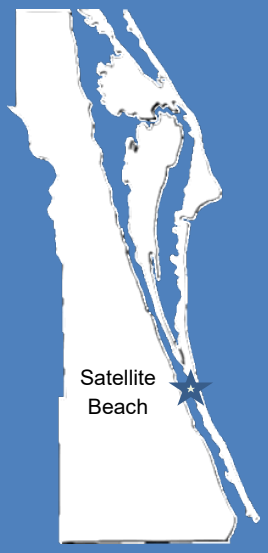
BREVARD COUNTY CANCER ASSESSMENT



Florida HEALTH

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Executive Summary

The Florida Department of Health takes health concerns brought forward by the community very seriously. This cancer assessment was undertaken to address community concerns received by the Florida Department of Health in Brevard County (DOH-Brevard). Citizens were questioning the number of cancers occurring in the community given a recent national report on usage of per- and polyfluoroalkyl substances (PFAS), one of which is a suspected cancer-causing agent, at the local military installation, Patrick Air Force Base. Moreover, concerned citizens questioned if there were high cancer rates in Brevard County among individuals younger than 40 years of age. The geographic areas identified were zip codes 32937 and 32940.

The Florida Department of Health reviewed data from the state cancer registry, the Florida Cancer Data System (FCDS), for two time periods: 1996 to 2005 and 2006 to 2015 for nine (9) cancer types in zip codes 32937 and 32940. Cancer data from the FCDS and population data from the United States (U.S.) Census were used to calculate a standardized incidence ratio (SIR) respectively for each cancer type, allowing comparisons between the number of observed cases versus the number of expected cases to determine if the occurrence of these cancer types are higher or lower than one would expect given the population size and demographics of the local area in question.

The cancer types reviewed were Hodgkin lymphoma, non-Hodgkin lymphoma, leukemia, and cancers of the kidney, liver, pancreas, testes, thyroid, and urinary bladder. These cancer types were selected and included based on relatively new scientific literature indicating there may be a slight risk but not definitive causal evidence for the occurrence of these cancer types from perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) (ATSDR, 2018).

For this assessment, specific cancers with potential associations with the contaminants of concern (PFAS), over two (2) ten-year periods, 1996 to 2005 and 2006 to 2015, were reviewed. For the time period from 1996 to 2005, a higher than expected number of cases was found for urinary bladder cancer for both females (expected 23-24 cancers, 37 cancers occurred) and males (expected 80 cancers, 106 cancers occurred) in zip code 32937, the primary area of concern, and leukemia (expected 28-29 cancers, 50 cancers occurred) and urinary bladder cancer (expected 72-73 cancers, 109 cancers occurred) for males in zip code 32940. Of note, during this time period there were fewer observed cases than expected for liver and thyroid cancers among females in zip code 32937. When analyzing data over the ten-year period from 2006 to 2015, a statistically significant higher number of observed cases than expected was found for non-Hodgkin lymphoma (expected 65-66 cancers, 84 cancers occurred) and pancreatic cancer (expected 38-39 cancers, 54 cancers occurred) among males, and pancreatic (expected 33-34 cancers, 49 cancers occurred) and urinary bladder cancer (expected 28-29 cancers, 50 cancers occurred) among females in zip code 32940. For the primary area of concern, zip code 32937, during this time period there were fewer number of observed cases than expected for liver (expected 6-7 cancers, 2 cancers occurred) and leukemia (expected 37-38 cancers, 24 cancers occurred). In some cases, an initial analysis can demonstrate statistical significance in the number of cancer cases occurring, but may not be linked with a statistically significant association with exposure to environmental contaminants. Also, a statistically significant excess of cancer cases can occur within a given population without a discernible cause and might be a chance or random occurrence (CDC, 2013).

The Florida Department of Health's Bureau of Environmental Health reviewed current environmental sample data on PFAS, the contaminant of concern, from multiple locations within the area of concern and found no evidence of PFAS currently impacting the public drinking water of the surrounding areas of Patrick Air Force Base. Irrigation water and surface water samples did reveal detections of PFAS but levels were below their respective screening values. Since there is no documented exposure, it is not possible to link any excess cancer cases with PFOS/PFOA exposure in the local community. For the cancer types in which the observed number of cases were statistically significantly higher than expected, there are various known biologic/genetic and behavioral/lifestyle risk factors that increases one's risk for developing these cancers which may explain the findings. Current findings from this cancer assessment review do not confirm a cancer cluster.

The Florida Department of Health takes health concerns brought forward by the community very seriously and will continue to take necessary and appropriate actions to ensure the health and safety of our residents. The Department will continue to monitor cancer incidence in the suspected areas of concern. Moreover, the Department and local cancer stakeholders will continue to provide education on cancer and the respective risk factors for that cancer type and the importance of maintaining a healthy lifestyle. The Department will continue to work with representatives from the Department of Environmental Protection and the local government of Brevard County to conduct environmental testing as indicated, and address any new, emerging community health concerns and questions.

Purpose

This cancer assessment was completed to address community concerns received by the Florida Department of Health in Brevard County (DOH-Brevard). Concerned citizens were questioning the number of cancers occurring in the community given a recent national report on usage of per- and polyfluoroalkyl substances (PFAS), one of which is a suspected cancer-causing agent, at the local military installation, Patrick Air Force Base. Moreover, concerned citizens questioned if there were high cancer rates in Brevard County among individuals younger than 40 years of age, specifying geographic areas of zip codes 32937 and 32940.

Background: Cancer and What Constitutes a Cancer Cluster

Although cancer is a serious and frightening diagnosis, it is a common disease. Current information shows that approximately one of three Americans will develop cancer in their lifetime, and cancer will affect three of four families. The risk of developing cancer increases with age, so as the population ages, more cases of cancer are expected in our communities. Florida, given its higher percentage of older residents has correspondingly higher cancer rates.

The term *cancer* covers not one but many diseases that share the common feature of abnormal cell growth. It can occur in almost any part of the body. Each cancer type develops differently and has different risk factors. For example, the main risk factor for lung cancer is cigarette smoking, but for skin cancer it is sun exposure. The causes of some common cancers such as breast cancer remain unknown.

Many people believe that something in the environment causes most cancers, but behavior and lifestyle accounts for most of the known cancer risks. Factors such as smoking, poor diet, obesity, heavy alcohol use, sexual and reproductive history, and genetic factors can all contribute to developing cancer. It is estimated that less than 10% of cancers are caused by environmental exposures, with many related directly to radon exposures. In contrast, cigarette smoking alone causes about 30% of cancers. In addition, family history is important and contributes to some types of cancer.

Most cancers take a long time to develop. It is usually decades from the time someone is exposed to something that might cause cancer until that cancer is discovered. The time that passes between exposure to the development of symptoms and disease is the latency period. This is one of the reasons that cancer is more common in older adults. In addition, the few chemicals that are linked to cancer have fairly long and/or concentrated exposures before they typically cause cancer. Additionally, most human exposures to carcinogens vary significantly over time, making a precise determination of cancer- related exposures and minimum latency periods difficult to identify.

A cancer cluster is defined as a greater than expected number of cancer cases that occurs within a group of people in a defined geographic area over a specified period of time. If a suspected cluster includes cancers of different types, it is probably not a “true” cancer cluster. For example, if someone reported that there were many people with cancer in their community, but the kinds of cancer included lung, breast, leukemia, and prostate which are cancers known to have different risk factors; this would not be considered a cancer cluster. A confirmed cancer cluster is a relatively rare occurrence and few documented clusters have been able to be linked to an environmental agent.

To be a cancer cluster, a group of cancer cases must meet the following criteria. Until all of these parameters are met, the group of cancer cases is often referred to as a **suspected cancer cluster**.

A greater than expected number:
A greater than expected number is when the observed number of cases is higher than one would typically observe in a similar setting (in a group with similar population, age, race, or gender). This may involve comparison with rates for comparable groups of people over a much larger geographic area (e.g., an entire state).

Of cancer cases:
All cases must involve the same type of cancer or types of cancer scientifically proven to have the same cause.

That occurs within a group of people:
The population in which the cancers are occurring is carefully defined by factors such as race/ethnicity, age, and gender, for purposes of calculating cancer rates.

In a geographic area:
Both the number of cancer cases included in the cluster and calculation of the expected number of cases can depend on how we define the geographic area where the cluster occurred. The boundaries must be defined carefully. It is possible to “create” or “obscure” a cluster by selection of a specific area.

Over a period of time:
The number of cases included in the cluster, and calculation of the expected number of cases, will depend on how we define the time period over which the cases occurred.

Methods

For this assessment, the number of observed cases for zip codes 32937 and 32940, and the state age-specific rates for the two time periods from 1996 to 2005 and 2006 to 2015 for the nine (9) selected cancer types were gathered from the Florida Cancer Data System (FCDS), Florida's statewide cancer registry. The cancer types reviewed were Hodgkin lymphoma, non-Hodgkin lymphoma, leukemia, and cancers of the kidney, liver, pancreas, testes, thyroid, and urinary bladder. These cancer types were selected and included based on relatively new scientific literature indicating there may be a slight risk, but not definitive causal evidence, for the occurrence of these cancer types from perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) (ATSDR, 2018).

The FCDS is legislatively mandated per Section 385.202 *Florida Statutes* to collect incidence data (i.e., the number of new cancers per year) on all cases diagnosed in the state of Florida. The FCDS has collected cancer incidence data from hospitals statewide since 1981 and from non-hospital sources (i.e., ambulatory surgical centers, radiation therapy centers, pathology laboratories, and private physician offices) since 1997. The main goal of the FCDS is to gather complete, accurate, and timely data to assist policy makers and researchers in developing policy and programs to reduce death and illness due to cancer by better understanding cancer trends and possible causes of cancer.

There are limitations to using FCDS data to address community cancer concerns. Although the FCDS data can be provided by select geographical areas (based at the time of diagnosis), these data represent a retrospective account of the burden of cancer for the area of concern. The FCDS collects outcome data; the FCDS does not collect environmental exposures, complete genetic data, nor residential history. However, the Florida Department of Health does have environmental health programs that can search for available environmental data and evaluate how the data may potentially tie into health concerns from the environment. For this assessment in Brevard County, current groundwater, irrigation water, surface water and public drinking water levels of PFAS were reviewed.

The data submitted by reporters to the FCDS describes "who", "what", "when", and "where" of the cancer case. However, the FCDS does not collect data as to "why" nor can analyses of FCDS data alone determine why the occurrence of cancer in a specific area or population is happening. Moreover, there is an inherent delay in collecting cancer incidence data. Reporting entities have up to six (6) months after the initial date of diagnosis to report the cancer case information to the Florida Department of Health. This six-month period permits the cancer case information to include the completed initial course of treatment. In addition, the FCDS must conduct external linkages with the Bureau of Vital Statistics and the Agency for Health Care Administration to ensure the completeness and accuracy for the diagnosis year. Therefore, cancer data from the FCDS is not available for official release until two years after the close of the diagnosis year. For this assessment, the most current, complete diagnosis year available for analysis is 2015.

While no documented environmental exposure link could be found currently for the area, the DOH did proceed to review cancer rates in two zip codes, 32937 and 32940, that community members provided due to the national report on usage of PFAS, one of which is a suspected cancer-causing agent, at the Patrick Air Force Base. For each zip code, the cancer count (i.e., the number of observed cases) among females and males for nine (9) selected cancer types were obtained from the FCDS in addition to the state age-specific rate for each cancer type respectively. Population data for the two zip codes

were obtained from the U.S. Census. An estimate of the total population for the time period from 1996 to 2005 for each zip code was calculated by multiplying the 2000 U.S. zip code tabulated area (ZCTA) population by 10. Similarly, an estimate of the total population for the time period from 2006 to 2015 for each zip code was calculated by multiplying the 2010 U.S. ZCTA population by 10. The standardized incidence ratio (SIR) was calculated to determine if there was a greater than expected number of cases for each cancer type for the two zip codes. A SIR was calculated for each cancer type for each time period for zip codes 32937 and 32940 respectively.

$$\text{Standardized Incidence Ratio (SIR)} = \frac{\text{Observed Incidence}}{\text{Expected Incidence}}$$

Observed cases are the number of cases found within the FCDS for the area of concern. Expected cases are calculated based on the age-sex population gathered from the 2010 U.S. Census for the area of concern and the state age-sex specific rates obtained from the FCDS.

Statistical tests were performed to determine the likelihood of difference between observed and expected cases. Statistical testing was based on the 95% confidence interval of the SIR calculated. If the 95% confidence interval contained the value ‘1’, then the difference between “observed” and “expected” numbers was likely due to chance or a random occurrence. If the 95% confidence interval did not contain the value ‘1’, then the difference in two numbers was unlikely due to random factors, and the numbers are “statistically significantly” different.

Environmental Assessment

Purpose

The Bureau of Environmental Health within the Florida Department of Health was requested to assist with evaluating a health concern regarding potential exposure to current levels of per- and poly-fluoroalkyl substances, also known as PFAS (particularly perfluorooctane sulfonate [PFOS] and perfluorooctanoic acid [PFOA]) in the City of Cocoa Beach, the City of Satellite Beach, and South Patrick Shores in Brevard County. Nearby activities at the Patrick Air Force Base may be a source of PFAS in local environmental samples; however, the Florida Department of Health's Bureau of Environmental Health cannot say with certainty what the source of PFAS is.

Human health risk assessors and public health toxicologists from the Bureau of Environmental Health evaluated the public health concerns using a stepwise approach of determining the possible PFAS exposure routes with guidance from the most current human health assessment tools from the Agency for Toxic Substances and Disease Registry (ATSDR) [See Florida Department of Health Environmental Health Consultation Letter, Tables 1-6]. The Department health assessors also collaborated with colleagues both within and outside the Department to develop a strategy for data evaluation. The collaborative effort and open communication provided quality information that was representative of actual conditions in Brevard County.

Background Information

Peer-reviewed studies have shown that two of the PFAS called PFOA and PFOS can cause reproductive, developmental, and immunological abnormalities, and cancer in animals (EPA, 2018). The most consistent finding from human epidemiology studies show increased cholesterol levels among exposed populations. More limited findings are seen affecting infant birth weights, the immune system, as well as causing cancer (for PFOA) and thyroid hormone disruption (for PFOS). It is important to understand that humans and animals metabolize and react to these chemicals differently (e.g., differing half-lives) thus more research is needed to fully understand how PFAS compounds affect human health and if findings in animals translate well to humans.

The health risks associated with PFAS exposure are a function of the dose of the chemicals that enter the human body, the rate they enter the body, the frequency and duration of the exposure, which PFAS (or mixture) individuals are being exposed to, as well as the health status of the exposed individual. PFAS are fairly water soluble and are known to accumulate during continuous exposures. In some species (humans) it is excreted relatively slowly and therefore may accumulate in the body. Therefore, as individuals get exposed to PFAS from different sources over time, the level of PFAS in their body may accumulate to the point where they have adverse health effects.

Workers at facilities producing PFAS can be exposed to higher PFAS levels than the general public. While levels of PFAS in communities that are not close to manufacturing sources are typically lower, people can be exposed to trace amounts from products that contain PFAS through drinking contaminated water or food. Research shows that PFAS do not volatilize (are not easy to breath in) or penetrate skin easily. Therefore, exposure through inhalation and dermal routes is negligible in comparison to direct ingestion.

Public Drinking Water

The public drinking water for the City of Satellite Beach comes from water pumped from Lake Washington and four Floridan Aquifer wells at 650 to 950 feet underground and located approximately 7-8 miles away. The water sent to customers is a blend of water from both sources: water samples from the lake and aquifer, tested for PFAS, were below the Environmental Protection Agency's Lifetime Health Advisory Level (EPA HAL) of 70 nanograms per liter (ng/L) for PFOA + PFOS, which represents the combined concentration of the substances in drinking water deemed to be safe for drinking over a lifetime without causing adverse health effects.

As part of the EPA's Unregulated Contaminant Monitoring Rule (UCMR3), the city took four samples at each water treatment plant between October 2013 and July 2014. The samples were collected after any treatment and analyzed for PFAS. There was no detection of PFAS in any of the samples and consequently, no pathway for individuals in the City of Satellite Beach to be exposed to harmful concentrations of PFAS through public drinking water (see Florida Department of Health Environmental Health Consultation Letter (Appendix E)).

The public drinking water for residents in the zip code 32940 comes from the City of Cocoa Water System. Cocoa's water system includes the wellfield and raw water collection system, Wewahootee Water Treatment Plant, transmission mains, and the Dyal Water Treatment Plant (WTP). Cocoa's drinking water sources include the Floridan Aquifer, Intermediate Aquifer, Taylor Creek Reservoir, and Aquifer Storage and Recovery (ASR) wells. All these facilities are in east Orange County. A distribution system and storage and pumping facilities are in Brevard County. Raw water comes from 48 wells in the Cocoa well field: 34 wells drilled 400 to 600 feet into the Floridan Aquifer and 14 wells drilled into the Intermediate Aquifer.

Reclaimed Water

People can be exposed to PFAS from reused water for irrigation purposes. However, there is no current data for home-grown vegetables that are watered by this reclaimed water. While PFAS have been found in reclaimed water in Cocoa Beach, people don't routinely drink reclaimed water used for irrigation and the exposure from inhalation or dermal contact is limited.

Private Wells

One private potable well was tested in the Satellite Beach area. The results showed PFOS at 42 ng/L, PFOA at 4.2 ng/L and PFOA + PFOS at 46.2 ng/L which is below the EPA HAL of 70 ng/L for PFOA+PFOS. Other PFAS were not included in the test results. This well has been disconnected and the home is now serviced by City of Melbourne Water Services as of February 2019. There were a few other potable wells identified on the same street but the those households are were connected to the city water system so they were not tested.

Irrigation Wells

People can be exposed to PFAS from reused water for irrigation purposes. However, there is no current data for home-grown vegetables that are watered by irrigation wells. Sample data for ground

water irrigation wells were taken by Brevard County Natural Resources, City of Cocoa Beach, and City of Satellite Beach. None of the levels of PFOA or PFOS were above the Florida Department of Environmental Protection's provisional screening value for irrigation water. While PFAS have been found in irrigation wells, people don't routinely drink irrigation water and the possibility of exposure from inhaling water or through dermal contact is limited.

Surface Water

Sample data for surface water in the Indian River Lagoon was taken by the Brevard County Natural Resources. When compared to a range of available screening values for surface water from different states, the combined PFOA + PFOS levels did not exceed those ranges. The surface water sampled is not a drinking water source and the exposure from inhalation or dermal contact is limited.

Fish Consumption

No fish tissue sample data were available for analysis. Once sample data are available and undergo quality assurance and quality control, this pathway can be evaluated further.

Table 1. Present Exposure Pathways Evaluated for PFAS Impact in Brevard County, Florida

Pathway Name	Exposure Pathway Element					Pathway Determination
	Source	Media	Point of Exposure	Route of Exposure	Receptor	
public drinking water	PFAS in drinking water system	water	water taps, showerheads, and indoor air for connected structure	ingestion, inhalation, and dermal contact	resident and visitors	**potential**
private well	PFAS in groundwater	water	water taps, showerheads, and indoor air for connected structure	ingestion, inhalation, and dermal contact	resident and visitors	**potential**
irrigation well	PFAS in groundwater	water	tap, outdoor air, and edible plants/fruits/vegetables	ingestion, inhalation, and dermal contact	resident and visitors	**potential**
reclaimed water	PFAS in wastewater treatment plant	water	tap and outdoor air	ingestion, inhalation, and dermal contact	resident and visitors	**potential**
surface water	PFAS in surface water	water	recreation bathing, boating, fishing in surface water bodies	ingestion, inhalation, and dermal contact	resident and visitors	**potential**
fish consumption	PFAS in fish	food	consuming PFAS contaminated fish	ingestion	recreational users	**potential**

Conclusions

DOH concludes that presently there is a minimal potential to harm human health via consuming drinking water from local water supplies as PFAS is below the EPA lifetime health advisory. In addition,

PFAS-contaminated irrigation, surface and reclaimed water in the Brevard County Area is below screening values and will have minimal impact if swallowed. Inhalation and dermal (skin) exposure are minor exposure pathways. PFAS tend to stay in water once they have dissolved. The uptake of PFAS through dermal contact is slow and not considered significant. While garden fruits and vegetables, as well as fish and seafood consumption concerns were considered, DOH currently has no data to evaluate but would consider evaluation once that becomes available. More detailed information regarding the environmental health assessment is found in Appendix E.

Uncertainties

All risk assessments, to varying degrees, require the use of assumptions, judgments, and incomplete data. These contribute to the uncertainty of the risk estimates. Some more important sources of uncertainty in this summary include environmental sampling and analyses, exposure parameter estimates, modeled exposure doses, and toxicological knowledge. This report uses the most recent understanding of PFAS toxicity and reference values derived from those studies to make its findings and conclusions.

Risk Factors by Cancer Types

A risk factor is anything that affects the chance of getting a disease, such as cancer (ACS, 2016). Different cancers have different risk factors. Cancer risk factors include exposure to chemicals or other substances, as well as certain behaviors. They also include things people cannot control, like age and family history. A family history of certain cancers can be a sign of a possible inherited cancer syndrome (NCI, 2015).

Risk factors do not tell us everything. Having a risk factor, or even several risk factors, does not mean that an individual will get the disease. Some people who get the disease may have few or no known risk factors (NCI, 2015).

Although some risk factors can be avoided, others—such as growing older—cannot. Limiting exposure to avoidable risk factors may lower the risk of developing certain cancers. These risk factors include the following: alcohol, cancer-causing substances, chronic Inflammation, diet, hormones, immunosuppression, infectious agents, obesity, radiation, sunlight, and tobacco.

Cancer Type	**Risk Factor**
Kidney	Gender, Race, Smoking, Obesity, Workplace Exposures, Family History, High Blood Pressure, Certain Medicines, Advanced Kidney Disease
Liver	Gender, Race, Cirrhosis, Hepatitis B, Hepatitis C, Obesity, Type 2 Diabetes, Heavy Alcohol Use, Tobacco Use
Pancreas	Age, Gender, Race Family History, Tobacco Use, Overweight/ Obesity, Chemical Exposure, Type 2 Diabetes, Cirrhosis
Testes	Family History, Undescended Testicle,
Urinary Bladder	Age, Gender, Race/Ethnicity, Genetics. Smoking, Chemical Exposure
Thyroid	Age, Gender, Family History
Hodgkin Lymphoma	Age, Gender, Family History
Non-Hodgkin Lymphoma	Age, Gender, Race, Family History, Chemical Exposure
Leukemia	Age, Gender, Tobacco Use, Family History, Chemical Exposure

Results

Table 1. Standardized Incidence Ratio (SIR) for Select Cancer Types among Females in ZIP 32937, Brevard County, Florida, 1996-2005

Cancer Type	Observed Cases*	Expected Cases**	SIR (95% CI)+	Significant++
Hodgkin				
Lymphoma	3	3.4	0.89 (-0.12, 1.90)	No
Kidney	18	18.7	0.96 (0.52, 1.41)	No
Leukemia	32	21.8	1.47 (0.96, 1.98)	No
Liver	1	4.8	0.21 (-0.20, 0.62)	Yes
Non-Hodgkin				
Lymphoma	44	34.6	1.27 (0.90, 1.65)	No
Pancreas	24	21.9	1.10 (0.66, 1.54)	No
Testes	N/A	N/A	N/A	N/A
Thyroid	9	16.1	0.56 (0.19, 0.92)	Yes
Urinary Bladder	37	23.8	1.55 (1.05, 2.05)	Yes

*Number of cancers found within the Florida Cancer Data System
**Number of expected cases are calculated using the state-specific rate for the respective cancer type and 2000 U.S. Census population for ZCTA 32937
+Confidence Interval
++If the confidence interval includes the value '1', then not statistically significant.

Table 2. Standardized Incidence Ratio (SIR) for Select Cancer Types among Males in ZIP 32937, Brevard County, Florida, 1996-2005

Cancer Type	Observed Cases*	Expected Cases**	SIR (95% CI)+	Significant++
Hodgkin				
Lymphoma	8	4.4	1.81 (0.55, 3.06)	No
Kidney	26	33.7	0.77 (0.48, 1.07)	No
Leukemia	39	33.0	1.18 (0.81, 1.55)	No
Liver	17	13.2	1.29 (0.67, 1.90)	No
Non-Hodgkin				
Lymphoma	37	42.3	0.88 (0.59, 1.16)	No
Pancreas	24	24.2	0.99 (0.60, 1.39)	No
Testes	10	5.9	1.68 (0.64, 2.72)	No
Thyroid	9	6.2	1.45 (0.50, 2.40)	No
Urinary Bladder	106	80.0	1.33 (1.07, 1.58)	Yes

*Number of cancers found within the Florida Cancer Data System
**Number of expected cases are calculated using the state-specific rate for the respective cancer type and 2000 U.S. Census population for ZCTA 32937
+Confidence Interval
++If the confidence interval includes the value '1', then not statistically significant.

Table 3. Standardized Incidence Ratio (SIR) for Select Cancer Types among Females in ZIP 32940, Brevard County, Florida, 1996-2005

Cancer Type	Observed Cases*	Expected Cases**	SIR (95% CI)+	Significant++
Hodgkin Lymphoma	1	2.4	0.42 (-0.40, 1.23)	No
Kidney	17	15.1	1.13 (0.59, 1.66)	No
Leukemia	22	17.6	1.25 (0.73, 1.77)	No
Liver	4	3.9	1.03 (0.02, 2.03)	No
Non-Hodgkin Lymphoma	36	28.0	1.29 (0.87, 1.71)	No
Pancreas	16	18.2	0.88 (0.45, 1.31)	No
Testes	N/A	N/A	N/A	N/A
Thyroid	8	11.7	0.68 (0.21, 1.16)	No
Urinary Bladder	25	19.8	1.26 (0.77, 1.75)	No

*Number of cancers found within the Florida Cancer Data System

**Number of expected cases are calculated using the state-specific rate for the respective cancer type and 2000 U.S. Census population for ZCTA 32940

+Confidence Interval

++If the confidence interval includes the value '1', then not statistically significant.

Table 4. Standardized Incidence Ratio (SIR) for Select Cancer Types among Males in ZIP 32940, Brevard County, Florida, 1996-2005

Cancer Type	Observed Cases*	Expected Cases**	SIR (95% CI)+	Significant++
Hodgkin Lymphoma	9	3.2	2.84 (0.98, 4.70)	No
Kidney	34	28.6	1.19 (0.79, 1.59)	No
Leukemia	50	28.6	1.75 (1.26, 2.23)	Yes
Liver	12	11.0	1.09 (0.47, 1.71)	No
Non-Hodgkin Lymphoma	43	35.8	1.20 (0.84, 1.56)	No
Pancreas	25	21.3	1.17 (0.71, 1.63)	No
Testes	8	3.9	2.05 (0.63, 3.47)	No
Thyroid	3	4.8	0.62 (-0.08, 1.33)	No
Urinary Bladder	109	72.6	1.50 (1.22, 1.78)	Yes

*Number of cancers found within the Florida Cancer Data System

**Number of expected cases are calculated using the state-specific rate for the respective cancer type and 2000 U.S. Census population for ZCTA 32940

+Confidence Interval

++If the confidence interval includes the value '1', then not statistically significant.

Table 5. Standardized Incidence Ratio (SIR) for Select Cancer Types among Females in ZIP 32937, Brevard County, Florida, 2006-2015

Cancer Type	Observed Cases*	Expected Cases**	SIR (95% CI)+	Significant++
Hodgkin Lymphoma	3	3.4	0.88 (-0.12, 1.88)	No
Kidney	21	20.8	1.01 (0.58, 1.44)	No
Leukemia	29	25.9	1.12 (0.71, 1.53)	No
Liver	2	6.7	0.30 (-0.12, 0.72)	Yes
Non-Hodgkin Lymphoma	38	38.7	0.98 (0.67, 1.29)	No
Pancreas	31	24.5	1.26 (0.82, 1.71)	No
Testes	N/A	N/A	N/A	N/A
Thyroid	25	26.2	0.95 (0.58, 1.33)	No
Urinary Bladder	31	20.7	1.50 (0.97, 2.03)	No

*Number of cancers found within the Florida Cancer Data System

**Number of expected cases are calculated using the state-specific rate for the respective cancer type and 2010 U.S. Census population for ZCTA 32937

+Confidence Interval

++If the confidence interval includes the value '1', then not statistically significant.

Table 6. Standardized Incidence Ratio (SIR) for Select Cancer Types among Males in ZIP 32937, Brevard County, Florida, 2006-2015

Cancer Type	Observed Cases*	Expected Cases**	SIR (95% CI)+	Significant++
Hodgkin lymphoma	7	4.5	1.57 (0.41, 2.74)	No
Kidney	41	38.6	1.06 (0.74, 1.39)	No
Leukemia	24	37.1	0.65 (0.39, 0.91)	Yes
Liver	24	20.6	1.16 (0.70, 1.63)	No
Non-Hodgkin lymphoma	45	48.0	0.94 (0.66, 1.21)	No
Pancreas	26	27.9	0.93 (0.57, 1.29)	No
Testes	6	5.1	1.18 (0.24, 2.13)	No
Thyroid	13	9.6	1.36 (0.62, 2.09)	No
Urinary Bladder	93	73.8	1.26 (1.00, 1.52)	No

*Number of cancers found within the Florida Cancer Data System

**Number of expected cases are calculated using the state-specific rate for the respective cancer type and 2010 U.S. Census population for ZCTA 32937

+Confidence Interval

++If the confidence interval includes the value '1', then not statistically significant.

Table 7. Standardized Incidence Ratio (SIR) for Select Cancer Types among Females in ZIP 32940, Brevard County, Florida, 2006-2015

Cancer Type	Observed Cases*	Expected Cases**	SIR (95% CI)+	Significant++
Hodgkin lymphoma	6	4.4	1.37 (0.27, 2.47)	No
Kidney	26	28.1	0.93 (0.57, 1.28)	No
Leukemia	44	35.2	1.24 (0.88, 1.62)	No
Liver	6	8.9	0.67 (0.13, 1.21)	No
Non-Hodgkin lymphoma	51	52.4	0.97 (0.71, 1.24)	No
Pancreas	49	33.5	1.46 (1.05, 1.87)	Yes
Testes	N/A	N/A	N/A	N/A
Thyroid	30	33.7	0.89 (0.57, 1.21)	No
Urinary Bladder	50	28.4	1.76 (1.27, 2.25)	Yes

*Number of cancers found within the Florida Cancer Data System
**Number of expected cases are calculated using the state-specific rate for the respective cancer type and 2010 U.S. Census population for ZCTA 32940
+Confidence Interval
++If the confidence interval includes the value '1', then not statistically significant.

Table 8. Standardized Incidence Ratio (SIR) for Select Cancer Types among Males in ZIP 32940, Brevard County, Florida, 2006-2015

Cancer Type	Observed Cases*	Expected Cases**	SIR (95% CI)+	Significant++
Hodgkin lymphoma	7	5.5	1.26 (0.33, 2.20)	No
Kidney	47	51.3	0.92 (0.65, 1.18)	No
Leukemia	52	51.5	1.01 (0.74, 1.29)	No
Liver	27	25.9	1.04 (0.65, 1.43)	No
Non-Hodgkin lymphoma	84	65.4	1.28 (1.01, 1.56)	Yes
Pancreas	54	38.5	1.40 (1.03, 1.78)	Yes
Testes	4	5.8	0.68 (0.01, 1.36)	No
Thyroid	11	12.2	0.90 (0.37, 1.43)	No
Urinary Bladder	125	105.4	1.18 (0.98, 1.39)	No

*Number of cancers found within the Florida Cancer Data System
**Number of expected cases are calculated using the state-specific rate for the respective cancer type and 2010 U.S. Census population for ZCTA 32940
+Confidence Interval
++If the confidence interval includes the value '1', then not statistically significant.

Conclusions and Next Steps

Cancer can occur randomly among populations. The number of cancer cases may vary from year to year even if there is no change in the population or environment. Overall, cancer occurrence in the two zip codes show a pattern in which most cancers occur over the age of 60 and for certain cancer types (e.g. urinary bladder), there is more cancers among males than females as expected. Likewise, thyroid cancer is more common among females than males as expected, which aligns with scientific literature.

For this assessment, specific cancers with potential associations with the contaminants of concern (PFAS), over two (2) ten-year periods, 1996 to 2005 and 2006 to 2015, were reviewed. For the time period from 1996 to 2005, a higher than expected number of cases was found for urinary bladder cancer for both females (expected 23-24 cancers, 37 cancers occurred) and males (expected 80 cancers, 106 cancers occurred) in zip code 32937, the primary area of concern, and leukemia (expected 28-29 cancers, 50 cancers occurred) and urinary bladder cancer (expected 72-73 cancers, 109 cancers occurred) for males in zip code 32940. Of note, during this time period there fewer observed cases than expected for liver and thyroid cancers among females in zip code 32937. When analyzing data over the ten-year period from 2006 to 2015, a statistically significant higher number of observed cases than expected was found for non-Hodgkin lymphoma (expected 65-66 cancers, 84 cancers occurred) and pancreatic cancer (expected 38-39 cancers, 54 cancers occurred) among males, and pancreatic (expected 33-34 cancers, 49 cancers occurred) and urinary bladder cancer (expected 28-29 cancers, 50 cancers occurred) among females in zip code 32940. For the primary area of concern, zip code 32937, during this time period there were fewer number of observed cases than expected for liver (expected 6-7 cancers, 2 cancers occurred) and leukemia (expected 37-38 cancers, 24 cancers occurred).

The data from the FCDS provides a background cancer rate of occurrence among the general population. Although FCDS data can be provided by select geographical area (based at the time of diagnosis), these data represent a retrospective account of the burden of cancer for the area(s) of concern. The FCDS collects outcome data; the FCDS does not collect environmental exposures, complete genetic information, nor residential history. The data submitted by health care providers to the FCDS describes “who”, “what”, “when”, and “where” of the cancer case. However, the FCDS does not collect data to determine why a particular person has developed cancer. Nor can analyses of FCDS data alone determine why cancers are more or less common than expected in a specific area or population. More information, if available, would need to be gathered to determine causality. This would require a complex, extensive investigation effort of local and state resources beyond public health. The present review of cancer and environmental data does not support conducting an extensive investigation.

Furthermore, there are important contributing factors that are not included in an analysis of the standardized incidence ratio (SIR), such as the lifestyle factors and personal and family medical history of persons diagnosed with the respective cancer type and the length of time the person was living within that zip code. The SIR was adjusted for age and stratified by gender, but other factors, such as personal and family history and lifestyle factors, that increase or decrease one’s risk of developing cancer could not be adjusted for.

At this time, there is no evidence of PFOS/PFOA in the drinking water in the surrounding areas of Patrick Air Force Base. This limits the opportunity for exposure of the local community from these chemicals. For the cancer types in which the observed number of cases were statistically significantly higher than expected, there are various known biologic/genetic and behavioral/lifestyle risk factors that increases one’s risk for developing cancer. Per the Centers for Disease Control and Prevention (CDC) guidance, in some cases an initial analysis can demonstrate statistical significance in the number of

cases but may not lead to a statistically significant association with exposure to environmental contaminants. Also, a statistically significant excess of cancer cases can occur within a given population without a discernible cause and might be a chance occurrence (CDC, 2013).

The Department will continue to monitor cancer incidence in the suspected areas of concern. Moreover, the Department and local cancer stakeholders will continue to provide education on cancer and the respective risk factors for that cancer type and the importance of maintaining a healthy lifestyle (i.e., healthy diet, regular physical activity, routine medical physical exam, etc.). Finally, the Department will continue to work with representatives of the Department of Environmental Protection and the local government of Brevard County to continue to perform environmental testing in the local area when indicated to determine health risks.

Frequently Asked Questions

What is a cancer cluster?

A cancer cluster is defined as a greater-than-expected number of cancer cases that occurs within a group of people in a defined geographica area over a specified period of time. When people learn that several friends, family members, or neighbors have found out they have cancer, cancer clusters are often suspected. Cancer clusters are also sometimes suspected when people who work at the same place or have other factors in common get cancer.

What are the criteria for a group of cancer cases to be considered a cluster?

To be a cancer cluster, a group of cancer cases must meet the following criteria:

- Includes a number of cases of one type of cancer or types of cancer scientifically proven to have the same cause or etiology, rather than several different cancer types.
- The observed number of cases is higher than one would typically observe in a similar setting, e.g. in a group with a similar population, age, race, or gender.

Other important factors in evaluating reports of cancer clusters are:

- A rare type of cancer, rather than common types.
- An increased number of cases of a certain type of cancer in an age group that is not usually affected by that type of cancer.
- The type of cancer involved is a primary (original) cancer not a metastasized (spread from another organ) cancer.

How are suspected cancer clusters investigated?

Not all community concerns of excess cancer require investigation; oftentimes, community concerns can be resolved by providing general cancer educational information, facts and resources.

When needed, a local or state health department gathers information about the suspected cancer cluster. This commonly includes the types of cancer, number of cases, age, sex, race, address, and age at diagnosis of the individuals with cancer. The department reviews this available information and determines if analysis of cancer rates and other investigative steps are needed to better understand the situation.

If the department determines that analysis is needed, this involves confirming the number and types of cancers in the community and comparing this to what might be expected based on state or county rates of cancer. Specific analysis (such as investigating just childhood rates, or just among women in the case of breast cancer) may also be needed depending upon the type of concern. The department communicates and discusses the results of the analysis with the community.

Where do I go for additional information?

Centers for Disease Control and Prevention (CDC) Cancer Clusters

Agency for Toxic Substances and Disease Registry

National Cancer Institute (NCI) Cancer Clusters

What is FCDS (Florida Cancer Data System)?

The FCDS is Florida's statewide cancer surveillance system. The FCDS is legislatively mandated to collect incidence data on all cancers diagnosed in Florida per Section 385.202 *Florida Statute.* The FCDS has been collecting the number of new cancers diagnosed each year statewide since 1981. The FCDS is used to observe cancer trends and provide a research base for studies into the possible causes of cancer.

What kind of cancer cases must be reported to FCDS?

Florida statute requires all malignant cancers reportable with the following *exceptions* - In situ carcinoma of the cervix (CIS), intraepithelial neoplasia grade III of the cervix (CIN III) and intraepithelial neoplasia of the prostate (PIN III) are *not reportable.* Basal and squamous cell carcinoma of non-genital skin sites are *not reportable* regardless of extent of disease at the time of diagnosis or the date of first contact with the reporting facility. *Reportable* on or after diagnosis date of 01/01/2001 are Intraepithelial neoplasia Grade III of vulva (VIN III), vagina (VAIN III) and anus (AIN III) and Myelodysplastic Syndrome (MDS). All patients with an active, benign or borderline brain or central nervous system (CNS) tumor, diagnosed on or after 01/01/2004, whether being treated or not *are reportable.* All cancer cases diagnosed and/or treated in Florida since 1981 must be reported to the FCDS.

What kind of data is collected?

The FCDS requires that the data collected include information which indicates diagnosis, stage of disease, patient demographics, laboratory data, tissue diagnosis and methods of diagnosis or treatment for each cancer diagnosed or treated in Florida.

Who is required to report cancer cases to FCDS?

All facilities licensed under Chapter 395 and each freestanding radiation therapy center under Section 408.07; All ambulatory surgical centers as specified by Rule 64D-3.034; Any licensed practitioner in the state of Florida that practices medicine, osteopathic, chiropractic medicine, naturopathy or veterinary medicine are required to report under Chapter 381 or any laboratory licensed under Chapter 483 that diagnoses or suspects the existence of a cancer.

Appendices

Appendix A: Number of Observed Cases by Cancer Type, Age Group, Gender, and Zip Code

Table A.1. Number of Observed Cases among Females by Cancer Type, Age Group, ZIP 32937, 2006-2015

	Hodgkin Disease	Leukemia	Liver	Non-Hodgkin Lymphoma	Pancreas	Testes	Thyroid	Urinary Bladder
0-4	0	1	0	0	0	0	0	0
5-9	0	1	0	0	0	0	0	0
10-14	0	0	0	0	0	0	0	0
15-19	1	0	0	0	0	0	0	0
20-24	0	0	0	0	0	0	1	0
25-29	1	0	0	0	0	0	0	0
30-34	1	0	0	0	0	0	1	0
35-39	0	0	0	0	0	0	2	0
40-44	0	0	0	0	0	0	2	0
45-49	0	0	0	1	1	0	4	1
50-54	0	1	0	3	0	0	1	1
55-59	0	2	0	2	4	0	2	0
60-64	0	5	0	3	1	0	5	3
65-69	0	0	0	8	4	0	1	1
70-74	0	5	1	5	5	0	1	4
75-79	0	5	1	7	5	0	2	3
80-84	0	4	0	7	1	0	3	8
85+	0	5	0	2	10	0	0	10
Total	3	29	2	38	31	0	25	31

Source: FCDS; Data extracted August 2018

Table A.2. Number of Observed Cases among Males by Cancer Type, Age Group, ZIP 32937, 2006-2015

	Hodgkin Disease	Leukemia	Liver	Non-Hodgkin Lymphoma	Pancreas	Testes	Thyroid	Urinary Bladder
0-4	0	0	0	0	0	0	0	0
5-9	0	0	0	0	0	0	0	0
10-14	1	0	0	0	0	0	0	0
15-19	1	0	0	0	0	0	0	0
20-24	1	0	0	1	0	0	0	0
25-29	2	0	0	0	0	2	2	0
30-34	0	1	0	1	0	2	0	0
35-39	0	0	0	1	0	0	1	0
40-44	1	0	1	0	1	1	1	0
45-49	0	0	0	1	1	0	0	5
50-54	0	0	4	6	0	0	1	5
55-59	0	1	5	4	3	0	1	9
60-64	1	2	7	5	1	0	0	6
65-69	0	5	1	9	6	0	2	9
70-74	0	3	2	3	1	0	3	15
75-79	0	3	2	4	6	1	2	19
80-84	0	5	1	5	4	0	0	11
85+	0	4	1	5	3	0	0	14
Total	7	24	24	45	26	6	13	93

Source: FCDS; Data extracted August 2018

Table A.3. Number of Observed Cases among Females by Cancer Type, Age Group, ZIP 32940, 2006-2015

	Hodgkin Disease	Leukemia	Liver	Non-Hodgkin Lymphoma	Pancreas	Testes	Thyroid	Urinary Bladder
0-4	0	2	0	0	0	0	0	0
5-9	0	0	0	1	0	0	0	0
10-14	0	1	0	0	0	0	1	0
15-19	0	1	0	0	0	0	0	0
20-24	2	0	0	0	0	0	0	0
25-29	1	0	0	2	0	0	1	0
30-34	0	1	0	0	0	0	1	0
35-39	1	0	0	0	0	0	5	0
40-44	1	0	0	0	0	0	0	0
45-49	0	1	0	0	1	0	5	0
50-54	0	2	0	0	1	0	6	3
55-59	0	0	1	6	2	0	2	3
60-64	1	5	0	8	1	0	3	4
65-69	0	3	1	5	8	0	4	6
70-74	0	5	2	7	10	0	1	6
75-79	0	8	2	10	11	0	1	12
80-84	0	5	0	7	5	0	0	3
85+	0	10	0	5	10	0	0	13
Total	6	44	6	51	49	0	30	50

Source: FCDS; Data extracted August 2018

Table A.4. Number of Observed Cases among Males by Cancer Type, Age Group, ZIP 32940, 2006-2015

	Hodgkin Disease	Leukemia	Liver	Non-Hodgkin Lymphoma	Pancreas	Testes	Thyroid	Urinary Bladder
0-4	0	1	0	0	0	0	0	0
5-9	0	1	0	0	0	0	0	0
10-14	0	0	0	0	0	0	0	0
15-19	0	0	0	0	0	0	0	0
20-24	0	0	0	0	0	0	0	0
25-29	0	0	0	2	0	1	0	0
30-34	1	1	0	0	0	1	0	0
35-39	1	1	0	1	0	0	0	0
40-44	1	2	0	4	0	0	1	0
45-49	1	1	2	6	1	0	1	0
50-54	0	3	3	2	2	1	1	3
55-59	0	2	1	6	1	0	0	10
60-64	0	3	6	4	1	0	0	5
65-69	1	7	3	13	6	0	3	16
70-74	0	9	2	12	10	1	3	26
75-79	2	8	3	15	14	0	0	29
80-84	0	8	5	9	10	0	2	21
85+	0	5	2	10	9	0	0	15
Total	7	52	27	84	54	4	11	125

Source: FCDS; Data extracted August 2018

Appendix B: Population

Table B.1. Population by Gender and ZCTA*

	Female		Male	
	32937	**32940**	**32937**	**32940**
0-4	451	606	466	668
5-9	549	871	579	888
10-14	651	1063	750	987
15-19	748	938	783	973
20-24	493	520	588	500
25-29	579	596	584	566
30-34	511	694	526	599
35-39	654	874	607	771
40-44	789	1184	715	994
45-49	1104	1322	962	1267
50-54	1163	1232	1156	1119
55-59	1052	1152	952	948
60-64	920	1208	892	1000
65-69	788	1219	708	1046
70-74	708	1061	620	913
75-79	643	912	562	859
80-84	521	707	412	587
85+	479	640	304	515

Data Source: 2010 U.S. Census Population
*ZCTA – Zip Code Tabulation Area

Table B.2. Florida Population, 2006 - 2015

Age Group	Male	Female
0-4	5,585,078	5,364,245
5-9	5,620,473	5,396,318
10-14	5,794,991	5,541,825
15-19	6,132,425	5,858,491
20-24	6,354,615	6,043,796
25-29	5,974,341	5,758,777
30-34	5,660,879	5,599,080
35-39	5,734,789	5,750,837
40-44	6,191,663	6,269,773
45-49	6,621,122	6,784,296
50-54	6,534,992	6,877,783
55-59	5,973,472	6,512,315
60-64	5,332,959	6,005,475
65-69	4,539,095	5,217,142
70-74	3,665,737	4,295,139
75-79	2,884,264	3,522,650
80-84	2,196,456	2,936,252
85+	1,722,817	2,951,935

Data Source: Florida Legislature's Office of Economic and Demographic Research (EDR)

Appendix C: Risk Factors

Information compiled from the American Cancer Society

Kidney Cancer

Gender
Renal cell carcinoma (RCC) is about twice as common in men as in women. Men are more likely to be smokers and are more likely to be exposed to cancer-causing chemicals at work, which may account for some of the difference.

Race
African Americans and American Indians/Alaska Natives have slightly higher rates of RCC than do Whites. The reasons for this are not clear.

Family history of kidney cancer
People with a strong family history of renal cell cancer (without one of the known inherited conditions listed below) have a higher chance of developing this cancer. This risk is highest for people who have a brother or sister with the cancer. It's not clear whether this is due to shared genes, something that both people were exposed to in the environment, or both.

Genetic and hereditary risk factors
Some people inherit a tendency to develop certain types of cancer. The DNA in each of your cells that you get from your parents may have changes that give you this tendency. Some rare inherited conditions can cause kidney cancer. It is important that people who have hereditary causes of RCC see their doctors often, especially if they have already been diagnosed with RCC. Some doctors recommend regular imaging tests (such as CT scans) to look for new kidney tumors in these people.

People who have the conditions listed here have a much higher risk for getting kidney cancer, although they account for only a small portion of cases overall.

von Hippel-Lindau (VHL) disease
People with this condition often develop several kinds of tumors and cysts (fluid-filled sacs) in different parts of the body. They have an increased risk for developing clear cell RCC, especially at a younger age. They may also have benign tumors in their eyes, brain, spinal cord, pancreas, and other organs; and a type of adrenal gland tumor called *pheochromocytoma*. This condition is caused by mutations (changes) in the *VHL* gene.

Hereditary papillary renal cell carcinoma
People with this condition have a tendency to develop one or more papillary RCCs, but they do not have tumors in other parts of the body, as is the case with the other inherited conditions listed here. This disorder is usually linked to changes in the *MET* gene.

Hereditary leiomyoma-renal cell carcinoma
People with this syndrome develop smooth muscle tumors called *leiomyomas* (fibroids) of the skin and uterus (in women) and have a higher risk for developing papillary RCCs. It has been linked to changes in the *FH* gene.

Birt-Hogg-Dube (BHD) syndrome
People with this syndrome develop many small benign skin tumors and have an increased risk of different kinds of kidney tumors, including RCCs and oncocytomas. They may also have benign or malignant tumors of several other tissues. The gene linked to BHD is known as *FLCN*.

Familial renal cancer
People with this condition develop tumors called *paragangliomas* of the head and neck region, as well as tumors known as *pheochromocytomas* of the adrenal glands and thyroid cancers. They also tend to get kidney cancer in both kidneys before age 40. It is caused by defects in the genes *SDHB* and *SDHD*.

Cowden syndrome
People with this syndrome have a high risk of breast, thyroid and kidney cancers. It is linked to changes in the *PTEN* gene.

Tuberous sclerosis
People with this syndrome develop many, usually benign (noncancerous) tumors in different parts of the body including the skin, brain, lungs, eyes, kidneys, and heart. Although the kidney tumors are most often benign, occasionally they can be clear cell RCC. It is caused by defects in the genes *TSC1* and *TSC2*.

Hereditary renal oncocytoma
Some people inherit the tendency to develop a kidney tumor called an *oncocytoma*, which is almost always benign (not cancer).

Smoking
Smoking increases the risk of developing renal cell carcinoma (RCC). The increased risk seems to be related to how much you smoke. The risk drops if you stop smoking, but it takes many years to get to the risk level of someone who never smoked.

Obesity
People who are very overweight have a higher risk of developing RCC. Obesity may cause changes in certain hormones that can lead to RCC.

Workplace exposures
Many studies have suggested that workplace exposure to certain substances increases the risk for RCC. Some of these substances are cadmium (a type of metal), some herbicides, and organic solvents, particularly trichloroethylene.

High blood pressure
The risk of kidney cancer is higher in people with high blood pressure. Some studies have suggested that certain medicines used to treat high blood pressure may raise the risk of kidney cancer, but it is hard to tell if it's the condition or the medicine (or both) that may be the cause of the increased risk.

Certain medicines
Phenacetin: This drug used to be a popular non-prescription pain reliever and has been linked to RCC in the past. But it has not been available in the United States for over 30 years, so it no longer appears to be a major risk factor.

Diuretics: Some studies have suggested that diuretics (water pills) may be linked to a small increase in the risk of RCC. It is not clear whether the cause is the drugs or the high blood pressure they treat. If you need to take diuretics, don't avoid them to try to reduce the risk of kidney cancer.

Advanced kidney disease
People with advanced kidney disease, especially those needing dialysis, have a higher risk of RCC. Dialysis is a treatment used to remove toxins from your body if the kidneys do not work properly.

Liver Cancer

Gender
Hepatocellular carcinoma (HCC) is much more common in males than in females. Much of this is probably because of behaviors affecting some of the risk factors. The fibrolamellar subtype of HCC is more common in women.

Race/ethnicity
In the United States, Asian Americans and Pacific Islanders have the highest rates of liver cancer, followed by American Indians/Alaska Natives and Hispanics/Latinos, Blacks, and Whites.

Chronic viral hepatitis (Hep-B or Hep-C)
Worldwide, the most common risk factor for liver cancer is chronic (long-term) infection with hepatitis B virus (HBV) or hepatitis C virus (HCV). These infections lead to cirrhosis of the liver and are responsible for making liver cancer the most common cancer in many parts of the world. In the United States, infection with hepatitis C is the more common cause of HCC. People infected with both viruses have a high risk of developing chronic hepatitis, cirrhosis, and liver cancer. The risk is even higher if they are heavy drinkers (at least 6 standard drinks a day).

HBV and HCV can spread from person to person through sharing contaminated needles (such as in drug use), unprotected sex, or childbirth. They can also be passed on through blood transfusions, although this is very rare in the United States since the start of blood product testing for these viruses. In developing countries, children sometimes contract hepatitis B infection from prolonged contact with family members who are infected.

HBV is more likely to cause symptoms, such as a flu-like illness and a yellowing of the eyes and skin (jaundice). But most people recover completely from HBV infection within a few months. Only a very small percentage of adults become chronic carriers (and have a higher risk for liver cancer). Infants and small children who become infected have a higher risk of becoming chronic carriers.

HCV, on the other hand, is less likely to cause symptoms. But most people with HCV develop chronic infections, which are more likely to lead to liver damage or even cancer. Other viruses, such as the hepatitis A virus and hepatitis E virus, can also cause hepatitis. But people infected with these viruses do not develop chronic hepatitis or cirrhosis, and do not have an increased risk of liver cancer.

Cirrhosis
Cirrhosis is a disease in which liver cells become damaged and are replaced by scar tissue. People with cirrhosis have an increased risk of liver cancer. Most (but not all) people who develop liver cancer already have some evidence of cirrhosis. There are several possible causes of cirrhosis. Most cases in the United States occur in people who abuse alcohol or have chronic HBV or HCV infections.

Non-alcoholic fatty liver disease
Non-alcoholic fatty liver disease, a condition in which people who consume little or no alcohol develop a fatty liver, is common in obese people. People with a type of this disease known as *non-alcoholic steatohepatitis* (NASH) might go on to develop cirrhosis.

Primary biliary cirrhosis
Some types of autoimmune diseases that affect the liver can also cause cirrhosis. For example, there is also a disease called *primary biliary cirrhosis (PBC)*. In PBC, the bile ducts in the liver are damaged and even destroyed which can lead to cirrhosis. People with advanced PBC have a high risk of liver cancer.

Inherited metabolic diseases
Certain inherited metabolic diseases can lead to cirrhosis. People with *hereditary hemochromatosis* absorb too much iron from their food. The iron settles in tissues throughout the body, including the liver. If enough iron builds up in the liver, it can lead to cirrhosis and liver cancer.

Heavy alcohol use
Alcohol abuse is a leading cause of cirrhosis in the United States, which in turn is linked with an increased risk of liver cancer.

Obesity
Being obese (very overweight) increases the risk of developing liver cancer. This is probably because it can result in fatty liver disease and cirrhosis.

Type 2 diabetes
Type 2 diabetes has been linked with an increased risk of liver cancer, usually in patients who also have other risk factors such as heavy alcohol use and/or chronic viral hepatitis. This risk may be increased because people with type 2 diabetes tend to be overweight or obese, which in turn can cause liver problems.

Certain rare diseases
Diseases that increase the risk of liver cancer include:

- Tyrosinemia
- Alpha1-antitrypsin deficiency
- Porphyria cutanea tarda
- Glycogen storage diseases
- Wilson disease

Aflatoxins
These cancer-causing substances are made by a fungus that contaminates peanuts, wheat, soybeans, ground nuts, corn, and rice. Storage in a moist, warm environment can lead to the growth of this fungus. Although this can occur almost anywhere in the world, it is more common in warmer and tropical countries. Developed countries such as the United States and those in Europe regulate the content of aflatoxins in foods through testing. Long-term exposure to these substances is a major risk factor for liver cancer. The risk is increased even more in people with hepatitis B or C infections.

Vinyl chloride and thorium dioxide (Thorotrast)
Exposure to these chemicals raises the risk of angiosarcoma of the liver. It also increases the risk of developing cholangiocarcinoma and hepatocellular cancer, but to a far lesser degree. Vinyl chloride is a chemical used in making some kinds of plastics. Thorotrast is a chemical that in the past was injected

into some patients as part of certain x-ray tests. When the cancer-causing properties of these chemicals were recognized, steps were taken to eliminate them or minimize exposure to them. Thorotrast is no longer used, and exposure of workers to vinyl chloride is strictly regulated.

Anabolic steroids
Anabolic steroids are male hormones used by some athletes to increase their strength and muscle mass. Long-term anabolic steroid use can slightly increase the risk of hepatocellular cancer. Cortisone-like steroids, such as hydrocortisone, prednisone, and dexamethasone, do not carry this same risk.

Arsenic
Drinking water contaminated with naturally occurring arsenic, such as that from some wells, over a long period of time increases the risk of some types of liver cancer. This is more common in parts of East Asia, but it might also be a concern in some areas of the United States.

Infection with parasites
Infection with the parasite that causes schistosomiasis can cause liver damage and is linked to liver cancer. This parasite is not found in the US, but infection can occur in Asia, Africa, and South America.

Tobacco use
Smoking increases the risk of liver cancer. Former smokers have a lower risk than current smokers, but both groups have a higher risk than those who never smoked.

Pancreatic Cancer

Tobacco use
Smoking is one of the most important risk factors for pancreatic cancer. The risk of getting pancreatic cancer is about twice as high among smokers compared to those who have never smoked. About 20% to 30% of pancreatic cancers are thought to be caused by cigarette smoking. Cigar and pipe smoking also increase risk, as does the use of smokeless tobacco products.

Overweight and obesity
Being overweight is a risk factor for pancreatic cancer. Very overweight (obese) people are about 20% more likely to develop pancreatic cancer. Carrying extra weight around the waistline may be a risk factor even in people who are not very overweight.

Workplace exposure to certain chemicals
Heavy exposure at work to certain chemicals used in the dry cleaning and metal working industries may raise a person's risk of pancreatic cancer.

Age
The risk of developing pancreatic cancer goes up as people age. Almost all patients are older than 45. About two-thirds are at least 65 years old. The average age at the time of diagnosis is 71.

Gender
Men are slightly more likely to develop pancreatic cancer than women. This may be due, at least in part, to higher tobacco use in men, which raises pancreatic cancer risk. The difference in pancreatic cancer risk was larger in the past (when tobacco use was much more common among men than women), but the gap has closed in recent years.

Race
Blacks are slightly more likely to develop pancreatic cancer than Whites. The reasons for this aren't clear, but it may be due in part to having higher rates of some other risk factors for pancreatic cancer, such as diabetes, smoking in men, and being overweight in women.

Family history
Pancreatic cancer seems to run in some families. In some of these families, the high risk is due to an inherited syndrome. In other families, the gene causing the increased risk is not known. Although family history is a risk factor, most people who get pancreatic cancer do not have a family history of it.

Inherited genetic syndromes
Inherited gene changes (mutations) can be passed from parent to child. These gene changes may cause as many as 10% of pancreatic cancers. Sometimes these changes result in syndromes that include increased risks of other cancers (or other health problems). Examples of genetic syndromes that can cause exocrine pancreatic cancer include:

- **Hereditary breast and ovarian cancer syndrome**, caused by mutations in the *BRCA1* or *BRCA2* genes
- **Familial atypical multiple mole melanoma (FAMMM) syndrome**, caused by mutations in the *p16/CDKN2A* gene
- **Familial pancreatitis**, usually caused by mutations in the *PRSS1* gene
- **Lynch syndrome**, also known as *hereditary non-polyposis colorectal cancer* (HNPCC), most often caused by a defect in the *MLH1* or *MSH2* genes.
- **Peutz-Jeghers syndrome**, caused by defects in the *STK11* gene. This syndrome is also linked with polyps in the digestive tract and several other cancers.
- **Von Hippel-Lindau syndrome**, caused by mutations in the *VHL* gene. It can lead to an increased risk of pancreatic cancer and carcinoma of the ampulla of Vater.

Pancreatic neuroendocrine tumors and cancers can also be caused by genetic syndromes, such as:

- **Neurofibromatosis, type 1**, which is caused by mutations in the *NF1* gene. This syndrome leads to an increased risk of many tumors, including somatostatinomas.
- **Multiple endocrine neoplasia, type I (MEN1)**, caused by mutations in the *MEN1* gene. This syndrome leads to an increased risk of tumors of the parathyroid gland, the pituitary gland, and the islet cells of the pancreas.

Changes in the genes that cause some of these syndromes can be found by genetic testing. For more information on genetic testing please contact your medical provider.

Diabetes
Pancreatic cancer is more common in people with diabetes. The reason for this is not known. Most of the risk is found in people with type 2 diabetes. This type of diabetes most often starts in adulthood and is often related to being overweight or obese. It's not clear if people with type 1 (juvenile) diabetes have a higher risk.

Chronic pancreatitis
Chronic pancreatitis, a long-term inflammation of the pancreas, is linked with an increased risk of pancreatic cancer (especially in smokers), but most people with pancreatitis never develop pancreatic cancer. Chronic pancreatitis is sometimes due to an inherited gene mutation. People with this inherited (familial) form of pancreatitis have a high lifetime risk of pancreatic cancer.

Cirrhosis of the liver
Cirrhosis is a scarring of the liver. It develops in people with liver damage from things like hepatitis and heavy alcohol use. People with cirrhosis seem to have an increased risk of pancreatic cancer.

Stomach problems
Infection of the stomach with the ulcer-causing bacteria *Helicobacter pylori* (*H. pylori*) may increase the risk of getting pancreatic cancer. Some research has suggested that excess stomach acid might also increase the risk.

Testicular Cancer

Undescended testicle
One of the main risk factors for testicular cancer is a condition called cryptorchidism, or undescended testicle(s). This means that one or both testicles fail to move from the abdomen (belly) into the scrotum before birth. Males with cryptorchidism are many times more likely to get testicular cancer than those with normally descended testicles.

Normally, the testicles develop inside the abdomen of the fetus and they go down (descend) into the scrotum before birth. But in about 3% of boys, the testicles do not make it all the way down before the child is born. Sometimes one or both testicles stay in the abdomen. In other cases, the testicles start to descend but stay in the groin area.

Most of the time, undescended testicles continue moving down into the scrotum during the child's first year of life. If the testicle hasn't descended by the time a child is a year old, it probably isn't going to do it on its own. If cancer does develop, it's usually in the undescended testicle, but about 1 out of 4 cases occur in the normally descended testicle. Because of this, some doctors conclude that cryptorchidism doesn't actually cause testicular cancer, but that there's something else that leads to both testicular cancer and abnormal positioning of one or both testicles.

Family history
Having a father or brother with testicular cancer increases the risk that you will get it, too. But only a small number of testicular cancers occur in families. Most men with testicular cancer do not have a family history of the disease. Kleinfelter's syndrome is an inherited disease that's also linked to an increased risk of testicular cancer.

HIV infection
Some evidence has shown that men infected with the human immunodeficiency virus (HIV), particularly those with AIDS, are at increased risk. No other infections have been shown to increase testicular cancer risk.

Carcinoma in situ
This condition, often doesn't cause a lump in the testicles or any other symptoms. It isn't clear how often carcinoma in situ (CIS) in the testicles progresses to cancer. In some cases, CIS is found in men who have a testicular biopsy to evaluate infertility or have a testicle removed because of cryptorchidism.

Cancer in the other testicle
A personal history of testicular cancer is another risk factor. About 3% or 4% of men who have been cured of cancer in one testicle will at some point develop cancer in the other testicle.

Age
About half of testicular cancers occur in men between the ages of 20 and 34. But this cancer can affect males of any age, including infants and elderly men.

Race and ethnicity
The risk of testicular cancer among white men is about 4 to 5 times that of Black and Asian-American men. The risk for American Indians falls between that of Asians and Whites. The reason for these differences is not known. Worldwide, the risk of developing this disease is highest among men living in the United States and Europe and lowest among men living in Africa or Asia.

Urinary Bladder Cancer

Smoking
Smoking is the most important risk factor for bladder cancer. Smokers are at least three times as likely to get bladder cancer as nonsmokers. Smoking causes about half of all bladder cancers in both men and women.

Workplace exposures
Certain industrial chemicals have been linked with bladder cancer. Chemicals called *a*romatic amines, such as benzidine and beta-naphthylamine, which are sometimes used in the dye industry, can cause bladder cancer.

Workers in other industries that use certain organic chemicals also may have a higher risk of bladder cancer. Industries carrying higher risks include makers of rubber, leather, textiles, and paint products as well as printing companies. Other workers with an increased risk of developing bladder cancer include painters, machinists, printers, hairdressers (probably because of heavy exposure to hair dyes), and truck drivers (likely because of exposure to diesel fumes).

Cigarette smoking and workplace exposures can act together to cause bladder cancer. Smokers who also work with cancer-causing chemicals have an especially high risk of bladder cancer.

Certain medicines or herbal supplements
According to the US Food and Drug Administration (FDA), use of the diabetes medicine pioglitazone (Actos) for more than one year may be linked with an increased risk of bladder cancer. This possible link is still an area of active research.

Dietary supplements containing aristolochic acid (mainly in herbs from the *Aristolochia* family) have been linked with an increased risk of urothelial cancers, including bladder cancer.

Arsenic in drinking water
Arsenic in drinking water has been linked with a higher risk of bladder cancer in some parts of the world. The chance of being exposed to arsenic depends on where you live and whether you get your water from a well or from a public water system that meets the standards for low arsenic content. For most Americans, drinking water is not a major source of arsenic.

Not drinking enough fluids
People who drink a lot of fluids, especially water, each day tend to have lower rates of bladder cancer. This might be because they empty their bladders more often, which could keep chemicals from lingering in their bladder.

Race and ethnicity
Whites are about twice as likely to develop bladder cancer as Blacks and Hispanics. Asian Americans and American Indians have slightly lower rates of bladder cancer. The reasons for these differences are not well understood.

Age
The risk of bladder cancer increases with age. About nine out of ten people with bladder cancer are older than 55.

Gender
Bladder cancer is much more common in men than in women.

Chronic bladder irritation and infections
Urinary infections, kidney and bladder stones, bladder catheters left in place a long time, and other causes of chronic bladder irritation have been linked with bladder cancer (especially squamous cell carcinoma of the bladder), but it's not clear if they actually cause bladder cancer.
Schistosomiasis (also known as *bilharziasis*), an infection with a parasitic worm that can get into the bladder, is also a risk factor for bladder cancer. In countries where this parasite is common (mainly in Africa and the Middle East), squamous cell cancers of the bladder are seen much more often. This is an extremely rare cause of bladder cancer in the United States.

Personal history of bladder or other urothelial cancer
Urothelial carcinomas can sometimes form in different areas in the bladder, as well as in the lining of the kidney, the ureters, and urethra. Having a cancer in the lining of any part of the urinary tract puts you at higher risk of having another cancer, either in the same area as before, or in another part of the urinary tract. This is true even when the first tumor is removed completely. For this reason, people who have had bladder cancer need careful follow-up to look for new cancers.

Bladder birth defects
Before birth, there is a connection between the belly button and the bladder. This is called the *urachus*. If part of this connection remains after birth, it could become cancerous. Cancers that start in the urachus are usually adenocarcinomas, which are made up of cancerous gland cells. About one-third of the adenocarcinomas of the bladder start here. However, this is still rare, accounting for less than half of 1% of all bladder cancers.

Another rare birth defect called *exstrophy* greatly increases a person's risk of bladder cancer. In bladder exstrophy, both the bladder and the abdominal wall in front of the bladder don't close completely during fetal development and are fused together. This leaves the inner lining of the bladder exposed outside the body. Surgery soon after birth can close the bladder and abdominal wall (and repair other related defects), but people who have this still have a higher risk for urinary infections and bladder cancer.

Genetics and family history
People who have family members with bladder cancer have a higher risk of getting it themselves. Sometimes this may be because the family members are exposed to the same cancer-causing chemicals (such as those in tobacco smoke). They may also share changes in some genes (like *GST* and *NAT*) that make it hard for their bodies to break down certain toxins, which can make them more likely to get bladder cancer. A small number of people inherit a gene syndrome that increases their risk for bladder cancer. For example:

- A mutation of the **retinoblastoma** (*RB1*) gene can cause cancer of the eye in infants, and also increases the risk of bladder cancer.
- **Cowden disease**, caused by mutations in the *PTEN* gene, is linked mainly to cancers of the breast and thyroid. People with this disease also have a higher risk of bladder cancer.
- **Lynch syndrome** (also known as hereditary non-polyposis colorectal cancer, or HNPCC) is linked mainly to colon and endometrial cancer. People with this syndrome might also have an increased risk of bladder cancer (as well as other cancers of the urinary tract).

Prior chemotherapy or radiation therapy
Taking the chemotherapy drug cyclophosphamide (Cytoxan) for a long time can irritate the bladder and increase the risk of bladder cancer. People taking this drug are often told to drink plenty of fluids to help protect the bladder from irritation.People who are treated with radiation to the pelvis are more likely to develop bladder cancer.

Thyroid Cancer

Gender and age
For unclear reasons, thyroid cancers (like almost all diseases of the thyroid) occur about three times more often in women than in men. Thyroid cancer can occur at any age, but the risk peaks earlier for women (who are most often in their 40s or 50s when diagnosed) than for men (who are usually in their 60s or 70s).

Hereditary conditions
Several inherited conditions have been linked to different types of thyroid cancer, as has family history. Still, most people who develop thyroid cancer do not have an inherited condition or a family history of the disease.

Medullary thyroid cancer: About two out of ten medullary thyroid carcinomas (MTCs) result from inheriting an abnormal gene. These cases are known as familial medullary thyroid carcinoma (FMTC). FMTC can occur alone, or it can be seen along with other tumors. The combination of FMTC and tumors of other endocrine glands is called *multiple endocrine neoplasia type 2* (MEN 2). Ask your doctor about having regular blood tests or ultrasound exams to look for problems and the possibility of genetic testing.

Other thyroid cancers: People with certain inherited medical conditions have a higher risk of more common forms of thyroid cancer. Higher rates of thyroid cancer occur among people with uncommon genetic conditions such as:

Familial adenomatous polyposis (FAP): People with this syndrome develop many colon polyps and have a very high risk of colon cancer. They also have an increased risk of some other cancers, including papillary thyroid cancer. *Gardner syndrome* is a subtype of FAP in which patients also get certain benign tumors. Both Gardner syndrome and FAP are caused by defects in the gene *APC*.

Cowden disease: People with this syndrome have an increased risk of thyroid problems and certain benign growths (including some called hamartomas). They also have an increased risk of cancers of the thyroid, uterus, breast, as well as some others. The thyroid cancers tend to be either the papillary or follicular type. This syndrome is most often caused by defects in the gene *PTEN*. It is also known as Multiple Hamartoma Syndrome and PTEN Hamartoma Tumor Syndrome

Carney complex, type I: People with this syndrome may develop a number of benign tumors and hormone problems. They also have an increased risk of papillary and follicular thyroid cancers. This syndrome is caused by defects in the gene *PRKAR1A*.

Familial nonmedullary thyroid carcinoma: Thyroid cancer occurs more often in some families and is often seen at an earlier age. The papillary type of thyroid cancer most often runs in families. Genes on chromosome 19 and chromosome 1 are suspected of causing these familial cancers.

If you suspect you might have a familial condition, talk with your doctor, who might recommend genetic counseling if your medical history warrants it.

Family history
Having a first-degree relative (parent, brother, sister, or child) with thyroid cancer, even without a known inherited syndrome in the family, increases your risk of thyroid cancer. The genetic basis for these cancers is not totally clear.

A diet low in iodine
Follicular thyroid cancers are more common in areas of the world where people's diets are low in iodine. In the United States, most people get enough iodine in their diet because it is added to table salt and other foods. A diet low in iodine may also increase the risk of papillary cancer if the person also is exposed to radioactivity.

Radiation
Radiation exposure is a proven risk factor for thyroid cancer. Sources of such radiation include certain medical treatments and radiation fallout from power plant accidents or nuclear weapons.
Having had head or neck radiation treatments in childhood is a risk factor for thyroid cancer. Risk depends on how much radiation is given and the age of the child. In general, the risk increases with larger doses and with younger age at treatment.

Before the 1960s, children were sometimes treated with low doses of radiation for things we wouldn't use radiation for now, like acne, fungus infections of the scalp (ringworm), or enlarged tonsils or adenoids. Years later, the people who had these treatments were found to have a higher risk of thyroid cancer. Radiation therapy in childhood for some cancers such as lymphoma, Wilms tumor, and neuroblastoma also increases risk. Thyroid cancers that develop after radiation therapy are not more serious than other thyroid cancers.

Imaging tests such as x-rays and CT scans also expose children to radiation, but at much lower doses, so it's not clear how much they might raise the risk of thyroid cancer (or other cancers). If there is an increased risk it is likely to be small, but to be safe, children should not have these tests unless they are absolutely needed. When they are needed, they should be done using the lowest dose of radiation that still provides a clear picture.

Several studies have pointed to an increased risk of thyroid cancer in children because of radioactive fallout from nuclear weapons or power plant accidents. Some radioactive fallout occurred over certain regions of the United States after nuclear weapons were tested in western states during the 1950s. A higher risk of thyroid cancer has not been proven at these low exposure levels. If you are concerned about possible exposure to radioactive fallout, discuss this with your doctor. Being exposed to radiation when you are an adult carries much less risk of thyroid cancer.

Hodgkins Lymphoma

Epstein-Barr virus infection/mononucleosis
The Epstein-Barr virus (EBV) causes infectious mononucleosis (often called mono). People who have had mono have an increased risk of Hodgkins Lymphoma (HL). But even though the risk is higher than for people who haven't had mono, it is still very small (about 1 in 1,000).

The exact role of EBV in the development of HL isn't clear. Many people are infected with EBV, but very few develop HL. Parts of the virus are found in Reed-Sternberg cells in about 1 out of 4 people with classic HL in the US. But most people with HL have no signs of EBV in their cancer cells.

Age
People can be diagnosed with HL at any age, but it's most common in early adulthood (especially in a person's 20s) and in late adulthood (after age 55).

Gender
HL occurs slightly more often in males than in females.

Family history
Brothers and sisters of young people with this disease have a higher risk for HL. The risk is very high for an identical twin of a person with HL. But a family link is still uncommon – most people with HL do not have a family history of it. It's not clear why family history might increase risk. It might be because family members have similar childhood exposures to certain infections (such as Epstein-Barr virus), because they share inherited gene changes that make them more likely to get HL, or some combination of these factors.

Weakened immune system
The risk of HL is increased in people infected with HIV, the virus that causes AIDS.
People who take medicines to suppress the immune system after an organ transplant and people with auto-immune diseases are also at higher than normal risk for HL.

Non-Hodgkin Lymphoma

Age
Getting older is a strong risk factor for lymphoma overall, with most cases occurring in people in their 60s or older. But some types of lymphoma are more common in younger people.

Gender
Overall, the risk of non-Hodgkins Lymphoma (NHL) is higher in men than in women, but there are certain types of NHL that are more common in women. The reasons for this are not known.

Race, ethnicity, and geography
In the United States, Whites are more likely than Blacks and Asian Americans to develop NHL.

Family History
Having a first-degree relative (parent, child, sibling) with NHL increases your risk of developing NHL.

Exposure to certain chemicals and drugs

Some studies have suggested that chemicals such as benzene and certain herbicides and insecticides (weed- and insect-killing substances) may be linked to an increased risk of NHL. Research to clarify these possible links is still in progress.

Some chemotherapy drugs used to treat other cancers may increase the risk of developing NHL many years later. For example, patients who have been treated for Hodgkin lymphoma have an increased risk of later developing NHL. But it's not totally clear if this is related to the disease itself or if it is an effect of the treatment.

Some studies have suggested that certain drugs used to treat rheumatoid arthritis (RA), such as methotrexate and the tumor necrosis factor (TNF) inhibitors, might increase the risk of NHL. But other studies have not found an increased risk. Determining if these drugs increase risk is complicated by the fact that people with RA, which is an autoimmune disease, already have a higher risk of NHL.

Radiation exposure
Studies of survivors of atomic bombs and nuclear reactor accidents have shown they have an increased risk of developing several types of cancer, including NHL, leukemia, and thyroid cancer.

Patients treated with radiation therapy for some other cancers, such as Hodgkin lymphoma, have a slightly increased risk of developing NHL later in life. This risk is greater for patients treated with both radiation therapy and chemotherapy.

Having a weakened immune system
People with weakened immune systems have an increased risk for NHL. For example:

- People who receive organ transplants are treated with drugs that suppress their immune system to prevent it from attacking the new organ. These people have a higher risk of developing NHL.
- The human immunodeficiency virus (HIV) can weaken the immune system, and people infected with HIV are at increased risk of NHL.
- In some genetic (inherited) syndromes, such as ataxia-telangiectasia (AT) and Wiskott-Aldrich syndrome, children are born with a deficient immune system. Along with an increased risk of serious infections, these children also have a higher risk of developing NHL.

Autoimmune diseases
Some autoimmune diseases such as rheumatoid arthritis, systemic lupus erythematosus (SLE or lupus), Sjogren (Sjögren) disease, celiac disease (gluten-sensitive enteropathy), and others have been linked with an increased risk of NHL.

In autoimmune diseases, the immune system mistakenly sees the body's own tissues as foreign and attacks them, as it would a germ. Lymphocytes (the cells from which lymphomas start) are part of the body's immune system. The overactive immune system in autoimmune diseases may make lymphocytes grow and divide more often than normal. This might increase the risk of them developing into lymphoma cells.

Certain infections
Some types of infections may raise the risk of NHL in different ways.

Infections that directly transform lymphocytes
Some viruses can directly affect the DNA of lymphocytes, helping to transform them into cancer cells:

- Infection with human T-cell lymphotropic virus (HTLV-1) increases a person's risk of certain types of T-cell lymphoma. This virus is most common in some parts of Japan and in the

Caribbean region, but it's found throughout the world. In the United States, it causes less than 1% of lymphomas. HTLV-1 spreads through sex and contaminated blood and can be passed to children through breast milk from an infected mother.

- Infection with the Epstein-Barr virus (EBV) is an important risk factor for Burkitt lymphoma in some parts of Africa. In developed countries such as the United States, EBV is more often linked with lymphomas in people also infected with HIV, the virus that causes AIDS. EBV has also been linked with some less common types of lymphoma.

- Human herpes virus 8 (HHV-8) can also infect lymphocytes, leading to a rare type of lymphoma called primary effusion lymphoma. This lymphoma is most often seen in patients who are infected with HIV. HHV-8 infection is also linked to another cancer, Kaposi sarcoma. For this reason, another name for this virus is *Kaposi sarcoma-associated herpes virus* (KSHV).

Infections that weaken the immune system
Infection with human immunodeficiency virus (HIV), also known as the AIDS virus, can weaken the immune system. HIV infection is a risk factor for developing certain types of NHL, such as primary CNS lymphoma, Burkitt lymphoma, and diffuse large B-cell lymphoma.

Infections that cause chronic immune stimulation
Some long-term infections may increase a person's risk of lymphoma by forcing their immune system to be constantly active. As more lymphocytes are made to fight the infection, there is a greater chance for mutations in key genes to occur, which might eventually lead to lymphoma. Some of the lymphomas linked with these infections get better when the infection is treated.

- *Helicobacter pylori*, a type of bacteria known to cause stomach ulcers, has also been linked to mucosa-associated lymphoid tissue (MALT) lymphoma of the stomach.

- *Chlamydophila psittaci* (formerly known as *Chlamydia psittaci*) is a type of bacteria that can cause a lung infection called *psittacosis*. It has been linked to MALT lymphoma in the tissues around the eye (called *ocular adnexal marginal zone lymphoma*).

- Infection with the bacterium *Campylobacter jejuni* has been linked to a type of MALT lymphoma called *immunoproliferative small intestinal disease*. This type of lymphoma, which is also sometimes called *Mediterranean abdominal lymphoma*, typically occurs in young adults in eastern Mediterranean countries.

- Long-term infection with the hepatitis C virus (HCV) seems to be a risk factor for certain types of lymphoma, such as splenic marginal zone lymphoma.

Body weight and diet
Some studies have suggested that being overweight or obese may increase your risk of NHL. Other studies have suggested that a diet high in fat and meats may raise your risk. More research is needed to confirm these findings. In any event, staying at a healthy weight and eating a healthy diet have many known health benefits outside of the possible effect on lymphoma risk.

Breast implants
Although it is rare, some women with breast implants develop a type of anaplastic large cell lymphoma (ALCL) in their breast. This seems to be more likely with implants that have textured (rough) surfaces (as opposed to smooth surfaces).

Leukemias

Acute Myeloid Leukemia (AML) can occur at any age, but it becomes more common as people get older. AML is more common in males than in females. The reason for this is not clear.

Smoking
The only proven lifestyle-related risk factor for AML is smoking. Many people know that smoking is linked to cancers of the lungs, mouth, and throat, but few realize that it can also affect cells that don't come into direct contact with tobacco smoke. Cancer-causing substances in tobacco smoke are absorbed by the lungs and spread through the bloodstream to many parts of the body.

Exposure to Chemicals
The risk of AML is increased if you have been exposed to certain chemicals. For example, long-term exposure to benzene is a risk factor for AML. Benzene is a solvent used in the rubber industry, oil refineries, chemical plants, shoe manufacturing, and gasoline-related industries, and is also found in cigarette smoke, gasoline and motor vehicle exhaust, and some glues, cleaning products, detergents, art supplies, and paints. Some studies have linked AML risk to heavy workplace exposure to formaldehyde, but this link has not been seen in some other studies.

Chemotherapy Drugs
Patients with cancer who are treated with certain chemotherapy (chemo) drugs are more likely to develop AML in the years following treatment. Drugs called alkylating agents are linked to an increased risk of AML. Often a patient will get a disease called a myelodysplastic syndrome before the AML. Examples of alkylating drugs include cyclophosphamide, mechlorethamine, procarbazine, chlorambucil, melphalan, busulfan, carmustine, cisplatin, and carboplatin.

Chemo drugs known as topoisomerase II inhibitors are also linked to AML. AML linked to these drugs tends to occur without myelodysplastic syndrome developing first. Examples of topoisomerase II inhibitors include etoposide, teniposide, mitoxantrone, epirubicin, and doxorubicin.

Radiation Exposure
High-dose radiation exposure (such as being a survivor of an atomic bomb blast or nuclear reactor accident) increases the risk of developing AML. Radiation treatment for cancer has also been linked to an increased risk of AML. The risk varies based on the amount of radiation given and what area is treated.

The possible risks of leukemia from exposure to lower levels of radiation, such as from imaging tests like x-rays or CT scans, are not well-defined. Exposure to such radiation, especially very early in life, might carry an increased risk of leukemia, but how much of a risk is not clear. If there is an increased risk it is likely to be small, but to be safe, most doctors try to limit radiation exposure from tests as much as possible, especially in children and pregnant women.

Blood Disorders
People with certain blood disorders seem to be at increased risk for getting AML. These include chronic myeloproliferative disorders such as polycythemia vera, essential thrombocythemia, and idiopathic myelofibrosis. The risk of AML increases if these disorders are treated with some types of chemotherapy or radiation.

Some people who have a myelodysplastic syndrome (MDS) may develop AML. Patients with MDS have low blood cell counts and abnormal cells in the blood and bone marrow. MDS can evolve over time into AML. AML that develops after MDS is often hard to treat.

Genetic Syndrome
Some syndromes that are caused by genetic mutations (abnormal changes) present at birth seem to raise the risk of AML. These include:

- Fanconi anemia
- Bloom syndrome
- Ataxia-telangiectasia
- Diamond-Blackfan anemia
- Schwachman-Diamond syndrome
- Li-Fraumeni syndrome
- Neurofibromatosis type 1
- Severe congenital neutropenia (also called Kostmann syndrome)

Chromosomes are long strands of DNA (genes) inside our cells. Some chromosome problems present at birth are also linked to a higher risk of AML, including:

- Down syndrome (being born with an extra copy of chromosome 21)
- Trisomy 8 (being born with an extra copy of chromosome 8)

Family History
Although most cases of AML are not thought to have a strong genetic link, having a close relative (such as a parent, brother, or sister) with AML increases your risk of getting the disease. Someone who has an identical twin who got AML before they were a year old has a very high risk of also getting AML.

Chronic Lymphocytic Leukemia (CLL)

Age
The risk of CLL goes up as you get older. About 9 out of 10 people with CLL are over age 50.

Certain chemical exposures
Some studies have linked exposure to Agent Orange, an herbicide used during the Vietnam War, to an increased risk of CLL. Some other studies have suggested that farming and long-term exposure to certain pesticides may be linked to an increased risk of CLL, but more research is needed to be sure. Radon exposure at home has been linked to an increased risk.

Family history
First-degree relatives (parents, siblings, or children) of people with CLL have more than twice the risk for this cancer.

Gender
CLL is slightly more common in males than females. The reasons for this are not known.

Race/ethnicity
CLL is more common in North America and Europe than in Asia. Asian people who live in the United States do not have a higher risk than those living in Asia. This is why experts think the differences in risk are related to genetics rather than environmental factors.

Chronic Myeloid Leukemia (CML)
The only risk factors for chronic myeloid leukemia (CML) are:

- **Radiation exposure**: Being exposed to high-dose radiation (such as being a survivor of an atomic bomb blast or nuclear reactor accident) increases the risk of getting CML
- **Age**: The risk of getting CML goes up with age
- **Gender**: This disease is slightly more common in males than females, but it's not known why

There are no other proven risk factors for CML. The risk of getting CML does not seem to be affected by smoking, diet, exposure to chemicals, or infections. And CML does not run in families.

Appendix D: PFAS Fact Sheet

PFAS Chemical Awareness

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Per-and polyfluorinated substances (PFAS) are a man-made family of chemicals, with PFOS (perfluorooctane sulfonic acid) and PFOA (perfluorooctanoic acid) being the most studied and understood.

What products contain PFAS?

PFAS have been manufactured and used worldwide since the 1940s and are in many products such as:

- **Fire-fighting foam**
- **Nonstick cookware**
- **Stain-resistant carpets**

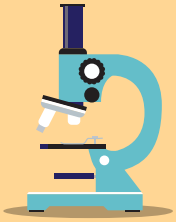
- **Paints and stains**
- **Water-resistant fabrics**
- **Food packaging**

Do PFAS cause cancer?

According to the U.S. EPA (Environmental Protection Agency), there is limited evidence that PFAS (PFOS and PFOA) cause cancer in humans.

The International Agency for Research on Cancer (IARC) has classified PFOA as possibly cancer causing.

Correlations between exposure to PFAS and human health effects have been inconsistent.

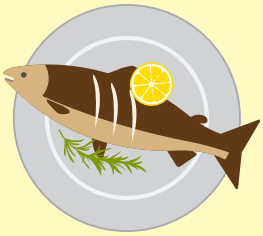


Where can PFAS be found?

PFAS can be found in the environment (air, water, soil) as well as produce products such as vegetables and fruits. PFAS can last a long time in the environment and may be carried over a great distance.

What are the sources of PFAS exposure?

People are most likely exposed by consuming PFAS-contaminated water or food. Exposure may also occur by using products that contain PFAS.



Inhalation and skin exposure are minor exposure pathways. Exposure through skin contact is slow and minor compared to other exposure routes.

Should I get my blood tested for PFAS?

It is not clear how PFAS in blood impacts human health. Having PFAS in your blood does not necessarily mean that you will become ill from PFAS.

A blood test will not provide information for treatment or identify how or where the PFAS exposure occurred. Any decision on testing or treatment should be discussed with your healthcare provider.

PFAS in People

CDC (Centers for Disease Control and Prevention) monitoring estimates that most people in the U.S. will have measurable amounts of PFAS in their blood.

Some PFAS stay in the body for a long time. There is no recommended medical treatment to reduce PFAS in the body.

How can PFAS affect my health?

Effects on health from exposure to low environmental levels of PFAS, such as PFOS and PFOA, are not well known.

Studies in humans and animals are inconclusive.

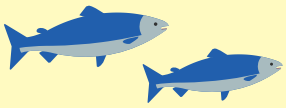
Findings are limited that exposure leads to increased risk of certain cancers such as prostate, kidney, or testicular cancer.

Non-cancer effects include increased cholesterol levels, impacts on human hormones and the immune system, and fetal and infant developmental effects.

How can I reduce my exposure to PFAS?

You can take the following steps to reduce your risk of exposure:

- Check for fish advisories for water bodies where you fish.
- Follow fish advisories that tell people to stop or limit eating fish from waters contaminated with PFAS or other compounds.
- Read consumer product labels and avoid using products with PFAS.



If your drinking water contains PFAS above the EPA Lifetime Health Advisory, consider using an alternative or treated water source for any activity in which you might swallow water:

- Drinking
- Preparing food
- Cooking
- Brushing teeth
- Preparing infant formula

What are the safe levels of PFAS in Florida's drinking water that do not cause a risk?

The Health Advisory Levels (HALs) for PFOS and PFOA is a combined maximum of **0.07 micrograms per liter** (0.07 µg/L) for both.

Contact

If you have questions, please contact the Hazardous Waste Site Health Assessment Team at:

phtoxicology@flhealth.gov

Or call toll free at:

877-798-2772

Learn More

Use the QR code to visit the Florida Department of Health's Hazardous Waste Site Risk Assessment webpage.

Additional information can be found online at:

ATSDR.CDC.gov/pfas

EPA.gov/pfas

Appendix E: Environmental Health Consultation Letter

Health Consultation Letter

Date: March 29, 2019

From: Kendra F. Goff, PhD, DABT, CPM, CEHP, State Toxicologist & Chief
Bureau for Environmental Health

Requested by: Brevard County Health Department

PURPOSE:
The Florida Department of Health (DOH) Public Health Toxicology Section received a request from the Brevard County Health Department (CHD) to evaluate groundwater data that were collected as part of a 2018 area-wide groundwater surveillance effort in Brevard County. The surveillance effort was conducted as a response to community concerns about the presence of per- and polyfluoroalkyl substances (PFAS) on local military bases (Patrick Air Force Base and Cape Canaveral Air Force Station). The request focuses on PFAS measured in irrigation water, surface water, reclaimed water and public drinking water systems as wells as its potential health threat.

The DOH Public Health Toxicology Section is committed to ensuring that people in and/or near contaminated sites have the best information available to understand the chemicals and the health risks.

METHOD:

Environmental Data Collection

Groundwater was sampled at 28 locations throughout the mainland and Barrier Islands in communities serviced by central sewer and communities including Titusville, Suntree, Palm Bay, South Patrick Shores and Satellite Beach (Figure 1). Out of the 28 locations, four shallow irrigation wells and three deep (Floridan Aquifer) irrigation wells were sampled in the Patrick Shores community, and one shallow irrigation well and three deep (Floridan Aquifer) irrigation wells were sampled in the Snug Harbor community. Four irrigation samples were collected in the Barrier Island community (Figure 1).

Surface Water samples were collected at 13 locations within the Grand Canal system, the Banana River (BR) Lagoon, and the North and Central Indian River Lagoon (IRL).

Data from the groundwater and surface water sampling effort were reviewed in various sources (Applied Ecology, Inc. 2018; U.S. EPA 2015a, b; SGS North America Inc, 2018 a, b; Universal Engineering Sciences, 2018; Mead&Hunt, 2018; FDEP, 2018; Test America, 2018) to evaluate possible health concerns when consuming and using water containing PFAS. The waters investigated included irrigation water, surface water, reclaimed water, public system water, school drinking water, private well drinking water, waste water and groundwater monitoring water (Table 1 to Table 6, Table A1 and A2).

Risk Evaluation

Human health risk assessors and public health toxicologists from DOH evaluated environmental data to assess a possible public health threat using a stepwise approach by first **screening** the data against current drinking water, irrigation water and/or surface water standards/levels (Table 1 through Table 6), and second by determining possible PFAS **exposure** routes following the most current human health assessment guidelines from the Agency for Toxic Substances and Disease Registry (ATSDR) (Table 7).

During the screening process DOH determines the contaminated media and the contamination levels by screening the site-related data against comparison values (CVs). Each CV is a concentration for a chemical in the environment (i.e. water or soil) below which DOH does not expect harm to the public health. DOH identifies contaminants higher than their CVs or those that are considered carcinogenic for further evaluation.

Once the first step of screening has been conducted, DOH studies ways people could be exposed to contaminated media, called exposure pathways. Chemical contamination in the environment can harm your health but only if you have contact with those contaminants (exposure). Without contact or exposure, there is no harm to health. If there is contact or exposure, how much of the contaminants you contact (concentration), how often you contact them (frequency), for how long you contact them (duration), and the hazard level of the contaminant (toxicity) determine the risk of harm. Exposures occur if a contamination source has all the following:

- an environmental medium to hold or transport it; like air, soil, or water
- an exposure point where people contact it
- an exposure route through which it enters the body
- an exposed population who contact it

The identification of an exposure pathway does not necessarily mean that harm to health will occur.

DOH health assessors also collaborated with colleagues both within and outside the Department to develop a strategy for sample collection and data evaluation. The collaborative effort and open communication provided quality information that was representative of actual conditions in Brevard County**.** The gathered information was compared, analyzed, and discussed using current knowledge of PFAS toxicity and mixture effects.

RESULTS AND FINDINGS:

Environmental Data Screening

The most common and well-studied PFAS are perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS). The U.S. Environmental Protection Agency (EPA) has developed a lifetime drinking water Health Advisory (HA) for PFOA+PFOS of 0.07 micrograms per liter (µg/L) [0.07 µg/L = 70ppt = 70 nanograms per liter (ng/L)]. This is the equivalent of a shot glass (1.5 oz) in approximately 150 million gallons of water. The EPA lifetime HA assumes that a person's daily water consumption could occur at or below the level for a lifetime and be unlikely to cause a health effect. The Agency for Toxic Substances and Disease Registry (ATSDR) has developed an intermediate drinking environmental media evaluation guide level (EMEG) of 0.021 µg/L (child) and 0.078 µg/L (adult) for PFOA, and of 0.014 µg/L (child) and 0.052 µg/L for PFOS. Irrigation and surface water screening values are currently being discussed for use in Florida.

We compared the concentrations in the irrigation wells to provisional screening values that the Florida Department of Environmental Protection (DEP) has proposed (Table 1). The levels are 6.7 µg/l for PFOA and 72.0 µg/L for PFOS. All measured concentrations were below these screening values (Table 1). Because PFOA is classified by the international agency for research (IARC) on cancer as possibly carcinogenic to humans, irrigation water exposure it was further evaluated (Table 7).

No current surface water screening values exist for Florida. Until a value is developed in Florida, DOH chooses to use a range of comparison values that other states have developed to look at potential surface water concerns (ITRC, 2018). For PFOA the screening value range is 0.42 - 24 µg/l and for PFOS the screening value range is 0.011 - 300 µg/l. The PFOA+PFOS range is 0.43-324 µg/l (ITRC, 2018) (Table 2). All measured PFOA+PFOS concentrations were below these comparison values Because PFOA is classified by the IARC as possibly carcinogenic to humans, surface water exposure was further evaluated (Table 7).

The reclaimed water results provided by the City of Cocoa Beach were compared with both, the provisional irrigation screening values that the DEP has proposed; 6.7 µg/l for PFOA and 72.0 µg/L for PFOS, and a range of surface water comparison values that other states have developed to look at potential surface water concerns (ITRC, 2018) (Table 3). All measured concentrations were below the provisional irrigation water screening values. The measured PFOA+PFOS concentration was below the surface water comparison value other states have developed (Table 3). Because PFOA is classified by the IARC as possibly carcinogenic to humans, reclaimed water exposure was further evaluated (Table 7).

The public drinking water in Satellite Beach is provided from the City of Melbourne and the drinking water in Cocoa Beach is provided by the City of Cocoa. PFAS+PFOS concentrations in water supplies from the City of Cocoa and City of Melbourne (Table 4), as well as from Brevard County schools (Table 5) and a private drinking water well (Table 6) were either non-detected and/or below the EPA lifetime HA of 0.07 µg/l (Applied Ecology, Inc, 2018; U.S. EPA 2015a,b).

The waste water results were compared with the provisional irrigation screening values that the DEP has proposed; 6.7 µg/l for PFOA and 72.0 µg/L for PFOS, a range of surface water comparison values that other states have developed to look at potential surface water concerns (ITRC, 2018), and EPA lifetime HA of 0.07 µg/l (Table A1). All measured PFOA+PFOS concentrations were below provisional irrigation water screening and other state surface water values.

Groundwater monitoring data were compared with the provisional irrigation screening values that the DEP has proposed; 6.7 µg/l for PFOA and 72.0 µg/L for PFOS, a range of surface water comparison values that other states have developed to look at potential surface water concerns (ITRC, 2018), and EPA lifetime HA of 0.07 µg/l (Table A2). All measured PFOA+PFOS concentrations were below provisional irrigation water screening and other state surface water values.

Exposure Analysis

As result of the screening analysis, DOH health risk assessors continued to evaluate possible exposure pathways for residents, visitors and recreational receptors exposed to irrigation water, surface water, and/or reclaimed water (Table 1 through 3, Table 6).

Reclaimed water is treated waste water often used as replacement of potable water for irrigation and industrial needs, and should not be used as replacement for drinking water. Since irrigation water and reclaimed water are not the direct or main source of daily water consumption but rather a small source through the sprinkling of edible foods, the probability of risk from this exposure is minimal. We do not have data for home-grown vegetables that may be watered with irrigation water but would consider evaluation once that becomes available. Other than direct ingestion of irrigation water, DOH also considered inhalation and dermal contact exposure routes, however, these pathways were eliminated for further investigations. Dermal exposure is limited as PFOA and PFOS are not absorbed effectively through the skin. Inhalation exposure routes are also very limited since the substances do not vaporize as they are non-volatile.

The risk for health effects when being exposed to PFAS contaminated surface water is minimal for direct ingestion since people don’t use the tested surface water for drinking water purposes. The risk for health effects from incidental ingestion through recreational activities is not expected to cause harm. Exposure risk of surface water from inhalation or dermal contact is limited due to the nature of the substances and limited inhalation and absorption.

The public drinking water is provided for most citizens in zip code 32937 and 32940 from either the City of Melbourne or the City of Cocoa. The public water in these two cities did not show any detection and/or very low detection of PFAS. Therefore, drinking public water is not expected to cause any harm to the human health.

One private drinking water well was found in Brevard county. Data from the private drinking water show very low detection of PFAS. PFOA+PFOS concentration was below the EPA lifetime drinking water HA of 0.07 µg/. Therefore, drinking from the sampled private well is not expected to cause any current or future harm to the human health.

The reclaimed water was below the provisional irrigation water screening and other state surface water values. Reclaimed water (water reuse) is not considered drinking water and should not be used for drinking or food preparation. Water reuse involves taking domestic wastewater, giving it a high degree of treatment, and using the resulting high-quality reclaimed water for a new, beneficial purpose (like irrigation). Therefore, wastewater is not considered a source of daily water drinking, irrigation or recreational uses and is not expected to cause any harm to the human health.

Waste water and data from groundwater monitoring wells was below the provisional irrigation water screening and other state surface water values. Waste water is not considered usable water and should not be used for drinking, irrigation or other uses. Groundwater monitoring wells are used for observation, quality checks, progress measure over time, and regular surveillance and are not used for daily water uses. Therefore, the water collected at waste water sampling locations and monitoring wells should not cause any harm to the human health.

Conclusions:

DOH concludes that presently there is minimal potential to harm human health via consuming drinking water from water supplied to the Brevard County area as PFAS is below the EPA lifetime HA. In addition, PFAS-contaminated irrigation, surface and reclaimed water in the Brevard County Area is below screening values and will have minimal impact if swallowed. Inhalation and dermal (skin) exposure are minor exposure pathways. PFAS tend to stay in water once they have dissolved. The uptake of PFAS through dermal contact is slow and not considered significant.

While garden fruits and vegetables, as well as fish and seafood consumption concerns were considered, DOH currently has no data to evaluate but would consider evaluation once that becomes available.

If you have any questions or comments concerning this letter, please contact Dr. Gladys A. Liehr, in the Health Risk Assessment Program at 877-798-2772.

Sincerely,

Kendra F. Goff, PhD, DABT, CPM, CEHP
State Toxicologist & Chief

KFG/gal
Enclosures

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Letter Preparation

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- Carina Blackmore, DVM, PhD, Division Director, Disease Control and Health Protection

Table 1: Irrigation Well PFAS results for Brevard County.

		Compound	PFOA	PFOS	PFOA+PFOS
Irrigation Water Lab Results (µg/L)	**Satellite Beach- South Patrick Shores Subdivision**	**PS-1**	0.0198	0.049	0.0688
		PS-2	0.00783 I	0.0371	0.04493 I
		PS-4	0.0348	0.0656	0.1004
		PS-6	0.00845 I	0.0359	0.04435 I
	Cocoa Beach- Sung Harbor Subdivision	**SH-4**	0.0156 I	0.0209	0.0365 I
	Cocoa Beach – various areas	**Private Well**	ND	ND	ND
		Artesian Well	ND	ND	ND
		Lori Wilson Dog Park	0.0213	0.102	0.1233
FL DEP provisional irrigation screening level (µg/L)			**6.7**	**72.0**	NA

[Source: Applied Ecology, Inc. (2018), Mead&Hunt (2018)]

FL DEP – Florida Department of Environmental Protection
I – qualified value indicates the analytical concentration is greater than or equal to the method detection limit but less than the practical quantitation limit
ND – not detected
µg/L – microgram per liter
PFOA – perfluorooctanoic acid
PFOS – perfluorooctanesulfonic acid
U – qualified value indicates the analytical concentration is below laboratory minimum detection limits (MDLs); vary depending on parameter and sample

Table 2: Surface Water PFAS results for Brevard County.

		Compound	PFOA	PFOS	PFOA+PFOS
Surface Water Lab Results (µg/L)	Banana River	BR-1	0.00777 I	0.0276	0.03537 I
		BR-2	0.00843 I	0.0275	0.03593 I
		BR-3	0.0119 I	0.115	0.1269 I
	Indian River Lagoon	IRL-1	0.00545 I	0.0978	0.10325 I
		IRL-2	0.00796 I	0.0987	0.10666 I
		IRL-3	0.0042 U	0.00496 I	0.00916 U I
		IRL-4	0.00614 I	0.0261	0.03224 I
	Grand Canal	GC-1	0.0053 I	0.0142 I	0.0195 I
		GC-2	0.00676 I	0.0157 I	0.02246 I
		GC-3	0.00586 I	0.0117 I	0.01756 I
		GC-4	0.00606 I	0.0156	0.02166 I
		GC-5	0.00542 I	0.0128 I	0.01822 I
Surface Water screening values µg/l (ITRC 2018)			**0.42 - 24**	**0.011 - 300**	**0.43-324**

[Source: Applied Ecology, Inc. (2018)]

I – qualified value indicates the analytical concentration is greater than or equal to the method detection limit but less than the practical quantitation limit
NA – not available
µg/L – microgram per liter
PFOA – perfluorooctanoic acid
PFOS – perfluorooctanesulfonic acid
U – qualified value indicates the analytical concentration is below laboratory minimum detection limits (MDLs); vary depending on parameter and sample

Table 3: Reclaimed water PFAS result for Brevard County, City of Cocoa Beach

			Date	PFOA	PFOS	PFOA+PFOS
Reclamation Water Lab Results (µg/L)	**Plant Effluent**	**EFF-1**	06/2018 (G)	0.0234	0.261	0.2844
			08/2018 (G)	0.0207	0.0835	0.1042
			08/2018 (C)	NS	NS	NS
			08/2018 (C)	0.018	0.067	0.085
	South Reuse Distribution		08/2018	NS	NS	NS
			08/2018	0.0228	0.092	0.1148
	North Reuse Distribution		08/2018	NS	NS	NS
			08/2018	0.0226	ND	0.0226
	Golf Course Outfall (reuse water runoff)		08/2018	NS	NS	NS
			08/2018	0.0309	0.13	0.1609
FL DEP provisional irrigation screening level (µg/L)				**6.7**	**72.0**	NA
Surface Water screening values µg/l (ITRC 2018)				**0.42 - 24**	**0.011 - 300**	**0.43-324**

[Source: SGS North America Inc. (2018b), Mead&Hunt (2018)]

C – composite
FL DEP – Florida Department of Environmental Protection
G – grab
ND – not detected
NS – not sampled
µg/L – microgram per liter
PFOA – perfluorooctanoic acid
PFOS – perfluorooctanesulfonic acid

Table 4: Public water system PFAS results in Satellite Beach.

	Public Water System	Facility	Type	PFOA	PFOS	PFOA+PFOS
Public Water System Lab Results (µg/L)	**City of Cocoa**	**Dyal WTP**	**surface water**	Below MRL		NA
	City of Melbourne	**Melbourne WTP**	**Mix of surface water, ground water, and ground water under the direct influence of surface water**			
Minimum Reporting Level defined by UCMR 3 (µg/L)				**0.02**	**0.04**	**NA**
U.S. EPA lifetime HA (µg/L)				**NA**	**NA**	**0.07**

[Source: U.S. EPA (2015a,b)]

U.S. EPA – U.S. Environmental Protection Agency
MRL – Minimum Reporting Level defined by UCMR 3
NA – not available
µg/L – microgram per liter
PFOA – perfluorooctanoic acid
PFOS – perfluorooctanesulfonic acid
UCMR – Unregulated Contaminant Monitoring Rule (U.S. EPA)
WTP – Water Treatment Plant

Table 5: Drinking water PFAS results for Brevard County Schools.

			Compound	PFOA	PFOS	PFOA+PFOS
School Drinking Water Lab Results (µg/L)	Gemini Elementary	Room 804	GE-1	ND		
	Indialantic Elementary	Room 10-056	IE-3			
	Hoover Middle School	Hallway	HM-1			
	Ocean Breeze Elementary	Hallway	OB-4			
	Surfside Elementary	Room 5-005	SE-1			
	Cape View Elementary	Room205	CV-1			
	Roosevelt Elementary	Hallway	RE-1			
	Cocoa Beach JR/SR High	Hallway	CB-1			
	Freedom 7 Elementary	Room 604	F7-2			
	Sea Park Elementary	Room 322	SP-1			
	Holland Elementary	Hallway	HE-1			
	DeLaura Middle	Room 103	DM-1			
	Satellite High	Hallway	SH-1			
U.S. EPA lifetime HA (µg/L)						**0.07**

[Source: SGS North America Inc. (2018a), Universal Engineering Sciences (2018)]

U.S. EPA – U.S. Environmental Protection Agency
ND – not detected
µg/L – microgram per liter
PFOA – perfluorooctanoic acid
PFOS – perfluorooctanesulfonic acid

Table 6: Drinking water PFAS results for Brevard County, Satellite Beach, Private Well.

		Compound	PFOA	PFOS	PFOA+PFOS
Potable Water Lab Results (µg/L)	**Satellite Beach**	**Sample**	0.00071	0.00045	0.00116
		Resampled	0.0042	0.042	0.0462
U.S. EPA lifetime HA (µg/L)			**NA**	**NA**	**0.07**

[Source: FDEP (2018), Test America (2018)]

U.S. EPA – U.S. Environmental Protection Agency
NA – not available
µg/L – microgram per liter
PFOA – perfluorooctanoic acid
PFOS – perfluorooctanesulfonic acid

Table 6: Overview of investigated exposure pathways, possible contamination source, effected media, exposure routes and population that might be at risk.

Pathway	Source	Media	Point of Exposure	Exposure Route	Receptor
Completed	None				
Potential	PFAS in groundwater	Irrigation water	Irrigation water taps+, outdoor air, edible plants/fruits/vegetables	ingestion, inhalation, and dermal contact	past, current and future recreational receptor
		Surface water	Recreational bathing, boating, and fishing	ingestion, inhalation, and dermal contact	past, current and future recreational receptor
	Reclaimed water	Irrigation water	Irrigation water taps+ edible plants/fruits/vegetables, outdoor air	ingestion, inhalation, and dermal contact	past, current and future recreational receptor
		Industrial water	Irrigation water taps+ edible plants/fruits/vegetables, cooling water taps+, toilets flushing, dust control, restoration of surface water	ingestion, inhalation, and dermal contact	past, current and future workers, visitors, consumers
	PFAS in groundwater	Private potable well	Drinking and irrigation water taps+, shower, indoor and outdoor air, edible plants/fruits/vegetables	ingestion, inhalation, and dermal contact	past resident and visitors
Eliminated	PFAS in public drinking water	Public drinking water system	Drinking water taps, shower, indoor air for connected structure	ingestion, inhalation, and dermal contact	past, current and future residents and visitors
	PFAS in public drinking water supplies at schools	Water fountain public drinking water	Drinking water taps, shower, indoor air for connected structure	ingestion, inhalation, and dermal contact	past, current and future students, workers and visitors
	PFAS in groundwater	Private potable well	Drinking and irrigation water taps+, shower, indoor and outdoor air, edible plants/fruits/vegetables	ingestion, inhalation, and dermal contact	current and future resident and visitors
	PFAS in Wastewater++	Wastewater	Drinking and irrigation water taps+, shower, indoor and outdoor air, edible plants/fruits/vegetables	ingestion, inhalation, and dermal contact	current and future resident and visitors

+ Irrigation water and reclaimed water are not considered drinking water and should not be used for drinking or food preparation.
++ Waste water are not considered usable water and should not be used for drinking, irrigation or other uses.

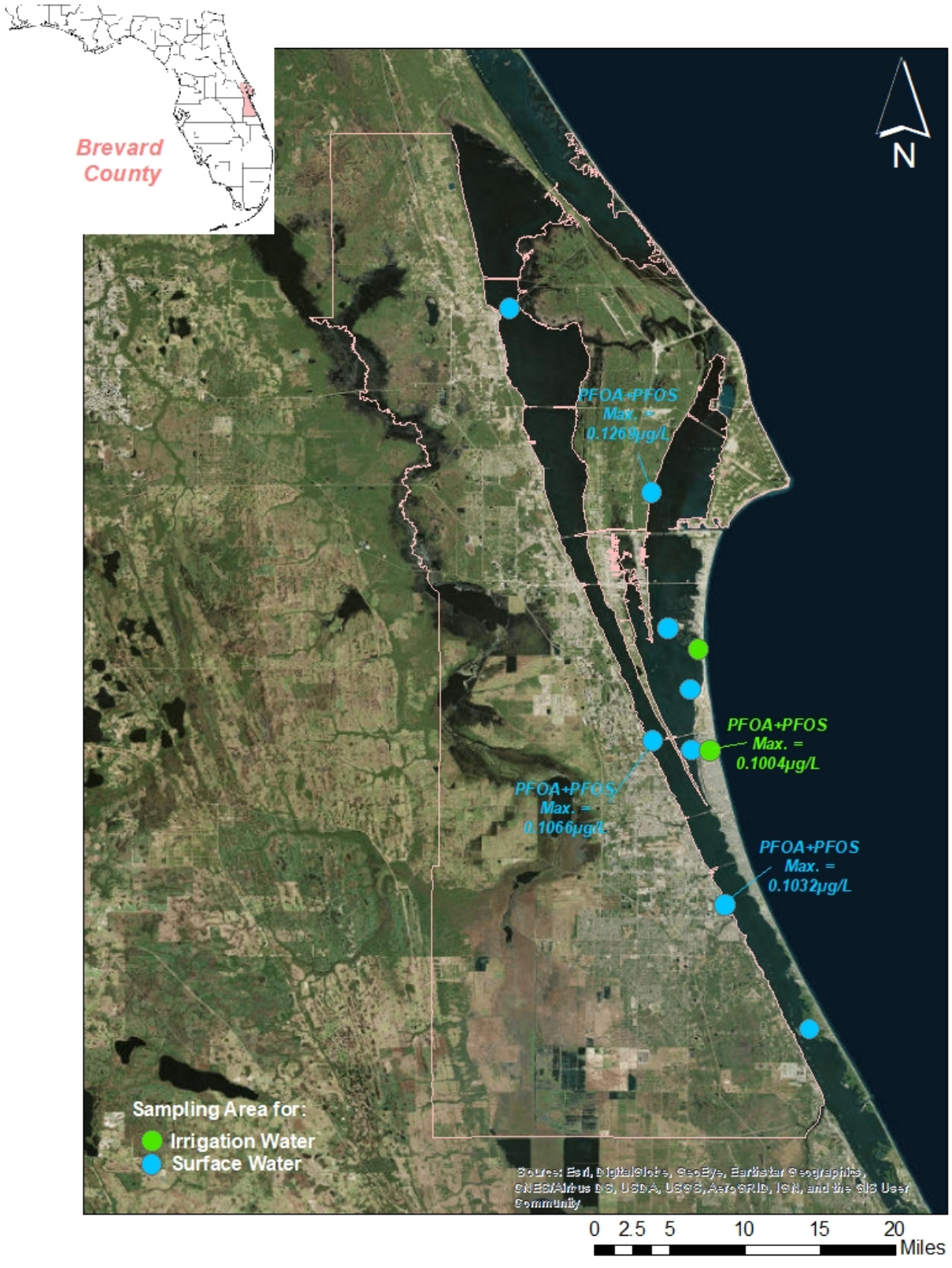

Figure 1: Irrigation water and surface water PFAS (PFOA+PFOS) sampling areas. Only areas that exceeded the EPA lifetime drinking water Health Advisory of 0.07 µg/L (PFOA+PFOS) are labeled with sample results.

APPENDIX A – ADDITIONAL TABLES

Table A1: Wastewater PFAS result for Brevard County, City of Cocoa Beach

			Date	PFOA	PFOS	PFOA+PFOS
Wastewater Lab Results (µg/L)	**AFB Sanitary Discharge**	**PAFB**	06/2018	0.0191	0.411	0.4301
			08/2018	0.016	0.278	0.294
	Plant Influent	**INF-2**	06/2018 (G)	0.0122	0.165	0.1772
			08/2018 (G)	0.0182	0.0936	0.1118
			06/2018 (C)	NS	NS	NS
			08/2018 (C)	0.0132	0.0896	0.1028
	Lift Stations	**#3**	06/2018	NS	NS	NS
			08/2018	0.0093	0.0313	0.0406
		#5	06/2018	NS	NS	NS
			08/2018	0.0124	0.0472	0.0596
		#10	06/2018	NS	NS	NS
			08/2018	0.0079	0.029	0.0369
		#17	06/2018	NS	NS	NS
			08/2018	0.0042	0.0192	0.0234
		Port Main Station	06/2018	NS	NS	NS
			08/2018	0.0112	0.0097	0.0208
FL DEP provisional irrigation screening level (µg/L)				**6.7**	**72.0**	NA
Surface Water screening values µg/l (ITRC 2018)				**0.42 - 24**	**0.011 - 300**	**0.43-324**

[Source: SGS North America Inc. (2018b), Mead&Hunt (2018)]

C – composite
FL DEP – Florida Department of Environmental Protection
G – grab
NS – not sampled
µg/L – microgram per liter
PFOA – perfluorooctanoic acid
PFOS – perfluorooctanesulfonic acid

Table A2: Groundwater Monitoring PFAS result for Brevard County, City of Cocoa Beach

			Date	PFOA	PFOS	PFOA+PFOS
Groundwater Monitoring Lab Results (µg/L)	**Golf course**	**MW-2**	06/2018	NS	NS	NS
			08/2018	0.0343	0.191	0.2253
		MW-3	06/2018	0.0313	0.217	0.2483
			08/2018	0.0304	0.188	0.2184
		MW-4	06/2018	0.0286	0.101	0.1296
			08/2018	0.0249	0.0628	0.0877
FL DEP provisional irrigation screening level (µg/L)				**6.7**	**72.0**	NA
Surface Water screening values µg/l (ITRC 2018)				**0.42 - 24**	**0.011 - 300**	**0.43-324**

[Source: SGS North America Inc. (2018b), Mead&Hunt (2018)]

FL DEP – Florida Department of Environmental Protection
NS – not sampled
µg/L – microgram per liter
PFOA – perfluorooctanoic acid
PFOS – perfluorooctanesulfonic acid

-FREQUENTLY ASKED QUESTIONS-

Household Water Sources

What is drinking water?

Drinking (or potable) water is water safe to drink or use for food preparation. Potable water is available either from a municipal utility company or from a private well on your property.

- If you pay a bill for water, you are served by a public utility that must meet the testing schedule and requirements under the U.S. Safe Drinking Water Act (https://www.epa.gov/sdwa). The water well that serves the utility that provides your potable water is often several miles away from your home.
- For private wells, water testing and upkeep of the water well is the responsibility of the owner. The Department of Health does have a program that can help a private owner determine whether their potable water is at risk. (http://www.floridahealth.gov/environmental-health/private-well-testing/index.html).

What is reuse water?

Reuse (or reclaimed) water is not considered drinking water and should not be used for drinking or food preparation. Water reuse involves taking domestic wastewater, giving it a high degree of treatment, and using the resulting high-quality reclaimed water for a new, beneficial purpose (like irrigation). The water reuse program is regulated by the Florida Department of Environmental Protection (https://floridadep.gov/water/domestic-wastewater/content/water-reuse-program).

What is irrigation water?

Irrigation water is not considered drinking water and should not be used for drinking or food preparation. Irrigation wells are installed by a well contractor to be a certain distance from a septic system (if it exists) and are constructed to protect the water below. There are no water quality testing requirements. Most irrigation wells in this area are over 100 feet deep and thus are somewhat protected from surface pollution.

What is monitoring well water?

Water from monitoring wells is not considered potable water and should not be used for drinking or food preparation. Monitoring wells are used to sample for water quality.

Per- and polyfluoroalkyl substances (PFAS)

General Facts

What are Per- and polyfluoroalkyl substances (PFAS)?

PFAS are man-made chemicals that do not occur naturally in the environment. They may be used in surface protection of non-stick cookware, stain resistant carpets and fabrics, waterproof mattress and clothes, and to make some food packaging resistant to grease absorption (such as microwave popcorn bags). PFAS are also used in some firefighting materials. Other industrial uses include photo imaging, metal plating, printers and copy machines.

The term PFAS encompass a wide universe of substances with very different physical and chemical properties.

What are the most common Per- and polyfluoroalkyl substances (PFAS)?

The most common and well-studied PFAS are perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS).

Why are PFAS a concern?

PFAS are widespread and global. Once released, they are very persistent in the environment and the human body. They can be found in air, soil, and water (ground and surface water). PFAS also can be found in blood, urine, breast milk and umbilical cord blood of humans.
The elimination of PFAS from the environment is slow.

What are the main sources of PFAS exposure?

Ingestion is the main source of exposure. You can be exposed to PFAS by:

- drinking contaminated water
- eating fish caught from waters contaminated with PFAS
- eating food packed in PFAS-containing material (e.g., popcorn bags)
- hand-to-mouth transfer from surfaces treated with PFAS, such as carpets. This is thought to be most significant for infants and toddlers.

What are other sources of PFAS exposure?

Inhalation and dermal (skin) exposure are minor exposure pathways. PFAS tend to stay in water once they have dissolved. The uptake of PFAS through dermal contact is slow and not considered significant.

How can PFAS potentially affect health?

Effects on health from exposure to low environmental levels of PFOS and PFOA are not well known. Studies in humans and animals are inconclusive but suggest that certain PFAS may cause health effects. Some studies have suggested an increased risk of certain cancers, such as prostate, kidney, or testicular cancer. However, non-cancer effects appear more common and include increased cholesterol levels, and impacts on human hormones and immune system, as well as fetal and infant developmental effects.

Correlations between exposure to PFAS and health effects have been inconsistent. More research is needed to understand health effects in humans.

Are health effects caused by PFAS in animals the same as in humans?
Humans and animals often react differently to chemicals, including PFAS and not all the effects observed in animal tests may occur in humans.

How long do PFAS remain in the body?
The duration of PFAS remaining in the body dependents on the physical and chemical properties of the individual PFAS compound. On average, PFAS can remain in the body between 2 and 9 years.

Do PFAS cause cancer?
The U.S. Environmental Protection Agency (EPA) has determined there is some evidence that PFOA and PFOS can cause cancer. The International Agency for Research on Cancer (IARC) has classified PFOA as possibly cancer causing, although, there is currently no consistent scientific evidence that PFOS and PFOA cause cancer in humans.

Some increases in kidney, prostate and testicular cancers have been seen in individuals exposed to higher PFAS levels, mostly in occupational exposures. Most of these exposures were in people who worked in, or lived near, PFAS manufacturing facilities.

How certain are the studies that showed health risks?
Correlations between exposure to PFAS and health effects have been inconsistent. More research is needed to fully understand any health effects in humans. Animals (mostly rats and mice) exposed to much higher levels than most people showed several health problems, such as liver damage, developmental and reproductive effects, and changes in hormone levels. Some human studies have found increases in prostate, kidney, and testicular cancers in workers exposed to PFAS and people living near facilities producing PFAS. However, other studies did not report a link between cancer and PFAS. These studies should be interpreted carefully, since the effects were not consistent across studies, there were contradictory findings among studies, and exposure levels were much higher than seen in the general population.

PFAS Regulation and Advisories

What is the current Health Advisory Level (HAL) for PFAS in drinking water?
The U.S. Environmental Protection Agency (EPA) has developed a lifetime drinking water HAL for PFOA+PFOS of 0.07 micrograms per liter (µg/L) [0.07 µg/L = 70ppt]. This is the equivalent of a shot glass (1.5 oz) in approximately 150 million gallons of water. Drinking water at or below this standard for a lifetime is not expected to cause any harm to your health.

If the drinking water is above the HAL, what should I do?
If the drinking water contains PFOA+PFOS above the EPA health advisory level, alternative water sources for drinking, food preparation, cooking, brushing teeth and other activities may be preferable. Because the HAL is based upon long term exposure, a short-term increase above the HAL should not increase risk.

If the drinking water is above the HAL, should my pets drink it?
Pets should be given drinking water. As with humans, if the drinking water contains PFOA+PFOS above the EPA HAL, alternative water sources for drinking, food preparation, brushing teeth and other activities may be preferable. Because the HAL is based upon long term exposure, a short-term increase above the HAL should not increase risk.

Should I use irrigation water with PFAS for watering the lawn?
Irrigation of a lawn with non-edible plants and grass poses little risk. PFOA and PFOS are not absorbed effectively through the skin, nor is inhalation of vapors from water with PFOA and PFOS likely to cause

health problems. Remember that irrigation water is not potable water. For this chemical, drinking is a main route of exposure.

Is it safe to use reuse water for irrigation of my home produce?

Reuse water should never be used for home-grown produce due to the concern for human microbial pathogens. Reuse water should not be used for drinking.

Is it ok to swim in my pool if it is contaminated with PFAS?

Skin contact and inhalation of PFAS is a minor concern due to the low dermal adsorption and inhalation potential. You should not drink swimming pool water. It is possible to drain and replace the pool with municipal utility water. However, if you take reasonable precautions to avoid drinking the pool water, the risk from swimming should be very low.

Biomonitoring and Blood Testing

Is there a test to determine whether a person has been exposed to PFAS?

PFAS can be measured in blood, serum, and urine. It is not a routine test used in doctor's offices to guide any diagnosis or treatment.

When is blood testing of PFAS useful and what can the results tell me?

Blood tests for PFAS can be useful when they are part of a scientific investigation or a health study. One such study is the National Health and Nutrition Examination Survey (NHANES). Blood tests can also be helpful when researching health effects from PFAS among persons who have been exposed to very high concentrations of the chemical, such as workers in industries where PFAS was used. It is possible to compare the PFAS results from individuals who have their blood tested with national averages established through these types of studies.

What can the results from blood testing for PFAS NOT tell me?

Most people in the US will have measurable amounts of PFAS in their blood. It is not clear how this impacts our health. Currently there is not an established PFAS screening blood level at which a health effect is known to occur nor is there a level that predicts future health problems.

The blood test will not:

- provide information to pinpoint whether PFAS caused a health problem nor will it provide information for treatment
- predict or rule-out the development of future health problems related to a PFAS exposure
- identify how or where the PFAS exposure occurred.

What is currently known about PFAS blood levels in U.S. population?

It has been reported in the National Report on Human Exposure to Environmental Chemicals Report that serum levels of PFAS appear to be higher in the U.S. than in some other countries.

For the average American the normal level of PFOA and PFOS is 2.1 and 6.3 micrograms per liter of blood, respectively. These levels have been shown to be higher if a person's drinking water source is contaminated with PFAS or if a person is exposed at a workplace that produces the PFAS product. More information can be found at https://www.atsdr.cdc.gov/pfc/docs/pfas_clinician_fact_sheet_508.pdf.

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