*At Risk from Contaminated Water:*

Humans are the primary carrier of *Shigella spp*. Therefore, the risk is contact with human feces or fecal contaminated water.	• Fish
	• Duck
	• Foods typically eaten raw:
	  o Berries
	  o Wild Leeks/Ramps

Shigella spp. is a group of pathogenic bacteria which travels in the fecal matter of people. Contaminated lakes and rivers are a source of exposure (CDC 2017). These bacteria are a concern in aquatic habitats however, they are not a concern in terrestrial habitats. Ingesting only a small amount of this bacteria can make a person ill. Route of exposure is through consuming contaminated substances including food and water. Another known route of exposure is food contaminated by flies which seek out human feces and human food.

According to the CDC, shigella causes about 500,000 cases of diarrhea annually in the United States. Consuming a substance contaminated with *shigella* can cause shigellosis. The onset of symptoms occurs between eight hours and two days after exposure and is characterized by moderate to severe diarrhea, stomach pain, and fever which can last for five to seven days. Anyone can become ill from *shigella* and all patients excrete the bacterial cells for several weeks after onset (CDC 2017). Harvesters interested in plants eaten raw, waterfowl, or fish that may be in fecal contaminated water are at the most risk of exposure to *shigella* bacteria.

C. jejuni Brief	
Habitat: Aquatic and Terrestrial	
Sources:	
• Bird Feces • Contaminated Soil	• Fecal Contaminated Water • Cross-contamination
Primary Carriers:	*At Risk from Contaminated Water/Soil:*

• Duck
• Turkey
	• Fish
	• Foods typically eaten raw:
	  o Berries
	  o Wild Leeks/Ramps

Campylobacter jejuni (*C. jejuni)* are bacteria found in the intestines of animals and birds. *C. jejuni* is most often associated with raw or under cooked poultry. In aquatic habitats, water becomes contaminated from feces through sewage overflow, after a storm or flood, and/or animal related agricultural runoff (Abraham 2012). In terrestrial habitats, it can be found in the intestinal tract and feces of cats, dogs, poultry, rodents, cattle, pigs, wild birds, and humans (FSIS 2013).

Consuming *C. jejuni* causes an illness known as campylobacteriosis, which infects approximately 1.3 million people annually. This illness is characterized by diarrhea (which may be bloody), fever, abdominal cramps, and nausea. Symptoms onset within two to five days after exposure and last about one week. Populations with weakened immune systems are at risk of the bacteria entering the bloodstream leading to life threatening conditions (CDC 2017).

Harvesters interested in plants eaten raw, waterfowl, or fish that may be in fecal contaminated water are at the most risk of exposure to *C. jejuni* bacteria.

***Y. enterocolitica* Brief**	
Habitat: Aquatic and Terrestrial	
Sources:	
• Animal Feces (including intestines) • Contaminated Soil • Vacuumed sealed foods	• Fecal Contaminated Water • Cross-contamination
Primary Carriers:	*At Risk from Contaminated Water/Soil:*

• Deer/Venison
• Rabbit/Hare
	• Fish
	• Duck
	• Foods typically eaten raw:
	  o Berries
	  o Wild Leeks/Ramps

Yersinia enterocolitica (*Y. enterocolitica*) are pathogenic bacteria that are associated with rabbit, hare, pig, and cattle intestines. In aquatic habitats, fecal matter can contaminate water through contact with contaminated feces or soil entering the water. For example, sewage overflow, after a storm or flood, and animal related agricultural runoff**.** In terrestrial habitats, *Y. enterocolitica* can be found in soils and feces from infected animals including: pigs, rodents, rabbits, hare, sheep, cattle, horses, dogs, and cats (CDC 2016).

People become infected with *Y. enterocolitica* when they consume contaminated foods or water, while person-to-person infection is rare. *Y. enterocolitica* causes a disease known

as enteric (intestinal) yersiniosis, which causes approximately 117,000 illnesses, 640 hospitalizations, and 35 deaths in the United States annually (CDC 2016a).

Onset of symptoms is usually 24 hours to 11 days after exposure, but can occur later, with symptoms persisting from a few days to a few weeks. In children, symptoms can include fever, abdominal pain, and vomiting. In healthy adults, symptoms are typically nonspecific, but includes diarrhea. In rare cases, immunologic diseases can occur, but this accounts for approximately two to three percent of all *Y. enterocolitica* infections. (Abraham 2012). Harvesters interested in game animals, plants eaten raw, waterfowl, or fish that may be in fecal contaminated water are at the most risk of exposure to *Y. enterocolitica* bacteria.

***F. tularensis* Brief**	
Habitat: Aquatic and Terrestrial	
Sources:	
• Rabbit meat	• Fecal Contaminated Water • Cross-contamination
Primary Carriers:	*At Risk from Contaminated Water/Soil:*
• Rabbit/Hare	• Fish • Foods typically eaten raw: o Berries o Wild Leeks/Ramps

Francisella tularensis are the bacteria which cause a disease termed tularemia or "rabbit fever".[11] There are several different ways to be exposed to tularemia, but 90 percent of people contract this disease through the cleaning and processing of rabbits (MI DNR 2018). Rabbit, hare, grouse, beaver, and muskrat[12] are all carriers of tularemia. As a result, people that harvest or trap these animals have a greater risk of contracting tularemia than the general public. Harvesters may be exposed to tularemia even without contact from visible open cuts or sores.

Individuals eating undercooked meat of infected cottontail rabbits, snowshoe hares,[13] or grouse are also at increased risk for exposure to tularemia. Tularemia cannot be killed

[11] There are two varieties of tularemia: Type A and Type B. Type A is the more serious version, however, Type B is more common. Certain subspecies of bacteria cause the different ailments (Conover and Vail 2015, 57).
[12] Ruffed grouse (*Bonasa umbellus*) and sharp-tailed grouse (*Tympanuchus phasianellus*) are both present in the ceded territories. However, ruffed grouse are more common.
[13] The scientific name of Eastern cottontail rabbit is *Sylvilagus floridanus*. The scientific name of snowshoe hare is *Lepus americanus*.

by freezing meat. Bites from ticks, deer flies, horse flies, midges, and mosquitoes are another mode of transmission of tularemia to humans (CDC 2018).[14]

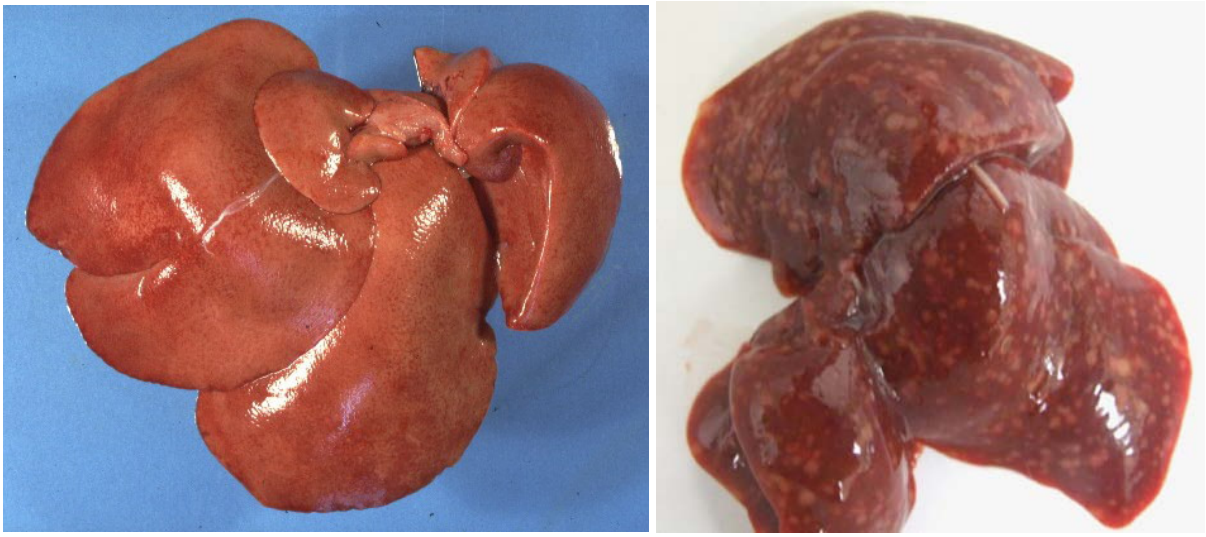


FIGURE 2: HEALTHY RABBIT LIVER (LEFT PHOTO) AND RABBIT LIVER INFECTED WITH TULAREMIA (RIGHT PHOTO). PHOTOS COURTESY OF IDAHO DEPARTMENT OF FISH AND GAME AND IOWA STATE UNIVERSITY

Two less common ways to contract tularemia include inhaling contaminated dust and drinking water containing the bacteria.[15] Although humans are not believed to transmit the disease to other humans, people can become infected with tularemia through infected pets, including cats and hamsters (Conover and Vail 2015; CDC 2018).

Symptoms of tularemia mimic other common sicknesses, therefore, it can be hard to diagnose at first. Symptoms usually appear anywhere from three to five days after exposure, which generally include: swollen lymph nodes, loss of appetite, fever, chills, muscle aches, headache, and fatigue. Some people also experience sore throat, chest pain, and a dry cough. However, symptoms vary depending on how a person was first exposed (Conover and Vail 2015). If the pathogen enters the eye, the eye often becomes watery, accompanied by a mucous discharge. If tularemia enters via skin, a solid, inflamed swelling often forms where the bacteria entered.

[14] Ticks in the United States that transmit tularemia to humans include the wood tick (*Dermacentor andersoni*), the dog tick (*Dermacentor variabilis*), and the lone star tick (*Amblyomma americanum*).
[15] Contaminated dust could get into the air by running over an infected animal or carcass with a lawnmower or tractor. Water could become contaminated by infected animals drinking from a particular area (CDC 2018x).

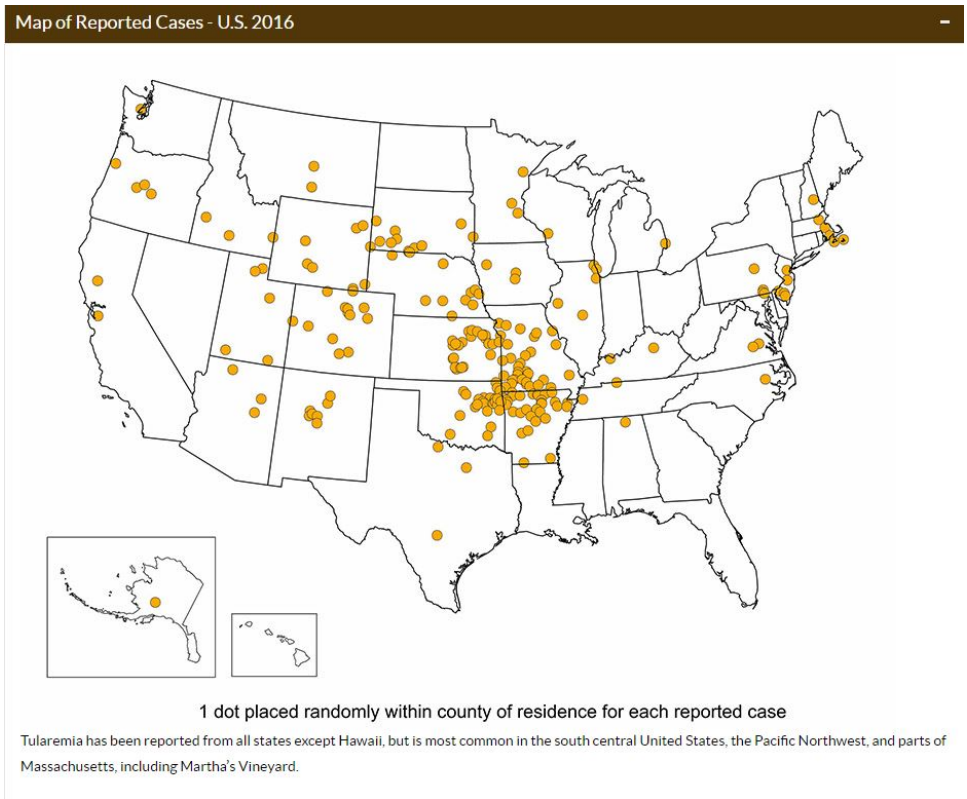


FIGURE 3: TULAREMIA CASES IN THE U.S. SINCE 2016 (CDC 2017).

Tularemia is not common in the ceded territories, with 13 cases being reported since 2016.[16] However, if left untreated[17], tularemia can become serious or life-threatening. Death from this disease is relatively rare, but typically occurs from secondary complications, such as pneumonia (Conover and Vail 2015). Harvesters interested in rabbits or hare are at an increased risk of exposure to tularemia.

Bacterial Pathogens Not Reasonably Likely to Occur

Highly-pathogenic avian influenza (bird flu), bovine tuberculosis, brucellosis, and Eastern Equine Encephalitis are diseases that have been documented in the ceded territories, but do not pose a reasonable human health hazard based upon their rarity of illness to humans in the area. For more information on these diseases, please see Appendix 2.

[16] Since 2016, five cases have been reported in Minnesota, four cases in Michigan, and three cases in Wisconsin (CDC 2018; Conover and Vail 2015, 59).
[17] Tularemia is treated with antibiotics (Conover and Vail 2015).

B. Food Pathogens

C. botulinum Brief	
Habitat: N/A - Becomes a Hazard After Processing	
Condition for Growth: Reduced oxygen-packaged Foods (Canned, Vacuum Packed, Oil Immersed)	
Sources:	
• Soil	• Fecal Contaminated Water
Processes of Concern:	*Examples:*
• Low Acid Canned Foods • Improperly Canned Foods • Vacuumed Packed Foods • Untreated Bulb-in-Oil Mixtures	• Home Canned Meat • Home Canned Vegetables • Fermented Meats • Fermented Fish • Vacuumed Packed Fish/Meat • Vacuumed Packed Vegetables • Untreated Infused Wild leek/Ramp oil

Clostridium botulinum *(C. botulinum)* is a bacteria found naturally in mammal intestines, fish intestines, and in soil (Center for Food Security and Public Health 2017). It is rare for the bacteria to cause illness, however, under the right conditions, it produces spores which create a powerful neurotoxin which can cause serious illness or death. Conditions that encourage the toxin formation are low or no oxygen, low sugar, low salt, low acid, and specific temperature ranges (CDC 2017a). These conditions are typically associated with improperly canned foods (especially home canned vegetables or meats), improperly prepared sausage, improperly fermented meats and fish, garlic in oil, foil wrapped baked potatoes, and improperly thawed vacuum packed foods (Center for Food Security and Public Health 2017; CDC 2017a). The *C. botulinum* bacteria can be killed through cooking but the spores are resistant to normal cooking temperatures. Toxins that are ingested must be treated with anti-toxin medication under medical supervision, as untreated botulism can be deadly. Commercially or home canned foods or vacuumed sealed foods which are bulging may be contaminated, however, foods often show no sign of contamination (Center for Food Security and Public Health 2017).

From 2009 to 2016, the CDC National Botulism Surveillance reported a total of 134 confirmed and probable cases of foodborne botulism, with 8 deaths. The onset of symptoms occurs within 4 hours or up to 8 days after exposure. Botulism symptoms include drooping eyelids, double or blurred vision, slurred speech, dry mouth, swallowing problems, muscle weakens, constipation, and swollen abdomen (Abraham 2012). To treat botulism, medical assistance is quickly needed (CDC 2017a). Recovery may include temporary paralysis or require the use of a ventilator and may take many weeks or months to fully recover (Abraham 2012). Individuals interested in consuming

vacuum packed, canned, or other reduced-oxygen packaged game, fish, or plants may be at an increased risk of exposure to the botulism toxin. Additionally, individuals interested in infusing oil with wild leeks/ramps or fermenting foods in oxygen free environments may be at an elevated risk.

***C. perfringens* Brief**	
Habitat: N/A - Becomes a hazard after processing	
Condition for Growth: Foods kept at temperatures between 41°-139°F for extended periods of time, includes cooked foods.	
Sources:	
• Soil	• Contaminated Water
Processes of Concern:	*Examples:*
• Dishes made with meat and poultry held at temperatures between 41°-139°F for extended periods of time.	• Venison Hot Dish • Smoked Fish/Game • Turkey Gravy

Clostridium perfringens (*C. perfringens*) are foodborne bacteria naturally found in the intestines of humans and animals, which survives only in the absence of air and produces heat resistant spores and toxins. Food sources of *C. perfringens* include meat and poultry, but is also found in soil and can easily contaminate foods through cross-contamination from humans and food contact surfaces. *C. perfringens* spores can survive normal cooking temperatures, and once the food temperature drops into the danger zone (41-139°F), the spores open, releasing bacteria that begin to multiply. *C. perfringens* multiplication occurs rapidly in temperature ranges of 60°-125°F (Division of Public Health 2004). *C. perfringens* is most often associated with large batches of food which are either improperly cooled, reheated, or held for service (CDC 2017a).

On average, one million cases are reported annually in the United States, making it a common foodborne illness. After consumption the bacteria can produce a toxin in the intestines. The onset of symptoms occurs six to 24 hours after exposure and typically begins suddenly lasting less than 24 hours. Symptoms include abdominal cramps and diarrhea, however, fever and vomiting are not a common symptoms (CDC 2017a). Individuals interested in consuming game, birds, or fish from dishes made in large batches may be at an elevated risk of exposure to *C. perfringens* bacteria.

B. cereus Brief	
Habitat: N/A - Becomes a hazard after processing	
Condition for Growth: Foods kept at temperatures between 41°-139°F for extended periods of time, includes cooked foods.	
Sources:	
• Soil	• Fecal Contaminated Water
Processes of Concern:	*Examples:*
• Dishes made with meat and poultry held at temperatures between 41°-139°F for extended periods of time. Especially dishes made with rice, including wild rice.	• Wild Rice Dishes • Cooked Wild Leeks/Ramps • Cooked Wild Beach Peas • Venison Hot Dish • Turkey Gravy

Bacillus cereus, including other *Bacillus* bacteria, are a source of foodborne illness in starchy foods such as cooked rice (including wild rice) and rice dishes. *B. cereus* forms two types of toxins that cause two different illnesses. The CDC estimates that approximately 63, 400 cases of *B. cereus* illness occur annually in the United States. The first toxin is linked to many foods, including turkey and fish, which causes the following symptoms: diarrhea, nausea, and abdominal cramps. The onset time is about six to 15 hours after exposure and lasts about 24-36 hours. The second toxin is most often associated with cooked rice dishes and causes symptoms such as nausea and vomiting. The onset time of this second illness take 30 minutes to six hours and typically lasts about 24 hours. *B. cereus* is best controlled through freezing, refrigeration, good hygiene, and proper cooking (Abraham 2012). Individuals interested in consuming dishes made with turkey, fish, and/or wild rice may be at an elevated risk of exposure to *B. cereus* bacteria.

S. aureus Brief	
Habitat: N/A - Becomes a hazard after processing	
Condition for Growth: Foods kept at temperatures between 41°-139°F for extended periods of time, includes cooked foods.	
Sources:	
• Humans (esp. hair, nose, and throat)	• Fecal Contaminated Water
Processes of Concern:	*Examples:*
• Dehydrated foods. • Foods requiring handling, especially foods that require assembling after cooking. E.g. tuna salad, sandwiches with deli meat.	• Venison Jerky • Smoked Fish

Staphylococcus aureus (*S. aureus*) is a group of bacteria that is commonly found on the skin and nose of approximately 25% people and animals. These bacteria are harmless on healthy people, however, under certain circumstances, it creates a toxin which is heat resistant (CDC 2016b). The CDC estimates approximately 241,188 illnesses, 1,064 hospitalizations, and 6 deaths which occur annually are due to food related *S. aureus* infections (Abraham 2012). The toxin usually causes a reaction within 30 minutes, however, it can take up to six hours after exposure before symptoms develop. Symptoms typically include vomiting, nausea, stomach cramps, and diarrhea lasting for about 24 hours (Food Safety Working Group n.d.). Individuals interested in consuming smoked fish or dehydrated game are at an elevated risk of exposure to *S. aureus* bacteria.

C. Parasitic Pathogens:

Parasites eaten in undercooked fish and meat can present a potential human health hazard.[18] Among parasites, protozoans[19], roundworms (nematodes), tapeworms (cestodes), and flukes (trematodes) are of most concern. Many of these parasites cause only mild illness in humans, however in some cases, severe illness can occur.

Protozoans

***T. gondii* Brief**	
Habitat: Aquatic and Terrestrial	
Sources:	
• Cat Feces (including intestines) • Deer Meat • Contaminated Soil	• Fecal Contaminated Water • Cross-contamination
Primary Carriers:	
• Deer/Venison	

Toxoplasma gondii *(T.gondii)* is a common parasite found in mammals, especially cats (both wild and domestic), and contaminated water throughout the United States. *T. gondii* can contaminate aquatic habitats through fecal contamination such as sewage overflow, after a storm or flood, and animal related agricultural runoff**.** In terrestrial habitats, *T. gondii* is known to infect most warm-blooded animals, but it must infect cats and their wild feline relatives in order to multiply. The most common food sources in

[18] Freshwater fishes are not advised to be consumed raw under any circumstance.
[19]Protozoa are one-celled eukaryotes. Eukaryotes have membrane-bound organelles. Protozoa are in a subkingdom of the kingdom Protista (Yaeger 1996).

the United States are meat from pigs, sheep, and deer (CDC 2017), all of which can be infected by *T. gondii*.

According to the CDC *T. gondii* is the second leading cause of death from foodborne illnesses in the United States, and accounts for an estimated 327 deaths annually. *T. gondii* creates many hospitalizations related to foodborne illnesses, accounting for an estimated 4,428 hospitalizations annually. Symptoms occur within one to three weeks of exposure, and while symptoms in healthy people are uncommon, they can include enlarged lymph nodes in the head and neck, mild illness with fever, and rarely eye disease (CDC 2017).

Pregnant women who become infected with *T. gondii* soon before becoming pregnant or during pregnancy, can pass the infection on to the baby. Infected newborns do not always show symptoms, but newborns with symptoms may have serious brain or eye damage. Infants may not exhibit symptoms at birth but can develop symptoms, such as blindness or mental disability, later in life (CDC 2017).

T. gondii can be limited, but not necessarily destroyed, by freezing temperatures. Freezing meat at 0°F (home freezer temperature) for several days can decrease the chance of infection. (CDC 2017) Harvesters interested in plants eaten raw, white-tail deer, fish or waterfowl that may be in fecal contaminated water are at the most risk of exposure to the *T. gondii* parasite.

***Cryptosporidium* Brief**	
Habitat: Aquatic	
Sources:	
• Human Feces	• Fecal Contaminated Water • Cross-contamination
Primary Carriers:	*At Risk from Contaminated Water:*
Humans are the primary carrier of *Cryptosporidium spp*. Therefore, the risk is from contact with human feces or fecal contaminated water.	• Fish • Duck • Foods typically eaten raw: o Berries o Wild Leeks/Ramps

Cryptosporidium spp. is a common parasite that can be found in water throughout the United States or surfaces contaminated with fecal matter from humans and wild or domestic animals. Aquatic habitats can be contaminated by fecal matter containing *Cryptosporidium spp*. through flood water or storm run-off, agricultural run-off, or sewage overflow. Aquatic habitats, are a common source of the parasite in humans participating in recreational water activities. In terrestrial habitats, *Cryptosporidium spp*. can be found in fecal matter from humans and animals and in soil. (CDC 2015).

Each year, nearly 748,000 people in the United States become sick with cryptosporidiosis, the illness caused by this parasite, via consuming the parasite (CDC 2015). Populations with weakened immune systems that come in contact with *Cryptosporidium* can potentially encounter life threatening symptoms, as the parasite migrates from the intestines to the liver or lungs (Abraham 2012). Cryptosporidiosis symptoms can vary, as some individuals who come in contact with this parasite will not become ill. For individuals who become ill, the onset of symptoms occurs with two to 10 days and last for one to two weeks, but may last up to four weeks. Cryptosporidiosis symptoms include: stomach cramps, extreme diarrhea, nausea, vomiting, fever, decreased appetite, and headache (May 2010). *Cryptosporidium spp.* is resistant to chlorine, such as bleach, but can be inactivated by boiling untreated water for several minutes (Abraham 2012).

Harvesters interested in plants eaten raw, waterfowl or fish that may be in fecal contaminated water are at the most risk of exposure to the *Cryptosporidium* parasite.

***Giardia* Brief**	
Habitat: Aquatic	
Sources:	
• Animal Feces (including intestines)	• Fecal Contaminated Water • Cross-contamination
Primary Carriers:	*At Risk from Contaminated Water:*
Generally, a concern for only the harvester and any person handling the raw food. Killed by cooking.	• Fish • Duck • Foods typically eaten raw: o Berries o Wild Leeks/Ramps

Giardia lamblia is a relatively common parasite in the ceded territories, typically found in aquatic habitats (MDH 2018; WDHS 2017; Michigan Disease Surveillance System 2018). *Giardia* is transmitted in water or surfaces contaminated with fecal matter from infected animals, including deer, beaver, muskrat, fish, or humans (EPA 2000). In terrestrial habitats, *giardia* can live for weeks or even months in soil, on surfaces, and in fecal matter. Illness due to the *giardia* parasite is called giardiasis and is contracted by consuming contaminated material.

The CDC estimates that approximately 20,305 individuals become ill from giardiasis annually. Some individuals who contract giardiasis will not exhibit symptoms. The onset of symptoms occurs within one to three weeks of infection and can last from two to six weeks (Abraham 2012). Giardiasis symptoms include: diarrhea, flatulence, greasy stool that can float, abdominal cramps, nausea, and/or dehydration (CDC 2015). In

children with severe giardiasis, developmental delays and malnutrition can occur (CDC 2015). Harvesters interested in plants eaten raw, waterfowl or fish that may be in fecal contaminated water are at the most risk of exposure to the *Giardia* parasite.

Nematodes (Roundworms)

Nematodes Brief	
Habitat: Aquatic	
Sources:	
• Fish	• Fecal Contaminated Water • Cross-contamination
Primary Carriers:	*At Risk from Contaminated Water:*
Roundworms are most often associated with raw and under cooked fish. Typically killed by cooking or freezing.	• Fish

Nematodes, commonly known as roundworms, are a class of parasite found in both aquatic and terrestrial habitats worldwide. Roundworms in food are most typically associated with fish, however, they can be found in soil, and the feces of wild and domestic animals. Some examples of fish roundworms include: *Anisakis spp.*, *Pseudoterranova spp.*, and *Eustrongylides spp.* Each can be killed by proper cooking or freezing (DHHS 2011).

Once consumed, roundworms can embed within the wall of the human stomach or intestine (DHHS 2011; Abraham 2012). The frequency of diseases caused by roundworms in the United States is unknown, as it is not a reportable disease by the

CDC's standards and is therefore not required to be reported. The onset of symptoms occurs within 24 hours, or can take up to two weeks, and last about 3 weeks. Symptoms include: abdominal pain, nausea, vomiting, and diarrhea. In some cases, the roundworms can cause an allergic reaction (Abraham 2012). Individuals interested consuming in fish are the most risk of exposure to roundworm parasites.

Cestodes (Tapeworms)

Diphyllobothrium Brief	
Habitat: Aquatic	
Sources:	
• Animal Feces (including intestines) • Bird Feces •	• Fecal Contaminated Water • Cross-contamination
Primary Carriers:	*At Risk from Contaminated Water:*
Diphyllobothrium spp. is most often associated with raw and under cooked fish. Typically killed by cooking or freezing.	• Fish • Duck • Foods typically eaten raw: o Berries o Wild Leeks/Ramps

Diphyllobothrium spp. is a parasite found in fish throughout the United States, more commonly in freshwater fish than saltwater, which can infect humans, mammals, and birds which consume fish. Sometimes called "broad tapeworms" or "fish tapeworms", these are the largest tapeworms that can infect humans, potentially infecting 100-200 people annually in the United States (Abraham 2012; CDC 2017).

Diphyllobothrium is considered a minor public health concern and records of infection are no longer kept. Infections occur when the larvae are consumed, which can be found in the meat or viscera of fresh fish or of raw or undercooked fish. The onset of symptoms typically occurs 15 days after exposure, however, without symptoms or treatment, the parasite can live for years in the body. Symptoms are typically mild and include: abdominal discomfort, changes in appetite, and diarrhea. Typically, patients become aware of an infection when pieces of the tapeworm appear in stool (Abraham 2012). Individual interested in consuming fish are the most risk of exposure to *Diphyllobothrium spp.* parasites.

E. multilocularis Brief	
Habitat: Terrestrial	
Sources:	
• Fox Feces	• Fecal Contaminated Water • Cross-contamination
Primary Carriers:	*At Risk from Fox Fecal Contact:*
Fox are the primary carrier of *E. multilocularis.* Therefore, the risk is from contact with fox feces. Killed by cooking.	• Foods typically eaten raw: o Berries o Wild Leeks/Ramps

Echinococcus multilocularis is a type of tapeworm whose prevalence in North America is slowly increasing. *E. multilocularis* is a potential concern to people eating berries or mushrooms which have been contaminated with fox feces (Poulle et al. 2017).

Trematodes (Flukes)

Trematodes Brief	
Habitat: Aquatic	
Sources:	
• Fish • Deer Liver	• Cross-contamination
Primary Carriers:	
Fish can contain a variety of Flukes which can impact human health. Flukes are killed by cooking.	Deer can carry liver flukes which can impact human health. Deer liver with flukes may have secondary bacterial infection (WDNR 2012).

Trematodes can come from fish or deer. In human, intestinal flukes from fish can cause diarrhea and abdominal pain. Some flukes can migrate to the heart and central nervous system, causing damage to these systems. Some examples of intestinal flukes in fish include: *Heterophyes spp., Metagonimus spp.,* and *Nanophyetes salmincola*.

In humans, liver and lung flukes from fish can damage respective vital organs. *Paragonimus spp.* is an example of a lung fluke found in fish. Some examples of liver flukes found in fish include *Chlonorchis sinensis* and *Opisthorchis spp.* (DHHS 2011).

Liver flukes in deer are flat, grayish-purple, and resemble leeches or "bloodsuckers." People can consume meat from white-tailed deer infected with liver flukes, however, it is recommended to avoid consuming the liver of these deer. Deer liver flukes may be infected with a bacteria caused after the fluke attaches to the liver, which may impact human health (WDNR 2012).

D. Viral Pathogens

West Nile Virus Brief	
Habitat: Terrestrial	
Sources:	
• Mosquitos	• Birds
Primary Carriers:	*At Risk from Food:*
• Mosquitos • Birds *Current research suggest that harvesters are at the greatest risk due to greater exposure, especially to mosquitos carrying the disease.	• Mothers who are breastfeeding can pass the virus on to the breastfeeding infants. • Little or no evidence for foodborne risk to consumer.

West Nile Virus (WNV)[20] is transmitted mainly by mosquito bites.[21] Mosquitoes become infected with West Nile Virus by first biting infected animals, like birds,[22] white-tailed deer[23], and black bears. Viral antibodies have been found in Wisconsin black bears and medium-sized mammals,[24] which indicates that these animals have been exposed to the virus (Katz et al. 2007). Some species of ticks have also been found to carry the virus. It has been determined that West Nile Virus can survive in salt water at 30°F for 4 days, however, there is no documented cases of waterborne transmission to humans (Lund et al. 2017).

Waterfowl, such as wood ducks and mallards, live in habitats where they may be subjected to higher mosquito populations. Although most ducks do not appear to play a large role in disease transmission[25], or are documented in largescale West Nile Virus

[20] A virus is a microscopic agent that infects normal, host cells, and multiplies exponentially (EOL 2018). WNV is comprised of a single, linear strand of RNA (genetic material) called *Flavivirus* (Conover and Vail 2015, 327).
[21] *Culex* mosquitoes are mostly responsible for transmitting WNV (Conover and Vail 2015, 339).
[22] American crows (*Corvus brachyrhynchos*), common ravens (*Corvus corax*), and blue jays (*Cyanocitta cristata*), particularly, are susceptible to massive die-offs during WNV outbreaks (CDC 2018). All three are members of the Corvidae family. Some hawks, owls, songbirds, and other non-game bird species are also susceptible to WNV mortality (CDC 2017).
[23] In the United States, there has been one documented WNV mortality in a white-tailed deer in Georgia (Miller et al. 2005).
[24] Raccoons, opossums, red fox, and coyote were all found to have WNV-specific antibodies. Viral antibodies are blood proteins formed as an immune response to a virus (Docherty et al. 2006)
[25] One study found that wild mallards, American widgeon, northern pintail, and wood ducks tested only at 10 percent for WNV antibodies, indicating that these species are not as exposed as initially expected (Hoffmeister et al. 2016).

mortalities, they may serve as a possible reservoir for the virus to infect humans and other types of birds (Hofmeister et al. 2015 ; 2016).

Wild turkeys do not appear to be uniquely susceptible to death from West Nile Virus (Swayne et al. 2000), however, some wild turkeys that have died tested positive for the virus (CDC 2017; Zhang et al. 2006). Recent research from Pennsylvania has indicated that West Nile Virus may be stressing ruffed grouse populations within the state (Stauffer et al. 2018).[26]

FIGURE 4: COMPARISON OF BREAST MEAT OF GROUSE SUFFERING FROM WEST NILE VIRUS (LEFT) AND HEALTHY GROUSE BREAST MEAT (RIGHT).

PHOTO CREDIT: MILWAUKEE JOURNAL SENTINEL

Currently, no evidence exists that people can become infected with West Nile Virus from consuming infected meat which is properly cooked. However, harvesters cleaning infected game can obtain the virus from blood-to-blood contact with the infected animal (NH DHHS 2006).

Eight out of ten people infected have no symptoms, with approximately one in five infected people develop flu-like symptoms, including fever. Recovery time can range from several weeks to months. Rarely, people infected with West Nile Virus can develop serious complications of the central nervous system, including brain or spinal cord swelling, which sometimes results in death (CDC 2018).

[26] State natural resources agencies present in the ceded territories will be collaborating in the fall of 2018 to examine possible population-level effects of the West Nile Virus on ruffed grouse. (MI DNR 2018).

Pregnant women infected with West Nile Virus can transfer the virus to their developing child. Additionally, breastfeeding mothers can transfer the virus to their nursing child, however, this is not common (Conover and Vail 2015, 337). Harvesters interested in duck, fish, wild rice, wild leeks/ ramps, or prefer to hunt in areas where mosquitoes are abundant may be at an elevated risk of exposure to West Nile Virus.

Norovirus Brief	
Habitat: Aquatic and Humans Contact	
Sources:	
• Human Feces	• Fecal Contaminated Water • Cross-contamination
Primary Carriers:	*At Risk from Contaminated Water:*
Humans are the primary carrier of Norovirus and can easily spread from food handler to prepared food.	• All Identified traditional foods are at risk due to the nature of this virus. Harvesters may contract the virus from contaminated water.

Norovirus is a highly contagious virus which is transmitted when a small amount of feces or vomit contaminate water or food (CDC 2018). Norovirus can enter aquatic habitats through sewage overflow from a flood or contaminated storm water. Norovirus is the leading cause of vomit and diarrhea from acute gastroenteritis[27] in the United States; causing between 19 and 21 million cases, 56,00 to 71,000 hospitalizations, and 570 to 800 deaths annually (CDC 2018). Individuals that are ill or have recently been ill are known to transmit the virus, which enter the body through consumption of contaminated material (NRAEF 2012).

The onset of Norovirus symptoms occur within 24 to 48 hours and include vomiting, diarrhea, nausea, and abdominal cramps, generally occurring a few hours after exposure (NRAEF 2012). Symptoms last from 12 to 60 hours in healthy people and 72 to 96 hours in hospitalized patients, immunocompromised, and elderly populations. Harvesters interested in plants eaten raw, waterfowl or fish that may be in fecal contaminated water are at the most risk of exposure to Norovirus.

[27] Acute gastroenteritis is inflammation of the stomach and intestines.

Hepatitis A Brief	
Habitat: Aquatic and Humans Contact	
Sources:	
• Human Feces	• Fecal Contaminated Water • Cross-contamination
Primary Carriers:	*At Risk from Contaminated Water:*
Humans are the primary carrier of Hepatitis A and can easily spread from food handler to prepared food.	• All Identified traditional foods are at risk due to the nature of this virus. Harvesters may contract the virus from contaminated water.

Hepatitis A is a contagious virus that affects the liver which is transmitted by fecal matter from individuals whom have contracted the virus. Hepatitis A can enter aquatic habitats through sewage overflow from a flood or contaminated storm water. Unfortunately, it only takes a little bit of the Hepatitis A virus to make someone ill (NRAEF 2012). Hepatitis A is typically associated with eating prepared, ready–to-eat (RTE)[28] foods, however it is important to note that this virus is not destroyed by typical cooking temperatures during the preparation process (CDC 2018).

Hepatitis A symptoms include: mild fever, general weakness, nausea, abdominal pain, and jaundice. The onset of symptoms typically occurs about 4 weeks after exposure and can last up to 2 months for most people and 6 months for 10-15% of people. Children under the age of 6 do not generally show symptoms once infected (CDC 2018). Unlike many of the previously mention pathogens, a vaccine is available to prevent the Hepatitis A virus. Harvesters interested in plants eaten raw, waterfowl or fish that may be in fecal contaminated water are at the most risk of exposure to the Hepatitis A virus.

E. Prions

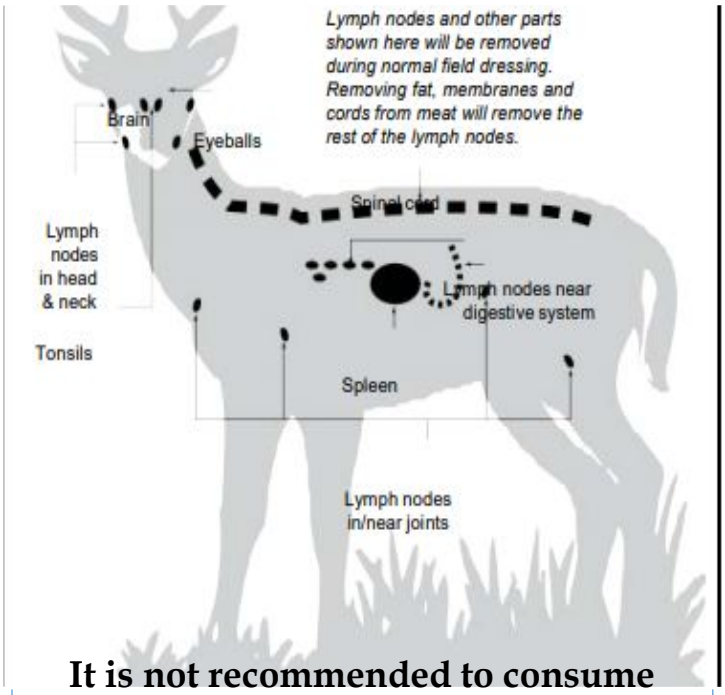
Chronic Wasting Disease (CWD) is the only prion disorder found within the ceded territories. A prion is an atypical, misfolded variety of a protein (Botzler and Brown 2014, 358). Prion disorders belong to a family of diseases called transmissible spongiform encephalopathies, or TSEs. TSEs are progressive disorders that are characterized by degeneration of the nervous system, including the brain. These types of diseases impact both humans and animals. TSEs have long incubation periods and cause the brain to have sponge-like holes in very advanced cases (CDC 2018).

[28] Ready-to-eat foods are foods that do not need cooking or have already been cooked and are ready for consumption.

Chronic Wasting Disease (CWD) is a fatal, prion disorder impacting wild and captive white-tailed deer, elk, and moose[29] (CDC 2018). Please see Appendix 3 for a map showing disease prevalence in the ceded territories. Prions tend to concentrate in tissues of the brain, lymph nodes, spinal cord, and spleen (Wisc. DPH 2018)[30] which are shed into the environment by infected animals through blood, saliva, feces, and urine (Geist et al. 2017, 2; CDC 2018; Mathiason 2015).[31]

A deer infected with CWD may not show any symptoms for several years. In time, CWD causes damage to the brain and other nervous system tissue. This results in significant behavioral changes, including isolation, head lowering, and repetitive walking patterns. Deer with advanced CWD become unusually thin, are constantly thirsty, copiously drool, and often grind their teeth. Elk with CWD have been known to become very nervous and hyperactive (CWD Alliance 2018).

The biological pathogens of CWD are not easily destroyed. The pathogens can remain present even after



It is not recommended to consume any meat from animals known to be infected with CWD (CDC 2018; CWD Alliance 2018).

FIGURE 5: CWD CONCENTRATES IN BRAIN, LYMPH NODES, SPINAL CORD, AND SPLEEN TISSUES. IN PARTICULAR, LYMPH NODES NEAR AND IN JOINTS ARE LIKELY TO BE ENCOUNTERED DURING PROCESSING (WISC. DPH 2018).

[29] The scientific names for white-tailed deer, elk, and moose, respectively, are *Odocoileus virginianus, Cervus canadensis, and Alces.*

[30] Prion numbers increase within a host, but do not multiply directly. Instead, prions cause the host's ordinary cell proteins to fold abnormally (Botzler and Brown 2014, 358).

[31] CWD is a highly infectious disease that persists on contaminated areas, in the soil, on plants, after passing through the digestive systems of CWD infected animals, and even after undergoing wastewater treatment (Hawkins et al. 2015; Johnson et al. 2007; Nichols et al. 2015; Hinckley et al. 2008; Geist et al. 2017,4).

treatments with common disinfectants, high temperatures, freezing, dehydration, and radiation (Geist et al. 2017, 2; Hughson et al. 2016; Brown et al. 2000).[32] Also, CWD prions can remain infectious in soil, bind to the outside of some plants, and even possibly be taken into plants via root systems (Geist et al. 2017, 4; Nichols 2016; Johnson et al. 2007).

To date, CWD infection in humans has not been found. Currently, research is conflicted about whether the disease is potentially transmissible to humans (Marsh et al. 2005; Waddell et al. 2018). Several studies have linked CWD in other mammals through eating infected meat, which indicates that the species barrier is able to be crossed by the pathogen (Geist et al. 2017, 3). However, other studies have contradicted this claim (Zabel and Ortega 2017; Race et al. 2018).

Due to this absence of knowledge, international, federal, and state agencies recommend that CWD positive meat be properly disposed of rather than consumed (Mathiason 2015; WDPH 2016; CDC 2018).[33] Additionally, human health impacts are not known concerning potential CWD exposure to small amounts of prions through eating contaminated plants or fungi in CWD-infected areas (Nichols 2016; Pritzkow et al. 2015).

Female White-tailed Deer

Photo credit:

Linda Freshwater Ardnt

It is believed among the Ojibwe that all animals, just like people, need to eat certain healing plants in order to maintain their overall health. CWD may be an indicator that white-tailed deer are not consuming enough white sage.[34] White sage on the ceded territories has declined over the years, mainly due to habitat loss (Enright and Doran n.d.). Tribal elders are encouraging tribal members to plant white sage in CWD-infected areas for wild deer. Sage grows best in prairies with dry to moderately moist soils (Meeker et al. 1993, 66).

[32] Biological inactivation by lichens, cleaning with relatively-inert hypochlorous acid (HOCl), and composting infectious materials all may help reduce infectivity of CWD prions. However, these methods do not completely eradicate them (Hughson et al. 2016; Rodriguez et al. 2012; Xu et al. 2014).
[33] Agencies include the World Health Organization, U.S. Centers for Disease Control and Prevention, the Wisconsin Division of Public Health, and other state natural resource agencies.
[34] The scientific name for white sage is *Artemisia ludoviciana* (Meeker et al. 1993, 66).

Many federal and state agencies have created herd certification plans for CWD management and monitoring on captive deer farms. Many of these regulations are intended to keep wild and captive deer separated to prevent the spread of disease from captive to wild deer populations and the spread of the disease between one captive deer herd to another.

In addition, Minnesota, Wisconsin, and Michigan have adopted regulations to restrict the movement of deer carcasses from known CWD-infected areas and carcass disposal regulations. There is more data that needs to be collected to understand the impact of these regulations on the spread of CWD.

Currently, the risk of CWD to humans is not fully understood. Other prion diseases, such as Mad Cow Disease (bovine spongiform encephalopathy), have caused disease in humans, therefore, it is reasonable to suspect that CWD may potentially have a similar impact to humans. Prions are very difficult to destroy during normal cooking methods and may be hard to remove from contaminated hands or clothing (Geist et al. 2017, 2; Hughson et al. 2016; Brown et al. 2000).

Sighting a Sick Deer:
If you observe or shoot a deer that appears to be sick, immediately report it to the nearest tribal registration clerk, GLIFWC biologist, conservation warden, or your local state DNR biologist (GLIFWC 2017).

CWD is a growing concern, yet, many counties in ceded territories have not been affected to date. Michigan, Minnesota, and Wisconsin's Natural Resource departments regularly update CWD affected counties list within their respective states.

Regulations

Regulations regarding CWD vary by state and jurisdiction. However, regulations generally apply to both deer and elk, where applicable, as both animals are affected by CWD and are in the cervid family. A compilation of regulations Michigan, Minnesota, Wisconsin, Colorado, Wyoming, and Canada has been provided in Appendix 4. The compilation includes regulations regarding the following for each jurisdiction:

- Baiting during hunting
- Deer farm fencing requirements
- Hunting preserve fencing requirements
- Intrastate movement of carcasses from CWD affected areas/management zones
- Action if CWD is verified – deer farm
- Action if CWD is verified – hunting preserve
- Testing requirements for hunting preserves

- Testing requirements for hunters in CWD Units
- Requirements for donated deer from non-CWD Units
- Requirements for donated deer from CWD Units

A table of deer farming regulations in Michigan, Minnesota, and Wisconsin has been provided in Appendix 5.

F. Allergens

Allergens are agents in food that cause or trigger an allergic reaction. There are more than 160 foods which can cause allergic reactions in people with food allergies, however, the FDA identifies the eight most common allergic foods. According to the FDA, these foods and their derivatives are implicated in approximately 90% of allergic reactions. The FDA requires food producers to specially label foods and or food ingredients containing one or more of these allergens (Nutrition 2017).

The eight most common allergens are:

- Milk
- Eggs
- Fish
- Crustacean shellfish
- Tree nuts
- Peanuts
- Wheat
- Soybeans

Of the 14 identified traditional foods, only three are major allergens: whitefish, walleye, and hazelnuts. It is important to note that allergic reactions can be triggered by foods that are not on the list. There are two additional foods from the identified traditional foods that are reasonably likely to cause allergic reactions: strawberries and morel mushrooms (fungi). These reactions are not as common as the reactions from the most common allergen above.

2. Chemical Hazards

Chemical hazards are non-biological contaminants which develop in or are added to foods that cause illness. These contaminants can be a naturally occurring chemical within the food, unnaturally occur due to tissue uptake from the environment, or are added to foods. The following sections outline natural toxins, heavy metal contaminants, pesticides, and industrial chemicals.

TABLE 5: AN OVERVIEW OF CHEMICAL HAZARDS OF IDENTIFIED TRADITIONAL FOODS. TO INCLUDE NATURAL TOXINS, HEAVY METALS, PESTICIDES AND INDUSTRIAL CHEMICALS.

Chemical Hazard Risk Overview	Deer	Rabbit/Hare	Duck	Turkey	Whitefish	Walleye	Berries	Wild Ramps	Beach Pea	Hazelnut	Morel Mushroom	Wild Rice	Maple Syrup	Berry Jam/Jelly	Animal Fats	Venison Jerky
NATURAL TOXINS																
Sambunigrin							X									
BOAA									X							
HEAVY METALS																
Lead	X		X	X					U		X	U	X			X
Mercury			X	X	X	X										
Arsenic							U				X	U				
Copper			X				X									
Zinc			X				X									
Manganese							X									
Cadmium																
Aluminum			X													
PESTICIDES & INDUSTRIAL CHEMICALS																
Toxaphene					X											
Dioxins & Furans—					X											
PCBs					X											
PFAs																
PFOs																
	Deer	Rabbit/ Hare	Duck	Turkey	Whitefish	Walleye	Berries	Wild Ramps	Beach Pea	Hazelnut	Morel Mushroom	Wild Rice	Maple Syrup	Berry Jam/Jelly	Animal Fats	Venison Jerky

'U' indicates unknown risk. See the companion document for more information.

A. Natural Toxins in Traditional Foods

Elderberries and wild beach peas contain natural plant toxins which could pose a human health hazard. Negative health effects would be exaggerated by consuming great quantities over a period of time. Moderate consumption and proper preparation are key to preventing hazardous impacts to human health.

Elderberries seeds, leaves, and twigs from elderberries[35] contain a cyanide-producing glycoside (CGG) called sambunigrin. In one study, elderberries at the bottom of hills were found to have slightly less CGGs in its berries overall, when compared to berries collected at the tops of hills (Senica et al. 2017). A toxic alkaloid, sambucine, is also present in unripe fruit and in its flowers.

The human digestive system can break down very small amounts of CGGs, however, overconsumption of elderberry seeds would result in symptoms of nausea, vomiting, and diarrhea. The sensitivity of people to these natural toxins will vary from person to person (Atkinson and Atkinson 2002).

Wild Beach Pea[36] Excessive and repeated consumption of *Lathryus spp.* leads to a disorder known since ancient times called lathyrism. This disease is caused by a neurotoxin, called beta-N-oxalyl-amino-L-alanine (BOAA). Lathyrism is characterized by painful muscle spasms, followed by permanent paralysis of the legs and buttocks in humans, and frequent urination. Young adult males are the most affected by excessive consumption of this neurotoxin. The onset of the illness can take anywhere from a few weeks or months from pea overconsumption. Lathyrism is also known to impact animals, causing animals to become crippled in extreme cases (Alm 2015; Manna et al. 1999).

From an ethnographic, or cultural, standpoint, the safety of beach pea consumption remains debated (Alm 2015; Chavan et al. 2003). Nutritionally speaking, however, the literature points toward the safety of beach pea as a wild, traditional food when consumed in moderation as part of a balanced diet. BOAA (neurotoxin) analysis of beach peas closely resembled that of the common soybean (Chavan et al. 2003).

[35] *Sambucus canadensis* and *Sambucus racemosa* are the two species of elderberries within ceded territories, with S. *canadensis* being more common overall (Meeker et al. 1993).
[36] *Lathyrus japonicus* is the scientific name for wild beach pea.

B. Heavy Metals

Heavy metals and metalloids are chemical elements; elements are chemicals that cannot be broken down further. Most heavy metals possess a high density or high weight compared to its volume. Once in the environment, heavy metals do not break down. Heavy metals are often released from human activities, such as manufacturing and mining (Lenntech 2018). Lead, a common metal used in gun ammunition, can contaminate game meat during the harvesting process. White-tailed deer, wild rice, maple syrup, rabbits, hare, whitefish, walleye, wild turkey, duck, beach pea, and some berries are all traditional foods that may be impacted by one or more heavy metals in some fashion.

Heavy metals are often toxic to the human body at relatively low concentrations, however, some metals are needed in trace amounts for proper body function, like copper and zinc. Others, like lead and cadmium, do not have a known nutritional function and can create problems when introduced into the body at all. Unfortunately, most of these elements bioaccumulate in tissues and biomagnify in the food web. Increasing concentrations of a harmful substance found within an individual is known as "bioaccumulation." When this happens when animals eat other animals, it is called "biomagnification" or "bioamplification (USGS n.d.)." Once in the human body, these elements often take a while to be reduced in concentration.

Human health effects from heavy metals exposure vary widely among specific elements and may be different depending on the length of exposure. However, impacts to the nervous system, liver, and kidneys are often associated with chronic, or long-term, heavy metals toxicity. Chronic exposure is much more likely to occur than high dose, acute (short-term) exposure from eating identified traditional foods. Certain metals, like mercury and lead, impact developing children, infants, and small children, more so than adults (ATSDR 2018).

Lead Brief	
At Risk Foods:	
• Deer/Venison (esp. ground) • Turkey • Wild Beach Pea — under review	• Morel Mushrooms • Maple Syrup • Wild Rice— under review
Maximum Allowable Limit (excerpt):	
0.1 ppm imported dried fruit	United States
0.5 ppm maple syrup	Canada
0.1 ppm poultry meat	European Union
0.3 ppm fish	World Health Organization

Lead is a naturally-occurring metal that creates a variety of health problems at even very low doses. [37] The nervous system, which includes the brain, is most damaged by lead exposure. This element becomes mobilizes primarily by human activities, such as manufacturing, mining, smelting, and the burning of fossil fuels (see Appendix 6 for a map of metallic mines in the ceded territories). Lead is commonly found in a wide variety of products, including fishing sinkers, weights, gun ammunition, and solder in spigots and valves. Once mobilized, lead persists and strongly binds to soil (Wani et al. 2015, 55; ATSDR 2018).

> Parts per million (ppm) basically means "one part in one million." It is also expressed as mg/L or mg/kg using the metric system. Parts per billion (ppb) is "one part in a billion." Ppb can be converted into ppm by moving the decimal place three places to the left to make the value smaller. For example, 1 ppb is also 0.001 ppm, or 0.001 mg/kg (Satterfield 2004).

For lead, no safe level of exposure exists (WHO 2018; EFSA 2010). Prevention and reduction of lead exposure remains the best course of action to reduce negative health impacts (US CDC 2017). Infants and young children are rapidly growing, making young children more sensitive to low concentrations of lead (Wani et al. 2015, 55; ATSDR 2018).[38] Lead exposure in children can cause devastating, lifelong impacts, such as brain damage that causes lowered I.Q. scores, violent outbursts, and learning problems (Wani et al. 2015, 55; ATSDR 2018).

Chronic lead exposure in adults can also result in noticeable health impacts, such as decreased mental function, anemia, and high blood pressure (Wani et al. 2015; ATSDR 2018). Kidney problems caused by lead can be worsened in people with Type 2 diabetes, a health issue that disproportionately affects Native Americans (Watson et al. 2009, 26). Ongoing lead exposure was also found to reduce male fertility (Wani et al. 2015).

High lead exposure may cause pregnant women to miscarry (Wani et al. 2015, Watson et al. 2009, 27). In both children and adults, severe brain and kidney damage from extreme lead exposure levels is linked to death (Wani et al. 2015; ATSDR 2018). However, the consumption of lead through traditional foods is unlikely to cause dramatic, acute health effects. Subtle impacts are more likely to occur from low dose, chronic ingestion of lead.

[37] The chemical symbol for lead is Pb (LANL 2018).
[38] Behavior, such as mouthing and crawling, puts infants and children in a position of potential increased exposure to lead (Watson et al. 2009).

Unfortunately, lead still has a wide variety of applications due to its abundance within the environment and its unique physical properties.[39] When products made with lead, such as gun ammunition and fishing line weights, are left or lost in the environment, it creates a perpetual cycle of contamination within that area (Watson et al. 2009).

TABLE 6: DIFFERENT ROUTES OF LEAD EXPOSURE POTENTIALLY IMPACTING IDENTIFIED TRADITIONAL FOODS.[40]

Identified Ojibwe Traditional Foods Potentially Impacted by Lead					
			Routes of Exposure		
			Ammo	*Processing Equipment*	*Tissue Uptake*
Common Name	**Scientific Name**	**Ojibwe Name**			
Big/Small Game					
White-tailed Deer	*Odocoileus virginiatus*	waawaashkeshiwi-wiiyaas	x		
Snowshoe Hare	*Lepus americanus*	waabooz	x		
Cottontail Rabbit	*Sylvilagus floridanus*	manidoo-waabooz	x		
Ducks/divers					
Scaup/Bluebill	*Aythya affinis; A. marila*	zhiishiib	x		x
Ring-necked Duck	*Aythya collaris*	dagwaagishiib	x		x
Duck/dabblers					
Blue-winged Teal	*Anas carolinensis*	kwishkwishibens	x		
Green-winged Teal	*Anas discors*	ozhaashkwinigwi-shibens	x		
Mallard	*Anas platyrhynchos*	ininishib	x		
Wood Duck	*Aix sponsa*	zii'amo	x		
Upland					
Wild Turkey	*Meleagris gallopavo*	mizise	x		
Ruffed Grouse	*Bonasa umbellus*	bine	x		
Plants					
Wild Rice	*Zizania palustris*	manoomin			x
Wild Beach Pea	*Lathyrus japonicus*	anijiimin (pea)			x
Fungi					
Morel Mushroom	*Morchella esculenta*	wazhashkwedoons (mushroom)			x
Value Added					
Maple Syrup	*Acer saccharum*	ziinzibaakwadwaaboo		x	
Venison Jerky	*Odocoileus virginiatus*	baatewaawaashkeshiwi-wiiyaas	x		

[39] Some unique physical properties include high density, malleability, corrosion resistance, and poor conductivity (Wani et al. 2015, 55).
[40] Ojibwe names were determined using the Ojibwe People's Dictionary from the University of Minnesota. These names are to be reviewed by GLIFWC's Advisory and Guidance Input Group of Elders in the Winter of 2019.

Routes of Exposure[41]

Ammunition

The most common cause of lead contamination in wild game meat occurs when hunters use lead ammunition shot, bullets, or slugs during an animal's harvest. Elevated exposure to lead from consuming game meat harvested with lead ammunition is widely documented as a potential human health hazard (Pain et al. 2010; MI DCH 2010; Fachehoun 2015; Hunt et al. 2009; Watson et al. 2009). In contrast, when rabbits are snared, the potential for lead exposure in the meat is virtually eliminated since there is no potential for ammunition containing the toxic metal to be used.

Lead fragments found in a whole cut of venison, weighing 982 mg.

Photo credit: Minnesota Department of Agriculture

Particular groups, including families with hunters, indigenous groups, and food pantry recipients, are more at risk for higher dietary exposure to lead (EFSA 2010; Knutsen et al. 2015; Hunt et al. 2009; ND DOH 2008). Blood lead levels are consistently higher in

[41] Lead can get deposited through the air by floating dust particles from fuel exhaust. The United States banned the use of lead as an additive in gasoline for passenger vehicles in 1996 (U.S. EPA 2016; Newell and Rogers 2003,6). Plants near roadways are exposed to lead contamination through accumulation of this dust. However, lead contamination of plants from road dust is not reasonably likely to accumulate to levels affecting human health (Trombulak and Frissell 2000, 22).

these groups than in the general population (Hunt et al. 2009; Watson et al. 2009; Johansen et al. 2006).

However, in a joint study by Fond du Lac Tribe and the Minnesota Department of Health (2014), only 3 out of 490 people tested had blood lead levels which exceeded the Level of Health Concern for lead, which is 5 micrograms/deciliter. Two of these respondents were likely exposed to this metal from their workplaces. In general, there did not appear to be a relationship between the consuming of game and blood lead levels. The exposure source remained largely undocumented, nonetheless, as information regarding a timeline of people consuming game meat in relation to giving blood samples was not gathered from participants.

Ammunition components separate differently depending on a variety of factors, including firearm design, metal composition, weight, and size. Rifle ammunition shatters more than shotgun ammunition, and muzzleloader bullets, respectively. Inexpensive lead bullets commonly used by center-fire rifles splinter far more than leaded-core and all-copper bullets (Cornicelli and Grund 2008). It is important to note that lead cannot be washed off a harvested animal. Rinsing the abdominal cavity may actually spread microscopic lead shards throughout the carcass, increasing the possibility of contamination (Grund et al. 2010).

Unfortunately, the type of ammo used also impacts the bioavailability of lead, meaning the smaller a lead fragment is, the easier it can be absorbed into the body (Barltrop and Meek 1979; Hunt et al. 2009). Additionally, the cooking method used to prepare harvested meat impacts the availability of lead for uptake into the body. Particularly in acidic recipes, lead from ammo in game meat becomes more accessible to the body after it is cooked (Mateo et al. 2011).

The cut of deer meat can also influence the amount of lead in venison. Ground venison is tested most often by agencies because it is a common way to consume white-tailed deer. However, the grinding processes mixes lead-contaminated meat with previously uncontaminated meat. Michigan Department of Community Health (2010) examined butterfly loins for lead, and found none. Lead ammunition placement would largely influence the presence of lead even in whole cuts of venison.

Deer jerky would contain the same levels of lead the raw meat originally contained. Ground meat jerky recipes would be at greater risk for higher lead levels than muscle jerky, if the deer was shot with a lead bullet.

Lead deposited in the environment by ammunition also likely harms animals, including those culturally significant to the Ojibwe people, such as the bald eagle. Northern-dwelling eagles scavenge more often during the cold, winter months. As scavengers,

bald eagles consume deer gut and organ (offal) piles, some of which can contain lead ammunition.

In an upper Midwest study, 36 % of offal piles sampled contained lead particles (Warner et al. 2014), and a study of Wisconsin bald eagle deaths found about 15 % died from lead poisoning. Death trends have been found to overlap with state hunting seasons (Watson et al. 2009, 194) with lead exposure rates for bald eagles remain consistently high (Warner et al. 2014, Watson et al. 2009).[42]

Bald Eagle. Photo credit: Linda Albor

Processing Equipment

Maple sap contains on average 1.1 microgram/L (ppb), or 0.001 ppm, of lead (Stilwell and Musante 1996). Lead contamination of **maple syrup** most often occurs through solder, spigots, and pumps containing lead (IMSI 2015).[43] However, any piece of older, lead-bearing processing equipment that the sap comes in contact with can leach into the finished product. This includes equipment, like buckets, pans, tanks, and drums (Willits and Tressler 1937; Stilwell and Musante 1996).

Tissue Uptake

Certain plants may uptake lead from the soil into their tissues (US FDA 2017). **Wild rice** has been documented to uptake elevated amounts of lead in its seeds (Bennett 2000; Pip 1993; Nriagu and Lin 1995). Wild rice in natural stands may potentially contain greater levels of lead compared to paddy-grown wild rice due to surrounding environmental factors (Nriagu and Lin 1995, 225; Pip 1993).

The roots of the grass pea[44] have the ability to uptake large amounts of lead into its roots. **Wild beach peas** are closely related to the grass pea, and are likely to possess this same quality. However, lead levels were not tested in the grass pea pod or seed (Brunet et al. 2008).

Lead uptake into **morel mushrooms** are a potential health hazard for people that consume this identified traditional food (Abdel-Aziz 2016).[45] Lead arsenate, a formerly-used insecticide containing both lead and arsenic, was widely used in commercial apple production. Soils in abandoned orchards still contain high amounts of lead from

[42] 38% of those bald eagles also had liver lead concentrations that suggest lead poisoning.
[43] Leaching of lead into maple syrup occurs more frequently during conditions of higher acidity and temperature (Willits and Tressler 1937; Stilwell and Musante 1996).
[44] The scientific name of grass pea is *Lathyrus sativus* (Brunet et al. 2008).
[45] The scientific name of morel mushroom is *Morchella esculenta*.

previous use of this insecticide. A relationship exists between higher levels of lead in the soil and higher amounts of lead found in morel fruiting bodies (Shavit and Shavit 2010).

Mercury Brief	
At Risk Foods:	
• Duck • Turkey	• Whitefish • Walleye
Maximum Allowable Limit (excerpt):	
1 ppm fish (edible portion)	United States
0.5 ppm; 1 ppm species dependent	Canada
0.01 duck meat	European Union
0.5 ppm; 1 ppm species dependent	World Health Organization

Mercury is a metal that occurs naturally in the environment and takes several forms, including the most toxic version is the organic (carbon-based) form: methylmercury. Methylmercury is most frequently encountered by people that consume a diet high in fish and shellfish. (ATSDR 2018).[46]

Mercury is bound in muscle tissue. It cannot be trimmed or cut off fish.

Inorganic mercury is released into the environment mostly through human activities, such as fossil fuel combustion, mining, metal smelting, waste incineration, and chlorine production. Smaller amounts of inorganic mercury can enter the environment through natural processes, such as erosion and volcanic activity (ATSDR 2018). Globally, it is estimated about 2/3 of mercury mobilized from the Earth's crust is due to the result of human activity (UNEP Chemicals Branch 2008).

Once mobilized, mercury does not break down, with inorganic forms transformed to organic methylmercury by bacteria in water and sediment.[47] The conversion of inorganic mercury to organic methylmercury occurs almost exclusively in aquatic environments. The more total mercury there is in the environment, the more methylmercury that can be created by bacteria (ATSDR 2018).

Developing fetuses, infants, and small children are the most sensitive to the effects of methylmercury exposure. Methylmercury in the mother's body passes to a developing child and can accumulate there. The compound can also be passed to a nursing infant through breast milk. In children, common symptoms of methylmercury exposure

[46] The chemical symbol for mercury is Hg. Methylmercury and metallic mercury vapor both can easily reach the brain (ATSDR 2018).
[47] This is a complex process involving the work of sulfate-reducing bacteria in mainly oxygen-poor conditions.

include: delays in fine motor skills, cognition, and speech and language development (ATSDR 2018).

Advanced methylmercury toxicity symptoms occur differently in adults. Those symptoms which include: clumsiness, muscle weakness, hearing and speech problems, nerve loss in hands and face, problems walking, and changes in vision. Other neurological effects include: depression, anxiety, irritability, problems with memory, shyness, numbness, and tremors. Substantial evidence also exists that methylmercury exposure increases the risk of cardiovascular disease (ATSDR 2018).

High-dose methylmercury exposure can cause permanent brain and kidney damage. However, acute methylmercury toxicity is very unlikely to result from traditional foods consumption.

Fish in the ceded territories have been extensively tested for methylmercury due to the important cultural and dietary role that fish have in Anishinaabe culture. Mercury is bound in muscle tissue, so trimming fat from fish fillets and removing skin do not reduce mercury levels.

Nearly 8,000 **walleye** fillets have been collected and tested for methylmercury since 1989 by GLIFWC. WDNR and tribes have also been collecting mercury data for walleye since the 1970s. A slight, regional decrease annually in walleye methylmercury of approximately 0.6 % annually was found between 1982 and 2005 (Madsen 2007). However, there was also evidence that the trend may have reversed in some areas, with mercury increasing (Monson et al. 2011).

Methylmercury-based walleye consumption advisories in the ceded territories are fairly common for inland lakes, especially for sensitive populations including infants, children, and women of childbearing age.

None of the Lake Superior fish sampled (**whitefish**, lake herring, lake trout, or siscowet trout) exceeded the United States FDA's methylmercury action limit of 1.0 ppm for commercially sold fish. However, the state of Michigan uses a lower level of 0.5 ppm (500 ppb). Siscowet samples in the 23-23 inch and 24.5-25.5-inch size groups exceeded Michigan's action level.[48]

Puddle, or dabbling, **ducks** do not accumulate substantial amounts of mercury. Dabblers averaged 0.09 ppm of total mercury in breast tissue, and 0.23 ppm of total mercury in livers. Correspondingly, diving ducks averaged 0.12 ppm in muscle, and 0.45 ppm in liver. The highest total mercury values observed came from certain duck livers, particularly diving ducks and grebes. These levels approached or exceeded 0.5 ppm (Kleinert and Degurse 1972, 6). In contrast, a 1987 study found male bluebills had

[48] GLIFWC memorandums from Matt Hudson, Dec. 30, 2005 and April 11, 2006.

higher mercury liver levels than females, but comparable, low levels to mallards and black ducks overall (Gochfeld and Burger 1987).

A study conducted by Fond du Lac Environmental Program tested mercury levels in duck breasts harvested by tribal members. Seventy percent of ducks tested were ring-necked ducks, a diving duck species. The study revealed 15% of sampled ducks exceeded 0.5 ppm in mercury concentrations. The Fond du Lac tribe recommended that children and women of childbearing age consume no more than one meal of duck per week. Other groups had no restrictions.[49]

[50]

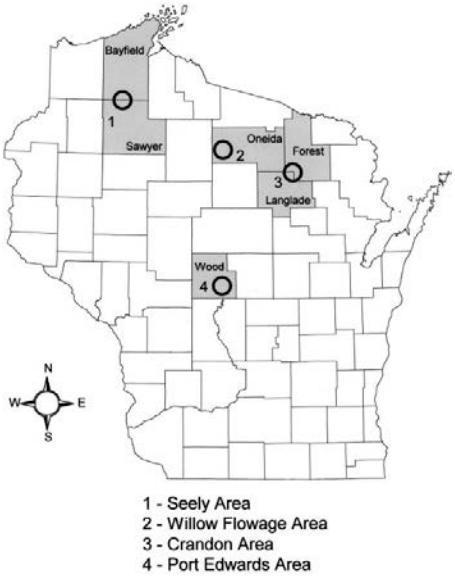


FIGURE 5: THIS MAP OF WISCONSIN SHOWS WHERE JAMES BENNETT AND HIS COLLEAGUES (2000) SAMPLED WILD RICE FOR VARIOUS HEAVY METALS, INCLUDING LEAD, MERCURY, AND ARSENIC.

Studies have consistently shown that wild rice does not uptake mercury into the seed head to any significant extent. One heavy metals study of Northern Wisconsin wild rice showed the highest total mercury average, 0.034 ppm, from the Seely-area rice beds (Bennett et al. 2000). The European Union has set a methylmercury consumption guideline for rice at 0.01 ppm. A study carried out by the Fond du Lac Environmental

[49] Personal communication with Nancy Schuldt, Fond du Lac Environmental Program, August 8, 2018.
[50] In general, diver ducks have large feet with short legs that are far back on the body in order to propel themselves underwater for food. In general, dabbler (puddle) ducks have smaller feet with legs situated farther forward on body. They often skim the water surface or tip forward to feed in the water (Ehrlich et al. 1988).

Program found no wild rice samples that exceeded 0.5 ppm, a fish consumption guideline set by United States EPA.[51] Thus, there is negligible risk of mercury exposure from consuming wild rice.

Arsenic Brief	
At Risk Foods:	
• Morel Mushrooms • Wild Rice — under review	• Berries — under review
Maximum Allowable Limit (excerpt):	
0.1 ppm infant rice cereal	United States
3.5 ppm fish protein	Canada
0.25 husked rice	European Union
0.1 edible fats and oils	World Health Organization

Arsenic is a metal-like element which is found naturally in the Earth's crust.[52] Two general types of arsenic exist: organic[53] and inorganic. Inorganic arsenic is more injurious to human health than organic arsenic. Compounds containing inorganic arsenic are mainly used in wood preservation and creation of pressure-treated lumber. Exposure to inorganic forms of arsenic are known to cause health problems in people, including: nausea, vomiting, decline in blood cell production, blood vessel damage, irregular heart rhythm, and prickling sensations on feet and hands. At very large doses of arsenic not likely to be associated with levels found in identified traditional foods, death can result. Limited research has been conducted on the human health effects of organic types of arsenic, but certain compounds caused diarrhea and kidney damage in laboratory animals (ATSDR 2018).

Wild rice and **blueberries** are two traditional Anishinaabe foods that may contain elevated amounts of arsenic in the ceded territories. Elevated amounts of total arsenic have been documented in wild rice seeds from natural areas (Pip 1993; Bennett et al. 2000).[54] However, amounts of the inorganic arsenic species, the variety more harmful to human health, was not evaluated in these studies. In addition, inorganic arsenic in cooked wild rice has not been examined to date (U.S. FDA 2013).

Wild blueberries in the Eagle Mine and Humboldt Mill area were found to contain amounts of total arsenic above the EPA's TDI value of 0.0003 ppm/day.[55] However, the

[51] Personal communication with Nancy Schuldt, Fond du Lac Environmental Program, August 8, 2018.
[52] The chemical symbol for arsenic is As (LANL 2018).
[53] The term 'organic' is defined as a chemical compound containing carbon.
[54] Personal communication with Abby Debiak, Superior Partnership & Land Trust, August 7, 2018.
[55] Eagle Mine and Humboldt Mill are in Marquette County of upper Michigan.

arsenic levels in the mining and mill area wild blueberries were approximately at the same arsenic level as in the control group.[56] This indicates that the geology of the area may naturally be high in arsenic overall.[57]

Arsenic uptake into **morel mushrooms** are a potential health hazard for people that consume this identified traditional food (Abdel-Aziz 2016). Lead arsenate, a formerly-used insecticide containing both lead and arsenic, was widely used in commercial apple production. Soils in abandoned orchards still contain high amounts of arsenic from past use of the insecticide. There is a relationship between higher levels of arsenic in the soil and higher amounts of arsenic found in morel fruiting bodies. About 94 percent of the arsenic stored in mushroom tissue was of the inorganic variety. Some morels exceeded consumption guidelines for long-term, oral ingestion of inorganic arsenic (Shavit and Shavit 2010).

Copper Brief	
At Risk Foods:	
• Duck (liver)	• Berries
Maximum Allowable Limit (excerpt):	
1 ppm bottled water	United States
1.3 ppm drinking water	Canada
2 ppm duck meat; 5 ppm duck fat	European Union
Limits have not been set	World Health Organization

Copper[58] is a naturally-occurring metal that is primarily released into the environment through mining and manufacturing operations. Smaller amounts of copper can also be released into the atmosphere from natural sources, like forest fires and decaying plants (ATSDR 2018). Trace levels of copper are essential for the proper function of the human body, such as cell energy generation and storage, wound healing, white blood cell formation, and enzyme function (Copper Development Association 2018).

Consuming excessive amounts of copper can cause negative health effects in both adults and children, such as diarrhea, vomiting, nausea, stomach cramps, and liver or kidney damage. At very large doses of copper not likely to be associated with levels found in identified traditional foods, death can result. (ATSDR 2018).

Blackberries sampled at the Eagle Mine and Humboldt Mill areas contained elevated copper concentrations of possible concern to human health, particularly at the mill site.

[56] Tolerable Daily Intake (TDI) refers to "an estimated amount of a potentially harmful substance in food or drinking water that can be ingested daily over the course of a lifetime without appreciable health risk."
[57] Personal communication with Abby Debiak, Superior Partnership & Land Trust, August 7, 2018.
[58] The chemical symbol for copper is Cu (LANL 2018).

Copper concentrations in blackberries during the 2-year study averaged around 10 ppm near the mill area. The control site berries were about half of this level. To compare, the European Union copper guideline is no more than 5 ppm in berries.

When examining copper exposure in **duck**, studies have shown that the livers of mallards, bluebills, and American black ducks from New Jersey all presented levels of possible concern to people who regularly consume duck liver. Male bluebills and female mallards have copper levels in their livers at 57 and 23 ppm wet weight, respectively. Copper levels in black ducks were slightly reduced compared to the other species, however, with males averaging 10 ppm in their livers (Gochfeld and Burger 1987). In contrast, the European Union's trigger levels are 5 ppm for duck fat, and 2 ppm for duck meat. Mallards and black ducks are considered dabbler ducks; bluebills are diving ducks. More recent studies of copper levels in duck liver in an area closer to the ceded territories have not been found.

Zinc Brief	
At Risk Foods:	
• Duck (liver)	• Berries
Maximum Allowable Limit (excerpt):	
Limits have not been established	United States
Limits have not been established	Canada
Limits have not been established	European Union
Limits have not been established	World Health Organization

Zinc[59] is a naturally-occurring metal and is frequently found in the crust of the Earth. It is present in many foods and needed in small amounts by the body for proper health. Most zinc is released as a result of from human activities, such as mining, steel production, and the burning of coal and industrial garbage. Zinc can form compounds with certain other elements, and can often be found near hazardous waste areas[60] (ATSDR 2018). Please see Appendix 7 for Comprehensive Environmental Response, Compensation and Liability Act (Superfund) areas.

When consumed in large amounts, zinc can cause nausea, stomach cramps, and vomiting. When zinc is consumed in excessive, long-term (chronic) amounts, it can decrease good cholesterol and cause anemia. While it is unknown if excess zinc can

[59] The chemical symbol for zinc is Zn (LANL 2018).
[60] Zinc compounds commonly found around hazardous waste areas are zinc sulfate, zinc sulfide, zinc oxide, and zinc chloride (ATSDR 2018).

cause any reproductive effects in humans, studies have shown infertility in rats that are fed very high concentrations of zinc (ATSDR 2018).

In 2015, **wild raspberries** sampled at the Eagle Mine and Humboldt Mill areas contained elevated zinc concentrations when compared to a control site, at values of 39, 43, and 26 ppm, respectively.[61] For comparison, the United States EPA's TDI value for oral zinc intake is 0.3 ppm/day. Wild raspberries sampled from the same areas the following year, however, did not demonstrate a consistent pattern of elevated zinc levels. Zinc concentrations in raspberries sampled from the mine, mill, and control areas were 25, 24, 25 ppm, respectively.[62]

When examining potential zinc exposure in **duck,** one study has shown that the livers of New Jersey mallards, bluebills,[63] and American black ducks all presented elevated levels of zinc that could be concerning for regular consumers of duck liver (Gochfeld and Burger 1987). For example, the average zinc levels in the livers of female mallards, male black ducks, and both sexes of bluebill were 92, 50, and 60 ppm wet weight. Mallards and American black ducks are considered dabbler ducks; bluebills are considered diving ducks.

Maple syrup is naturally high in zinc. Seasonal variation in zinc content included slightly higher levels in Canadian maple sap as the syrup season progressed. Zinc concentrations in resulting syrup reflected the change. The levels were not at amounts possibly harmful to human health (Robinson et al. 1989). However, zinc-coated processing equipment could potentially introduce excess zinc into maple syrup.

Manganese Brief	
At Risk Foods:	
• Berries	
Maximum Allowable Limit (excerpt):	
Limits have not been established	United States
Limits have not been established	Canada
Limits have not been established	European Union
Limits have not been established	World Health Organization

Manganese[64] is a metal commonly found in rocks, which is not able to break down in the environment (ATSDR 2018). Manganese is widely used in steel production and can

[61] The control site was located about 26 miles northeast of Humboldt Mill, and 28 miles southeast of Eagle Mine.
[62] Personal communication with Abby Debiak, Superior Partnership & Land Trust, August 7, 2018.
[63] Greater scaup was the specific bluebill species.
[64] The chemical symbol for manganese is Mn (LANL 2018).

also be found in modern gasoline additives (ATSDR 2018; US EPA 2016). Metallic sulfide mining activities can create acidic conditions that can mobilize this metal from the Earth as well (GLIFWC 2016). Please see Appendix 6 for a map showing metallic mining operations in the ceded territories.

Manganese is needed by the human body in trace amounts, and is naturally present in some foods, including shellfish and pine nuts. However, excess amounts of manganese negatively impact the nervous system. Some evidence suggests that children are more sensitive, especially to its effects on the nervous system. At toxic levels not likely to be found in traditional foods, manganese can cause brain damage, regardless of age. Individuals with iron deficiencies may uptake more of manganese than normally absorbed by healthy individuals (ATSDR 2018).[65] In animals that consume very high levels of manganese, reproductive effects have also been observed.

Wild blueberries, raspberries, and blackberries sampled around Eagle Mine or Humboldt Mill were consistently higher in manganese when compared to levels found in berries from the control site. The manganese levels in these berries were potentially harmful to human health over a period of time. The EPA's TDI manganese level for oral ingestion of berries is 0.14 ppm/day.

Wild blueberries from the Eagle Mine area possessed an average of 187 ppm of manganese, compared to the average at the control site, 160 ppm.[66] The difference between the manganese averages in raspberries was even more pronounced for the mine and control site at 245 and 34 ppm, respectively. Blackberries collected at Humboldt Mill site had much higher average manganese levels than berries at the control site, 217 and 78 ppm, respectively.

[65] Teenage girls, pregnant women, and women of childbearing years are more likely to be deficient in iron (ATSDR 2018).

[66] The control site was located about 26 miles northeast of Humboldt Mill, and about 28 miles southeast of Eagle Mine.

TABLE 6: THESE CALCULATIONS FOR AVERAGE MANGANESE LEVELS IN WILD RASPBERRIES ARE BASED UPON A PERSON WEIGHING 70 KG, OR ABOUT 154 POUNDS. ONE CUP OF RASPBERRIES EQUALS 125 GRAMS, OR 0.125 KG. TDI MEANS "TOLERABLE DAILY INTAKE," AND REFERS TO THE U.S. EPA STANDARD FOR ORAL INGESTION OF MANGANESE IN BERRIES. [67]

Superior Watershed Berry Monitoring Study: Manganese (Mn) Results for Raspberry			
2015			
	Eagle Mine	Humboldt Mill	Control Site
Ppm (mg/kg)	320	98	39
Kg to Exceed TDI	0	0.1	0.25
Equivalence in cups of berries	0.2	0.8	2.01
2016			
Ppm (mg/kg)	170	110	29
Kg to Exceed TDI	0.1	8.91	0.34
Equivalence in cups of berries	0.46	0.7	2.7

Cadmium Brief	
At Risk Foods:	
• Organ Meat (duck)	
Maximum Allowable Limit (excerpt):	
0.005 ppm bottled water	United States
0.005 ppm drinking water	Canada
0.05 poultry liver	European Union
0.01 ppm legumes	World Health Organization

Cadmium[68] is a soft, silvery metal associated with copper and zinc ores. Cadmium is released into the atmosphere from metallic sulfide mines, refineries, combustion of fossil fuels, and during waste disposal and incineration (ATSDR 2018). Mining activities also create acidic conditions which can mobilize cadmium into living organisms from the soil (Levit 2010). Please see Appendix 6 for a map showing metallic mining operations in the ceded territories.

[67] Personal communication with Abby Debiak, Superior Partnership & Land Trust, August 7, 2018.
[68] The scientific symbol for cadmium is Cd (LANL 2018).

Cadmium is known to accumulate in some crops, like Asian rice,[69] and aquatic organisms, such as birds, fish, and shellfish (ATSDR 2018). Cadmium is excreted from the body of aquatic organisms very slowly and has been documented to bioaccumulate in the food web (chain). People that consume contaminated foods from higher on the food web would, as a result, be exposed to higher levels of this heavy metal (Levit 2010). Also, people that regularly eat organ meat typically have higher rates of exposure (FdL and MN DoH 2014).

Currently, there is no known beneficial function of cadmium in the human body (Levit 2010). Consuming small amounts of cadmium over time can lead to buildup in the kidneys, with this high concentrations leading to damage in the kidneys (ATSDR 2018).[70] Additionally, fragile bones are another side effect of low-dose cadmium exposure. When lactating mothers are exposed to cadmium, small amounts are transferred to the infant from breastfeeding.

Ingestion of very high cadmium levels would not be very likely from eating identified traditional foods. However, those acute symptoms include: irritation of the stomach lining, causes vomiting and diarrhea, and even death, in some cases (ATSDR 2018). Individuals with iron deficiencies may absorb more cadmium from food sources more so than healthy individuals.

Animals that ingest cadmium develop liver disease, anemia, and brain and nerve damage. A few studies indicate that younger animals absorb more of cadmium than adult animals, which may result in bone loss from and have shown negative effects on learning and behavior (ATSDR 2018).

A study of New Jersey **ducks** found elevated cadmium concentrations in both diver and dabbler duck livers. The European Union has set a maximum level for poultry liver at 0.05 ppm. Not surprisingly, since diving ducks are higher on the food chain (web), greater scaup (bluebill) had the highest cadmium levels in their livers out of the three species, 1.09 and 0.897 ppm for female and male bluebill, respectively. In contrast, 0.565 ppm comprised the average for male American black duck livers, with averages for female livers being lower. Female mallards had an average of 0.566 ppm of cadmium in their livers, with male mallards having lower concentrations in their livers (Gochfeld and Burger 1987).

Forty-three people sampled in the Fond du Lac and Minnesota Department of Health (2014) human biomonitoring study exceeded the Level of Health Concern for cadmium

[69] *Oryza sativa* is the scientific name of Asian rice.
[70] High dietary cadmium exposure from Asian rice and *Tilapia* fish is the culprit for chronic kidney failure in a Sri Lankan farming region. As of 2008, more than 5,000 residents of the area were undergoing medical treatments for kidney failure (Levit 2010).

in blood, which is 1.7 micrograms/liter of blood. These results appeared to be related to cigarette smoking, however. There did not appear to be a relationship between blood cadmium levels and consumption of game meat or organs, yet only a few people reported eating organ meats.

There have been a number of studies concerning the potential cadmium exposure of **wild rice**, specifically to determine if cadmium affected the wild rice plant and/or the wild rice seed. Most studies have concluded that wild rice does not accumulate cadmium in its seeds to any significant extent.

Within one study, the majority of wild rice samples tested for cadmium came from Port Edwards and Crandon rice beds of northern Wisconsin, which are embedded within the ceded territory. This study determined that harmful concentrations of cadmium were not found within wild rice (Bennett et al. 2000). The median concentrations for cadmium were 0.016 ppm. The WHO consumption level recommendation for cadmium in rice is no more than 0.4 ppm.

Another study conducted on wild rice seeds in natural wild rice beds in Canada found cadmium concentrations no greater than 0.006 ppm. Within the study, it is also noted that the increased amount of cadmium found in wild rice typically means the wild rice will contain less copper, and could potentially affect the rice's nutritional composition (Pip 1993, 180).

Aluminum Brief	
At Risk Foods:	
• Blueberries	
Maximum Allowable Limit (excerpt):	
Limits have not been established	United States
Limits have not been established	Canada
Limits have not been established	European Union
Limits have not been established	World Health Organization

Aluminum[71] is the most abundant lightweight metal and is widely distributed in the Earth's crust. Aluminum is consistently found in combination with other elements in nature, such as oxygen. Aluminum can be mobilized by incinerators and coal-burning power plants (ATSDR 2018). Mining activities can create acidic conditions which can free aluminum from the soil to interact with the surrounding natural environment (Cronan and Schofield 1990).

[71] The chemical symbol for aluminum is Al (LANL 2018).

Individuals with kidney disease store aluminum within their bodies, as it cannot be effectively removed through urine. These people sometimes developed brain or bone diseases caused by high levels of this element. People with Type II diabetes may, consequently, be at a more elevated risk for aluminum exposure (ATSDR 2018).

In animal studies, test animals with aluminum toxicity performed lower than other test animals in grip-strength tests and presented with less mobility, showing that the nervous system is particularly sensitive to aluminum (ATSDR 2018).

Wild blueberries growing near Eagle Mine area were consistently higher in aluminum when compared to levels found in blueberries from the control site. The United States EPA's TDI for aluminum ingestion is 1 ppm/day. The average aluminum level for Eagle Mine blueberries was 29 ppm. The Humboldt Mill site levels were comparable to the control at about 16 ppm.

Elevated aluminum concentrations in **wintergreen** plants were found around the Eagle Mine area, of which the Ojibwe people use the plants and berries to make tea (Meeker et al. 1993). However, aluminum levels found in the wintergreen plants were not at a sufficient level to reasonably exceed the recommended EPA's TDI value of 1 ppm/day.[72] This is consistent with findings of elevated aluminum levels in tea made from other plants (Fung et al. 2009).[73]

Heavy Metal Safety Levels - Multijurisdictional

Safety and health of food supplies is a top priority for the United States, Canada, European Union member countries, and the World Health Organization. Each of these jurisdictions have set levels or limits to the amount or concentration of heavy metals in specific foods or food contact surfaces. Typically referred to as Maximum Level (ML), these levels are often based on the best available science and aimed at protecting human health. By setting limits, jurisdictions are creating parameters for food industries which are actionable and enforceable.

Appendix 8 provides tables of the current maximum allowable levels of heavy metals per country[74]. MLs vary from jurisdiction to jurisdiction and from food to food. Some countries have limits on only a few items per heavy metal, while others have set limits for a large quantity of foods. The MLs related or possibly related to traditional foods

[72] Superior Partnership did not infuse wintergreen tea, and only tested the whole plant. In the mine area, 1.76 pounds of wintergreen plant would have to be ingested to exceed TDI. In the control area, 2.43 pounds of plant would have had to be ingested. Personal communication with Abbie Debiak,Superior Partnership and Land Trust, August 7, 2018

[73] The tea plant, *Camellia sinensis,* has been widely researched in regards to aluminum levels.

[74] Maximum levels are revised often by each jurisdiction. For the most up to date information, refer to the jurisdiction's website or contact their outreach offices directly.

have been **bolded** for easier reference. For jurisdictions that have MLs for more than 5 non-traditional foods, only the traditional foods have been represented.

D. Pesticides & Industrial Chemicals

Pesticides and industrial chemicals can pose potential human health hazards, as they may contaminate animals living in the natural environment that people eat. Some of these chemicals, such as toxaphene, PCBs, dioxins, and furans, remain in the environment for long periods of time, biomagnify in the food web, and tend to accumulate in fat tissue. Fish and ducks, and the people that consume them, are often the most at risk from exposure to these types of pollutants. Some plants have been documented to uptake smaller amounts of certain chemicals from soil, but they are not likely to accumulate in plants at levels that would be hazardous to human health.

In a joint study conducted by Fond du Lac Environmental Program and Minnesota Department of Health (2015), people were assessed for exposure to certain organochlorine[75] chemicals. The chemicals tested for were DDT, DDE, hexachlorobenzene, mirex, PCBs, and toxaphene. In general, people that consumed wild rice, berries, fish, and game had similar chemical levels to people that did not consume traditional foods. However, people eating toxaphene from Lake Superior fish had small, but slightly elevated levels compared to the general population.

Older people in the study displayed higher concentrations of these chemicals than younger people, likely due to their extreme persistence. Since these pollutants are now banned in the United States, therefore, future generations will be less exposed than in the past (FdL and MN DOH 2015). However, lower levels of exposure can still occur when some of these chemicals are used elsewhere and travel around the world.

Perfluoroalkyl contaminants (PFCs), such as perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA), are chemicals of emerging concern with regards to their potential impacts on human health. The possible side effects of human exposure to these chemicals are largely unknown to date. However, animals exposed to these substances have experienced negative health effects.

Chlordane, DDT/DDE, mirex, chlordecone, aldrin, dieldrin, heptachlor, heptachlor epoxide, and hexachlorobenzene are organochlorine pesticides that no longer pose a threat to human health in the ceded territories, overall. They have significantly degraded over time to negligible levels. Glyphosate, 2,4-D, endothall, diquat dibromide, and chlorothalonil, although commonly used herbicides in the ceded territories, also

[75] Chemicals containing chlorine atoms on aromatic rings of carbon and hydrogen are called organochlorines or chlorinated hydrocarbons (U.S. EPA 2018).

generally do not present a significant threat to human health mainly due to lack of biomagnifying abilities. For more information about these chemicals, please see Appendix 9.

Organochlorine Pesticides

Toxaphene Brief	
At Risk Foods:	
• Ducks (particularly divers)	
Maximum Allowable Limit (excerpt):	
Limits have not been established	United States
0.1 ppm (default)	Canada
0.01 ppm (wild vertebrates)	European Union
Limits have not been established	World Health Organization

Toxaphene was an insecticide used heavily in the United States throughout the 1970s and into the early 1980s.[76] It is made up of a mixture of hundreds of specific chlorinated compounds. Most uses for toxaphene ceased in 1982, and it was completely banned in 1990 (ATSDR 2018). Toxaphene residues continues to persist in the environment, namely in Lake Superior (Xia et al. 2012).

Toxaphene can travel long distances in the air, contaminating sites where the chemical was not directly applied. This chemical strongly binds to soil, and in water, it tends to settle out onto the sediment as it does not dissolve well. Different types (congeners) of toxaphene break down at different rates, but all types break down quite slowly.

A common route of toxaphene exposure is by consuming contaminated food. Like many other persistent chemicals, toxaphene accumulates in the fat of fish, birds, and mammals. Studies in animals consuming toxaphene over time showed negative impacts to the kidneys, liver, and immune system. Several agencies have agreed that toxaphene likely causes cancer in humans (ATSDR 2018).

Consuming extremely high amounts of toxaphene would injure the liver, kidneys, and nervous system, and could cause death (ATSDR 2018). However, concentrations of toxaphene are not normally high enough to cause these acute illnesses from consuming traditional foods.

Although toxaphene levels are gradually declining over time (Xia et al. 2012), open-lake levels of toxaphene in Lake Superior continue to exceed water quality standards for

[76] Toxaphene was often used in the southern United States on cotton crops (ATSDR 2018).

certain jurisdictions. Higher concentrations of this chemical in Lake Superior compared to other Great Lakes can be explained by notable differences its unique physical properties, including large volume and cold temperature (Xia et al. 2011). Inland waters do not appear to have accumulation issues with this contaminant.

Since toxaphene biomagnifies in the environment, concern exists regarding the impacts in concentrations at higher trophic levels encompassing Lake Superior.[77] In particular, lake trout from Lake Superior generally contain the most total toxaphene out of fish species tested from the Great Lakes (Xia et al. 2012). This information is not entirely surprising considering the predatory status of the lake trout and higher trophic levels of these fishes.

Diving ducks collected in the southern Great Lakes were tested for this chemical and it was not detected in those samples. However, the southern Great Lakes are not afflicted with toxaphene persistence (Custer and Custer 2000). It is not entirely understood how toxaphene accumulates in the Lake Superior food web with respect to diving ducks, in particular, as testing has been primarily focused on fish.

Toxaphene 26 and 50 are the forms of the chemical commonly found in people. People that consume Lake Superior fish have elevated levels of toxaphene in their blood. Concentrations assessed in the blood were quite small overall, however. Both toxaphene congeners were more commonly found in people over 60 years of age. Median toxaphene 26 values for the 60+ year-old age group were 0.72 nanograms/gram of blood lipid. Median toxaphene 50 values for the 60+ year-old age group were 1.55 nanograms/gram of blood lipid (FdL and MN DOH 2015).

Dioxins & Dioxin-Like Compounds

Dioxins & Furans Brief	
At Risk Foods:	
• Whitefish	
Maximum Allowable Limit (excerpt):	
Limits have not been established	United States
20,000 ppm (under review)	Canada
0.000001 ppm (fish)	European Union
Limits may not be set at this time	World Health Organization

[77] A trophic level is an organisms' position in a food chain (web).

Dioxins and furans are contaminants that are created unintentionally through burning of garbage or coal. [78] In the United States, the greatest dioxin emission sources are electricity and heat-generating plants (Kanan and Samara 2018). They are also created as byproducts when the paper industry bleaches wood pulp, during herbicide manufacture, and in the creation of steel. Dioxins can be released during forest fires. These chemicals strongly bind to soil and sediment, but smaller amounts can evaporate (ATSDR 2018).

Dioxins and furans bioaccumulate and biomagnify up the food chain, primarily concentrating in fatty tissues of animals and humans. However, these chemicals have also been found on the surface of some produce. In addition, dioxins and furans can be taken up into squash family[79] plants in amounts smaller than found in animal-derived foods.[80] Consuming contaminated foods accounts for over 90 percent of human exposure to dioxins and related compounds.

Groups of people that are particularly sensitive to the effects of dioxins and dioxin-like compounds are developing fetuses, infants, and young children. They are also more highly exposed when their food intake per body mass is factored in. Other subgroups that are more highly exposed through the food supply include indigenous North Americans, people that fish, people in isolated areas whose food supplies are locally contaminated, and breastfed infants (IOM 2003).

The most toxic version of all the dioxins and furans is called TCDD.[81] Dioxin exposure at chronic levels in animals found negative effects, like liver damage, endocrine disruption,[82] and weakening of the immune system. Other studies in animals found birth defects and reproductive damage. TCDD is a known human carcinogen. High-dose TCDD exposure in people has caused acne-like lesions, skin rashes, excessive body hair, and changes in blood sugar metabolism. However, acute dioxin exposure is unlikely to occur from consumption of traditional foods.

In a GLIFWC study, measurable concentrations of dioxins and furans were found in fillet tissues of the largest sizes of tribal, commercially-harvested Lake Superior fish (lake herring, lake trout, **lake whitefish**, and siscowet trout). However, these concentrations were not particularly concerning, except for the untrimmed large siscowet.[83]

[78] There are 2010 different congeners, or varieties, of dioxins and furans. Seventeen of these have been found to be toxic (Kanan and Samara 2018).
[79] These foods were zucchini and pumpkin, specifically (IOM 2003).
[80] Carrots and peas also uptake small amounts of dioxin. However, most of the contaminant is found in the peel of the carrot and the pod of the pea (IOM 2003).
[81] The chemical name for TCDD is 2,3,7,8-tetrachloro-p-dibenzo-dioxin.
[82] The endocrine system regulates hormone use and production throughout the body.
[83] GLIFWC memorandums from Matt Hudson, Dec. 30, 2005 and April 11, 2006.

The sport fish dioxin and furan advisory level for the state of Michigan is 10 ppt toxic equivalency (TEQ).[84] The lowest TEQ concentrations were found in cisco and whitefish. Sampled lake herring averaged less than 0.5 ppt TEQ when the fillets were untrimmed and the skin was taken off. Comparatively, the sampled Lake whitefish averaged 1.8 ppt TEQ when the fillets were untrimmed and the skin was taken off, however, the whitefish muscle tissue averaged less than 0.6 ppt TEQ. Noticeably higher concentrations were found in both lake trout and sicowet. Lake trout averaged less than 5 ppt TEQ when the fillets were untrimmed and the skin was removed, while the lake trout muscle tissue averaged less than 4 ppt TEQ. Siscowet trout, which had the highest average of dioxins and furans concentrations, averaged 21 ppt TEQ when the fillets were untrimmed and the skin was taken off, with the siscowet muscle tissue averaging up to 7 ppt TEQ (Groetsch and Hudson 2005).

Fish consumption advisories for dioxin continue in Lake Superior. WHO, European agencies, and various states have established exposure guidelines for dioxins and furans in food to protect human health. However, FDA has not established Tolerance Levels or Action Levels for dioxin at this time.

PCBs Brief	
At Risk Foods:	
• Whitefish	
Maximum Allowable Limit (excerpt):	
2 ppm for fish	United States
2 ppm for fish — under review	Canada
0.00000175 ppm (cattle meat)	European Union
Limits may not be set at this time	World Health Organization

Polychlorinated biphenyls, or PCBs, are members of a broad chemical family, and are also known as dioxin-like compounds (DLCs).[85] They are a mixture of individual, manmade chemicals[86] that are no longer produced in the United States. PCBs were used mainly as coolants and lubricants in electrical equipment.[87] Other uses for these chemicals were in industrial adhesives, carbonless copy paper, hydraulic and motor oil, paint, plastics, floor finishes, and insulation. They were banned from use and

[84] TEQs are a weighted measure used to assess the relative toxicity of dioxins and dioxin-like compounds compared to the most toxic compound, TCDD (U.S. EPA 2016).
[85] PCBs are chemically composed of hydrocarbon rings with attached chlorines (US EPA 2018).
[86] These specific, identifiable chemicals are called congeners. There are 209 PCB congeners that can be subdivided even further into homologs that indicate the specific positions of chlorine on the ring (US EPA 2018).
[87] PCBs can be found in old fridges and televisions. They were used for this application because of unique physical properties, such as inflammability, thermal stability and resistance to corrosion.

production in 1979. Most people are exposed to PCBs through food, and fish are a common dietary source of PCBs (ATSDR 2018).

Developing fetuses and infants are believed to be more sensitive to the effects of PCBs. Children born to women that were exposed to higher amounts of the chemical had babies weighed less and showed abnormal responses in behavior and memory. Sometimes these symptoms lasted for several years. Immune system function in these children may have also been impacted. PCBs can be passed down from a nursing mother to an infant. However, prenatal exposure to these chemicals did not appear to cause structural birth defects.

Animals consuming smaller amounts of PCBs developed a variety of health effects, such as acne-like skin lesions, anemia, as well as stomach, liver, and thyroid gland injuries. Other chemical effects in animals include impairments to the immune system, behavior, and reproduction. Exposed workers had blood and urine results indicating impaired liver function. People exposed to large amounts of these chemicals often had acne[88] and rashes (ATSDR 2018). Recent research has also shown a link between PCBs and Type II diabetes (Faroon and Ruiz 2016).

Studies in workers[89] exposed to PCBs found an increased incidence of cancers in the bile duct,[90] liver, intestines, and skin. Rats eating high chemical amounts for two years got liver cancer. Based upon this information, the U.S. EPA has declared that PCBs likely cause cancer in humans. The International Agency for Research on Cancer has unquestionably decided that is a human carcinogen (ATSDR 2018).

Aquatic areas known to have high amounts of PCB contamination often have fish consumption advisories to protect the health of people eating fish from those areas. Tribal or state consumption advisories can sometimes be more restrictive than federal guidelines set by U.S. FDA (ATSDR 2018). Fish consumption advisories are sometimes in combination with other chemicals, such as mercury, dioxins, and toxaphene.

In a GLIFWC study, all Lake Superior fish sampled were did not exceed the FDA's action limit for PCBs, which is 2.0 ppm. The Lake Superior fish sampled during this study included **whitefish**, lake herring, lake trout, and siscowet. [91] Fish consumption advisories for this chemical continue in Lake Superior. However, people that consume

[88] This is a special type of severe acne called chloracne. It is caused by chlorinated chemical exposure (ATSDR 2018).
[89]In particular, male workers of capacitor manufacturing and/or repair facilities were examined after employment of greater than five years (ATSDR 2018).
[90] Biliary cancer is rare, and affects the tubes that take bile from the liver to the small intestine during the digestion of food (AGITG 2017)
[91] GLIFWC memorandums from Matt Hudson, Dec. 30, 2005 and April 11, 2006.

Lake Superior fish generally do not have concerning levels of PCBs in their blood (FdL and MN DOH 2015).

PCB levels can be reduced by trimming the fat from fillets, where PCBs are stored, which reduced PCB levels by 12 to 40 percent, depending on the fish species. Specifically, PCB levels in whitefish were reduced 32 percent, and 23-25 percent in lake trout. PCB concentrations in siscowet trout were lowered by 12-40 percent depending on the fish length. Removing skin from fillets further reduced concentrations of this chemical in whitefish, lake trout, and siscowet.[92]

Overall, harvested **waterfowl** do not uptake concerning amounts of PCBs in their breast muscle (Tsuji et al. 2007; Braune and Malone 2006). However, certain lakes are impacted more by past industrial use. For example, in one study, 80 percent of bluebill meat from Lakes Erie and Michigan exceeded the U.S. FDA's action level[93] during the years 1991 to 1993 (Custer and Custer 2000). PCB levels in the Laurentian Great Lakes continue to go down over time, decreasing impacts to human health for future generations (ECCC and U.S. EPA 2017).

Chemicals of Emerging Concern

Some persistent, manmade chemicals are being detected in increasing amounts within the environment. A few of these chemicals include perfluoroalkyl contaminants (PFCs), such as perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA). The potential side effects of human exposure to these chemicals are largely unknown, and more research is currently being conducted on them. Possible human health exposure from polybrominated diphenyl ethers (PBDEs) in fish was evaluated, but the risk to human health via traditional foods is negligible. For more information on PBDE, please see Appendix 11.

PFOS & PFOA Brief	
At Risk Foods:	
• Whitefish	
Maximum Allowable Limit (excerpt):	
Limits have not been established	United States
Limits have not been established	Canada
Limits have not been established	European Union
Limits have not been established	World Health Organization

[92] Ibid.
[93] The FDA action level was specific to consumption of poultry (3.0 mg/g lipid weight).

Perfluorooctane sulfonate (PFOS) and **perfluorooctanoic acid** (PFOA) are two specific compounds in a class of perfluorinated chemicals (PFCs). Out of the PFCs, PFOS and PFOA are the most widely studied. They are used in a wide variety of applications, predominately used in nonstick cookware, stain resistance for carpets and sofas, and waterproofing mattresses and clothes (NIH 2016). Other uses include paper coatings, cardboard packaging, leather products, industrial additives and coatings, and fire and chemical resistant tubing. PFOS and PFOA enter the environment from the manufacture and use of products that contain the chemicals. Degradation of similar chemicals can also turn into PFOS and PFOA (U.S. EPA 2017).

Studies of PFOA-exposed humans found links between exposure and increased liver enzymes, high cholesterol, thyroid disorders, preeclampsia, pregnancy-induced hypertension, kidney and testicular cancer, and decreased vaccination response. The WHO's International Agency for Research on Cancer (IARC) has categorized PFOA as possibly carcinogenic to humans. Research involving humans found connections between PFOS exposure and adverse developmental and reproductive effects, and high cholesterol. Laboratory animals exposed to PFOS and PFOA have exhibited detrimental developmental and reproductive effects (U.S. EPA 2017).

Although PFCs are not stored in body fat, they are persistent as they are resistant to environmental degradation (NIH 2016). PFOS and PFOA have been found in remote areas of the world, indicating that these chemicals are capable of long-range transport. In particular, PFOS is likely a bioaccumulating chemical and also able to biomagnify in the natural environment, such as in fish (U.S. EPA 2017). PFOS cannot be cooked or trimmed from fish, as it is bound in tissue (MDHHS 2016). PFOA can accumulate in species that breathe air, but not in fish (Vierke et al. 2012).

Wisconsin DNR evaluated various PFC congeners in different fish species from both the Laurentian Great Lakes and major rivers. PFOS was present in the highest concentrations and found in most samples. PFCs in fish sampled from the Great Lakes were lower overall than fish sampled from rivers, in particular the Mississippi River, which suggests that closeness to a PFC source is a major factor impacting contaminant levels. White bass and panfishes typically contain higher concentrations of PFOS than **walleye**. One of the lowest PFOS concentrations (2.0 ng/g, or 0.000002 ppm) was measured in Menominee River walleye (Williams and Schrank 2016).

Another study examined PFOS concentrations in the eggs and livers of **lake whitefish.** Average PFOS egg and liver concentrations in whitefish were 0.000263 and 0.000067 ppm wet weight, respectively (Kannan et al. 2005). Whitefish liver concentrations were below this standard, but whitefish eggs were nearly equal to the EPA's guideline. The United States EPA's drinking water maximum level for PFOS is 0.0002 ppm.

This study did not examine PFOS levels in whitefish tissue due to the negligible amount anticipated. The whitefish in this study were collected from Thunder Bay in Lake Huron, and PFOS levels differ between the Great Lakes (Kannan et al. 2005). For perspective, PFOS concentrations in lake trout from Lakes Huron, Michigan, and Superior are 0.000039, 0.000016, and 0.000005 ppm (Furdui 2007). Lake trout are situated within a higher trophic level than whitefish, therefore, it can be reasonably surmised that whitefish PFOS concentrations would be lower in Lake Superior than in Lake Huron. However, the interaction of factors that influence contamination from PFOS are still being investigated, and could be more complicated than expected (U.S. EPA 2017).

Pesticide and Industrial Chemical Safety Levels - Multijurisdictional

Safety and health of food supplies is a top priority for the United States, Canada, European Union member countries, and the World Health Organization. Each of these jurisdictions have set levels or limits to the amount or concentration of industrial chemicals in drinking but may not exist for foods. Available level or limits have been provided in Appendix 11 and Appendix 12. These levels are often based on the best available science and aimed at protecting human health. By setting limits, jurisdictions are creating parameters for food industries which are actionable and enforceable.

3. Physical Hazards

Physical hazards in food are foreign objects or materials that can enter food cause harm when consumed. Symptoms generally include but are not limited to: choking, dental damage, and laceration of the mouth or throat. Physical hazards specifically related to the identified traditional foods and outlined in this report include bullet fragments and shot pellets.

Physical Hazard Risk Overview	
	Bullet Fragmentation/ Shot Pellets
Deer	X
Rabbit/ Hare	X
Duck	X
Turkey	X
Whitefish	
Walleye	
Berries	
Wild Ramps	
Beach Pea	
Hazelnut	
Morel Mushroom	
Wild Rice	
Maple Syrup	
Berry Jam/Jelly	
Animal Fats	
Venison Jerky	

Bullet Fragmentation/Shot Pellets

A concern exists encountering metal fragments while consuming cooked game and or fowl meat. During consumption of meat, pellets or large bullet fragments could pose a choking hazard, cause damage to teeth, laceration hazard to the mouth or throat, and possibly damage the intestines (DHHS 2011). The FDA size standard for adulteration due to metal fragments is a minimum size of 0.3 inches (7 mm), however, it is unlikely that wild game killed with ammunition would exceed this threshold.

The potential for this hazard exists when using either non-lead or lead ammunition. However, it has been more widely studied in animals harvested with lead ammunition, mainly due to the significant health hazards associated with that metal. Agencies in Michigan, Wisconsin, Minnesota, and North Dakota X-rayed venison and all studies found some metal fragments, mostly in the form of lead (MI DCH 2010; WI DHFS 2008;

DNR 2008; ND DOH 2008). [94] The x-rays conducted by the Minnesota DNR found that metal shards spread as much as 18 inches,[95] or 1.5 feet, from where the bullet passed through. However, for the most part, citizens report that they rarely find metal fragments or shot in the wild game meat they consume (FSAS 2012).[96]

The largest concern would likely be in the form of pellets embedded in meat of fowl which are missed during cleaning and processing. As would be expected, harvesters perform a visual inspection to remove as much shot as possible from meat either during processing and before cooking (FSAS 2012). However, it is possible that pellets may be missed during the inspection process. Please see Figure 6 on page 67 for common pellet sizes for shotgun ammunition.

FIGURE 6 SHOT SIZES OF COMMON HUNTING AMMUNITION

Photo credit: IHEA-USA (https://www.hunter-ed.com/images/pdfs/ammo_shot_sizes.pdf)

[94] In Wisconsin, about 4 percent of x-ray results tested positive for lead. In N.D., about 12 percent tested positive for foreign material, with nearly 6 percent of that testing positive for lead. In the North Dakota study, other foreign particles found could have been bone shards, plastic, or metal fragments of other types, like copper. However, only lead was tested for due to funding constraints.
[95] This is the maximum distance that could be measured with x-ray (MN DNR 2008).
[96] This survey was conducted in Scotland.

BIBLIOGRAPHY

Abdel-Aziz, Shadia M., et al. 2016. "Edible Mushrooms: A Green Biotechnology and Great Nutritional Value for Improving Human Health." *International Journal of Science and Engineering* 1(8).

Abraham, A. et al. 2012. "Bad Bug Book Handbook of Foodborne Pathogenic Microorganisms and Natural Toxins." https://www.fda.gov/downloads/Food/FoodborneIllnessContaminants/UCM297627.pdf.

Agency for Toxic Substances & Disease Registry. 2018. "Toxic Substances Portal." *United States Centers for Disease Control.* https://www.atsdr.cdc.gov/toxprofiles/index.asp.

———. 2018. "Per- and Polyfluoroalkyl Substances (PFAS) and Your Health." https://www.atsdr.cdc.gov/pfas/index.html

Alm, Torbjorn. 2015. "Plant Species Introduced by Foreigners According to Folk Tradition in Norway and Some Other European Countries: Xenophobic Tales or Not?" *Journal of Ethnobotany and Ethnomedicine* 11(72).

Atkinson, Mark D., and Elaine Atkinson. 2002. "*Sambucus nigra* L." Journal of Ecology 90: 895-923. https://besjournals.onlinelibrary.wiley.com/doi/epdf/10.1046/j.1365-2745.2002.00698.x.

Australasian Gastro-Intestinal Cancer Trials Group. 2017. "Gallbladder & Biliary Tract Cancer." https://gicancer.org.au/cancer/gallbladder-and-biliary-cancer/.

Barcus, Adrian L, Steven D Burdette, and Thomas E Herchline. 2005. "Intestinal Invasion and Disseminated Disease Associated with Penicillium Chrysogenum." *Annals of Clinical Microbiology and Antimicrobials* 4 (December). BioMed Central:21. https://doi.org/10.1186/1476-0711-4-21.

Barltrop, D. and F. Meek. 1979. "Effect of Particle Size on Lead Absorption from the Gut." *Archives of Environmental Health* 34(4): 280-5.

Bennett, James P., Esteban Chiriboga, John Coleman, and Donald M. Waller. 2000. "Heavy Metals in Wild Rice from Northern Wisconsin." *The Science of the Total Environment* 246: 261-69.

Berzak, Laura, and Lauren Kingsley. 2003. "Graphite Furnace Atomic Absorption

Spectrometry." www.gmu.edu/departments/SRIF/tutrial/aas/aas.htm.

Bibi, Fahmeeda, Shafqat Nawaz Qaisrani, Atif Nisar Ahmad, Masood Akhtar, Bushra Nisar Khan, and Zulfiqar Ali. 2015. "Occurrence of Salmonella in Freshwater Fishes: A Review." *Journal of Animal and Plant Sciences* 25 (3):303–10.

Botzler, Richard G., and Richard N. Brown. 2014. *Foundations of Wildlife Diseases.* Oakland, CA: University of California Press.

Brown, Paul, et al. 2000. "New Studies on the Heat Resistance of Hamster-Adapted Scrapie Agent: Threshold Survival After Ashing at 600°C Suggests an Inorganic Template of Replication." Proceedings of the National Academy of Sciences 97(7). https://www.ncbi.nlm.nih.gov/pmc/articles/PMC16254/.

Carstensen, Michelle, and Michael W. DonCarlos. 2011. "Preventing the Establishment of a Wildlife Disease Reservoir: A Case Study of Bovine Tuberculosis in Wild Deer in Minnesota, USA." *Veterinary Medicine International* 2. https://www.researchgate.net/publication/51197444_Preventing_the_Establishment_of_a_Wildlife_Disease_Reservoir_A_Case_Study_of_Bovine_Tuberculosis_in_Wild_Deer_in_Minnesota_USA.

Centers for Disease Control and Prevention. 2018. "Chronic Wasting Disease." United States Department of Health & Human Services. https://www.cdc.gov/prions/cwd/index.html.

Center for Food Security and Public Health. 2017. "Fast Facts: Botulism." *Fast Facts*. http://www.cfsph.iastate.edu/FastFacts/pdfs/botulism_F.pdf.

Centers for Disease Control & Prevention. 2017. "Lead: What Do Parents Need to Know to Protect Their Children?" https://www.cdc.go Centers for Disease Control & Prevention. 2017. "Lead: What Do Parents Need to Know to Protect Their Children? "https://www.cdc.gov/nceh/lead/acclpp/blood_lead_levels.htm.v/nceh/lead/aclpp/blood_lead_levels.htm.

Centers for Diseases Control and Prevention. 2016. "Escherichia Coli (E. Coli) Factsheet." https://www.cdc.gov/ecoli/pdfs/CDC-E.-coli-Factsheet.pdf.

Centers for Disease Control and Prevention. 2015. "Cryptosporidiosis Surveillance — United States, 2011–2012." Morbitity and Mortality Weekly Report. 2015. https://www.cdc.gov/mmwr/preview/mmwrhtml/ss6403a1.htm.

Centers for Disease Control and Prevention. 2015. "Enteroviruses and Drinking Water from Private Wells." Www.Cdc.Gov2. 2015. http://www.cdc.gov/healthywater/drinking/private/wells/disease/enterovirus.

html.

———. 2011. "*Mycobacterium bovis* (Bovine Tuberculosis) in Humans." https://www.cdc.gov/tb/publications/factsheets/general/mbovis.pdf.

———. 2015. "Enteroviruses and Drinking Water from Private Wells." Www.Cdc.Gov2. 2015. http://www.cdc.gov/healthywater/drinking/private/wells/disease/enterovirus.html.

———. 2015. "General Information| Giardia | Parasites." 2015. https://www.cdc.gov/parasites/giardia/general-info.html.

———. 2015. "Giardia Illness and Symptoms." 2015. https://www.cdc.gov/parasites/giardia/illness.html#thirteen.

———. 2016. "Questions and Answers | Yersinia." 2016. https://www.cdc.gov/yersinia/faq.html.

———. 2016. "Staphylococcal Food Poisoning | Food Safety." 2016. https://www.cdc.gov/foodsafety/diseases/staphylococcal.html.

———. 2017. "About Botulism | Botulism." 2017. https://www.cdc.gov/botulism/general.html.

———. 2017. "Meningitis | About Bacterial Meningitis Infection | CDC." https://www.cdc.gov/meningitis/bacterial.html.

———. 2017. "Parasites - Diphyllobothrium Infection." 2017. https://www.cdc.gov/parasites/diphyllobothrium/index.html.

———. 2017. "Questions & Answers | Shigella – Shigellosis | CDC." 2017. https://www.cdc.gov/shigella/general-information.html.

———. 2017. "Questions and Answers | Campylobacter." 2017. https://www.cdc.gov/campylobacter/faq.html.

———. 2017. "Toxoplasmosis - General Information - Frequently Asked Questions (FAQs)." 2017. https://www.cdc.gov/parasites/toxoplasmosis/gen_info/faqs.html.

———. 2017. "Outbreaks of North American Lineage Avian Influenza Viruses." https://www.cdc.gov/flu/avianflu/north-american-lineage.htm.

———. 2018. "Hepatitis A Questions and Answers for the Public." 2018. https://www.cdc.gov/hepatitis/hav/afaq.htm.

———. 2018. "Norovirus | About Norovirus | CDC." 2018. https://www.cdc.gov/norovirus/about/index.html.

———. 2018. "Questions and Answers | E. Coli." 2018. https://www.cdc.gov/ecoli/general/index.html.

———. 2018. "Questions and Answers | Listeria." 2018. https://www.cdc.gov/listeria/faq.html.

———. 2018. "Questions and Answers | Salmonella." 2018. https://www.cdc.gov/salmonella/general/index.html.

———. 2018. "E. Coli Infection." https://www.cdc.gov/healthypets/diseases/ecoli.html.

———. 2018. "Eastern Equine Encephalitis." https://www.cdc.gov/easternequineencephalitis/index.html.

———. 2018. "Tularemia." https://www.cdc.gov/tularemia/.

———. 2018. "West Nile Virus." https://www.cdc.gov/westnile/.

Chavan, U.D., et al. 2003. "Phytochemical Components of Beach Pea." *Food Chemistry* 81: 61-71.

Clarke, K.R., et al. 2006. "Experimental Inoculation of Wild Turkeys with *Mycobacterium bovis*." Avian Diseases 50: 131-4.

Conover, Michael R. and Rosanna M. Vail. 2015. *Human Diseases from Wildlife.* Boca Raton, FL: CRC Press.

Copper Development Association. 2018. "Copper: Essential for Human Health." *Copper Alliance*. https://copperalliance.org.uk/knowledge-base/education/education-resources/copper-essential-human-health/.

Cornell Lab of Ornithology. 2018. "Wild Birds." *Cornell University.* http://www.birds.cornell.edu/birdflu/impact-on-birds/impact-on-wild-birds.

Cornicelli, Lou, and Marrett Grund. 2008. "Examining Variability Associated with Bullet Fragmentation and Deposition in White-Tailed Deer and Domestic Sheep: Preliminary Results." *Minnesota Department of Natural Resources.* https://files.dnr.state.mn.us/fish_wildlife/lead/bulletstudy/resources/publicsummary.pdf.

Cronan, Christopher S., and Carl L. Schofield. 1990. "Relationships Between Aqueous Aluminum and Acidic Deposition in Forested Watersheds of North America and Northern Europe." *Environmental Science & Technology* 24: 1100-05.

http://www.esf.edu/efb/mitchell/2005-Biogeo/New%20Readings/Cronan%20and%20Schofield%201990%20EST.pdf.

Custer, Christine M., and Thomas W. Custer. 2000. "Organochlorine and trace element contamination in wintering and migrating diving ducks in the southern great lakes, USA, since the zebra mussel invasion." *Environmental Toxicology and Chemistry* 19(11): 2821-29.

DeFarge, N., et al. 2018. "Toxicity of Formulants and Heavy Metals in Glyphosate-Based Herbicides & Other Pesticides." *Toxicology Reports* 5: 156-63.

Docherty, Douglas E., et al. 2006. "West Nile Virus Antibody Prevalence in Wild Mammals, Southern Wisconsin." *Emerging Infectious Diseases* 12(12). https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3291340/

ECCC and U. S. EPA. 2017. State of the Great Lakes 2017 Technical Report. (https://binational.net/wp-content/uploads/2017/09/SOGL_2017_Technical_Report-EN.pdf).

Ehrlich, Paul R., et al. 1988. "Dabblers vs. Divers." https://web.stanford.edu/group/stanfordbirds/text/essays/Dabblers_vs._Divers.html.

Encyclopaedia Britannica. 2018. "Septicemia | Infection | Britannica.Com." 2018. https://www.britannica.com/science/septicemia.

Encyclopedia of Life. 2018. "Viruses." http://eol.org/pages/5006/overview.

Enright, Rachel and Caitlin Doran. n.d. "Habitat Suitability of an Ojibwe Sacred Plant: White Sage *(Artemisia ludoviciana).* "University of Wisconsin-Madison. https://solim.geography.wisc.edu/axing/teaching/geog578/projects/y2003/SacredPlants_Enright.htm.

Environmental Protection Agency. 2000. "GIARDIA: DRINKING WATER FACT SHEET." https://www.epa.gov/sites/production/files/2015-10/documents/giardia-factsheet.pdf.

European Food Safety Authority. 2010. "Scientific Opinion on Lead in Food." EFSA Journal 8(4). https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2010.1570.

Extension Toxicology Network. 1995. "Endothall." http://pmep.cce.cornell.edu/profiles/extoxnet/dienochlor-glyphosate/endothall-ext.html.

———. 1994. "Chlorothalonil." http://pmep.cce.cornell.edu/profiles/extoxnet/carbaryl-dicrotophos/chlorothalonil-ext.html.

———. 1993. "Diquat Dibromide." http://pmep.cce.cornell.edu/profiles/extoxnet/dienochlor-glyphosate/diquat-ext.html.

Fachehoun, Richard Coovi, et al. 2015. "Lead Exposure Through Consumption of Big Game Meat in Quebec, Canada: Risk Assessment and Perception." *Food Additives & Contaminants-Part A* 32 (9): 1501-11.

Faroon, Obaid, and Patricia Ruiz. 2016. "Polychlorinated Biphenyls: New Evidence from the Last Decade." *Toxicology & Industrial Health* 32 (11): 1825-1847.

Felsot, Allan S. 2011. *Pesticides & Health: Myths vs. Realities*. New York: American Council on Science & Health.

Fisher, Art. 2016. "DDT and DDE: Sources of Exposure and How to Avoid Them." https://www.unce.unr.edu/publications/files/nr/2003/sp0316.pdf.

Food Safety Inspection Service.

Fond du Lac and Minnesota Department of Health. 2015. *Fond du Lac Community Biomonitoring Study: Community Report for Persistent, Bioaccumulative Chemicals.* ATSDR.

———. 2014. *Fond du Lac Community Biomonitoring Study: Community Report for Cadmium, Lead, & Mercury.* ATSDR.

Food and Drug Administration. 2017. "Control of Listeria Monocytogenes in Ready-To-Eat Foods: Guidance for Industry Draft Guidance." http://www.regulations.gov.

Food Safety and Inspection Service. 2013. "Campylobacter Questions and Answers." 2013. https://www.fsis.usda.gov/wps/portal/fsis/topics/food-safety-education/get-answers/food-safety-fact-sheets/foodborne-illness-and-disease/campylobacter-questions-and-answers/CT_Index.

———. 2011. "Jerky and Food Safety." *USDA.* https://www.fsis.usda.gov/wps/wcm/connect/32da4779-ba5e-4d7b-ad5a-2ad8a13aad1e/Jerky_and_Food_Safety.pdf?MOD=AJPERES.

Food Safety Working Group. n.d. *Listeria*. Washington D.C.: U.S. Department of Health and Human Services. Accessed August 13, 2018. https://www.foodsafety.gov/poisoning/causes/bacteriaviruses/listeria/index.ht

ml

Food Safety Working Group. n.d. *Staphylococcus aureus*. Washington D.C.: U.S. Department of Health and Human Services. Accessed August 13, 2018. https://www.foodsafety.gov/poisoning/causes/bacteriaviruses/staphylococcus/index.html

Food Standards Agency in Scotland. 2012. "Habits and Behaviours of High-Level Consumers of Lead-Shot Wild-Game Meat in Scotland." *Harris Interactive*. https://www.food.gov.uk/sites/default/files/media/document/776-1-1353_FS421005_Habits_and_behaviours_of_high-level_consumers_of_lead-shot_wild-game_meat_in_Scotland_FINAL.pdf.

Fung, K.F, et al. 2009. "A Comparison of Aluminum Levels in Tea Products from Hong Kong Markets and in Varieties of Tea Plants from Hong Kong and India." *Chemosphere* 75: 955-62. https://ac.els-cdn.com/S0045653509000071/1-s2.0-S0045653509000071-main.pdf?_tid=a447e37c-7cf4-4406-8ae1-672d2e0df900&acdnat=1533917781_fe072aa99ecdc91f226daf31e1323606.

Furdui, Vasile I., et al. 2007. "Spatial Distribution of Perfluoroalkyl Contaminants in Lake Trout from the Great Lakes." *Environmental Science and Technology* 41 1554-59.

Gandhi, Nilima, et al. 2017. "Polybrominated Diphenyl Ethers (PBDEs) in Great Lakes Fish: Levels, Patterns, Trends and Implications for Human Exposure." Science of the Total Environment 576: 907-916.

Geist, Valerius, David Clausen, Vince Crichton, and Darrel Rowledge. 2017. *The Challenge of CWD: Insidious and Dire.* Alliance for Public Wildlife: Living Legacy White Paper. https://mwf.mb.ca/wp-content/uploads/2017/03/CWD-Comprehensive-Analysis-LR-14March.single-page-versionpdf.pdf.

Gochfeld, Michael, and Joanna Burger. 1987. "Heavy Metal Concentrations in the Liver of Three Ducks Species: Influence of Species and Sex." *Environmental Pollution* 45: 1-15. https://ac.els-cdn.com/0269749187900121/1-s2.0-0269749187900121-main.pdf?_tid=18102c0a-c12f-4d26-b10e-4d61e6fee663&acdnat=1535159096_60ceee19ce7dcf91a3cf3a7846c5af97.

Great Lakes Indian Fish & Wildlife Commission. 2018a. "Chronic Wasting Disease (CWD)." https://data.glifwc.org/cwd/.

———. 2018b. "Treaty Rights." http://www.glifwc.org/TreatyRights/.

———. 2017. "Chronic Wasting Disease (CWD): Information for Tribal Hunters about the Threat to Waawaashkeshi (White-Tailed Deer) in the Ceded Territories." Odanah, WI.

———. 2016. "Metallic Mineral Mining: The Process & the Price." https://www.glifwc.org/publications/pdf/2016Process.pdf.

———. 2016. *Ojibwe Treaty Rights*. Odanah, WI.

———. 2014. "Mercury Maps." Guidance for Safe Consumption of Walleye from Inland Lakes within the Ceded Territories of Wisconsin, Michigan, and Minnesota. 2014. http://glifwc.org/Mercury/index.html.

Groetsch, Kory J., and Matthew J. Hudson. 2005. "Polychlorinated Dibenzo-p-Dioxin (CDD) and Polychlorinated Dibenzofuran (CDF) Concentrations in Fillet Tissue of Five Species of Lake Superior Fish." GLIFWC. https://www.glifwc.org/lakesuperiorwhitefish/PDF/Dioxin_Concentrations_Lake_Superior_Fish.pdf.

Grund, Marrett D., et al. 2010. "Bullet Fragmentation and Lead Deposition in White-Tailed Deer and Domestic Sheep." *Human-Wildlife Interactions* 4(2): 257-65. https://pdfs.semanticscholar.org/56ab/a970b00b70194c7f160d42320883fb2bd822.pdf.

Hanning, Irene B., J.D. Nutt, and Steven C. Ricke. 2009. "Salmonellosis Outbreaks in the United States Due to Fresh Produce: Sources and Potential Intervention Measures." *Foodborne Pathogens and Disease* 6 (6):635–48. https://doi.org/10.1089/fpd.2008.0232.

Hawkins, Steve A.C., Hugh A. Simmons, Kevin C. Gough, and Ben C. Maddison. 2015. "Persistence of Scrapie Infectivity Within a Farm Environment After Cleaning & Decontamination." *Veterinary Record* 176(4). http://eprints.nottingham.ac.uk/30243/1/Persistence%20of%20scrapie%20infectivity%20within%20a%20farm%20environment%20after%20cleaning%20and%20decontamination_Hawkins_etal.pdf.

Henderson, A. M.; Gervais, J. A.; Luukinen, B.; Buhl, K.; Stone, D. 2010. *Glyphosate Technical Fact Sheet*; National Pesticide Information Center, Oregon State University Extension Services. http://npic.orst.edu/factsheets/archive/glyphotech.html.

Hinckley, Glen T., et al. 2008. "Persistence of Pathogenic Prion Protein During Simulated Wastewater Treatment Processes." *Environmental Science & Technology* 42(14). https://www.ncbi.nlm.nih.gov/pubmed/18754377.

Hoffmeister, Erik, et al. 2016. "Survey for West Nile Virus Antibodies in Wild Ducks, 2004-06, USA." *Journal of Wildlife Diseases* 52(2). https://www.ncbi.nlm.nih.gov/pubmed/26981693.

Hoffmeister, Erik, Robert E. Porter, and J. Christian Franson. 2015. "Experimental

Susceptibility of Wood Ducks for West Nile Virus." *Journal of Wildlife Diseases* 51(2): 411-8. http://www.jwildlifedis.org/doi/full/10.7589/2014-08-216?code=wdas-site.

Hughson, Andrew G., Brent Race, Allison Kraus, Laura R. Sangare, Lori Robins, Bradley R. Groveman, Eri Saijo, Katie Phillips, Luis Contreras, Virkamal Dhaliwal, Matteo Manca, Gianluigi Zanusso, Daniel Terry, Jeffrey F. Williams, and Byron Caughey. 2016. "Inactivation of Prions and Amyloid Seeds with Hypochlorous Acid." *PLOS Pathogens* 12(9). https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5042475/.

Hunt, Grainger et al. 2009. "Lead Bullet Fragments in Venison from Rifle-Killed Deer: Potential for Human Dietary Exposure." Reproduced in Richard T. Watson, Mark Fuller, Mark Pokras, and Grainger Hunt, editors. *Ingestion of Lead from Spent Ammunition: Implications for Wildlife and Humans.* Boise, ID: The Peregrine Fund.

Hunt, W.G., et al. 2009. "Lead Bullet Fragments in Venison from Rifle-Killed Deer: Potential for Human Dietary Exposure." PLoSOne. http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0005330.

Institute of Medicine. 2003. *Dioxins and Dioxin-Like Compounds in the Food Supply: Strategies to Decrease Exposure.* National Academy of Sciences. Washington, D.C.: The National Academies Press.

International Maple Syrup Institute. 2015. "Potential Sources of Lead Contamination in Maple Syrup Production & Processing by Exhibit Category." https://andersonsmaplesyrup.com/licensing/lead_reduction_chart.pdf.

Jennelle, Christopher S., et al. 2017. "Surveillance for Highly Pathogenic Avian Influenza in Wild Turkeys of Minnesota, USA, during Outbreaks in Domestic Poultry." *Journal of Wildlife Diseases* 53(3): 616-20.

Jervais, G.; Luukinen, B.; Buhl, K.; Stone, D. 2008. *2,4-D Technical Fact Sheet*; National Pesticide Information Center, Oregon State University Extension Services. http://npic.orst.edu/factsheets/archive/2,4-DTech.html.

Johansen, Poul, et al. 2006. "Lead Shot from Hunting as a Source of Lead in Human Blood." *Environmental Pollution* 142: 93-7. https://www.biologicaldiversity.org/campaigns/get_the_lead_out/pdfs/health/Johansen_et_al_2005.pdf.

Johnson, Christopher J., Joel A. Pedersen, Rick J. Chappell, Debbie McKensie, and Judd M. Aiken. 2007. "Oral Transmissibility of Prion Disease Is Enhanced by

Binding to Soil Particles." *PLOS Pathogens* 3(7). https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1904474/.

Kamerud, Kristin L., Kevin A. Hobbie, and Kim A. Anderson. 2013. "Stainless Steel Leaches Nickel and Chromium into Foods during Cooking." *Journal of Agricultural and Food Chemistry* 61 (39):9495–9501. https://doi.org/10.1021/jf402400v.

Kanan, Sofian, and Fatin Samara. 2018. "Dioxins & Furans: A Review from Chemical and Environmental Perspectives." *Trends in Environmental Analytical Chemistry* 17: 1-13. https://www.sciencedirect.com/science/article/pii/S2214158817300624.

Katz, Christopher J., et al. 2007. "West Nile Virus Exposure in Black Bears of Northeastern Wisconsin." *Journal of Wildlife Management* 71(2): 634-6.

Keawcharoen, Juthatip, et al. 2008. "Wild Ducks as Long-Distance Vectors of Highly Pathogenic Avian Influenza Virus (H5N1)." *Emerging Infectious Diseases* 14(4): 600-607. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2570914/.

Kleinert, Stanton J., and Paul E. Degurse. 1972. "Mercury Levels in Wisconsin Fish & Wildlife." WDNR, Technical Bulletin No. 52. https://dnr.wi.gov/files/PDF/pubs/ss/SS0052.pdf.

Knutsen, Helle K., et al. 2015. "Associations between Consumption of Large Game Animals and Blood Lead Levels in Humans in Europe: The Norwegian Experience. In: *Proceedings of the Oxford Lead Symposium,* Delahay R.J, and C.J. Spray, eds., Edward Gray Institute: Oxford University, 44-50.

Kolaja, G.J., and D.E. Hinton. 1977. "Effects of DDT on Eggshell Quality & Calcium Adenosine Triphosphatase." *Journal of Toxicology and Environmental Health* 3(4): 699-704.

Koschnick, Tyler J., et al. 2005. "Turf and Ornamental Plant Tolerances to Endothall in Irrigation Water." HortTechnology 15(2): 324-329.

Kumar, Pradeep, et al. 2016. "Aflatoxins: A Global Concern for Food Safety, Human Health and Their Management." *Frontier in Microbiology* 7. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5240007/.

Levit, Stuart M. 2010. "A Literature Review of Effects of Cadmium on Fish." *Center for Science in Public Participation and The Nature Conservancy.* https://www.conservationgateway.org/ConservationByGeography/NorthAmerica/UnitedStates/alaska/sw/cpa/Documents/L2010CadmiumLR122010.pdf.

Los Alamos National Laboratory. 2018. "Periodic Table of Elements." *United States*

Department of Energy. http://periodic.lanl.gov/index.shtml.

Lund, Melissa, et al. 2017. "Potential for Water Borne and Invertebrate Transmission of West Nile Virus in the Great Salt Lake, Utah." *Applied and Environmental Microbiology*. DOI: 10.1128/AEM.00705-17.

Madsen, E.R. and H.S. Stern. 2007. "Time Trends of Methylmercury in Walleye in Northern Wisconsin: A Hierachical Bayesian Analysis". *Environmental Science and Technology*. 41(13); 4568-4573.

Makarewicz, J.C., et al. 2003. "Trend Analysis Reveals a Recent Reduction in Mirex Concentrations in Coho and Chinook Salmon from Lake Ontario." *Environmental Science & Technology* 37(8): 1521-7.

Mallin, M. A., et al. 2002. "Impacts and Recovery from Multiple Hurricanes in a Piedmont–Coastal Plain River System: Human development of floodplains greatly compounds the impacts of hurricanes on water quality and aquatic life." *BioScience* 52 (11). 999–1010.

Manna, P.K., et al. 1999. "Lathyrus & Lathyrism: A Review." International Journal of Food Properties 2(3): 197-203.

Marsh, Richard F., et al. 2005. "Interspecies Transmission of Chronic Wasting Disease to Squirrel Monkeys (*Saimiri sciureus*)." *Journal of Virology* 79(21). http://jvi.asm.org/content/79/21/13794.full?site=JVirol&utm_source=TrendMDJVirol&utm_medium=TrendMDJVirol&utm_campaign=trendmdalljournals_0.

Mateo, Rafael, et al. 2011. "Bioaccessibility of Pb from Ammunition in Game Meat is Affected by Cooking Treatment." *PLoSOne* 6. http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0015892.

Mathiason, Candace K. 2015. "Silent Prions & Covert Prion Transmission. *PLOS Pathogens* 11(12). https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4684347/pdf/ppat.1005249.pdf

May, Kimberly. 2010. "Disease Precautions for Hunters." https://dnr.wi.gov/topic/wildlifehabitat/documents/diseaseguide.pdf.

Meeker, James E., Joan E. Elias, John A. Heim. 1993. *Plants Used by the Great Lakes Ojibwa.* Odanah, WI: Great Lakes Indian Fish & Wildlife Commission.

Michigan Department of Community Health. 2010. "Lead in Venison in Michigan." United States Department of Health & Human Services Agency for Toxic Substances and Disease Registry. https://www.atsdr.cdc.gov/hac/pha/LeadinVenisoninMichigan/LeadinVenisoni

nMichiganLHC03182010.pdf.

Michigan Department of Natural Resources. 2018. “Blackhead.” https://www.michigan.gov/dnr/0,4570,7-350-79136_79608_85016-26481--,00.html.

Michigan Disease Surveillance System. 2018. “Weekly Disease Report.” http://www.malph.org.

Michigan Department of Health and Human Services (MDHHS). 2016. “Eat Safe Fish Guides.” https://www.michigan.gov/mdhhs/0,5885,7-339-71548_54783_54784_54785_58671-296074--,00.html.

Michigan Department of Natural Resources. 2018. “Brucellosis.” https://www.michigan.gov/dnr/0,4570,7-350-79136_79608_85016-26503--,00.html.

———. 2018x. “Eastern Equine Encephalitis (EEE).” https://www.michigan.gov/dnr/0,4570,7-350-79136_79608_85016-241967--,00.html.

———. 2014. “Eastern Equine Encephalitis (EEE): Addressing Hunter Concerns.” https://www.michigan.gov/documents/emergingdiseases/EEE_huntersconcerns_472166_7.pdf.

Mihucz, Victor G., Eniko˝ Tatár, István Virág, Chen Zang, Yun Jao, and Gyula Záray. 2007. “Arsenic Removal from Rice by Washing and Cooking with Water.” *Food Chemistry* 105 (4). Elsevier:1718–25. https://doi.org/10.1016/J.FOODCHEM.2007.04.057.

Miller, Debra L., et al. 2005. “Fatal West Nile Virus Infection in a White-Tailed Deer.” Journal of Wildlife Diseases 41(1): 246-9. http://www.jwildlifedis.org/doi/pdf/10.7589/0090-3558-41.1.246.

Monson BA, DF Staples, SP Bhaysar, TM Holsen, CS Schrank, SK Moses, DJ McGoldrick, SM Backus, and KA Williams. 2011. "Spatiotemporal Trends of Mercury in Walleye and Largemouth Bass from the Laurentian Great Lakes Region". *Ecotoxicology*. 20: 1555-1567

National Center for Home Food Preservation. n.d. “Frequently Asked Jam and Jelly Questions.”. https://nchfp.uga.edu/questions/FAQ_jellied.html.

NRAEF. 2012. *Servsafe Manager 6th Edition*. 6th ed. Chicago, IL: National Restaurant Association Educational Foundation.

National Institute on Aging. 2018. “What is Alzheimer’s Disease?” *U.S. Department of Health & Human Services.* https://www.nia.nih.gov/health/what-alzheimers-

disease.

Newell, Richard G. and Kristian Rogers. 2003. "The U.S. Experience with the Phasedown of Lead in Gasoline." *Resources for the Future*. http://web.mit.edu/ckolstad/www/Newell.pdf.

New Hampshire Department of Health & Human Services. 2006. "West Nile Virus, Eastern Equine Encephalitis and Hunters." Division of Public Health Services. https://www.dhhs.nh.gov/dphs/cdcs/arboviral/documents/hunters.pdf.

New York Department of Conservation. 2018. "Ammunition: Non-Lead or Lead?" https://www.dec.ny.gov/outdoor/48420.html.

Nichols, Tracy A. 2016. "Plants, Prions and Possibilities: Current Understanding & Significance of Prion Uptake into Plants." USDA National Wildlife Research Center - Staff Publications. https://digitalcommons.unl.edu/cgi/viewcontent.cgi?article=2807&context=icwdm_usdanwrc.

Nichols, Tracy A., Justin W. Fischer, Terry R. Spraker, Qingzhong Kong, and Kurt C. VerCauteren. 2015. "CWD Prions Remain Infectious After Passage Through the Digestive System of Coyotes (*Canis latrans*)." *Prion* 9(5). https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4964857/.

North Dakota Department of Health. 2008. "2008/09 Lead Fragments in Ground Venison Processors Study." https://www.ndhealth.gov/lead/venison/2008-2009LeadFragmentsInGroundVenisonProcessorsStudy.pdf.

Noyes, Pamela D., and Heather M Stapleton. 2014. "PBDE flame retardants: Toxicokinetics and thyroid hormone endocrine disruption in fish." *Endocrine Disruptors* 2(1).

Nriagu, Jerome O. and Tser-Sheng Lin. 1995. "Trace Metals in Wild Rice Sold in the United States." *The Science of the Total Environment* 172: 223-28.

Ooi, S. T., and B. Lorber. 2005. "Gastroenteritis Due to Listeria Monocytogenes." *Clinical Infectious Diseases* 40 (9). Oxford University Press:1327–32. https://doi.org/10.1086/429324.

Pain, D.J., et al. 2010. "Potential Hazard to Human Health from Exposure to Fragments of Lead Bullets and Shot in the Tissues of Game Animals." *PLoS One* 5(4). https://www.ncbi.nlm.nih.gov/pubmed/20436670.

Pip, Eva. 1993."Cadmium, Copper, and Lead in Wild Rice from Central Canada." *Archives of Environmental Contamination and Toxicology* 24: 179-81.

Porter, Richard D., and Stanley N. Wiemeyer. 1969. "Dieldrin and DDT: Effects on Sparrow Hawk Eggshells and Reproduction." *Science* 165: 199-200.

Poulle, Marie-Lazarine, et al. 2017. "Detection of Echinococcus multiocularis and other foodborne parasites in fox, cat, and dog faeces collected in kitchen gardens in a highly endemic area for alveolar echinococcosis." *Parasite* 24 (29). https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5530766/.

Prey, J., et al. n.d. "The Wisconsin Stormwater Manual Part One: Overview." Publication Number: WR-349-94. 6.

Pritzkow, S., et al. 2015. "Grass Plants Bind, Retain, Uptake, and Transport Infectious Prions." *Cell Report* 11(8). https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4449294/.

Raab, Andrea, Christina Baskaran, Joerg Feldmann, and Andrew A. Meharg. 2009. "Cooking Rice in a High Water to Rice Ratio Reduces Inorganic Arsenic Content." *J. Environ. Monit.* 11 (1). Royal Society of Chemistry:41–44. https://doi.org/10.1039/B816906C.

Race, Brent, Katie Williams, Christina D. Orru, Andrew G. Hughson, Lori Lubke, and Bruce Chesebro. 2018. "Lack of Transmission of Chronic Wasting Disease to Cynomolgus Macaques." *Journal of Virology* 92(13). http://jvi.asm.org/content/early/2018/04/19/JVI.00550-18.abstract.

Robinson, Allan R. et al. 1989. "Heavy Metal, pH, and Total Solid Content of Maple Sap and Syrup Produced in Eastern Canada." *Journal - Association of Official Analytical* Chemists 72(4): 674-9.

Rodriguez, Cynthia M., James P. Bennett, and Christopher J. Johnson. 2012. "Degradation of the Disease-Associated Prion Protein by a Serine Protease from Lichens." PLOS ONE 6(5). http://journals.plos.org/plosone/article/citation?id=10.1371/journal.pone.0019836.

Roy, D.N., S. K. Konar, S. Banerjee, D. A. Charles, D. G. Thompson, and R. Prasad. 1989. "Uptake and Persistence of the Herbicide Glyphosate in Fruit of Wild Blueberry & Red Raspberry." *Canadian Journal of Forest Research* 19:842-847.

Satterfield, Zane. 2004. "What Does PPM or PPB Mean?" *On Tap.*

Saunders, Lyndsay E., and Reza Pezeshki. 2015. "Glyphosate in Runoff Water and in the Root-Zone: A Review." *Toxics* 3(4): 462-80. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5606642/.

Senica, M., et al. 2017. "The higher the better? Differences in phenolics and cyanogenic glycosides in *Sambucus nigra* leaves, flowers and berries from different altitudes." *Journal of the Science of Food and Agriculture* 97(8): 2623-32.

Shavit, Elinoar, and Efrat Shavit. 2010. "Lead and Arsenic in *Morchella esculenta* Fruitbodies Collected in Lead Arsenate Contaminated Apple Orchards in the Northeastern U.S.: A Preliminary Study." Fungi 3(2): 11-18.

Siddiqi, Muhammad Akmal, et al. 2003. "Polybrominated Diphenyl Ethers (PBDEs): New Pollutants-Old Diseases." *Clinical Medical Research* 1(4): 281-290.

Smith, Gerald R. 2010. *Guide to Great Lakes Fishes.* Ann Arbor, MI: University of Michigan Press.

Stauffer, Glenn E., et al. 2018. "Ruffed Grouse Population Declines After Introduction of West Nile Virus." *The Journal of Wildlife Management* 82(1): 165-72. https://onlinelibrary.wiley.com/doi/pdf/10.1002/jwmg.21347.

Stilwell, David E. and Craig L. Musante. 1996. "Lead in Maple Syrup Produced in Connecticut." *Journal of Agricultural and Food Chemistry* 44(10): 3153-3158.

Swayne, D.E., J.R. Beck, and S. Zaki. 2000. "Pathogenicity of West Nile Virus for Turkeys." *Avian Diseases* 44(4): 932-7.

Swift, Bryan L., et al. 1993. "Organochlorines in Common Goldeneyes Wintering in New York." *Wildlife Society Bulletin* 21: 52-6.

Trombulak, Stephen C. and Christopher A. Frissell. 2000. "Review of Ecological Effects of Roads on Terrestrial and Aquatic Communities." *Conservation Biology* 14(1). https://onlinelibrary.wiley.com/doi/epdf/10.1046/j.1523-1739.2000.99084.x.

Tsuji, Leonard J.S., et al. 2007. "Spring-Harvested Game Birds from the Western James Bay Region of Northern Ontario, Canada: Organochlorine Concentrations in Breast Muscle." *Science of the Total Environment* 385: 160-71.

Turyk, Mary E., et al. 2012. "Risks & Benefits of Consumption of Great Lakes Fish." *Environmental Health Perspectives* 120(1): 11-8. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3261933/.

United Nations Environmental Programme Chemicals Branch. 2008. "The Global Atmospheric Mercury Assessment: Sources, Emissions, and Transport." https://wedocs.unep.org/bitstream/handle/20.500.11822/11517/UNEP_GlobalAtmosphericMercuryAssessment_May2009.pdf.

United States Department of Agriculture. 2017. "Wisconsin Ag News-Chemical Use." https://www.nass.usda.gov/Statistics_by_State/Wisconsin/Publications/Miscellaneous/2017/WI_Ag_Chem_Corn_Potatoes_05_17.pdf.

United States Department of Health and Human Services. 2011. "Fish and Fishery Products Hazards and Controls Guidance." www.ifasbooks.com.

United States Environmental Protection Agency. 2018. "Learn More About Polychlorinated Biphenyls (PCBs)." https://www.epa.gov/pcbs/learn-about-polychlorinated-biphenyls-pcbs.

———. 2017. "DDT-A Brief History & Status." https://www.epa.gov/ingredients-used-pesticide-products/ddt-brief-history-and-status.

———. 2016. "EPA Comments on the Gasoline Additive MMT." https://www.epa.gov/gasoline-standards/epa-comments-gasoline-additive-mmt.

———. 2016. "EPA Takes Final Step in Phaseout of Leaded Gasoline." https://archive.epa.gov/epa/aboutepa/epa-takes-final-step-phaseout-leaded-gasoline.html.

———. 1992. "Mine Site Visit: Copper Range Company, White Pine Mine." *Office of Solid Waste.* https://archive.epa.gov/epawaste/nonhaz/industrial/special/web/pdf/copper4.pdf.

———. 1987. "Chlorothalonil: Health Advisory." *Office of Drinking Water.* Draft Report.

United States Food and Drug Administration. 2017. "Control of Listeria Monocytogenes in Ready-To-Eat Foods: Guidance for Industry Draft Guidance." http://www.regulations.gov.

———. 2017. "Questions & Answers on Lead in Foods." https://www.fda.gov/Food/FoodborneIllnessContaminants/Metals/ucm557424.htm.

———. 2013. "Analytical Results from Inorganic Arsenic in Rice & Rice Products Sampling."

———. 2011. *Fish & Fisheries Products Hazards & Controls Guidance.* 4th ed. US Department of Health and Human Services.

United States Geological Survey. n.d. "Food Webs & Bioaccumulation Activity."

https://www.pwrc.usgs.gov/contaminants-online/pages/ToolsTeachers/TTfiles/lesson2/Activity%202_FoodWeb_KEY.pdf.

Vierke, L., Staude, C., Biegel-Engler, A., Drost, W., and C. Schulte. 2012. "Perfluorooctanoic acid (PFOA) — main concerns and regulatory developments in Europe from an environmental point of view." *Environmental Sciences Europe* 24 (16).

Waddell, L., et al. 2018. "Current Evidence on the Transmissibility of Chronic Wasting Disease Prions to Humans-A Systematic Review." *Transboundary & Emerging Diseases* 65(1). https://onlinelibrary.wiley.com/doi/full/10.1111/tbed.12612.

Wani, Ab Latif, Anjum Ara, and Jawed Ahmad Usmani. 2015. "Lead Toxicity: A Review." *Interdisciplinary Toxicology* 8(2). https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4961898/.

Warner, Sarah E., et al. 2014. "Bald Eagle Lead Exposure in the Upper Midwest." Journal of Fish and Wildlife Management 5(2): 208-16. https://www.researchgate.net/publication/274772384/download.

Institute Final Copy." www.internationalmaplesyrupinstitute.com/uploads/7/0/9/2/7092109/__lmea_-_standards_on_maple_equipment.pdf.

Willits, C.O. and C.J. Tressler Jr. 1937. "Sources of Lead in Maple Syrup and a Method for Its Removal." *Food Research* 3 (5): 449-452.

Wilson, P. Chris, et al. 2010. "Nontarget Deposition and Losses of Chlorothalonil in Irrigation Runoff Water from a Commercial Foliage Plant Nursery." *Journal of Environmental Quality* 39(6): 2130-7.

Wisconsin Department of Natural Resources. 2012. 2,4-D Chemical Fact Sheet. http://dnr.wi.gov/lakes/plants/factsheets/ 2,4-DFactSheet.pdf.

———. 2012. "Diquat Chemical Fact Sheet." https://dnr.wi.gov/lakes/plants/factsheets/DiquatFactsheet.pdf.

Wisconsin Department of Health and Human Services. "Health Consultation: The Potential for Ingestion Exposure to Lead Fragments in Venison in Wisconsin." https://www.atsdr.cdc.gov/hac/pha/LeadFragmentsinVenison/Venison%20and%20Lead%20HC%20110408.pdf.

Wisconsin Division of Public Health. 2018. "Venison and CWD-What Hunters Should Know." Wisconsin Department of Health Services.

http://dnr.wi.gov/topic/wildlifehabitat/documents/Venison_CWD.pdf.

———. 2004. "Disease Fact Sheet Series: *Clostridium perfringens*." https://www.dhs.wisconsin.gov/publications/p4/p42171.pdf.

World Health Organization. 2018. "Lead Poisoning and Health." http://www.who.int/news-room/fact-sheets/detail/lead-poisoning-and-health.

———. 2005. "No Bird Flu Risk for Consumers from Properly Cooked Poultry and Eggs." http://www.who.int/mediacentre/news/releases/2005/pr66/en/.

Xia, X., P. K. Hopke, B. S. Crimmins, J. J. Pagano, M. S. Milligan and T. M. Holsen 2012. "Toxaphene Trends in the Great Lakes Fish." *Journal of Great Lakes Research* 38(1): 31-38.

Xia, X., P. K. Hopke, T. M. Holsen and B. S. Crimmins 2011. "Modeling Toxaphene Behavior in the Great Lakes." *Science of The Total Environment* 409(4): 792-799.

Xu, S., et al. 2014. "Biodegradation of Prions in Compost." Environmental Science & Technology 48(12). https://www.ncbi.nlm.nih.gov/pubmed/24819143.

Yaeger R.G. 1996. Protozoa: Structure, Classification, Growth, and Development. In: Baron S, editor. *Medical Microbiology*. 4th edition. Galveston (TX): University of Texas Medical Branch at Galveston, Chapter 77. Available from: https://www.ncbi.nlm.nih.gov/books/NBK8325/.

Zabel, Mark and Aimee Ortega. 2017. "The Ecology of Prions." *Microbiology and Molecular Biology Reviews* 81(3). http://mmbr.asm.org/content/81/3/e00001-17.full.

Zhang, Z., et al. 2006. "Detection and Characterization of a Naturally Acquired West Nile Virus Infection in a Female Wild Turkey." *Journal of Veterinary Diagnostic Investigation* 18: 204-8. http://journals.sagepub.com/doi/pdf/10.1177/104063870601800212.

APPENDIX

Appendix 1: Excerpt from the Traditional Food Interest Survey

* 6. If safely available in your tribal community, what **large and small game** traditional foods would you be interested in having access to?

Examples: white-tail deer, black bear, moose, elk, bison, beaver, muskrat, hare, rabbit, badger, porcupine, raccoon, otter, lynx, woodchuck, etc.

	Would **STRONGLY** LIKE access to this food	Would **OCCASIONALLY** LIKE access to this food	Would **RARELY** LIKE access to this food	Would rather **NOT** access this food
White-tail Deer (Venison)	○	○	○	○
Black Bear	○	○	○	○
Moose	○	○	○	○
Elk	○	○	○	○
Rabbit/Hare	○	○	○	○
Porcupine	○	○	○	○
Beaver	○	○	○	○
Muskrat	○	○	○	○

Other large and small game I'd like access to:

* 7. If safely available in your tribal community, what **migratory birds** would you be interested in having access to?

Examples: ducks (mallard, wood duck), crane, swan, geese, snipe, mudhen/coot, sora & Virginia rails, doves, woodcock, etc.

	Would **STRONGLY** like access to this food	Would **OCCASIONALLY** like acces to this food	Would **RARELY** like acces to this food	Would rather **NOT** access this food
Geese	○	○	○	○
Duck	○	○	○	○
Crane (sandhill)	○	○	○	○
Swan	○	○	○	○
Snipe	○	○	○	○
Mudhen/coot	○	○	○	○
Rails (Sora & Virginia)	○	○	○	○
Mergansers	○	○	○	○

Other migratory birds I'd like to access:

APPENDIX 2 – Rare Zoonotic Diseases in the Ceded Territories

Brucellosis

Brucellosis in white-tailed deer, elk, and moose is caused mainly by a contagious bacterium called *Brucella abortus*.[1] The most common transmission of brucellosis is from ingesting undercooked meat or contact with skin wounds or mucous membranes. Additionally, harvesters can also catch brucellosis from inhaling bacteria while cleaning game (MI DNR 2018; CDC 2018). Rarely, brucellosis can be transmitted from person-to-person, also breastfeeding mothers can pass the illness to their nursing child (CDC 2018).

Symptoms of brucellosis vary, but typically include: fever, sweating, tiredness, weight loss, headache, backache, and muscle or joint pain. Recurrent symptoms may also result, such as depression, chronic fatigue, arthritis, organ swelling, and painful swelling of the testicles in males. Pregnant women who contract the illness may experience spontaneous abortion, therefore it is recommended to seek medical treatment for appropriate antibiotics (Conover and Vail 2015; CDC 2018).

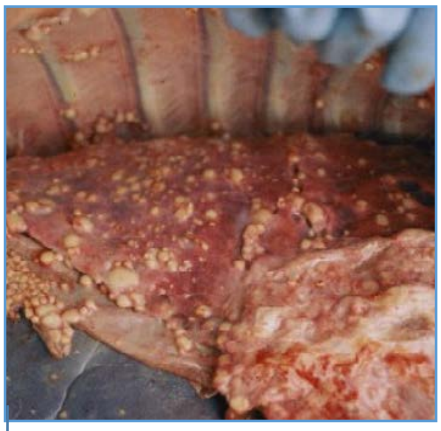
In the United States, brucellosis is relatively rare. In the ceded territories, only one human case has been reported since 2011 in Michigan (CDC 2018).[2] Brucellosis is not a very common disease in white-tailed deer. Michigan has extensively sampled the harvested deer populations and has found no evidence of brucellosis. In a study conducted nationwide, out of more than 17,000 white-tailed and mule deer[3] sampled, only 20 white-tailed deer tested positive for brucellosis (MI DNR 2018).

Bovine Tuberculosis (bTB)

Bovine tuberculosis (bTB) is a bacterial illness called *Mycobacterium bovis.*[4] It has been found in white-tailed deer,[5] elk, moose, and black bear.[6] People with the bovine version of TB have likely eaten contaminated meat or were infected through transmission via

[1] There are several species in the *Brucella* genus. Other animals able to become infected with *Brucella spp.*
[2] The case was found in Michigan. As this is a state we serve tribes in, I think it is important that this is mentioned in the sentence, not in a footnote.
[3] The scientific name for mule deer is *Odocoileus hemionus*.
[4] Most human tuberculosis (TB) cases are caused by *Mycobacterium tuberculosis,* not *M. bovis*.
[5] Due to its known infectivity of white-tailed deer and people, researchers experimentally inoculated wild turkeys (*Meleagris gallopavo*) with *M. bovis* to see if they serve as reservoir for the pathogen. They determined that young turkeys are resistant to *M. bovis* infection, and likely do not serve as a spillover host (Clarke et al. 2006, 131).
[6] The scientific name for the American black bear is *Ursus americanus.*

open wounds.[7] Direct animal-to-human transmission is believed to be quite rare. In these rare instances, people can become infected by inhaling air exhaled by animals that have the disease. Transmission of bTB can occur when an infected person coughs or sneezes onto an uninfected person (CDC 2011).



White-tailed deer chest cavity and lung infected with Bovine Tuberculosis. Photo credit: Michigan Department of Natural Resources.

Symptoms of tuberculosis vary depending on how a person was first exposed, and whether or not they have a latent form of the disease (Conover and Vail 2015, 45).[8] People that are exposed to bTB via ingestion or skin contact are more likely to have problems in the spleen, kidneys, liver, and digestive tracts, rather than issues within the lungs. Common universal symptoms include: unexplained weight loss, fever, excessive night sweating, and a constant cough (Conover and Vail 2015, 45).

Bovine tuberculosis is relatively uncommon, accounting for under 230 reported cases per year in the United States (CDC 2011), with incidents of tuberculosis higher in New England, Hawaii, and the southern United States. Throughout the ceded territories, incident rates of tuberculosis are less than 3.4 cases each year, per 100,000 people (Conover and Vail 2015, 43).

Eastern Equine Encephalitis (EEE)

Eastern Equine Encephalitis (EEE) in humans is very rare, more so than West Nile Virus. Humans typically become infected by EEE from a mosquito bite carrying the virus.[9] Mosquitos often get infected when feeding on carrier bird hosts, which are usually unaffected by the virus. White-tailed deer can become infected by EEE, but death from illness is rare. In Michigan, relatively higher mortality rates from EEE virus

[7] Bovine TB prevalence in Minnesota deer declined from 1.2 percent in 2005 to a level not detectable in 2010. Sharpshooting, a recreational deer feeding ban, and intensified hunting all helped decrease the disease in Minnesota (Carstensen and DonCarlos 2011).
[8] People who are infected, but have not developed symptoms, are termed as having a latent TB infection (CDC 2011). Infected individuals may get symptoms years later or not at all (Conover and Vail 2015, 45).
[9] EEE virus is one-stranded RNA (genetic material) with a spherical shape.

in deer has occurred, with seven dying in 2005, one in 2009, and two in 2010. One deer in Wisconsin died from this disease in 2004 (MI DNR 2018).

The last human case of this disease in Michigan occurred in 2014 in the southwest portion of lower Michigan. There were three cases reported, with no fatalities. The last reported EEE-related death occurred in 2001 in Michigan (MI DNR 2014). No specific information could be found regarding which portion of Michigan where this death occurred, but it was presumably in lower Michigan.

The majority of individuals that are infected exhibit no symptoms, or very mild symptoms that mimic other illnesses, including headache, tiredness, muscle aches, and fever (Conover and Vail 2015, 315). Only around 4 to 5 % of people infected with the virus develop serious brain swelling issues, typically affecting individuals under 15 years old or over age 50. Unfortunately, around a third of people who develop brain swelling issues die. Survivors of serious EEE infections often will have permanent neurologic damage, varying from mild to severe damage. Following the infection, affected individuals have immunity against the disease (CDC 2018).

People that hunt, fish, live, and/or work in hardwood swamps near the Great Lakes are at increased risk of infection with EEE (CDC 2018).[10] Although unlikely, harvesters could possibly become infected with the virus when field dressing and cleaning an infected deer. It is important for harvesters to avoid brain or spinal cord matter from the deer, taking extreme care to avoid with contact their eyes or open wounds during field dressing. Harvesters should also avoid inhaling droplets from the brain matter or spinal fluids (MI DNR 2018). Freezing meat will not kill the virus, so it should be cooked to the proper temperature (MI DNR 2014).

Highly-Pathogenic Avian Influenza (HPAI), or "Bird Flu"

Influenza is separated into three different classes, specifically A, B, C, and D.[11] "Bird flu," or "avian flu," is one specific type of influenza virus A. There are a number of different strains of HPAI in the United States which are different than strains that have caused outbreaks overseas.[12]. HPAI reached the United States from overlapping

[10] People that live or work in hardwood, freshwater swamps in the Gulf Coast and near the Atlantic Ocean are also at risk (CDC 2018).

[11] Influenza A is the class of viruses that can make people seriously ill or cause death. It also is a class of virus known to morph, or change, fairly easily. Viruses A, B, and C can infect humans with Viruses B and C typically cause short-lived, mild sickness. Virus D infects cattle, and is not known to make people ill (CDC 2017X).

[12] The deadliest strain is Asian H5N1, which is different than the North American H5N1 strain (CDC 2017X)

migratory bird flight pathways. These viruses do not usually infect humans, especially the North American varieties, however, in rare instances humans have become infected with bird flu (CDC 2017X).

Transmission of bird flu to humans is not completely understood, however, it seems to occur from close and continuous contact with infected birds. People can get infected when enough of the virus is present in air from water droplets, or dust, which is inhaled (Conover and Vail 2015; CDC 2017). Additionally, people can touch something infected, and can then transfer the virus to their eyes, nose, or mouth. However, in a few cases, direct contact with sick or dead birds was not thought to have occurred. The biggest concern exists regarding the ability of bird flu to morph into a form that is transmissible between humans (CDC 2017X).

Symptoms of bird flu include: eye inflammation, fever, cough, muscle aches, and sore throat, with symptoms ranging from mild to severe. In severe cases, symptoms include: diarrhea, vomiting, nausea, abdominal pain, shortness of breath, difficulty breathing, or seizures (CDC 2017).

It is important to note that bird meat or eggs containing HPAI are safe to eat if it is cooked to a proper temperature. However, there is the possibility of the HPAI virus contaminating the food after cooking, including exposure from contaminated hands, surfaces, or utensils (WHO 2005; Conover and Vail 2015). World Health Organization (2005) recommends that bird meat known to be diseased not be consumed, however.

In 2017, wild turkeys in Minnesota were assessed and found to not carry HPAI (Jennelle et al. 2017). However, different species of wild ducks[13] were experimentally infected with HPAI virus in a Dutch experiment (Keawcharoen et al. 2008, 600).[14] Researchers were interested in whether certain ducks were able to shed the virus while appearing unaffected, which would potentially allow these birds to carry the virus into areas further away.[15] Their findings suggested that some wild duck species, particularly

[13] Six species were chosen that are high-risk for HPAI, as well as common to Europe, Asia, and Africa. The species were mallard, gadwall, Eurasian widgeon, tufted ducks, Eurasian pochard, and common teal. Mallards and gadwall are species also found in the U.S. Tufted ducks and Eurasian pochards are closely related to bluebills. Eurasian widgeon is closely related to American widgeon, and common teal are closely related to blue-and green-winged teal.
[14] The subtype of HPAI virus used was Asian H5N1 (Keawcharoen et al. 2008, 600).

[15] Researchers found that Eurasian pochards, tufted ducks, and mallards shed much more of the virus than Eurasian wigeons, gadwalls, and common teals. However, only the ducks representing the diving duck family (tufted duck and Eurasian pochard) became gravely ill or died. Tufted duck had greater mortality than the pochard.

mallards, may be able to carry HPAI to previously uninfected areas (Keawcharoen et al. 2008, 600).

Deer Parapoxvirus is part of a viral group which can be found worldwide. While deer parapoxvirus typically infects sheep or goats, deer and other ungulates can become infected and potentially infect others. Deer with scabby, crusty lesions or sores in or around the mouth and on the face or antlers may have this virus, however, not all deer show symptoms (May 2010). Precaution should be made while field dressing deer, as the virus typically enters the body through open wounds in the skin on the hands or arms. To date, very few harvesters in the United States have been infected with this lesion-causing virus. Symptoms appear within 3 to 7 days, consisting of a discolored rash on the skin and which may continue to grow and swell (Roess et al. 2010).

Appendix 3: Maps of CWD prevalence in ceded territory

FIGURE 1: OVERVIEW OF CWD PREVALENCE IN CEDED TERRITORY.

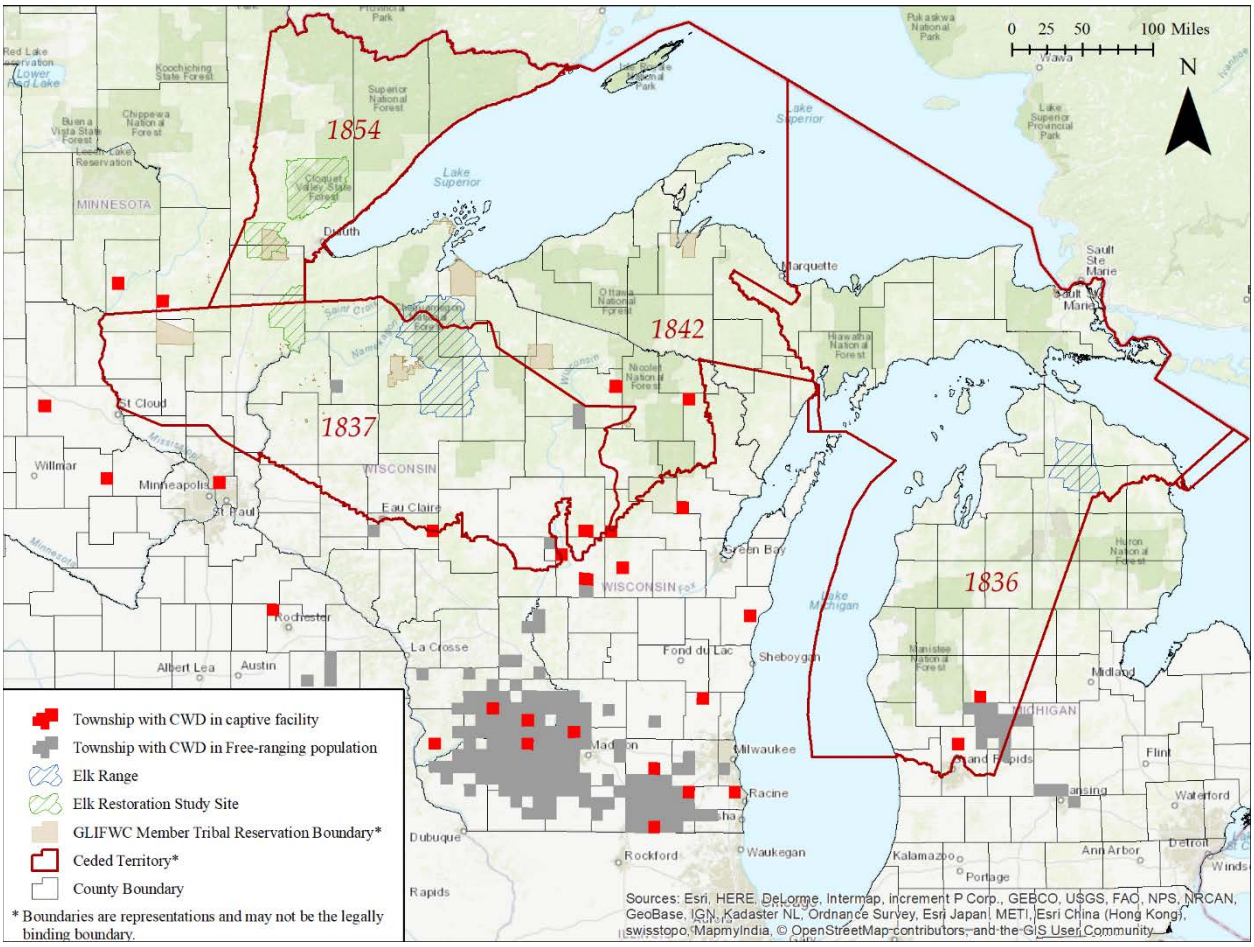


FIGURE 2: PREVALENCE OF CWD IN WISCONSIN.

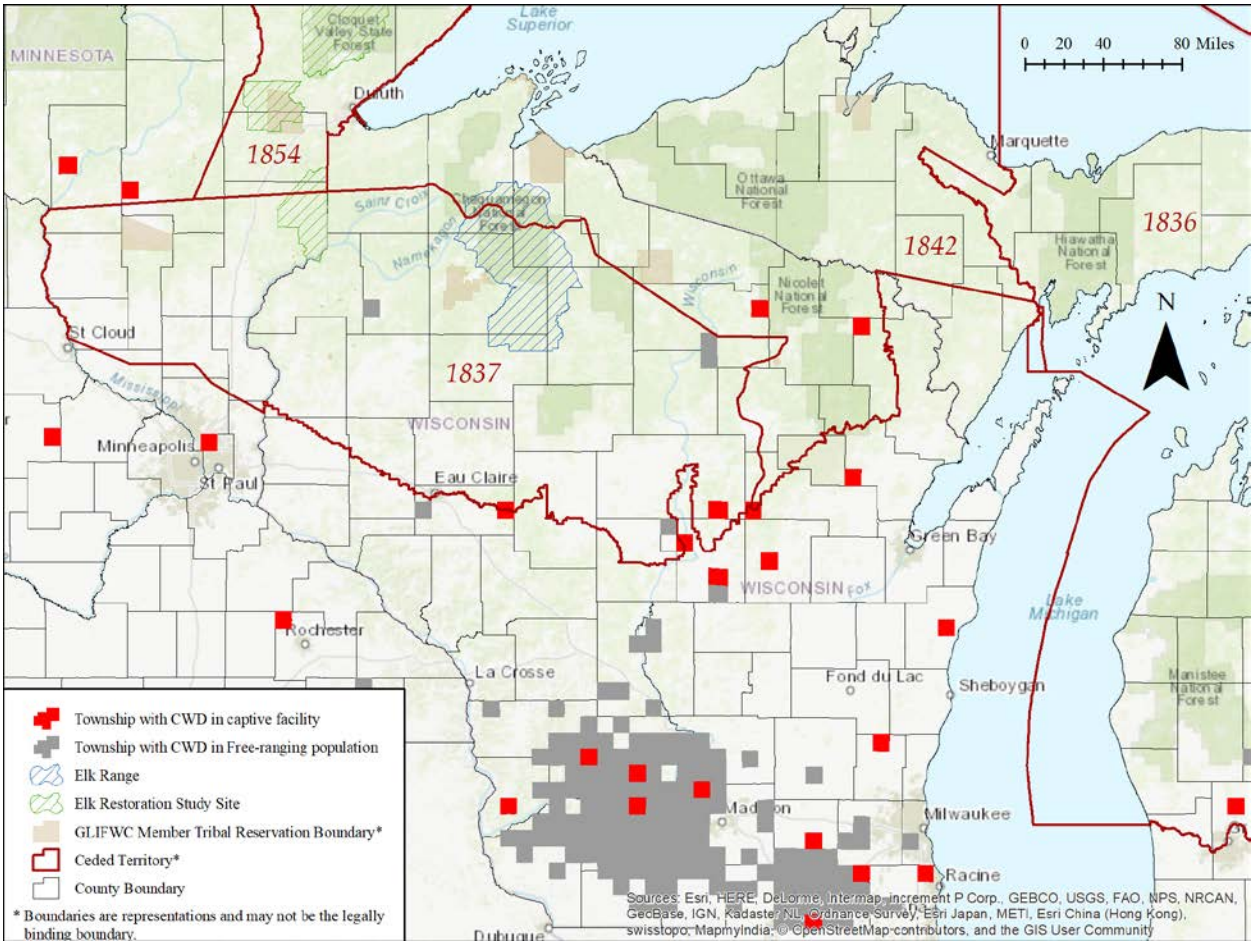


FIGURE 3: PREVALENCE OF CWD IN MICHIGAN.

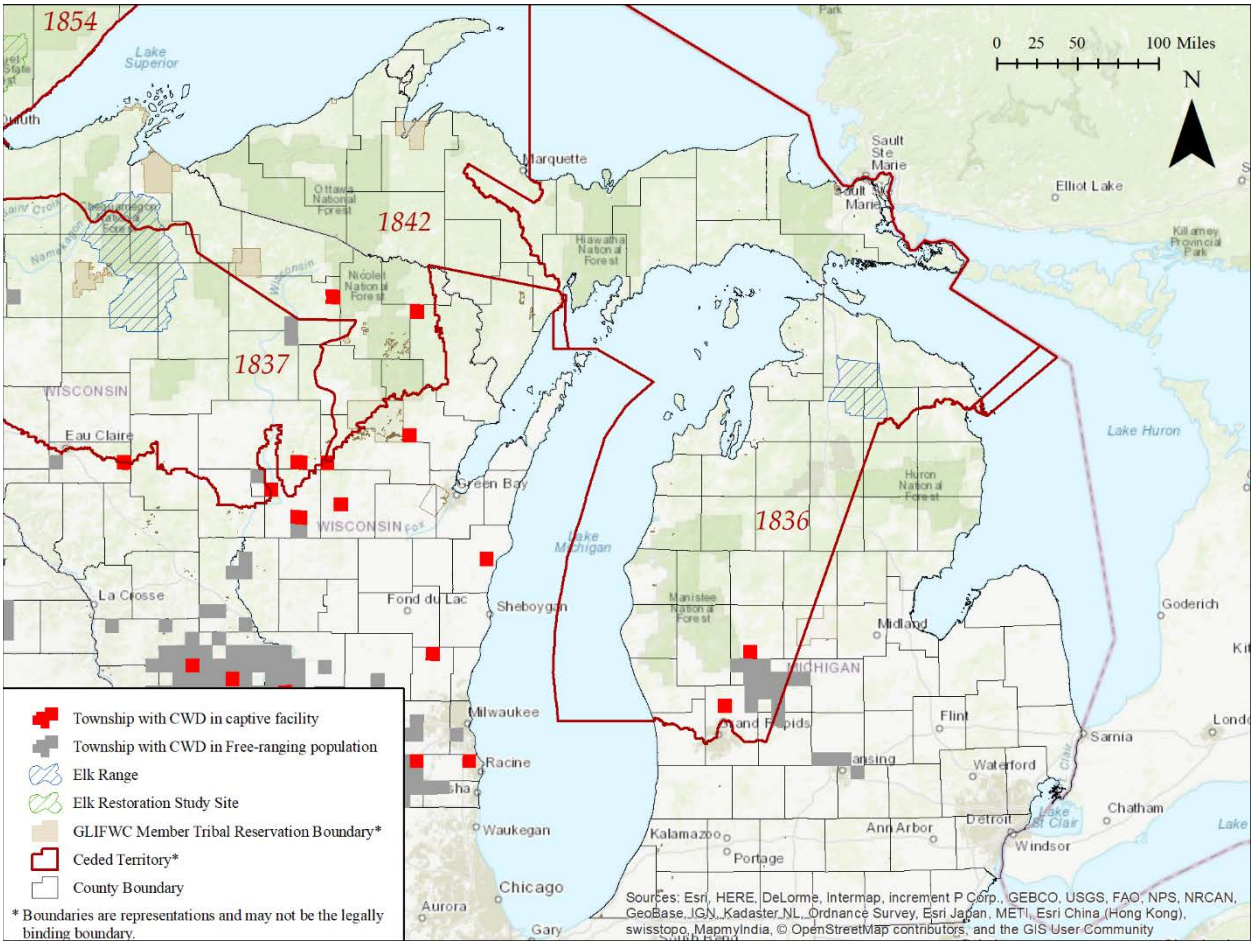
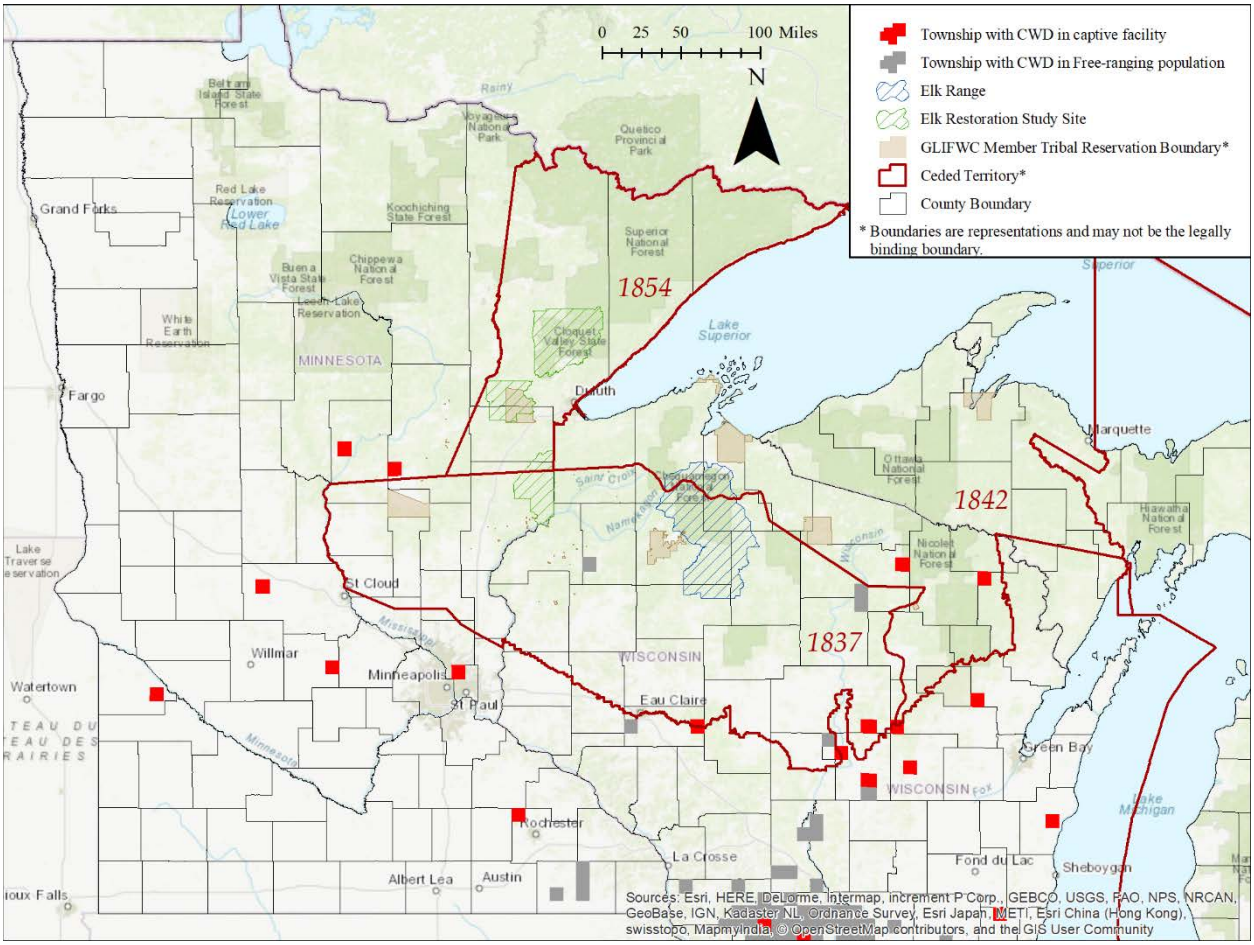


FIGURE 4: PREVALENCE OF CWD IN MINNESOTA.



Appendix 4: Regulations relating to CWD management in MN, WI, MI, WY, CO, and Canada.

First Documented CWD[1]	2002- on elk farm; 2011- confirmed in wild deer[1]	2002 – confirmed in wild deer[1]	2008- captive deer; 2015- confirmed in wild deer[1]	1979- in captive deer; 1985- confirmed in wild deer[2]	1967- captive deer; 1981- wild elk **first documented case of CWD in wild cervid;** 1985- first documented CWD in wild deer[1]	1981- in captive deer in zoo; [1]
Jurisdictional Agencies	MN DNR- wild cervid Board of Animal Health (BAH) – captive cervid	WI DNR- wild cervid and fencing DATCP- captive cervid	MI DNR- wild and captive cervid MI Agriculture and Rural Development (ARD): disease management and movement of captive cervid	Wyoming Game and Fish Dept. (WGFD)- wild cervid	Colorado Parks and Wildlife (CPW)- wild captive cervid; Dept. of Ag (CDA)- disease management of captive cervid	Canadian Food Inspection Agency: Voluntary Herd Certification Program (VHCP)
Baiting	**Banned** statewide[3]	**Partial Ban**: 43 counties banned; 29	**Partial Ban:** 11 counties banned; 72	**Banned** statewide with the exception	Big Game baiting is	3 provinces have baiting bans;

[1] Chronic Wasting Disease Alliance "Timeline" http://cwd-info.org/timeline/ accessed 7/3/2018
[2] Wyoming Game and Fish Department https://wgfd.wyo.gov/Wildlife-in-Wyoming/More-Wildlife/Wildlife-Disease/Chronic-Wasting-Disease Accessed 7/3/2018
[3] Chronic Wasting Disease Alliance "Chronic Wasting Disease and Cervidae Regulations in North America" Accessed 7/3/2018

		counties allowed (2 gallons/day archery season) Ban impacts is placed on any county within 10 miles of a captive or free-roaming deer that tests positive for either CWD or Bovine Tuberculosis (bTb). [4]	allowed (2 gallons/day from Sept. 15 - Jan 1)[3]	of permits provided by the WGFD[3]	**banned** statewide[3]	5 allow baiting; Baiting is an uncommon practice in the remaining 2 provinces; [3]
Fencing Requirements – Deer Farms	DNR-A **single fence is required.** Fencing must be 8 feet high and constructed "in a way that	DNR-a **single fence is required**. Since 2003, fence must be 8 feet tall, if build prior to 2003 it	DNR-A **single fence is required.** Must be 10 feet tall if newly constructed. Requires weekly	Deer farms are illegal in Wyoming.	CPW- **Double fencing is required**. Fences must be 8 feet tall. Fences must separated for ease of	National- **Single fence** must be 8 feet tall and in compliance with Provincial code.[9]

[4] Wisconsin Department of Natural Resources. "Wisconsin Deer Baiting and Wildlife Feeding Regulations (WM-456-2017)". https://dnr.wi.gov/topic/hunt/bait.html Accessed 7/3/2018

[9] Canadian Food Inspection Service. "National Standards for the CWD VHCP" document. Section 4.4.2 http://www1.agric.gov.ab.ca/$Department/deptdocs.nsf/all/rsb7193/$FILE/CWD_VHCP_NationalStandards2017.pdf Accessed 7/9/2018

	prevents the escape of farmed crevidae or entry… by free roaming crevidae"[5]	may be 7 feet, 10 inch tall. DATCP- **Double fence is required**. Specs: both solid barriers, 8 feet tall and no more than 16 feet apart.[6]	perimeter checks.[7] Michigan House Bill 5770 would require a second fence if passed. Submitted April 2018.		maintenance and vegetation should be controlled so as not to attract wild cervids. All gates shall be locked, consecutive, or self-closing.[8]	
Fencing Requirements – Hunting Preserves	MBAH- Under Minnesota law 35.153(3) ""Farmed cervidae" means cervidae that are: (1) **raised for any purpose**; and (2) registered in a manner	DNR- Must comply with the DNR regulations list for Deer Farms (see above). Preserves are a min. of 80 continuous acres.[10]	Same requirements as deer farms but the addition of a **double gating is also required**.[7]	Deer farms are illegal in Wyoming.	CPW- *Ranching for Wildlife:* 10,000 continuous acres privately owned. Must enter into a Cooperative Agreement with the State which includes a management	Requirements are set by each Province.

[5] Minnesota Statue 35.155 (4)
[6] Wisconsin ATCP 10.58
[7] Michigan Department of Natural Resources. "OPERATIONAL STANDARDS FOR REGISTERED PRIVATELY OWNEDCERVIDAE FACILITIES" document. Page 2. https://www.michigan.gov/documents/dnr/POC_OP_Standards_07_191455_7.pdf Accessed 7/5/2018
[8] Colorado Parks and Wildlife. "Chapter W-11 - Wildlife Parks and Unregulated Wildlife" Article III Section 1108 Subsection C
[10] Wisconsin ATCP 95.55 (5) (b) https://docs.legis.wisconsin.gov/statutes/statutes/95/55 7/5/2018

	approved by the Board of Animal Health."				plan. Must be open to the public. [11]	
Intrastate Movement of Carcasses from CWD affected areas or management units	DNR-1) *Whole Carcass:* Adult carcasses cannot be moved from CWD areas **until a negative CWD test is reported**. Fawns (1/2 year old) may only be moved after registered and tagged by a DNR official. 2) *Partial Carcass:* **Only the specific meat cuts and partial**	DNR- 1) *Professional Processing:* All parts and whole carcass may be transported from a CWD county to anywhere in the state as long as **they are transported to a licensed taxidermist or meat processor** within 72 hours of registering a deer or	DNR- **Whole carcasses and parts thereof may be moved within the state** after submitting the head to DNR official for testing (2017).[14]	WGFD- 1) *Whole Carcass:* **The spinal column and head must be left** at the kill site or disposed of at an approved landfill. 2)*Partial Carcass:* **Spinal column and head must be removed**. See next row for list.[15]	CPW- no regulations at this time	Requirements are set by each Province.

[11] Colorado Parks and Wildlife. Chapter W-2 "Big Game" Article 1 Section 210 Subsection B-D.
[14] Michigan Department of Natural Resources. "2017 Michigan Hunting and Trapping Digest". Page 62
[15] Wyoming Game & Fish Department. "Chapter 2: General Hunting Regulations". Section 15 (a)-(f) https://wgfd.wyo.gov/WGFD/media/content/REGULATIONS_CH2.pdf Accessed 7/4/2018

	carcasses may be moved prior to a negative test result. See next row for list.[12]	entering the state. 1) *Home Processing:* Whole carcass and specified parts **cannot be moved outside of CWD affect counties.** See next row for list.[13]				
Parts of a carcass that can be moved (within state): • Quarter without head or spinal column • Other portions of meat without head or	Carcass and partial carcass movement is the same as listed in first column.	Carcass and partial carcass movement is the same as listed in first column. *Teeth- allows only upper canines.	Carcass and partial carcass movement is the same as listed in first column.	Carcass and partial carcass movement is the same as listed in first column.	Carcass and partial carcass movement is the same as listed in first column.	Requirements are set by each Province.

[12] Minnesota Department of Natural Resources. Minnesota Hunting and Trapping Regulations. Page 65
[13] Wisconsin Department of Natural Resources webpage "Carcass Movement Restrictions". https://dnr.wi.gov/topic/wildlifehabitat/carcassmovement.html Accessed 7/4/2018

spinal column • Meat the is deboned and wrapped • Finished taxidermy heads • Teeth* • Hides cleaned of tissues • Antlers cleaned of tissues • Skull plate cleaned of tissues • Skulls cleaned of tissues						
Action if CWD is Verified – Deer Farm	BAH- Animal diagnose in late stage CWD is **euthanized**. [16]	DATCP- **Quarantine** the herd for up to 5 years after last positive	ARD- 1) **Complete epidemiological study to determine**	N/A	CAD: *During Slaughter-* carcass of suspected CWD will be	National- *HCP Enrolled Farms*: All cervids exposed to CWD positive

[16] Minnesota Statutes 1721.0370 (6)(b)

	Herds infected or exposed to CWD must be quarantined by BAH immediately. Movement from quarantine is only allowed by permit from BAH if determined not to endanger the health of other animals in the state. Quarantine is released based on level of environmental contamination and CWD	CWD result. After epidemiologic al study, a herd or individual deer can be quarantined for up to 5 years. DATCP may order **slaughter** or destruction of deer.[18]DATCP can **suspend** or **revoke** CWD Herd Plan status if a deer in the plan test positive for CWD[19]	**cause**; 2) **Quarantine** facility with depopulation and CWD testing; 3) "Trace Forward of exposed animal": exposed animals are depopulated and tested. If found positive the whole herd is positive; 4)"Traced back of exposed animals" Quarantine of remaining herd for 5 years from last		held for testing. If test is positive for CWD, carcass will be destroyed. CWD positive facility will be quarantined and a herd plan will be developed by the State Veterinarian and the Wildlife Division. No restocking will be allowed. Quarantine will remain until 5 years free of CWD is established.	deer are killed. Of the killed, all over 1-year-old will be tested for CWD. Positive carcasses must be disposed of by burial, incineration, or approved specified-risk material streams. *Non-Enrolled Farms:* Only and Epidemiologic al investigation will take place.[22]

[18] Wisconsin ATCP 10.52 (7)(8)
[19] Wisconsin ATCP 10.53 (8)(9)
[22] Canadian Food Inspection Agency. "Chronic wasting disease - What to expect if your animals may be infected" webpage. http://www.inspection.gc.ca/animals/terrestrial-animals/diseases/reportable/cwd/if-your-animals-may-be-infected/eng/1330188848236/1330189018195 Accessed 7/9/2018

	testing results. Quarantine can last up to 5 years and includes depopulation options.[17]		trace back date and monthly inspection by state or federal personnel.[20]		Depopulation will be required if the Division of Wildlife and CAD agree that there is substantial risk. [21]	
What is done if CWD is found – Hunting Preserve	See results for deer farm above	The results must be sent to the hunter.[23]	See results for deer farm above	N/A	No information available at this time.	Requirements are set by each Province.
Testing Requirements for Hunting Preserves	MBAH- See Testing Requirements for Deer Farms above.	DATCP- See Testing Requirements for Deer Farms above.	Same requirements as deer farms	WGFD- See Testing Requirements for Hunters below.	No information available at this time.	Requirements are set by each Province.
Testing Requirements for Hunters in CWD Units	DNR- "**Mandatory CWD testing continues** in southeast	DNR- **Voluntary.** Sample collection is offered at ~78	DNR- "If a deer is taken from a Core CWD Area, you **must**	WGFD- **Voluntary in 2018 but recently WGFD**	CPW- **Mandatory for some CWD Units due to lack of funds**	Requirements are set by each Province.

[17] Minnesota Rules: Deer and Elk 1721.0420 (2)
[20] Michigan Department of Natural Resources and Michigan Department of Agriculture and Rural Development. "MICHIGAN SURVEILLANCE AND RESPONSE PLAN FOR CHRONIC WASTING DISEASE (CWD) OF FREE-RANGING AND PRIVATELY OWNED CERVID" July 12.2012
[21] Code of Colorado Regulations 8 CCR 1201-17 (3.2), (3.4), and (3.5)
[23] Wisconsin ATCP 10.47 (4)

	Minnesota's disease management zone through … a late-season hunt that begins Saturday, Jan. 6."[24], [25]	stations throughout the state, however, the stations are concentrated in CWD affected counties. Testing is provided free of charge.[26]	**present the head at a deer check station** within the business hours of the next 72 hours after killing the deer."[27]	**received legal authority to mandate testing.[28]** Sample collection is offered at various stations throughout the state. Testing is provided free of charge.[29]	**not all can be tested.** New measures may be released in September 2018.[30] "All rifle season buck hunters licensed for Game Management Units (GMUs) … who harvest a buck are required to submit their	

[24] Minnesota Department of Natural Resources. "Deer Hunting Season Information" webpage. https://www.dnr.state.mn.us/hunting/deer/index.html Accessed 7/4/2018

[25] Minnesota Department of Natural Resources. "Testing required through Jan. 14, 2018" webpage. https://www.dnr.state.mn.us/cwd/603/index.html Accessed 7/5/2018

[26] Wisconsin Department of Natural Resources. "Sampling for chronic wasting disease" webpage. https://dnr.wi.gov/topic/wildlifehabitat/registersample.html Accessed 7/4/2018

[27] Michigan Department of Natural Resources webpage. "Deer". https://www.michigan.gov/dnr/0,4570,7-350-79119_79147_81438---,00.html Accessed 7/4/2018

[28] Wyoming Game and Fish Department. "New regulation to help measure effectiveness of future CWD management" https://wgfd.wyo.gov/News/New-regulation-to-help-measure-effectiveness-of-fu Accessed 7/9/2018

[29] Wyoming Game & Fish Department. "CWD Testing" webpage. https://wgfd.wyo.gov/Wildlife-in-Wyoming/More-Wildlife/Wildlife-Disease/CWD-in-Wyoming-Wildlife/CWD-Testing Accessed 7/4/2018

[30] Denver Post. "A disease that attacks the brains of deer, elk and moose has hit 16 percent of male animals tested in Colorado — and hunters need to be cautious". Published May 21, 2018. https://www.denverpost.com/2018/05/21/chronic-wasting-disease-deer-elk-moose-colorado/ Accessed 7/4/2018

					deer head or have a CWD sample taken. In addition, all deer hunters in GMU 33 who harvest a deer of either sex are required to submit their deer head or have a CWD sample taken."[31]	
Requirements for Donated Deer from Non-CWD Units	MDA- *Processor:* **All Firearm harvested venison product must be X-rayed** before serving. Any containing lead will be disposed of.	DNR- *Processor:* Accept only carcasses that appear clean and wholesome upon processor inspection.	Michigan Sportsmen Against Hunger- *Processor:* May accept whole carcass or customer may decide to donate 1-2 pounds from a	Currently there in not a statewide donation program.	Farmers and Hunters Feeding the Hungry: *Processor:* No testing is required.[37] CPW: *Hunter:* No testing is required. Must	Requirements are set by each Province.

[31] Colorado Parks and Wildlife Department. "" https://cpw.state.co.us/Documents/Research/CWD/Mandatory-CWD-FAQ-2017.pdf Accessed 7/4/2018
[37] Farmers and Hunters Feeding the Hungry. "BUTCHER & FEEDING PROGRAM PARTICIPATION" webpage. https://www.fhfh.org/butchers-and-feeding-programs.html Accessed 7/5/2018

	Accept only carcasses that appear clean and wholesome upon processor inspection.[32] *Hunter:* Must adhere to field dressing guidelines, sign donation form, provide proof of legal harvest, and hide must be intact. Processing is done at no cost to the hunter.[33]	*Hunter:* Deer must be legally harvested in WI and registered. Field dressed deer must be taken to a participating meat processor. The whole carcass (except the head and antlers) must be donated. Appears clean and wholesome upon processor inspection Complete a	deer the customer is having processed for the customer's personal use. Customer pays for the processing of the 1-2 pounds of donated meat. Accept only carcasses that appear clean and wholesome upon processor inspection. *Hunter:* No requirements were stated. Processing is		have proof of legal harvest.[38]	

[32] Minnesota Department of Agriculture. "Processor Participation Requirements - 2009 Venison Donation Program" webpage. http://www.mda.state.mn.us/licensing/inspections/meatpoultryegg/venisondonation/processorinfo.aspx Accessed 7/5/2108

[33]Minnesota Department of Agriculture. "Guide to donating hunter-harvested deer in Minnesota" brochure. https://files.dnr.state.mn.us/recreation/hunting/deer/venison-donation-guide.pdf Accessed 7/5/2018

[38] Colorado Parks and Wildlife. "2017 Colorado Big Game 2017" brochure. http://www.eregulations.com/wp-content/uploads/2017/08/biggame.pdf Accessed 7/5/2018

		donation form. Processing and donation are free to the hunter. Processing is done at no cost to the hunter.[34],[35]	done at no cost to the hunter if whole carcass is donated.[36]			
Testing Requirements for Donated Deer from CWD Units	There are no additional requirements at this time.	DNR- **Adult deer** harvested from 12[a] counties affected by CWD must be tested. **Adult deer and fawns** from the remaining 7[b] counties must be tested. The processed venison will be held until	There are no additional requirements at this time.	There are no additional requirements at this time.	There are no additional requirements at this time.	Requirements are set by each Province.

[34] Wisconsin Natural Resource Department. "Wisconsin Deer Donation" webpage. https://dnr.wi.gov/topic/hunt/donation.html Accessed 7/5/2018
[35] Wisconsin Statute 29.89 "Venison and wild turkey processing and donation program." Accessed 7/5/2018
[36] Michigan Sportsmen Against Hunger. "MSAH AFFILIATED PROCESSOR GUIDELINES MANUAL 2018" brochure. https://docs.wixstatic.com/ugd/a0a290_168f33569a0446e49fd48d32e1485af5.pdf Accessed 7/5/2018

		results are known. All other requirements are the same for the remainder of the state as outlined above.[32]				

[a] Adams, Crawford, Grant, Green, Jefferson, Juneau, Kenosha, Lafayette, Portage, Racine, Vernon, and Waukesha counties.

b Columbia, Dane, Iowa, Richland, Rock, Sauk and Walworth counties.

Appendix 5: Deer Farm regulations in Minnesota, Wisconsin, Michigan, and Canada

CWD Testing Requirements by State for Farm-Raised Deer	
Minnesota	Annual Requirements For Operation:
	• An animal inventory must be submitted annually (every 12 months) for each farmed cervidae[1] herd and include all identification, the age, sex, and type of animal. All tag numbers, letters, and colors must be included as they appear on each tag. • The accuracy of the inventory must be verified by the owner and an accredited veterinarian. • Fees: All cervidae producers are required to pay an annual inspection fee of $10 per animal in the herd, up to a $100 maximum by January 1. • Inspection: Each farmed cervidae facility must be inspected by an agent of the Board of Animal Health at least once each year to verify compliance with Minnesota statutes and rules. [2] CWD Herd Certification Program Enrollment: **Mandatory**
	CWD Testing Requirements:
	"**All farmed cervidae producers are required to test their herds for CWD**. From each herd, all farmed cervidae 12 months of age and older that die or are slaughtered, must be tested for CWD. Tissue samples are tested for CWD at the University of Minnesota Veterinary Diagnostic Laboratory" " All farmed cervid herds at a Level 6 Certified status must now submit both obex and lymph node tissues for testing."
Wisconsin	Annual Requirements For Operation:
	Farm Raised Deer (FRD) in Wisconsin, FRD Keeper (FRDK) must: • Register as a FRD Keeper (FRDK) with DATCP every year (registration expires March 15) and include any partial owner information on the registration • Register farm, free of charge, as a livestock premises every three years

[1] Cervidae is the biological Family classification for deer and elk. Cervid is a mammal from the cervidae family.
[2] Minnesota Board of Animal Health. "Minnesota Farmed Cervidae Handbook. Second Edition. 2017" booklet. https://www.bah.state.mn.us/media/FarmedCervidaeHandbk-Accessible.pdf Accessed 7/5/2018

	• Allow DATCP staff access to herd for disease testing and inspection upon request • Test FRD for CWD
CWD Testing Requirements by State for Farm-Raised Deer (continued)	
	• Report all escapes to DATCP and local DNR warden within 24 hours • Obtain a DNR fencing certificate if white-tailed deer are in the registered herd <u>Fees:</u> • 15 or fewer deer not enrolled in in CWD Herd Status Program that only move to slaughter: $85 • 15 or fewer deer enrolled in the CWD Herd Status Program: $162.50 • More than 15 deer: $325[3] CWD Herd Certification Program Enrollment: **Optional**
Wisconsin (cont.)	CWD Testing Requirements: "CWD testing of farm-raised deer is required regardless of whether or not the herd is enrolled in the CWD Herd Status Program, as follows: Herds enrolled in the CWD Herd Status Program with **LESS THAN 5 YEARS** of status must test the following deer that are <u>at least 12 months of age:</u> • 100% that die or are killed, including escapes • 100% that are shipped directly to a slaughtering establishment Herds enrolled in the CWD Herd Status Program with **AT LEAST 5 YEARS** of status must test the following deer that are <u>at least 12 months of age:</u> • 100% that die or are killed, including escapees • 25% that are shipped directly to a slaughtering establishment

[3] Wisconsin Department of Agriculture, Trade, and Consumer Protection. "Deer Farms" webpage. https://datcp.wi.gov/Pages/Programs_Services/DeerFarms.aspx Accessed 7/5/2018

	Herds **NOT ENROLLED** in the CWD Herd Status Program must test the following deer that are at least 16 months of age: • 100% that die by accident, natural causes, or escape
CWD Testing Requirements by State for Farm-Raised Deer (continued)	
	• 50% that are killed by hunt on a hunting ranch • 50% that are killed intentionally 25% that are shipped directly to a slaughtering establishment"[4]
Michigan	Annual Requirements For Operation:
	Every 3 years: • Register with Michigan Natural Resource Department in one of 4 classes (Hobby, Exhibit, Ranch, Full) • Records of animal identification, gender, age, test certificates, date and method of disposition from the herd and details, and escape details. Must be kept for 7 years. • Inventories must be kept Fees: • Class 1 (Hobby) unknown • Class 2 (Exhibition) $450 • Class 3 (Ranch) $750 • Class 4 (Full) $750 CWD Herd Certification Program Enrollment: **Optional**
	CWD Testing Requirements:
	• Upon the death of an animal in the herd, 12 months and older, or of an age determined by the state veterinarian, for any reason, the owner shall have the animal tested for CWD. • The tissue samples to be collected shall include obex and medial retropharyngeal lymph node. • The tissue samples must be collected by one of the following: an accredited veterinarian, an official laboratory, a veterinarian employed by the state or federal government.

[4] Wisconsin Department of Agriculture, Trade, and Consumer Protection. "CWD Testing Requirements" webpage. https://datcp.wi.gov/Pages/Programs_Services/CWDTestingRqmts.aspx Accessed 7/5/2018

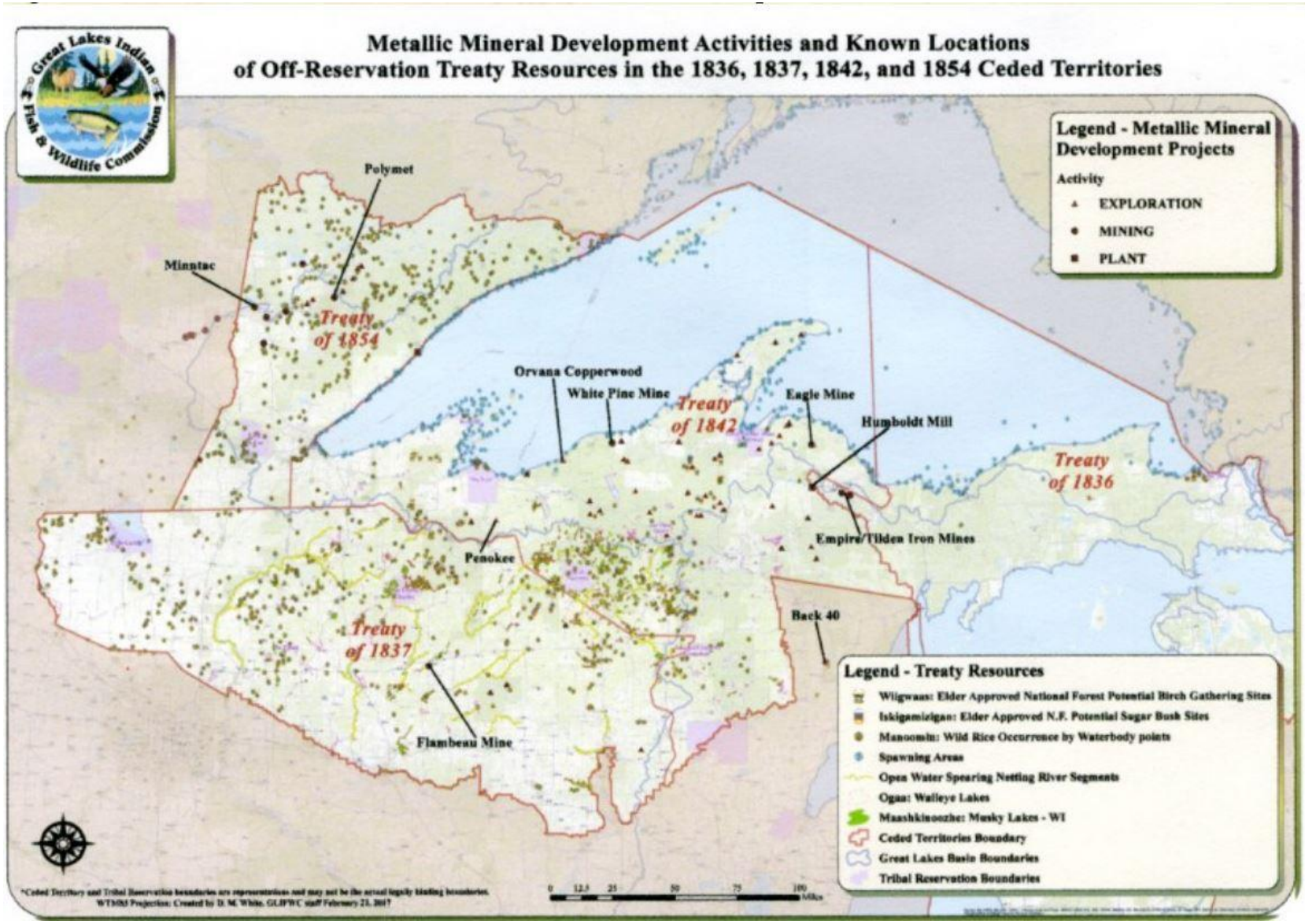
CWD Testing Requirements by State for Farm-Raised Deer (continued)	
Canada's, National Voluntary Herd Certification Program	Annual Requirements For Operation:

	• Voluntary enrollment in the program
	• Annual inspection by accredited veterinarian
	• Annual reports sign by owner, accredited veterinarian or qualified National Herd Certification staff.
	  • Annual inspection report
	  • Inventory reconciliation
	  • Documents for animal movement off of premise
	  • Necessary lab report

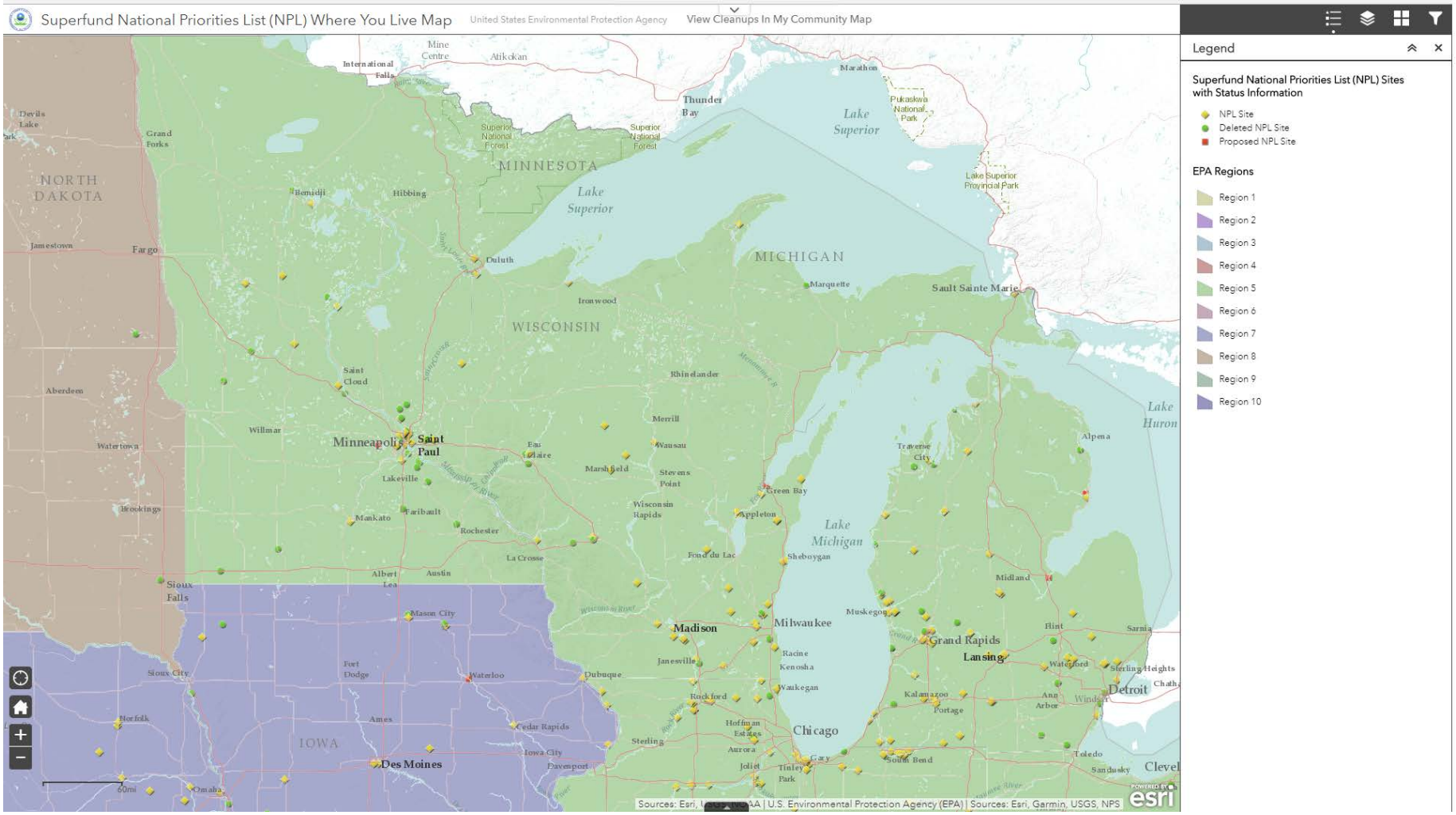
	• Perimeter fence report
	CWD Testing Requirements:
	• All deer 12 months and older that die of any cause, must be tested for CWD. • Beginning January 1, 2018, the requirement to submit samples for CWD testing will also include 50% of any cervids on the premises slaughtered at any abattoir (including US abattoirs) or on farm. • January 1, 2019, the slaughter requirement for CWD testing will increase to 75%, • January 1, 2020, the slaughter requirement for CWD testing will increase to 100%. • The obex and the RPLN must both be submitted for all farmed cervids tested for CWD[5]

[5] Canadian Food Inspection Service. "National Standards for the CWD VHCP" document. Section 4.2.1 http://www1.agric.gov.ab.ca/$Department/deptdocs.nsf/all/rsb7193/$FILE/CWD_VHCP_NationalStandards2017.pdf Accessed 7/9/2018

Appendix 6: Metallic Mineral Development in the Ceded Territories



APPENDIX 7: Map of Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) sites. These areas are commonly referred to as "Superfund" sites.



Appendix 8: Heavy Metal Limits in the United States, Canada, the European Union, and the World Health Organization.

Safety and health of food supplies is a top priority for the US, Canada, European Union member countries, and the World Health Organization. Each of these jurisdictions have set levels or limits to the amount or concentration of heavy metals in specific foods or food contact surfaces. Typically referred to as Maximum Level (ML), these levels are often based on the best available science and aimed at protecting human health. By setting limits, jurisdictions are creating parameters for food industries that are actionable and enforceable. Foods that are above the set limit are to be excluded from the food supply.

The following tables represent the maximum allowable levels of heavy metals per country at time of publication[1]. MLs vary from jurisdiction to jurisdiction and from food to food. Some countries have limits on only a few items per heavy metal and others have set limits for a large quantity of foods. The MLs related or possibly related to traditional foods have been bolded for easier reference. For jurisdictions that have MLs for more than 5 non-traditional foods only the traditional foods have been represented.

Note: Unless otherwise noted, the levels listed for the United States are set by the FDA unless otherwise listed.

Lead	
UNITED STATES	
0.1 ppm	Candy (likely to be consumed frequently by small children)[2]
0.1 ppm	**Imported Dried Fruits** (mostly raisins, dates, prunes/plums)
0.005 ppm	Bottled Water
0.05 ppm	**Fruit juice**
CANADA	
10 ppm	**Edible bone meal**
0.5 ppm	**Maple Syrup**
1.5 ppm	Tomato paste; Tomato sauce
0.5 ppm	**Fish protein**; Whole tomatoes
0.2 ppm	Beverages
0.15 ppm	Evaporated milk; Condensed milk; Concentrated infant formula
0.08 ppm	Infant formula when ready to serve

[1] Maximum levels are revised often by each jurisdiction. For the most up to date information refer to the jurisdiction's website or contact their outreach offices directly.
[2] https://www.fda.gov/Food/FoodborneIllnessContaminants/Metals/ucm557424.htm Accessed 6/20/2018

Lead (continued)

CANADA	
0.05 ppm	**Fruit juice; Fruit nectar**
0.01 ppm	Water sealed in containers
EUROPEAN UNION[3]	
0.02 ppm	Infant formula and follow-on formula
0.1 ppm	**Meat (excluding offal) of** bovine animals, sheep, pig, and **poultry**
0.5 ppm	**Offal of** bovine, sheep, pig, and **poultry**
0.3 ppm	**Muscle meat of fish**
0.2 ppm	**Legumes** and **pulses**
0.2 ppm	**Berries** and small fruit
0.1 ppm	**Fats and oils,** including milk fat
WORLD HEALTH ORGANIZATION[4]	
0.1 ppm	**Berries** and other small fruits
0.2 ppm	**Cranberries**
0.2 ppm	**Elderberries**
0.1 ppm	**Canned fruits**
0.4 ppm	**Jams, Jellies, Marmalades**
0.03 ppm	**Fruit juices and nectars**
0.1 ppm	**Bulb vegetables**
0.1 ppm	**Legumes** and **pulses**
0.1 ppm	Meat of cattle, pigs, and sheep
0.1 ppm	**Meat and fat of poultry**
0.5 ppm	**Edible Offal** (cattle, pigs, and **poultry**)
0.1 ppm	**Edible Fats and Oils**
0.3 ppm	**Fish**
2 ppm	Salt, food grade

[3] Commission Regulation (EU) 2015/1006 of 25 June 2015 amending Regulation (EC) No 1881/2006 as regards maximum levels of inorganic arsenic in foodstuffs
[4] Codex Alimentarius: International Food Standards. "General Standards for Contaminants and Toxins in Food and Feed" CXS 193-1995. Amended 2017.

Mercury	
UNITED STATES	
1 ppm	**methyl mercury in edible portion of fish**, shellfish, and crustaceans (CPG 540.600)
CANADA	
0.5 ppm	**In the edible portion of all retail fish**, with six exceptions (see the 1 ppm maximum level below).
1 ppm	The edible portion of escolar, orange roughy, marlin, fresh and frozen tuna, shark, and swordfish
0.5 ppm	**All fish products** (except swordfish, shark, fresh and frozen tuna, escolar, orange roughy and marlin)[5]
1 ppm	Swordfish, shark, fresh and frozen tuna, escolar, orange roughy and marlin[11]
EUROPEAN UNION[6]	
0.5 ppm	**Fishery products and muscle meat of fish**, excluding species listed in the following section. The maximum level for crustaceans applies to muscle meat from appendages and abdomen. In case of crabs and crab-like crustaceans (Brachyura and Anomura) it applies to muscle meat from appendages.
1 ppm	Muscle meat of the following fish: anglerfish (Lophius species) Atlantic catfish (Anarhichas lupus) bonito (Sarda sarda) eel (Anguilla species) emperor, orange roughy, rosy soldierfish (Hoplostethus species) grenadier (Coryphaenoides rupestris) halibut (Hippoglossus hippoglossus) kingklip (Genypterus capensis) marlin (Makaira species) megrim (Lepidorhombus species) mullet (Mullus species) pink cusk eel (Genypterus blacodes) **pike** (Esox lucius) plain bonito (Orcynopsis unicolor) poor cod (Tricopterus minutes) Portuguese dogfish (Centroscymnus coelolepis) rays (Raja species) redfish (Sebastes marinus, S. mentella, S. viviparus) sail fish (Istiophorus platypterus) scabbard fish (Lepidopus caudatus, Aphanopus carbo) seabream, pandora (Pagellus species) shark (all species) snake mackerel or butterfish (Lepidocybium flavobrunneum, Ruvettus pretiosus, Gempylus serpens) sturgeon (Acipenser

[5] http://www.inspection.gc.ca/DAM/DAM-food-aliments/STAGING/text-texte/fish_man_standardsmethods_appendix3_1406403090196_eng.pdf
[6] Commission Regulation (EU) 2015/1006 of 25 June 2015 amending Regulation (EC) No 1881/2006 as regards maximum levels of inorganic arsenic in foodstuffs

	species) swordfish (Xiphias gladius) tuna (Thunnus species, Euthynnus species, Katsuwonus pelamis)
Mercury (continued)	
EUROPEAN UNION[7]	
0.02 ppm	Other farmed terrestrial animals (Alpaca, Bactrian camel, Capybara, Cottontail/**American rabbit**, Dromedary, Eland, Elk/moose, Emu, Fallow deer, Guinea pig, Hare (farmed), Llama, Nandu/greater rhea, Ostrich, Peccari (collared), Rabbit, Red deer, Reindeer, Roe deer, **Other farmed terrestrial animals**,)[13]
0.01 ppm	**Duck**[13]
0.01 ppm	**Berries and small fruits**[13]
0.01 ppm	(b) strawberries (Musky strawberries, wild strawberries,)[13]
0.01 ppm	**Cranberries** (Cloudberries, Crowberries, Crowberries, Crowberries, Crowberries, Muntries, Partridge berries, Small cranberries/European cranberries,)[13]
0.01 ppm	SUGAR PLANTS: Others (2) (Birches (trunk sap), Manna ashes (trunk sap), **Maples (trunk sap**), Palms (trunk sap), Palms (trunk sap), Other sugar plants,)[13]
0.01 ppm	Rice (African rice, Hybrid Nerica®, Indian rice/**wild rice**,)[13]
0.02 ppm	**Hazelnuts**/cobnuts (Acorns, Filberts,)[13]
0.02 ppm	**Peas (with pods)** (Asparagus peas, Chickling vetches, Chickpeas/Bengal gram, Garden peas/green peas/mangetout/snow peas/split peas/sugar peas, Moringa/drumstick tree pods,)[13]
0.05 ppm	Cultivated fungi (Common mushrooms/button mushrooms/champignons mushrooms, Corn smuts/ Mexican truffles, Enokitake/winter mushrooms, Fusarium venenatum, Horse mushrooms, , Oyster mushrooms, Paddy straw mushroom, Pom-pom blancs/lion's mane mushrooms/monkeyhead mushrooms, Shiitake, Shimeji/bunashimeji/beach mushrooms, Snow mushrooms/white jelly mushrooms, Wood blewits/pied bleus, Other cultivated fungi, Other species of genus Pleurotus, not elsewhere mentioned,)[13]
0.01 ppm	**Bulb vegetables**[13]

[7] Commission Regulation (EU) 2015/1006 of 25 June 2015 amending Regulation (EC) No 1881/2006 as regards maximum levels of inorganic arsenic in foodstuffs

Mercury (continued)	
WORLD HEALTH ORGANIZATION	
0.5 ppm	**Fish**
1 ppm	**Predatory Fish** (shark, swordfish, tuna, pike, and others)

Arsenic (Inorganic)	
UNITED STATES	
0.1 ppm	**Infant Rice Cereal** (draft guidance)[8]
0.01 ppm	Apple juice[9]
0.35 ppm	Citrus Fruit (EPA)[10]
0.01 ppm	Bottled Drinking Water[11]
CANADA	
3.5 ppm	**Fish Protein**
1 ppm	**Edible Bone Meal**
0.1 ppm	Beverages; Fruit juice; Fruit nectar
EUROPEAN UNION (inorganic)[12]	
0.2 ppm	Non-parboiled milled rice (polished or white rice)
0.25 ppm	Parboiled rice and **husked rice**
0.3 ppm	Rice waffles, rice wafers, rice crackers and rice cakes
0.1 ppm	**Rice destined for the production of food for infants and young children**
WORLD HEALTH ORGANIZATION	
0.1 ppm	**Edible Fats and Oils**
0.35 ppm	**Rice, hulled**
0.5 ppm	Salt, food grade

[8] https://www.fda.gov/Food/FoodborneIllnessContaminants/Metals/ucm319870.htm Accessed 6/20/2018

[9] https://www.fda.gov/Food/GuidanceRegulation/GuidanceDocumentsRegulatoryInformation/ucm360020.htm#action_level Accessed 6/20/2018

[10] EPA 2005j 40 CFR 180.289

[11] 21 CFR 165.110

[12] European Union. "COMMISSION REGULATION (EU) 2015/1006 of 25 June 2015 amending Regulation (EC) No 1881/2006 as regards maximum levels of inorganic arsenic in foodstuffs" 2015.

Copper	
USA	
1.3 ppm	Water (EPA)[13]
1 ppm	Bottled Water[14]
CANADA	
1.3 ppm	Drinking water[15]
EUROPEAN UNION[16]	
2 ppm	Others (2) (Bactrian camel, Dromedary, **Elk/moose**, Reindeer, Other milk producer animals,)[15] (Listed below "Horse (Species listed with code numbers 1015000-xxx,)")
5 ppm	**Duck fat[15]**
2 ppm	**Duck[15]**
5 ppm	**Strawberries (Musky strawberries, wild strawberries)[15]**
5 ppm	**Cranberries (Cloudberries, Crowberries, Crowberries, Crowberries, Crowberries, Muntries, Partridge berries, Small cranberries/European cranberries,)[15]**
5 ppm	SUGAR PLANTS: Others (2) (Birches (trunk sap), Manna ashes (trunk sap), **Maples (trunk sap)**, Palms (trunk sap), Palms (trunk sap), Other sugar plants,)[15]
10 ppm	Rice (African rice, Hybrid Nerica®, Indian rice/**wild rice**)[15]
30 ppm	**Hazelnuts**/cobnuts (Acorns, Filberts,)[15]
20 ppm	Peas (with pods) (Asparagus peas, Chickling vetches, Chickpeas/Bengal gram, Garden peas/green peas/mangetout/snow peas/split peas/sugar peas, Moringa/drumstick tree pods, Pigeon peas,)[15]
20 ppm	**Wild fungi** (Ceps/porcino mushrooms, Chanterelles, Hedgehog mushrooms, Horns of plenty/black trumpets, Morels, Périgord black truffles, Piemont white truffles, Saint George's mushrooms, Scotch bonnet mushrooms, Summer truffles, Other wild fungi, Other species of genus Tuber, not elsewhere mentioned,)[15]
5 ppm	**Bulb vegetables** [15]
WORLD HEALTH ORGANIZATION	
Maximum limits have not been developed for these copper in food.	

[13] EPA 2002d 40CFR141.51(b
[14] FDA 2001a 21CFR165.110
[15] http://www.odwac.gov.on.ca/reports/Lead%20Workshop/09%20lcr_revisions_white_paper_final_10.26.16.pdf
[16] Commission Regulation (EU) 2015/1006 of 25 June 2015 amending Regulation (EC) No 1881/2006 as regards maximum levels of inorganic arsenic in foodstuffs

Cadmium

UNITED STATES[17]	
0.005 ppm	Bottle Water[18]
	Pottery (ceramics)
0.5 ppm leaching solution	Flatware (average of 6 units)
0.5 ppm leaching solution	Small hollowware (any 1 of 6 units)
0.25 ppm leaching solution	Large hollowware (any 1 of 6 units)
CANADA	
0.005 ppm	Drinking water
EUROPEAN UNION[4]	
0.05 ppm	Vegetables and **fruit**, excluding root and tuber vegetables, leaf vegetables, fresh herbs, leafy brassica, stem vegetables
1 ppm	**Fungi**
0.05 ppm	**Meat** (excluding offal) of bovine, sheep, pig and **poultry**
0.05 ppm	**Liver** of bovine animals, sheep, pig, **poultry** and horse
1 ppm	**Kidney** of bovine animals, sheep, pig**, poultry** and horse
0.05 ppm	**Muscle meat of fish**, excluding mackerel (Scomber species), tuna (Thunnus species, Katsuwonus pelamis, Euthynnus species), bichique (Sicyopterus lagocephalus), bullet tuna (Auxis species), anchovy (Engraulis species), swordfish (Xiphias gladius), sardine (Sardina pilchardus)
	Infant formulae and follow on-formula:
0.01 ppm	powdered formulae manufactured from cows' milk proteins or protein hydrolysates
0.005 ppm	liquid formulae manufactured from cows' milk proteins or protein hydrolysates
0.02 ppm	powdered formulae manufactured from soya protein isolates, alone or in a mixture with cows' milk proteins
0.01 ppm	liquid formulae manufactured from soya protein isolates, alone or in a mixture with cows' milk proteins

[17] FDA. "Guidance for Industry: Action Levels for Poisonous or Deleterious Substances in Human Food and Animal Feed" 2000.
[18] CDC. "Cadmium Toxicity: What Are the U.S. Standards for Cadmium Exposure?" 2008. https://www.atsdr.cdc.gov/csem/csem.asp?csem=6&po=7

Cadmium (continued)	
WORLD HEALTH ORGANIZATION	
0.4 ppm	Polished, rice
0.05 ppm	**Blub Vegetables**
0.1 ppm	**Legumes** and **pulses**
0.5 ppm	Salt, food grade

Manganese, Zinc, & Aluminum
UNITED STATES
Maximum limits have not been developed for these metals in food.
CANADA
Maximum limits have not been developed for these metals in food.
EUROPEAN UNION[19]
Maximum limits have not been developed for these metals in food.
WORLD HEALTH ORGANIZATION
Maximum limits have not been developed for these metals in food.

µ Micro

[19] Commission Regulation (EU) 2015/1006 of 25 June 2015 amending Regulation (EC) No 1881/2006 as regards maximum levels of inorganic arsenic in foodstuffs

Appendix 9: Chemicals not of reasonable or likely concern.

Chlordane

Chlordane is a composed of a mixture of about 10 different chemicals.[1] It was used as a pesticide from 1948 to 1988 in the United States. Although it is banned in the United States, it is still used in other countries.

Chlordane strongly binds to soil's upper layers. Due to its insolubility in water, it is not likely to enter into groundwater. Breakdown of chlordane occurs in either water or sediment, but it is unknown where most of the degrading occurs. If this chemical breaks down in the soil, it happens very slowly (ATSDR 2018). Open-lake concentrations of chlordane continue exceeding water quality standards in certain areas (Lake Superior Partnership 2018).

Chlordane can remain in some types of soil for more than 20 years. Persistence of chlordane is less in sandy soil and greater in heavy soils or soils rich in organic matter. Volatilization is more rapid from light, sandy soils than from heavy soils. The half-life of this chemical from the soil surface is 2 to 3 days (ATSDR 2018).

A half-life is "the calculated time for loss of the first 50 percent of a substance." It is important to keep in mind, however, that the time needed for the loss of the remaining half of the substance could be longer. In other words, the remaining portion may degrade somewhat slower than the first half did (ATSDR 2018).

Most chlordane is lost by soil through evaporation. This chemical degrades easiest in the atmosphere where it reacts with light. However, it does stay intact enough to travel long distances and contaminate water or soil far from its source (ATSDR 2018).

Eating food contaminated with chlordane, such as fish, is the most common route of exposure to this chemical (ATSDR 2018).[2] This chemical bioaccummulates in fatty tissues, and also biomagnifies in the food web. As a result, chlordane strongly persists in the environment. Due to its former widespread use and long half-life in soil, it can still be found in animals and humans. However, levels continue to decline the longer chlordane has been banned (Venier et al. 2014).

Liver cancer was detected in rodents exposed to small amounts of chlordane through food over time. As a result, it has been classified as a probable human carcinogen. Humans that ingested large amounts of chlordane experienced digestive and nervous

[1] Major chemical components include beta-chlordene, trans-chlordane, cis-chlordane, and trans-nonachlor (ATSDR 2018).

[2] People that live in homes treated for termites using chlordane before it was banned are exposed to this chemical at higher rates than eating contaminated food (ATSDR 2018).

system problems, like seizures, confusion, stomach cramps, and diarrhea. Problems with the liver indicated by jaundice in these people were also detected (ATSDR 2018).

Lake Superior whitefish, lake herring, and lake trout sampled in a GLIFWC study did not exceed FDA's chlordane action limit of 0.3 ppm. However, siscowet from the 22-23 inch and 24.5-25.5-inch size groups did exceed the FDA's action limit. Action levels were not exceeded in other siscowet size groups.[3]

Trimming fillets reduced chlordane concentrations by 13 to 38 percent, depending on the fish species. Specifically, trimming reduced levels of this chemical 33 percent in whitefish and 34 percent in lake trout. Chlordane concentrations in siscowet were lowered between 13-38 percent, depending on the fish length. Overall, chlordane is no longer considered a consumption-limiting contaminant in Lake Superior fishes. [4]

Harvested waterfowl do not uptake chlordane levels in their breast muscle that would be concerning to human health (Tsuji et al. 2007; Braune and Malone 2006).

DDT & Related Compounds (DDE & DDD)

DDT is one of the first manmade insecticides developed in the modern day.[5] It had a wide usage in combating diseases carried by mosquitoes. DDT was also used extensively in insect control for farms, businesses, and home gardens. In 1972, its application as a pesticide was banned due to its adverse impacts to wildlife[6] and possible risks to human health (US EPA 2017).[7]

DDT is the most abundant, formerly-used organochlorine pesticide in all of the Great Lakes, with the exception of Lake Superior. Since the 1972 U.S. EPA ban, however, concentrations of DDT are steadily declining (ECCC and U.S. EPA 2017).

[3] GLIFWC memorandums from Matt Hudson, Dec. 30, 2005 and April 11, 2006.
[4] Ibid.
[5] The chemical name for DDT is dichloro-diphenyl-trichloroethane. Its widespread use started in the 1940s (US EPA 2017).
[6] DDT has been implicated in thinning of eggshells, interfering with the reproduction of large birds (Porter and Wiemeyer 1969). The thinning is caused by a disruption in enzyme activity involving calcium transport (Kolaja and Hinton 1977).
[7] In America, DDT is completely banned for all uses. In African countries, it is still used to control malaria. It is still used as an agricultural insecticide in some non-industrialized countries (EPA 2017).

DDE and DDD are chemically similar to DDT (ATSDR 2018).[8] DDE is created when DDT breaks down. DDD is a banned insecticide, and also a breakdown product of DDT (ATSDR 2018).

Eating foods that contain small amounts of DDT and related chemicals is how most people are exposed to this contaminant. Fish can contain low levels of DDT. This chemical bioaccummulates in fatty tissues, and also biomagnifies in the food web.

DDT and its related compounds bind strongly to sediment.[9] One scientist calculated DDT's half-life in American temperate soils to be 5.3 years. Other studies have calculated anywhere from 837 to 6,087 days. Highest residues of DDT are found in mucky soils and in unflooded fields that are deeply plowed (ATSDR 2018).

In animal studies, long-term exposure to small amounts of DDT caused liver dysfunction. DDE can raise the chance of a mother having an infant prematurely and cause a reduction in the length of milk production in lactating women. Harmful effects on reproduction were observed in animals that ingested small amounts of DDT over time (ATSDR 2018). DDE exposure has also been associated with instances of diabetes (Turyk et al. 2009).

High concentrations of DDT can affect the nervous system in humans, and cause moodiness, tremors, or seizures. However, the symptoms stop when exposure is removed (ATSDR 2018). These acute symptoms are not likely to occur from DDT or DDE exposure to levels that could be found in some wild, traditional foods. DDT is now considered to be a probable human carcinogen (U.S. EPA 2017).

People that eat Lake Superior fish generally do not have higher levels of DDT in their blood (FdL and MN DOH 2015). Harvested waterfowl do not uptake concerning amounts of DDT in their breast muscle, the portion of the duck most commonly consumed (Tsuji et al. 2007; Braune and Malone 2006).

Mirex and Chlordecone

Mirex and chlordecone and are chemically similar, man-made insecticides that have been banned for use in the U.S. since 1978.[10] These chemicals break down in the environment very slowly, staying for years in soil and water. They have a long half-life estimated to be about 10 years (ATSDR 2018).

[8] The chemical name for DDE is dichloro-diphenyl-dichloroethylene, and the chemical name for DDD is dichloro-diphenyl-dichloroethane (ATSDR 2018).
[9] The half-life of DDT in air exposed to sun is 2 days (ATSDR 2018).
[10] Mirex was also used sometimes as a flame retardant. (ATSDR 2018).

Mirex and chlordecone are not soluble in water, but bind closely to soil particles. As a result, they do not travel very far down into the soil. Also, these chemicals do not volatilize very easily from water or soil. Both mirex and chlordecone bioaccummulate and biomagnify in the environment. However, mirex breaks down more quickly than chlordecone (ATSDR 2018).[11]

A common route of low-level exposure to mirex and chlordane is by eating contaminated animals, particularly fish. These chemicals could occur in the environment from past use or be released from hazardous waste sites. Also, breastfeeding mothers may pass mirex through their milk to nursing infants (ATSDR 2018). Please see Appendix 7 for map of hazardous waste sites in the Ceded Territories.

Chlordecone has a tendency to bind to blood plasma proteins rather than fat tissues, which is unusual for an organochlorine pesticide. Mirex's biological half-life is estimated to be around 435 days. This chemical behaves as expected, however, and attaches to fat.

Animal studies have shown that ingestion of mirex and chlordecone can cause tumors in the liver, adrenal gland, and kidneys. However, short-term, irregular exposures to fairly low levels of mirex did not injure kidneys. Nevertheless, these chemicals are reasonably thought to be a human carcinogen based upon these studies. Kidney problems and negative impacts on development and reproduction in animals were also reported. Studies in workers exposed to chlordecone have found toxic effects on the nervous and reproductive systems, as well as on the liver (ATSDR 2018).

Mirex residues in various animals are much lower than those originally reported around the peak years of the chemical's production and use. All Lake Superior fish sampled by GLIFWC were far below the U.S. FDA's action limit for mirex, which is 0.1 ppm. Fish sampled were lake whitefish, lake herring, lake trout, and siscowet trout.[12]

Harvested waterfowl do not uptake mirex levels in their breast muscle that would be concerning for human health (Tsuji et al. 2007; Braune and Malone 2006).

Aldrin & Dieldrin

[11] Mirex degrades into photomirex, which also can cause harmful health effects. Photomirex is more poisonous than the parent chemical, mirex. Chlordecone breaks down into chlordecone alcohol, which less harmful (ATSDR 2018).
[12] GLIFWC memorandums from Matt Hudson, Dec. 30, 2005 and April 11, 2006.

Aldrin and dieldrin[13] are manmade compounds that have a similar chemical structure. Since 1970, these chemicals are no longer used as an agricultural pesticide, although they were commonly used in the past. In 1987, its use as a termite killer was also discontinued. Aldrin and dieldrin are still present in the environment from when they were commonly used as an insecticide. However, they can also leak from storage containers at waste areas. Please see Appendix 7 for Superfund sites.

Aldrin easily changes into dieldrin upon entering the environment or the human body. Bacteria and sunlight can transform aldrin into dieldrin. Dieldrin strongly binds to soil. It takes a long time for this chemical to break down in either water or soil. Dieldrin does not dissolve well in water. As a result, high concentrations are not found in water. However, it can travel through the air as dust, and settle on areas where it was not found before (ATSDR 2018).

Fish and birds that eat something contaminated with this chemical store it mostly in fat. Plants can uptake smaller amounts of dieldrin from the soil and store it in their roots and leaves.[14] As a result, dieldrin bioaccummulates and biomagnifies in the environment.[15]

All Lake Superior fish sampled in a GLIFWC study were far below the FDA's action levels for these chemical contaminants. Fish sampled in Lake Superior were whitefish, lake herring, lake trout, and siscowet.[16]

Harvested waterfowl do not seem to uptake concerning amounts of dieldrin in their breast muscle (Tsuji et al. 2007; Braune and Malone 2006).

Heptachlor & Heptachlor Epoxide

Heptachlor is a manmade insecticide used for agricultural, commercial, and residential applications until 1988.[17] Bacteria in the environment break down heptachlor into heptachlor epoxide, which is the form of the chemical most likely to be found. Heptachlor does not dissolve easily in water. Heptachlor epoxide is a more water-soluble than its parent chemical. Both chemicals bind strongly to soil, and slowly

[13] Aldrin's chemical name is 1,2,3,4,10,10-hexachloro-1,4,4α,5,8,8α-hexahydro-1,4-endo, exo-5,8-dimethanonaphthalene. The abbreviation of this name is HHDN. Dieldrin's chemical name is 1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4α,5,6,7,8,8α-octahydro-1,4-endo,exo-5,8-dimethanonaphthalene. The abbreviation for this name is HEOD (ATSDR 2018).
[14] Barley and wheat seemed to have higher rates of chemical uptake (ATSDR 2018).
[15] Dieldrin has also been shown to thin eggshells (Porter and Wiemeyer 1969).
[16] GLIFWC memorandums from Matt Hudson, Dec. 30, 2005 and April 11, 2006.
[17] Heptachlor is now approved for only one use now: controlling fire ants in underground power transformers (ATSDR 2018).

evaporate into the air. The epoxide of heptachlor can remain in soil and water for many years (ATSDR 2018).

Eating fish or meat from other heptachlor-exposed animals is the most common way people come into contact with this chemical. However, certain types of plants, mostly root vegetables, have been found to uptake smaller amounts of heptachlor epoxide from the soil.[18] Nursing mothers that have been exposed to high concentrations of this chemical can also expose their infant through breast milk (ATSDR 2018).

The health effects to humans from heptachlor and its epoxide are largely unknown. Nervousness, liver damage, and fertility declines have been observed in animals that have ingested heptachlor. The effects worsened with high and long-term exposure, exposure not likely to occur from consumption of wild, traditional foods. Lifetime heptachlor exposure resulted in liver tumors in animals, which is a reason that heptachlor and heptachlor epoxide are classified as possible human carcinogens (ATSDR 2018).

All Lake Superior fish sampled by GLIFWC were far below the FDA's action levels for heptachlor and heptachlor epoxide. Fish sampled in Lake Superior were whitefish, lake herring, lake trout, and siscowet.[19]

Hexachlorobenzene (HCB)

Hexachlorobenzene (HCB) is a man-made fungicide used in the United States until it was banned in 1984.[20] It is formed as a waste product as other chemicals are manufactured. Smaller amounts of HCB can also be created during the large-scale combustion of household waste (ATSDR 2018).

HCB does not break down well in the air, and as a result, can be transported long distances in the atmosphere. It is insoluble in water, and binds strongly to soil. HCB's half-life in soil and water is 3 to 6 years. This chemical has been documented to bioaccummulate and biomagnify in aquatic environments (ATSDR 2018).

Most exposure to HCB occurs when people eat small amounts present in contaminated food. Long-term exposure to this chemical can cause liver disease, and negative impacts to the reproductive system. Mothers who are breastfeeding can pass HCB to their infant through milk. Infants and small children are particularly sensitive to the effects of

[18] Potatoes, carrots, beets, radishes, cucumbers, and tomatoes have all been found to uptake heptachlor epoxide to varying degrees (ATSDR 2018).
[19] GLIFWC memorandums from Matt Hudson, Dec. 30, 2005 and April 11, 2006.
[20] Commercial HCB production in the United States stopped in the late 1970s (ATSDR 2018).

highly-elevated HCB concentrations. Animal studies suggest that eating HCB-containing food for a long time can cause cancer of the kidney, liver, or thyroid. This chemical likely causes cancer in people (ATSDR 2018).

All Lake Superior fish sampled by GLIFWC were far below the FDA's action levels for HCB. Fish sampled in Lake Superior were whitefish, lake herring, lake trout, and siscowet. [21]

Harvested waterfowl do not uptake HCB levels in their breast muscle that would be concerning for human health (Tsuji et al. 2007; Braune and Malone 2006).

Chlorothalonil

Chlorothalonil is an organochlorine fungicide commonly used in commercial potato farming (USDA 2017).[22] A large swath of northeastern and west-central Wisconsin within the ceded territory falls within potato-growing country.[23] Overall, it is the most widely used pesticide for this crop, and was applied to 99 percent of the acres for planted potatoes within the state (USDA 2017).

The half-life of chlorothalonil in aerobic, or oxygen-abundant, soil is 1 to 3 months. Increased soil temperature or moisture increases the breakdown of this chemical, but it is not degraded by direct sunlight. Chlorothalonil binds more easily to soils containing a higher degree of organic matter. In sandier soils, this chemical is moderately mobile.

Chlorothalonil is not very soluble in water. Nearly 65 percent was degraded after 2.5 months in high pH water.[24] This chemical was found in one sample of surface water in Michigan at 6.5 ppm, which exceeded established levels. The EPA's reference dose is currently 0.015 ppm (U.S. EPA 1987).

Chlorothalonil is quickly excreted from the body, mostly in an unchanged form. Studies in rodents and dogs showed the chemical leaving the body via urine, feces, and in respiration. At low levels, it was found to be excreted within 24 hours. Therefore, it is not reasonably believed to accumulate in animal tissues.

Rats fed a range of chlorothalonil doses showed no impacts on physical appearance, behavior, and survival, overall. However, kidney enlargement of the rodents was common, which indicates organ dysfunction from large doses. The lowest amount of

[21] GLIFWC memorandums from Matt Hudson, Dec. 30, 2005 and April 11, 2006.
[22] Chlorothalonil is classified as an aromatic halogen compound.
[23] Wisconsin's Florence and Menominee counties do not engage in large-scale, commercial potato agriculture, as indicated on the map provided.
[24] The water was pH 9 (U.S. EPA 1987).

this chemical that was given to rats to eat without adverse symptoms was 60 ppm (US EPA 1987).

However, chlorothalonil is highly toxic to fish and aquatic insects, even at concentrations less than 1 ppm. Fishes, like rainbow trout, bluegill, and channel catfish,[4] exhibit noticeable effects (U.S. EPA 1987).[25] One study found 29 percent of the active ingredient was applied to non-target ground surfaces using typical applicator procedures. Small amounts of the chemical, 0.25 to 0.53 percent, were detected in runoff from sprayed area. Chemical concentrations ranged anywhere from 0.0012 to 0.5 mg/L (1.2 to 500 microgram/L) following the first runoff events (Wilson et al. 2010). However, runoff into a water body would be further diluted upon entrance.

Human health impacts from ingestion of chlorothalonil on wild, traditional foods are projected to be quite low overall. It does not accumulate in fish or animal meat. Direct application to a non-target plant would be unlikely, unless growing directly adjacent to a commercial potato field. This would be potential habitat for some types of berries, like blueberries and raspberries. However, non-target spraying at normal rates of application would likely not be at levels of concern to human health.

Glyphosate

Glyphosate is a widely-used, non-selective[26] herbicide first registered for American use in 1974 (Felsot 2011; Henderson et al. 2010). It is used in agriculture and forestry, and to kill weeds in commercial and residential settings. The largest applications are to corn and soybean fields, as well as pastureland and hay fields. It is also commonly applied to golf courses.

Glyphosate comes in a variety of formulations for either land or water applications.[27] In the U.S., over 750 glyphosate-containing formulations are sold (Henderson et al. 2010). This chemical kills plants by stopping the production of the EPSPS enzyme, which is essential for plant growth (Felsot 2011; Henderson et al. 2010).[28]

[25] LD is an abbreviation for "Lethal Dose". LD_{50} is the amount of a pollutant, given at one time, that causes 50 percent of a group of test animals to die. It is a common measure of acute, or short-term, toxicity. Rainbow trout's LD50 is 0.25 mg/L. Bluegill's LD50 is 0.3 mg/L. Channel catfish's LD50 is 0.43 mg/L. For reference, LD50 for mallards is 5,000 mg/L (U.S. EPA 1987).
[26] This means it will kill most plants it comes in contact with, that is, it does not select between plants.

[28] An enzyme is also a protein, increasing biological reaction times. The shikimic acid pathway requires the EPSPS enzyme, which is killed by glyphosate.

Glyphosate binds strongly to soil with high organic matter content. Glyphosate's half-life in soil is 1 to 174 days. However, the herbicide could be swept along by erosion and carried into water, if not directly applied to waterways. In ponds, the half-life of glyphosate is 12 days to 10 weeks. This chemical is broken down mainly by microbes. Its major metabolite is aminomethylphosphonic acid, or AMPA.

Studies were conducted that fed glyphosate to animals for long periods of time. Chickens, mice, rats, dogs, and rabbits were fed the chemical for as long as two years. There were, for the most part, no easily observable symptoms. Cell function, blood chemistry, and organ function were essentially not affected, even at the highest chemical concentrations tested (Henderson et al. 2010).

Researchers have debated for years whether glyphosate causes cancer. The evidence largely indicates that it does not cause cancer. Nonetheless, in 2015, this chemical was classified as "probably carcinogenic to humans" by IARC despite limited available evidence due to the ongoing debate.

Rats given the maximum glyphosate dose of 31 ppm/day tested did not exhibit toxic effects. Likewise, no ill effects were observed in a study of dogs eating the maximum dose tested, which was 500 ppm/day. Very high glyphosate amounts administered over the course of animals' lifetimes produced a minor reduction in body weight. However, slight changes to liver and kidney were observed in some animals (Henderson et al. 2010).

Two people that ingested large amounts of glyphosate had peak concentrations of the chemical in their blood plasma within 4 hours of ingestion. After 12 hours, glyphosate was nearly undetectable. Similar results have been found for AMPA, glyphosate's major metabolite. These findings indicate fairly rapid excretion of the actual chemical and AMPA from the human body.

Glyphosate can run off fields where it has been applied repeatedly, and at normal rates of application. It can also be taken up into the plant from roots. In this manner, glyphosate can impact plant functioning in plants found in agricultural ditches, including plant mortality. Some plants, however, are more tolerant of glyphosate residues (Saunders and Pezeshki. 2015).

Glyphosate is not the only chemical found in its formulations. Many formulations contain petroleum-based oxidized molecules, like POEA.[29] These chemicals are used in enhancing the ability of the herbicide to be applied to the plant, like as a surfactant.

[29] POEA stands for polyoxyethylene tallow amine. This chemical is a common surfactant in glyphosate formulations, and exhibits toxicity to aquatic organisms. (USGS 2018)

Surfactants help evenly spread the pesticide across the plant. Additional chemicals in the formulas are often considered inert, or chemically inactive. However, emerging evidence suggests that formulants may have more toxic effects on human cells than glyphosate itself. Traces of arsenic, cobalt, chromium, lead and nickel have also been found in some glyphosate-based herbicides (Defarge et al. 2018).

Glyphosate[30] uptake studies in forest blueberries and raspberries showed that less than 10 percent of the chemical entered fruit nine hours following application. A gradual decline in chemical residue levels over time were observed. The half-life of glyphosate on the berries were less than 20 days and less than 13 days for blueberry and raspberry fruit, in that order. Initial residue concentrations lowered to about 4 and 6 percent after 61 and 33 days for the blueberry and raspberry, respectively (Roy et al. 1989).

Glyphosate is not believed to bioaccumulate in animals or plants to any significant extent. Also, negative impacts in animals were at extremely high, repeated doses, which would not be typical at all in the human diet consisting of wild, traditional foods. However, people harvesting plants, rabbits, or hares next to golf courses and areas of intensive agriculture during the growing season are at increased risk of exposure. Wild turkeys are not typically eating green plant material when this chemical would be applied, so this species would be unlikely to be impacted.

2, 4-D

2,4-D is a selective, systemic[31] herbicide used to control plants with relatively broad, flat leaves. Major pesticide uses include residential lawns, roadways, pasture and rangeland, and cropland. It is also commonly applied to golf courses to control dandelions (Extension Toxicology Network 1993).

Once inside the plant, 2,4-D mimics a growth hormone called auxin, which causes uncontrolled and disorganized cell division within the target plant, typically within the stem region. This chemical can be found in several different forms depending on its specific formulation.[32] It has both land and water uses. Over 46 million pounds are applied annually in the U.S. for aquatic and terrestrial applications (Jervais et al. 2008).

2,4-D is one of the most common herbicides for aquatic invasive plant removal in the U.S. This chemical is commonly used in area lakes to control invasive Eurasian

[30] Vision® was the formulation used (Roy et al. 1989).
[31] Systemic herbicides are moved to other parts of the plant after first being absorbed by leaves or roots. 2,4-D has been registered for pesticide use since the 1940s.
[32] It can be an ester, amine or salt.

watermilfoil.[33] It is commonly applied for this application during spring or early summer when it is most effective (WDNR 2012).

As an aquatic herbicide, dimethylamine (DMA) salt and butoxyethyl ester (BEE) salt forms are used. The BEE formulation is more toxic to fishes and other aquatic organisms compared to the DMA form. Both forms are water soluble and break down rapidly to the 2,4-D acid in aquatic systems. The DMA salt is broken down to 2,4-D acid in less than 3 minutes. The BEE salt breaks down to 2,4-D acid within 1 to 3 days (WDNR 2012).

The 2,4-D acid itself is broken down in the aquatic environment primarily by microorganisms. Microbial degradation occurs mainly in sediments, with little taking place in the water column. The half-lives for 2,4-D acid in the field range from approximately 3 to 40 days. Half-lives under anaerobic conditions were over 300 days. Breakdown time increases with higher water and sediment temperatures, elevated dissolved oxygen levels, and increased nutrient loading. Breakdown rates for the chemical are slower in lakes that have not previously been treated, since bacteria present in the waterbody need to be able to break the chemical down (WDNR 2012).

Two low-dose (0.5 and 0.275 ppm), whole-lake treatments by the WDNR in Bayfield County found the 2,4-D (DMA) half-life to be 34 and 41 days in the water column, representing the upper end of recorded half-lives. 2,4-D remained at measurable concentrations in one lake for greater than 166 days after treatment (166 days after treatment was the final sample taken). Other studies have reported 2,4-D time to disappearance to be more typically 1 week to 2 months in water, and 2 weeks to 3 months in sediment. However, residues in sediment have been observed from 6 to 9 months (WDNR 2012).

2,4-D persistence in waterbodies is physically governed by transport of treated waters away from the treated area via water movement and circulation of water. This chemical does not bind strongly to sediments, though it can bind to a moderate degree to floating sediments with high organic content, especially in acidic lakes. This makes 2,4-D highly mobile and raises concerns about groundwater contamination. In fact, 2,4-D has been detected in groundwater throughout the U.S. and Canada, especially in rural areas where it is used as a terrestrial pesticide (WDNR 2012).

Liquid formulations will result in a higher initial 2,4-D water concentration. Granular (solid, grain-like) formulations have a longer persistence due to slow release and lead to lower water concentrations. Granules sink and release 2,4-D at the lake bottom. Liquid treatments take place at or near the surface of the water. As a result, sediment

[33] The scientific name for Eurasian watermilfoil is *Myriophyllum spicatum.*

concentrations are generally lower following liquid versus granular formulation treatments. Applications of granular formulations can result in excessive 2,4-D concentrations in sediment (WDNR 2012).

Some, but not all, studies have found an association between 2,4-D and non-Hodgkin's lymphoma in highly exposed populations. IARC determined the class of chemicals it is in (chlorophenoxy herbicides) to be possibly carcinogenic to humans in 1987. Based on limited evidence, EPA chose to place 2,4-D in the Group D category, meaning "not classifiable as to human carcinogenicity" in 2004 (Jervais et al. 2008).

There is also some evidence that 2,4-D may be an endocrine disruptor, having estrogen-like effects in humans. Two of the breakdown products of 2,4-D may also potentially affect male reproductive development. These impacts would not be expected at exposure levels occurring from occasional contact with treated water or consumption of fish from treated waters. However, exposure should be kept to a minimum since people could come in contact with this chemical from other sources, such as drinking water (Jervais et al. 2008; WDNR 2012).

The EPA states that there is no significant risk to recreational users of treated waters, though there is a 24-hour waiting period for swimming when the BEE formulation of 2,4-D is applied. There are no restrictions on fishing or boating in treated waters. Due to the low potential for bioaccumulation in animals, fish consumption from treated waters is not restricted. 2,4-D increases in edible fish tissue for a few days following application and is then rapidly eliminated (WDNR 2012).

Concern has arisen that 2,4-D may uptake into wild rice. No evidence exists that its systemic uptake into plants is harmful to human health, since the substance is not believed to bioaccumulate. However, wild rice is submergent during the application period for this chemical, and may be inadvertently injured or killed upon application. People harvesting berries would not have to worry about this chemical since the growing season is typically later than recommended timing of chemical application. Also, any berry or grape plant would likely be killed or significantly damaged by harvest time if accidentally sprayed.

Endothall

Endothall is the common name of endothal acid.[34] This chemical is a nonselective, contact herbicide.[35] In other words, it kills nearly all plants it comes into direct contact with.

Endothall is used in a variety of formulations to control both terrestrial and aquatic plants. Granular (solid) and liquid types of endothall are commonly used for aquatic weed control. It is also used on agricultural crops, like potato, sugar beet, hops, and alfalfa. On turf, it is used to annual grasses and broadleaf weeds (Extension Toxiology Network 1995). Annual grasses are more sensitive to the effects of endothall compared to perennial grasses (Koschnick et al. 2005). It is not really understood how endothall kills plants, but it is believed to interfere with plant respiration (Extension Toxiology Network 1995).

In waterways, this chemical is used mostly to treat invasive curly-leaf pondweed (*Potamogeton crispus*), although it is also effective against Eurasian watermilfoil (*Myriophyllum spicatum*) and a number of other invasive aquatic plants. It is normally applied in late spring and early summer, during active periods of growth (WDNR 2012).

Two types of endothall are available to use in aquatic applications: dipotassium salt and monoamine salts. The monomethylamine salt formulation of endothall is more toxic to fish and aquatic organisms than the dipotassium salt version (WDNR 2012).

The dipotassium and monomethylamine salt forms of endothall behave similarly in the environment. The dipotassium salt breaks down to the free acid immediately upon addition to water. In contrast, the monomethylamine salt can persist as the salt form to some degree in treated waters. This leads to the higher toxicity of the monomethylamine salt. Liquid formulations will result in a higher initial water concentration, whereas granular formulations have a longer persistence (WDNR 2012).

Endothall is dispersed by water movement. The herbicide is exceptionally stable in water, but is broken down in the environment by microorganisms. Low concentrations can persist in a treatment area for several days to several weeks, depending on environmental conditions. Persistence of greater 62 days has been reported. Its half-life ranges from less than 1 to 10 days. Complete microbial degradation takes between 30 and 60 days. Breakdown time increases with higher water and sediment temperatures, elevated dissolved oxygen levels, and increased nutrient loading (WDNR 2012).

[34] Endothaic acid's chemical name is 7-oxabicyclo[2,2,1] heptane-2,3-dicarboxylic acid.
[35] One formulation is approved for use as an algaecide.

The residence time of endothall in sediments is longer than in water, but sediment concentrations rarely reach levels as high as those in the water column. Because granules sink and release endothall at the lake bottom, in contrast to liquid treatments that take place at or near the surface of the water, sediment concentrations are generally lower following liquid versus granular formulation treatments. Endothall has a low probability of leaching to groundwater, but has been detected in groundwater where recharge areas with sandy or gravel bottoms have been treated (WDNR 2012).

Labels for products containing endothall specify wait times of up to 25 days before treated waters should be used for domestic purposes, for spraying food crops, or as livestock water. There are no restrictions on swimming, fishing, or boating in treated waters. Due to the low potential for endothall bioaccumulation, fish consumption from treated waters is not restricted (WDNR 2012).

Endothall would likely kill or injure wild rice seedlings during an optimal period of growth, but not be uptaken into the plant. Accordingly, low potential exists for significant human exposure via fish consumption from treated areas. Rabbits snared near large-scale potato growing operations during the growing season may be at increased exposure to endothall, but exposure is not at levels likely to impact human health.

Diquat Dibromide

Diquat, or diquat dibromide, is a swift-acting, contact herbicide. A contact herbicide kills just the area where it was applied, and is not taken up into the plant. The mode of action it uses to kill plants is by desiccation, or extreme drying of the plant. It is not selective, meaning it will kill most plants it comes in contact with. This chemical is commonly used in terrestrial and aquatic applications, including aquatic weed control, golf courses, and right-of-ways. It is also used on crops, including control of potato vines (Extension Toxicology Network 1993).

Diquat does not break down from bacterial activity, like many other pesticides used today. It is strongly bonded to silt or clay particles in soil or water, and accumulates in sediment. Its half-life in water is 1 to 35 days. In a waterbody with sandy sediments, it will remain in water longer (WDNR 2012).

A trace contaminant found in diquat products is ethylene dibromide (EDB). It comes about from the manufacturing process. EDB has been classified as a carcinogen. The EPA has set the maximum EDB level in diquat at 10 ppb. This contaminant degrades over time, and it does not persist like diquat itself (Extension Toxicology Network 1993)

Experiments show that walleye, particularly, are very sensitive to diquat, even at standard concentrations. In water treated with diquat at typical application rates, walleye displayed toxic symptoms. Other fish, like bass, northern pike, and bluegills, were not affected at normal rates of application. Bioaccummulation of diquat in fishes is minimal. In many fishes, the half-life of diquat is less than 21 days (Extension Toxicology Network 1993).

No restrictions exist for swimming or eating fish from diquat-treated aquatic areas. Water should not be used for drinking for 1 to 3 days following application, depending on the amount used. Water given to pets or livestock treated with diquat must be held for at least 1 day after treatment. For food crops, the irrigation restriction is 5 days. For lawn applications, it varies from 1 to 3 days depending on the amount used (WDNR 2012).

Long-term diquat exposure can produce cataracts, a clouding of the lens of the eye. When higher amounts were fed to test cats and dogs over a period of time, the instance of cataracts cases increased. In very large amounts not typically found in traditional foods, diquat can be fatal to humans. Studies in monkeys and massive human ingestions show that this chemical is mostly harmful to the digestive system, and the kidneys and liver (Extension Toxicology Network 1993).

Due to the low bioaccumulation potential, diquat ingestion from fishes is not of imminent concern (WDNR 2012). However, this chemical may be toxic to wild rice and walleyes during critical periods of growth. Rabbits snared near large-scale potato growing operations during the growing season are at increased exposure to diquat, but exposure is not likely to be at levels high enough to impact human health.

Appendix 10: Emerging chemicals not reasonably likely to be of concern

PBDE (Polybrominated Diphenyl Ethers)

Polybrominated diphenyl ethers (PBDEs) are a class of persistent, bioaccumulative compounds that are now understood to be environmental pollutants. These chemicals are used as flame-retardants in construction materials, electrical equipment, coatings, textiles and furniture padding. They enter the environment from manufacture and use of PBDE-containing products. PBDEs resist environmental degradation, as they are similar in chemical structure to PCBs (Siddiqi 2003).

PBDE toxicity is not entirely understood, yet these chemicals have been associated with neurodevelopmental delays, thyroid and liver problems, as well as tumors (Siddiqi 2003; ATSDR 2018). Negative neurological effects from PBDE exposure are similar to those observed for PCBs. Children exposed to PBDEs are prone to subtle, yet, measurable developmental problems (Siddiqi 2003). Studies of rodents and fish indicates that PBDE is likely an endocrine disruptor (Noyes and Stapleton 2014).

The tetra- and penta-BDE congeners are likely the most toxic of the PBDE compounds. The pentabromo formula is a mixture of tetra- and penta-PBDEs in nearly equal amounts. Pentabromo is banned by the European Union, but still widely used in North America. The United States is the top producer and user of pentabromo. However, the state of California has phased it out entirely (Siddiqi 2003).

Scientists evaluated PBDE concentrations in Great Lakes fishes to better understand the potential for human exposure through food. Overall, white sucker and common carp exhibited the highest total PBDE concentrations at 27–71 ng/g. Lake Superior's lake trout and whitefish contained higher levels than those from the other Great Lakes. However, fishes from Lake Ontario often had higher levels in tissue when not accounting for these outlying patterns. Whole body and egg contaminant loads are greater than in a corresponding fillet. Since PBDE levels have significantly declined since 2006, it will likely not result in appreciable fish accumulation of PBDEs (Gandhi et al. 2017). Therefore, it is not a significant human health hazard.

Appendix 11: Legally regulated limits of pesticide residues in foods in the United States, Canada, the European Union, and the World Health Organization.

Note: Canada has a default limit of 0.1 ppm for pesticide residues unless otherwise stated.

REGULATED PESTICIDE RESIDUE LIMITS IN FOODS		
Pesticide	**Maximum Residue Level**	**Agency**
Chlordane	0.3 ppm (fish)	United States
	0.1 ppm (berries)	United States
	0.1 ppm (bulb vegetables)	United States
	0.1 ppm (legumes)	United States
	0.1 ppm (default)	Canada
	0.01 ppm (berries)	European Union
	0.01 ppm (bulb vegetables)	European Union
	0.01 ppm (legumes)	European Union
	0.02 ppm (berries)	World Health Organization
	0.02 ppm (bulb vegetables)	World Health Organization
	0.02 ppm (legumes)	World Health Organization
DDT (DDE & DDD)	5 ppm (fish)	United States
	0.1 ppm (berries)	United States
	0.2 ppm (bulb vegetable)	United States
	0.2 ppm (legumes)	United States
	0.5 ppm (mushroom)	United States
	0.1 ppm (default)	Canada
	5 ppm (fish)	Canada
	0.5 ppm (bulb vegetable)	Canada
	0.5 ppm (legume)	Canada
	0.05 ppm (berries)	European Union
	0.01 ppm (wild raspberry)	European Union
	0.05 ppm (bulb vegetable)	European Union
	0.05 ppm (legume)	European Union
	0.05 ppm (rice)	European Union
	0.05 ppm (berries)	European Union
	0.1 ppm (rice)	World Health Organization
Mirex	0.1 ppm (fish)	United States
	0.1 ppm (fish)	Canada
	Limits have not been established at this time	European Union
		World Health Organization
Chlordecone	0.3 ppm (fish)	United States
	0.1 ppm (default)	Canada
	Limits have not been established at this time	European Union
		World Health Organization

REGULATED PESTICIDE RESIDUE LIMITS IN FOODS

Pesticide	Maximum Residue Level	Agency
Aldrin/ Dieldrin	0.3 ppm (fish-edible portion)	United States
	0.05 ppm (berries)	United States
	0.1 ppm (bulb vegetables)	United States
	0.03 ppm (legume)	United States
	0.1 ppm (default)	Canada
	0.01 ppm (berries)	European Union
	0.01 ppm (bulb vegetables)	European Union
	0.01 ppm (legumes)	European Union
	0.01 ppm (rice)	European Union
	0.05 ppm (bulb vegetable)	World Health Organization
	0.05 ppm (legume)	World Health Organization
	1 ppm (garden pea)	World Health Organization
	0.02 ppm (rice)	World Health Organization
Heptachlor	0.3 ppm (fish- edible portion)	United States
	0.05 ppm (berries)	United States
	0.03 ppm (rice)	United States
	0.1 ppm (default)	Canada
	0.01 ppm (berries)	European Union
	0.01 ppm (legumes)	European Union
	01 ppm (rice)	European Union
	0.02 ppm (rice)	World Health Organization
Hexachlorobenzene	0.05 ppm (berries)	United States
	0.05 ppm (bulb vegetable)	United States
	0.1 ppm (default)	Canada
	0.01 ppm (berries)	European Union
	0.01 ppm (bulb vegetables)	European Union
	0.01 ppm (legumes)	European Union
	0.01 ppm (rice)	European Union
	Limits have not been established at this time	World Health Organization
Glyphosate	0.25 ppm (fish)	United States
	0.2 ppm (berries)	United States
	0.2 ppm (wild leek)	United States
	0.1 ppm (wild rice)	United States
	310 ppm (aspirated fractions, wild rice)	United States
	8 ppm (pea)	United States
	1 ppm (tree nut)	United States
	5 ppm (liver- cattle)	United States
	0.1 ppm (meat- poultry)	United States
	1 ppm (liver- poultry)	United States

REGULATED PESTICIDE RESIDUE LIMITS IN FOODS		
Pesticide	**Maximum Residue Level**	**Agency**
Glyphosate (continued)	0.1 ppm (default)	Canada
	0.2 ppm (liver- cattle)	Canada
	0.08 ppm (meat- poultry)	Canada
	5 ppm (pea)	Canada
	0.1 ppm (berries)	European Union
	0.1 ppm (bulb vegetables)	European Union
	0.1 ppm (legumes)	European Union
	0.1 ppm (tree nut)	European Union
	0.2 ppm (liver- cattle)	European Union
	0.05 ppm (meat- polutry)	European Union
	0.05 ppm (liver- polutry)	European Union
	20 ppm (wild rice)	World Health Organization
	5 ppm (meat- mammalian)	World Health Organization
	5 ppm (offal- mammalian)	World Health Organization
	0.05 ppm (meat- polutry)	World Health Organization
	0.5 ppm (offal- poultry)	World Health Organization
2,4-D	0.1 ppm (fish)	United States
	0.2 ppm (berries)	United States
	0.2 ppm (tree nut)	United States
	0.3 ppm (meat- cattle)	United States
	40 ppm (wild rice)	United States
	0.1 ppm (default)	Canada
	0.3 ppm (meat- cattle)	Canada
	0.1 ppm (berries)	European Union
	0.2 ppm (tree nut)	European Union
	0.2 ppm (meat- cattle)	European Union
	0.1 ppm (wild rice)	European Union
	0.2 ppm (tree nut)	World Health Organization
	0.2 ppm (meat- mammalian)	World Health Organization
	5 ppm (offal-mammalian)	World Health Organization
Endothall	0.1 ppm (fish)	United States
	0.05 ppm (rice)	United States
	0.1 ppm (fish)	Canada
	0.1 ppm (default)	Canada
	0.01 ppm for all foods	European Union
	Limits have not been established at this time	World Health Organization

REGULATED PESTICIDE RESIDUE LIMITS IN FOODS		
Pesticide	**Maximum Residue Level**	**Agency**
Diquat dibromide	2 ppm (fish)	United States
	0.5 ppm (berries)	United States
	0.02 ppm (tree nut)	United States
	0.05 ppm (meat- cattle)	United States
	0.05 ppm (offal- cattle)	United States
	0.05 ppm (meat- poultry)	United States
	0.05 ppm (offal- poultry)	United States
	0.02 ppm (wild rice)	United States
	0.2 ppm (legumes)	Canada
	0.05 ppm (meat- cattle)	Canada
	0.05 ppm (offal- cattle)	Canada
	0.05 ppm (meat- poultry)	Canada
	0.01 ppm (berries)	European Union
	0.01 ppm (legumes)	European Union
	0.02 ppm (tree nut)	European Union
	0.05 ppm (meat- cattle)	European Union
	0.05 ppm (meat- cattle)	European Union
	0.05 ppm (meat- poultry)	European Union
	0.05 ppm (offal- poultry)	European Union
	0.02 ppm (wild rice)	European Union
	0.05 (berries)	World Health Organization
	0.3 ppm (legumes)	World Health Organization
	0.02 ppm (tree nut)	World Health Organization
	0.05 ppm (meat- mammalian)	World Health Organization
Chlorothalonil	1 ppm (blueberry)	United States
	5 ppm (berries)	United States
	5 ppm (wild leek)	United States
	0.1 ppm (legumes)	United States
	0.03 ppm (meat- cattle)	United States
	0.05 ppm (liver- cattle)	United States
	1 ppm (mushroom)	United States
	0.1 ppm (meat products)	Canada
	2 ppm (cranberries)	Canada
	0.6 ppm (berries)	Canada
	5 ppm (onion)	Canada
	0.1 ppm (legumes)	Canada
	1 ppm (mushroom)	Canada
	5 ppm (berries)	European Union
	0.01 ppm (bulb vegetable)	European Union
	3 ppm (legumes)	European Union

REGULATED PESTICIDE RESIDUE LIMITS IN FOODS		
Pesticide	**Maximum Residue Level**	**Agency**
Chlorothalonil (continued)	0.01 ppm (tree nut)	European Union
	0.15 ppm (meat- cattle)	European Union
	0.02 ppm (liver- cattle)	European Union
	5 ppm (berries)	World Health Organization
	10 ppm (spring onion)	World Health Organization
	1 ppm (legumes)	World Health Organization
	0.02 ppm (meat- mammalian)	World Health Organization
	0.2 ppm (offal- mammalian)	World Health Organization
Toxaphene	There are no established levels for food at this time.	United States
	0.1 ppm (default)	Canada
	0.01 ppm (wild vertebrates)	European Union
	0.01 ppm (poultry)	European Union
	0.01 ppm (poultry fat)	European Union
	0.01 ppm (berries)	European Union
	0.01 ppm (legumes)	European Union
	0.02 ppm (hazelnuts)	European Union
	0.01 ppm (bulb vegetables)	European Union
	0.01 ppm (wild fungi)	European Union
	0.01 ppm (cereals)	European Union
	There are no established levels for food at this time.	World Health Organization

Appendix 12: Legally regulated limits of industrial chemicals in foods in the United States, Canada, the European Union, and the World Health Organization

REGULATED INDUSTRIAL CHEMICAL RESIDUE LIMITS		
FOOD		
Pesticide	**Maximum Residue Level**	**Agency**
Dioxin & Furans	There are no established levels for food at this	United States
	20,000 ppm (under review)	Canada
	0.000001 ppm (fish)	European Union
	0.00000175 ppm (cattle meat)	European Union
	0.00000125 ppm (poultry meat)	European Union
	0.0000003 ppm (fruits and vegetables)	European Union
	No estabilished levels could be found at this time.	World Health Organization
Polychlorinated biphenyls (PCBs)	3 ppm fat basis (red meat)	United States
	3 ppm (poultry)	United States
	2 ppm (fish)	United States
	10 ppm paper food packaging	United States
	2 ppm (under review)	Canada
	0.0000025 ppm (fish)	European Union
	0.00000175 ppm (cattle meat)	European Union
	0.0000075 ppm (poultry meat)	European Union
	0.0000001 ppm (fruits and vegetables)	European Union
	No estabilished levels could be found at this time.	World Health Organization
Perfluorooctanoic acid (PFOA)	**There are no established levels for food at this time.**	United States
		Canada
		European Union
		World Health Organization
Perfluorooctane sulfonic acid (PFOS)	**There are no established levels for food at this time.**	United States
		Canada
		European Union
		World Health Organization

References:

Agency for Toxic Substances and Disease Registry. 2012. "Addendum to the Toxicological Profile for Chlorinated Dibenzo-p-dioxins (CDDs)". 66-69.

Code of Federal Regulation. Tolerances for polychlorinated biphenyls (PCB's). Title 21, Volume 2, Part 109.30 Subpart B.

Canada Food Inspection Agency. 2014. "Appendix 3 Canadian Guidelines for Chemical Contaminants and Toxins in Fish and Fish Products". 1-4.

European Union. 2013. "2013/711/EU: Commission Recommendation of 3 December 2013 on the reduction of the presence of dioxins, furans and PCBs in feed and food Text with EEA relevance".