Health Consultation

OATMAN WATER COMPANY

OATMAN, MOHAVE COUNTY, ARIZONA

Prepared by Arizona Department of Health Services

NOVEMBER 14, 2016

Prepared under a Cooperative Agreement with the U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES Agency for Toxic Substances and Disease Registry Division of Community Health Investigations Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

A health consultation is a verbal or written response from ATSDR or ATSDR's Cooperative Agreement Partners to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR or ATSDR's Cooperative Agreement Partner which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

You May Contact ATSDR Toll Free at 1-800-CDC-INFO or Visit our Home Page at: http://www.atsdr.cdc.gov

HEALTH CONSULTATION

OATMAN WATER COMPANY OATMAN, MOHAVE COUNTY, ARIZONA

Prepared By:

Arizona Department of Health Services Office of Environmental Health Environmental Health Consultation Services Under a Cooperative Agreement with the Agency for Toxic Substances and Disease Registry

Summary						
INTRODUCTION	The top priority of the Arizona Department of Health Services (ADHS) is to safeguard the health of the Oatman, Arizona community.					
	This report was written in response to a request from the Arizona Department of Environmental Quality (ADEQ) to evaluate the per- and polyfluoroalkyl substances (PFAS) detected in the Oatman Water Company (OWC) public water system (PWS ID AZ0408001) in Oatman, Arizona. PFAS were found in samples collected in April and October 2014. According to the ADEQ Drinking Water Section, OWC has only one well in operation, and OWC believes that PFAS contamination may be from certain types of fire-fighting foam. ADHS evaluated the potential health risks associated with exposure to the reported PFAS levels in the OWC drinking water system. The samples were collected prior to the entry point to the distribution system (EPDS) which is after processing and storage in the water treatment plant, but prior to reaching the first customer. The samples represent delivered water since no post-storage treatment is applied. The following PFAS were found in the water supply: Perfluorooctanoic acid (PFOA), perfluorooctane sulfonic acid (PFOS), perfluoroheptanoic acid (PFHpA), and perfluorohexane sulfonic acid (PFHxS).					
	After review of the available information using a weight of evidence approach, ADHS reached the following conclusion:					
Conclusion	Consuming the drinking water at the reported PFAS concentrations, especially for infants and young children, combined with other sources of exposure (i.e. background exposures from sources besides water and the body burden from pre-existing exposures), and pre-existing risk factors could increase the risk for health effects.					
	Additionally, PFHpA and PFHxS compounds were found at elevated levels in the water, have reported health effects (albeit limited toxicity data), and PFHxS also has a long half-life in the body. However, no EPA health advisories currently exist for these compounds. Currently, ATSDR considers effects from all PFAS exposures to be additive, including PFHpA and PFHxS. Therefore, detection of PFHpA and PFHxS at this site adds additional risk to the PFAS exposure.					
RECOMMENDATIONS	 ADHS recommends that OWC implement a long-term remedy to reduce the PFAS water levels in the public drinking water supply. 					

	 ADHS recommends that health education information about PFAS be provided to community members and the health professionals serving the area.
	 ADHS recommends that pregnant and lactating women, caregivers preparing formula for bottle-fed infants, and women of child bearing age use alternate water until OWC reduces PFAS in the water.
NEXT STEPS	 ADHS will notify ADEQ of the findings of this report and work with ADEQ to evaluate the protectiveness of mitigation action plans.
	 ADHS will continue to review and evaluate data provided for this site upon request.
	 ADHS will attend public meetings to discuss community concerns. ADHS will make presentations, develop handouts, and provide additional assistance as necessary to notify Oatman community members of the findings of this health consultation.
For More Information	If you have concerns about your health, you should contact your health care provider. Please call ADHS at 602-364-3118 if you have questions about this report.

Purpose

On August 18, 2016, the Arizona Department of Health Services (ADHS) received a request from the Arizona Department of Environmental Quality (ADEQ) to evaluate whether exposure to per- and polyfluoroalkyl substances (PFAS) detected in the public water system owned by Oatman Water Company (OWC) may harm the health of residents consuming the water.

Background and Statement of Issue

According to the ADEQ Drinking Water Section, the OWC is a small, privately owned drinking water company that serves 536 persons, including full time residents and visitors. In 2014, under U.S. Environmental Protection Agency's (EPA) third unregulated contaminant monitoring rule (UCMR 3), OWC was required to monitor the PFAS levels. Results from the two sampling events showed that four of the six tested PFAS exceeded UCMR 3 minimum reporting levels¹. The PFAS compounds that exceeded UCMR 3 minimum reporting levels¹. The PFAS compounds that exceeded UCMR 3 minimum reporting levels were perfluorooctanoic acid (PFOA), perfluorooctane sulfonic acid (PFOS), perfluoroheptanoic acid (PFHpA), and perfluorohexane sulfonic acid (PFHxS).

PFAS are a class of chemicals that are not currently regulated in public drinking water systems. In May of 2016, the EPA published a Lifetime Health Advisory (LTHA) for PFOA and PFOS in drinking water, either individually or combined, of 0.07 parts per billion (ppb, or 70 parts per trillion (ppt)). This value is based on a lifetime exposure (70 years) and assumes a 20% relative source contribution of contaminant exposure from drinking water. The LTHA provides a margin of protection from adverse health effects for individuals throughout their lifetime, including the most sensitive populations (for example, developing fetuses and breastfed infants).

Under EPA's UCMR3, about 1% of approximately 5,000 public water systems monitored from 2013 to 2015 have combined PFOA and PFOS concentrations above 0.07 ppb (USEPA 2016b). PFOA and PFOS in the OWC water samples from April and October 2014 exceeded EPA's LTHA. On May 19th, 2016, consumers were provided the information about the levels of PFOS and PFOA in their drinking water by OWC. The notice also was posted at the post office (Appendix A). In August, the water company sent a notice to customers with their water bills. On August 18, 2016, ADEQ requested a public health evaluation from ADHS on the PFAS detected in OWC.

PFAS are a family of man-made chemicals that have been used in the production of commercial and consumer products because they are resistant to heat, oil, stains, grease and water. Some examples are nonstick cookware, stain-resistant carpets, fabric coatings, some food packings, makeup and personal care products. PFAS can also be found in many industrial applications such as aqueous film forming foam (AFFF) fire-fighting agent for flammable liquids, floor care and cleaning products (ATSDR 2015; EPA2016c).

¹ EPA's UCMR 3 minimum reporting levels are found on their website: <u>https://www.epa.gov/dwucmr/third-unregulated-contaminant-monitoring-rule</u>

PFAS are very persistent in the environment and have the potential to bioaccumulate and biomagnify in wildlife. Studies show that nearly all people have some PFAS in their blood, regardless of age (Wu et al. 2015; Kato et al. 2011; CDC 2015). People are most likely exposed to PFAS by consuming contaminated drinking water and food, and by using consumer products containing PFAS (Fromme et al. 2009). PFAS are readily absorbed by the body after ingestion. It takes the human body about 5.4 years, 3.8 years, and 8.5 years for PFOS, PFOA and PFHxS, respectively, to lower the levels in the blood to half of the original concentrations if no more is taken into the body (Bartell et al. 2010; ATSDR 2015; USEPA 2014). The largest manufacturer of PFOS voluntarily stopped producing it in 2002. However, other countries still produce PFOS, and it can be imported into the United States in limited quantities. In 2006, EPA and major companies in the PFAS industry launched the 2010/2015 PFOA Stewardship Program. Companies participating in the program are working to stop producing PFOA and related chemicals by 2015.

Health Assessment Methods and Data

General Assessment Methodology

ADHS conducted a three step process to evaluate the public health implications of the PFAS contamination in drinking water supplies in this community. ADHS evaluated current/ongoing and past PFAS drinking water exposures. First, ADHS conducted an exposure pathway analysis to identify how people may be exposed. Second, ADHS conducted a screening analysis by comparing the water sampling data to the EPA's LTHA. Third, ADHS conducted a more detailed public health evaluation of contaminants of concern identified in the screening analysis (ATSDR 2005).

Available Environmental Data

ADHS evaluated drinking water exposures using the water sampling data collected in 2014 under EPA's UCMR 3 (Table 1, Appendix B). The sampling data were provided to ADHS by ADEQ. The OWC does not have any treatment for PFAS and only has one well in operation. Therefore, the sampling results are most likely representative of the water being delivered.

Exposure Pathway and Screening Analyses

Exposure Pathway Analysis

Identifying exposure pathways is important in a health consultation because adverse health impacts from contaminants can only happen if people are exposed to them. The presence of a contaminant in the environment does not necessarily mean that people are actually coming into contact with it. Exposure pathways have been divided into three categories: completed, potential, and eliminated.

There are five elements considered in the evaluation of exposure pathways:

- 1) a <u>source</u> of contamination
- 2) a <u>medium</u> such as soil or ground water through which the contaminant is transported
- 3) a <u>point of exposure</u> where people come into contact with the contaminant
- 4) a <u>route of exposure</u> by which the contaminant enters or comes into contact with the body
- 5) a <u>receptor</u> population (i.e. exposed population)

Completed pathways exist when all five elements are present and indicate that exposure to a contaminant has occurred in the past and/or is occurring presently. In a potential exposure pathway, one or more elements of the pathway cannot be identified, but it is possible that the element might be present or might have been present. In eliminated pathways, at least one of the five elements is or was missing, and is not expected to be present in the future.

Table 2 (Appendix B) shows that residents (receptor population) can be exposed to PFAS from contaminated groundwater (potential contamination source: fire-fighting foam), in public drinking water (exposure medium) while they are using the tap water (exposure point) for drinking, cooking, and preparing formula (via ingestion exposure route). This is a completed exposure pathway. ADHS further evaluated the completed ingestion exposure pathway to determine whether exposures were at high enough concentrations, often enough, and for a long enough period of time to result in adverse health effects.

Exposures to contaminants in drinking water include ingestion exposure (from drinking water, cooking with water and incidental ingestion during showering), dermal exposure (from bathing, showering or dishwashing), and inhalation exposure (from bathing or showering). Ingestion is considered the most significant exposure pathway. PFAS do not easily volatilize during bathing and showering, and absorption of PFAS through skin is slow. Breathing in PFAS and absorbing PFAS through the skin are not significant routes of exposure (USEPA 2016b; ATSDR 2015). As a result, inhalation and dermal exposures were not considered in the evaluation.

Screening Analysis: Comparison to Health-based Comparison Values

Following identification of a completed/potential exposure pathway, ADHS conducted a screening analysis of detected chemicals against health-based comparison values (CVs). These comparison values are conservative, and include uncertainty factors that account for the most sensitive populations. Adverse health effects are not expected to occur if an exposure concentration is below a CV. However, an exposure concentration at or above the CV doesn't automatically mean adverse effects will occur. Rather, it means that there is a need to conduct a site-specific exposure evaluation. The health risk for an individual depends on individual human factors (e.g. personal habits, occupation, and overall health), and site-specific environmental exposure factors (e.g. duration and amount of exposure). Therefore, the comparison values are not used to predict the occurrence of adverse health effects without looking at site-specific conditions.

To be health protective, for this evaluation ADHS used the maximum detected concentrations as the exposure concentrations. EPA's LTHA of 0.07 ppb is the recommended CV for PFOA and PFOS in drinking water, individually or combined. During the initial screening process, ADHS compared the combined PFOA and PFOS water concentration to the EPA LTHA level of 0.07 ppb since both were found in the water samples. The information is summarized in Table 3 (Appendix B). The combined concentration of PFOA and PFOS was found to exceed the comparison value and was selected for further evaluation. There is only limited toxicological information available to further evaluate PFHpA and PFHxS.

Review of Public Health Implications related to PFAS Exposure

Perfluorooctane sulfonate (PFOS)

PFOS is water- and lipid-resistant due to its chemical properties. PFOS can be found in carpets, leathers, paper packaging, coating additives, AFFF, and is used as a water- or stain- proofing agent. Most PFOS manufacturing was discontinued voluntarily in the US by 2002 (ATSDR 2015; EPA 2016b). However, exposures to PFOS are still possible because it was widely used, and is very persistent in the environment.

Food ingestion was an important exposure route because PFAS were widely used in food packaging and consumer products. PFOS was detected in a variety of food sources and processed food products including snack foods, vegetables, meat, dairy products, fish, and human breast milk (Van Asselt et al. 2011). In a survey that included multiple food types, PFOS was the most frequently detected PFAS and was present at higher concentrations than other related compounds (Hlouskova et al. 2013).

Some epidemiology studies showed associations between PFOS exposures and high cholesterol and reproductive and developmental effects. The strongest association was related to serum lipids with increased total cholesterol and high density lipoproteins (USEPA 2016a; ATSDR 2015). Results for mean birth weight, low birth weight, and small for gestational age were inconclusive due to study limitations such as exposure misclassification and confounders (USEPA 2014).

In most animal studies of PFOS exposures, changes in absolute and/or relative liver weight is the most common effect observed. Developmental effects were also observed in animal studies. Exposure to PFOS has significantly decreased birth weight and survival in neonatal rats exposed in utero (USEPA 2016a). PFOS exposure resulted in delayed development of mouse and rat pups, but this did not occur in animals exposed to perfluorobutanoic acid (PFBA) or PFHxS. Alterations in motor activity have also been observed in mouse pups exposed to PFOA, PFOS, or PFHxS, but not perfluorodecanoic acid (PFDeA) (ATSDR 2015).

Presently, EPA has not derived a cancer slope factor for PFOS. A chronic oral toxicity and carcinogenicity study identified liver, thyroid, and mammary fibroadenomas in rats but no dose-response relationship was identified. Under the EPA cancer guidelines, the evidence for

the carcinogenicity of PFOS is considered "suggestive of carcinogenicity," but it is not sufficient to assess human carcinogenicity (USEPA 2014; USEPA 2016a).

Perfluorooctanoic acid (PFOA, or C8)

Similar to PFOS, PFOA has been used to make household and commercial products that resist heat and chemical reactions, and repel oil, stains, grease and water. It can be found in nonstick cookware, stain-resistant carpet and fabrics, and fire-fighting foam. PFOA does not break down easily and persists for a very long time in the environment, especially in water. Major U.S. manufacturers voluntarily agreed to phase out production of PFOA by the end of 2015 (ATSDR 2015; EPA 2016d).

Adverse health effects due to PFOA exposures are the same or similar to PFOS. From 2005 to 2013, a series of epidemiology studies, the C8 Health Project², were conducted to assess the links between PFOA exposures from contaminated drinking water and diseases among 69,030 individuals in West Virginia. The C8 Science Panel found a probable link between PFOA exposures and high cholesterol, ulcerative colitis, thyroid disease, testicular and kidney cancers, and pregnancy-induced hypertension (ATSDR 2015). Epidemiology studies showed effects on serum lipids, serum uric acid and serum antibodies. A positive association was found between serum PFOA concentration and higher cholesterol in both general (Fitz-Simon et al. 2013; Frisbee et al. 2010; Steenland et al. 2009) and worker populations (Costa et al. 2009; Olsen et al. 2000, 2003; Olsen and Zobel, 2007; Steenland et al. 2015). Increases in uric acid levels have been observed in individuals with higher PFOA levels (Shankar et al. 2011; Steenland et al. 2010). Maternal or child plasma levels of PFOA were found to be positively associated with decreased antibody titers in children after vaccination (Grandjean et al. 2012; Granum et al. 2013), and obesogenic effects in female children at 20 years of age (Halldorsson et al. 2012). A positive association has been shown between serum PFOA concentrations and increased liver enzymes and/or decreased bilirubin in both worker (Costa et al. 2009; Olsen et al 2000, 2003; Olsen and Zobel, 2007) and general populations (Emmett et al. 2006; Gallo et al. 2012), chronic kidney disease in the general population (Shankar et al. 2011), and the odds of experiencing early menopause (Knox et al. 2011).

In animal studies of PFOA exposure, increased liver weight is one of the most observed effects. Other common effects include changes in spleen, thymus, and liver endpoints. Animal studies also showed developmental effects based on low birth weights, skeletal effects, and delayed onset of puberty (Butenhoff et al. 2004; Lau et al. 2006, Wolf et al. 2007; USEPA 2016d).

There is suggestive evidence for carcinogenic potential for PFOA. Epidemiology studies demonstrate an association of serum PFOA with kidney and testicular tumors among highly exposed members of the general population (ATSDR 2015; USEPA 2014; USEPA 2016d). EPA estimated a cancer slope factor of 0.07 per milligram per kilogram-day (mg/kg/day)⁻¹ based on

² See <u>http://www.c8sciencepanel.org/prob_link.html</u> for more information.

testicular tumors (USEPA 2016d). Further, EPA stated that the LTHA based on noncancer effects is protective for cancer effects, too (USEPA 2016d).

Perfluoroheptanoic acid (PFHpA)

PFHpA is part of a family of perfluoroalkyl carboxylates, which have similar structures but different carbon chain lengths. There is very limited toxicological research on PFHpA. ADHS did not identify specific toxicity information regarding this chemical.

Perfluorohexane sulfonic acid (PFHxS)

PFHxS is a type of PFAS that was used in certain fire-fighting foams and post-market carpet treatment applications. There are limited studies on PFHxS. Results from animal studies showed that administration of PFHxS to female and male rats from premating until postnatal day (PND) 21 did not result in any reproductive or developmental effects in dams or offspring; however, effects were seen in parental male rats including increased time for blood to clot, increased liver weights and the size of liver cells, and enlarged thyroid follicular cell (Butenhoff et al. 2009a). No reproductive or developmental effects were reported in these studies. In another study, altered spontaneous activity and habituation were observed in adult mice administered 9.2 mg/kg/day on PND 10 (Viberg et al. 2013).

Results of Site-specific Evaluation

ADHS used EPA's chronic Reference Doses (RfDs) for PFOS and PFOA, and ATSDR's default exposure scenario assumptions (ATSDR 2015; ATSDR 2005). An RfD is an estimate of a daily human exposure to the human population (including sensitive subgroups) that is likely to be without an adverse effect during a lifetime. EPA's RfDs were selected for estimating exposure doses in this community because these values are protective of both short-term and long-term adverse health effects (USEPA 2016d). This community is believed to have been exposed to PFOA and PFOS in drinking water for many years.

ATSDR's default exposure assumptions are defined by specific age groups with corresponding estimated exposure doses for each age group. ADHS estimated a central tendency exposure (CTE) and a reasonable maximum exposure (RME) that may occur for each age group according to the following equation (Table 4, Appendix B).

$$Exposure Dose\left(\frac{mg}{kg/day}\right) = \frac{Drinking Water Intake Rate\left(\frac{L}{day}\right) \times Drinking Water Concentration\left(\frac{mg}{L}\right)}{Body Weight (kg)}$$

By calculating estimated exposure doses, ADHS can better assess the possible public health implications for site-specific conditions among different age populations under different exposure durations. CTE estimates assumed typical (or average) water intake levels among each age group. As a result, it provides estimates of the average exposure dose for each age group. RME estimates assumed reasonable maximum water intake levels (i.e. the 95th percentile, USEPA 2011) for each age group. Therefore, the RME is higher than average but still within a realistic exposure range. ADHS used the highest concentration from the 2014 sampling results to calculate the CTE and RME doses. Body weights were selected based on data from NHANES 1999 – 2006, as reported in the USEPA Exposure Factors Handbook (USEPA 2011).

EPA has developed an RfD of $2x10^{-5}$ (0.00002) mg/kg/day for PFOS based on reduced pup body weight in a two-generation rat study (Luebker et al. 2005). EPA derived the human equivalent dose (HED) for PFOS from the no observed adverse effect level (NOAEL) identified in Luebker et al. (2005) (NOAEL_{HED} of $5.1x10^{-4}$ mg/kg/day). EPA then applied an uncertainty factor of 30 (10 for human variability and 3 for animal to human extrapolation) to derive the RfD of $2x10^{-5}$ mg/kg/day. This RfD is also protective for developmental effects, which is supported by the results from Butenhoff et al. (2009b). EPA has also developed an RfD of $2x10^{-5}$ mg/kg/day for PFOA based on reduced ossification of the proximal phalanges (forelimb and hindlimb) and accelerated puberty in male rat pups from Lau et al. (2006). This RfD is determined based on a lowest observed adverse effect level (LOAEL) derived from the human equivalent dose (LOAEL_{HED}) of 5.3×10^{-3} mg/kg/day and an uncertainty factor of 300 (10 for human variability, 10 for using LOAEL, and 3 for animal to human extrapolation).

The maximum reported concentrations of PFOA and PFOS in finished water from the Oatman water system were 0.032 and 0.3 ppb, respectively. Estimated exposure doses using the maximum PFOA and PFOS water concentrations and the RME and CTE water intake scenarios were calculated and compared to the RfDs described above (Table 4, Appendix B).

- None of the estimated CTE and RME doses were above the RfD for PFOA.
- None of the estimated CTE doses were above the RfD for PFOS.
- Estimated RME doses were above the RfD for PFOS for infants and young children.

In order to evaluate the potential risk of cumulative exposure to PFOA and PFOS, ADHS calculated a hazard index (HI). The hazard index approach uses the assumption of dose additivity to assess the non-cancer health effects of a mixture from the data of the components. The hazard index is the sum of the quotients of the estimated dose of a chemical divided by its RfD. If the hazard index is less than 1.0, it is highly unlikely that significant additive or toxic interactions would occur, so no further evaluation is necessary. If the hazard index is greater than 1.0, further evaluation is necessary.

As shown in Table 4 (Appendix B), infants and young children whose primary drinking water source was the Oatman Water Company and who drank average or above average quantities of this water at the maximum concentrations detected may have an increased risk of harmful effects resulting from exposure to PFOA and PFOS.

Uncertainties and Limitations

Only two sampling events were conducted in 2014. These concentrations may not well represent the actual exposure concentrations for a long period of time. In addition, ADHS did not have information about the health status of people consuming OWC water. The available epidemiology data identify several potential targets of toxicity of PFAS, and individuals with pre-existing conditions may be unusually susceptible. For example, it appears that exposure to PFOA or PFOS may result in increases in serum lipid levels, particularly cholesterol levels. Thus, an increase in serum cholesterol may result in a greater health impact in individuals with high levels of cholesterol or with other existing cardiovascular risk factors. Similarly, increases in uric acid levels have been observed in individuals with higher perfluoroalkyl levels; increased uric acid may be associated with an increased risk of high blood pressure. Thus, individuals with hypertension may be at greater risk. The liver has been shown to be a sensitive target in a number of animal species and there is some indication that it is also a target in humans. Therefore, individuals with compromised liver function may represent a susceptible population (ATSDR 2015).

There is uncertainty about whether community members have had additional exposures to PFAS from other sources such as PFAS contaminated food (certain types of fish and shellfish if nearby streams, rivers, lakes, etc. are impacted), hand-to-mouth transfer from surfaces previously treated with PFAS-containing stain protectants (carpet, most significant for infants and toddlers), or eating food packaged in material containing PFAS (popcorn bags, fast food container, or pizza boxes). There is not enough information to identify the individual exposure sources and to estimate the background exposure level in the OWC water users. The EPA LTHA assumes that PFOA and PFOS exposures from other sources (e.g., dust, diet, air) make up 80% of the overall exposure.

At the present time, ADHS and ATSDR do not have an approach for evaluating the public health implication of exposures to PFAS other than PFOA and PFOS. Currently, ATSDR considers effects from all PFAS exposures to be additive, including PFHpA and PFHxS. Therefore, detection of PFHpA and PFHxS at this site adds additional risk to the PFAS exposure.

Child Health Concerns

ADHS recognizes that the unique vulnerabilities of infants and children demand special emphasis in communities affected by environmental contamination. A child's developing body systems can sustain permanent damage if toxic exposures occur during critical growth stages. Children ingest a larger amount of water relative to body weight than adults, resulting in a higher burden of pollutants in proportion to body size. Furthermore, making them more sensitive to pollution than healthy adults.

PFAS can be transferred to nursing infants. Studies showed variable correlations among measured PFAS in maternal serum and breast milk in matched mother-infant pairs (ATSDR

2015). Some epidemiology studies showed that higher PFOA or PFOS in serum is associated with a slight decrease in infant birth weight (ATSDR 2015). Birth defects such as delayed lung maturation and lung atelectasis were seen in mice born to females that ingested relatively high amounts of PFOS (10 times higher than the highest PFOS serum levels measured in workers) during pregnancy (ATSDR 2015).

Conclusions

This health consultation evaluated the potential health risks associated with exposure to PFAS in the OWC public drinking water. The water samples were collected prior to the entry point to the distribution system which is after processing and storage in the water treatment plant, but prior to reaching the first customer. ADHS reached the following conclusions:

- Consuming the drinking water at the reported PFAS concentrations, especially for infants and young children, combined with other sources of exposure (i.e. background exposures from sources besides water and the body burden from pre-existing exposures), and pre-existing risk factors could increase the risk for health effects.
- PFHpA and PFHxS compounds were found at elevated levels in the water, have reported health effects (albeit limited toxicity data), and PFHxS also has a long half-life in the body. However, no EPA health advisories currently exist for these compounds. Currently, ATSDR considers effects from all PFAS exposures to be additive, including PFHpA and PFHxS. Therefore, detection of PFHpA and PFHxS at this site adds additional risk to the PFAS exposure.

Recommendations

- ADHS recommends that OWC implement a long-term remedy to reduce the PFAS water levels in the public drinking water supply.
- ADHS recommends that health education information about PFAS be provided to community members and the health professionals serving the area.
- ADHS recommends that pregnant and lactating women, caregivers preparing formula for bottle-fed infants, and women of child bearing age use alternate water until OWC reduces PFAS in the water.

Public Health Action Plan

- ADHS will attend public meetings to discuss the process of preparing health consultations and community concerns upon the community's request. ADHS will make presentations, develop handouts, and provide additional assistance as necessary to notify Oatman community members of the findings of this health consultation.
- ADHS will notify ADEQ regarding the findings of this report and work with ADEQ to evaluate the protectiveness of mitigation action plans.

• ADHS will continue to review and evaluate data provided for this site upon request.

References

ATSDR (Agency for Toxic Substances and Disease Registry). 2005. Public Health Guidance Manual (Update). Agency for Toxic Substances and Disease Registry, Public Health Service, United States Department of Health and Human Services, Atlanta, GA. Accessed: October 2016. <u>http://www.atsdr.cdc.gov/hac/phamanual/pdfs/phagm_final1-27-05.pdf</u>

ATSDR (Agency for Toxic Substances and Disease Registry). 2015. Toxicological Profile for Perfluoroalkyls. Draft for Public Comment. Agency for Toxic Substances and Disease Registry, Public Health Service, United States Department of Health and Human Services, Atlanta, GA. Accessed: September 2016. <u>http://www.atsdr.cdc.gov/ToxProfiles/tp200.pdf</u>

Bartell, S.M., et al., Rate of decline in serum PFOA concentrations after granular activated carbon filtration at two public water systems in Ohio and West Virginia. Environ Health Perspect, 2010. 118(2): p222-8.

Butenhoff, J.L., G.L. Kennedy, Jr., S.R. Frame, J.C. O'Connor, and R.G. York. 2004. The reproductive toxicology of ammonium perfluorooctanoate (APFO) in the rat. Toxicology 196:95–116

Butenhoff JL, Chang S, Ehresman DJ, et al. 2009a. Evaluation of potential reproductive and developmental toxicity of potassium perfluorohexanesulfonate in Sprague Dawley rats. Reproductive Toxicology 27:331-341

Butenhoff, J.L., D.J. Ehresman, S.-C.Chang, G.A. Parker, and D.G. Stump. 2009b. Gestational and lactational exposure to potassium perfluorooctanesulfonate (K+PFOS) in rats: developmental neurotoxicity. Reproductive Toxicology 27:319–330

CDC (Center for Disease Control and Prevention). 2015. Fourth National Report on Human Exposure To Environmental Chemicals, Updated Tables. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. Accessed: October 2016: <u>http://www.cdc.gov/biomonitoring/pdf/FourthReport_UpdatedTables_Feb2015.pdf</u>

Costa G., S. Sartori, and D. Consonni. 2009. Thirty years of medical surveillance in perfluorooctanoic acid production workers. Journal of Occupational and Environmental Medicine 51:364–372

Emmett, E.A., H. Zhang, F.S. Shofer, D. Freeman, N.V. Rodway, C. Desai, and L.M. Shaw. 2006. Community Exposure to Perfluorooctanoate: Relationships between Serum Concentrations and Certain Health Parameters. Journal of Occupational Medicine 48:771–779

Fitz-Simon, N., T. Fletcher, M.I. Luster, K. Steenland, A.M. Calafat, K. Kato, and B. Armstrong. 2013. Reductions in serum lipids with a 4-year decline in serum perfluorooctanoic acid and perfluorooctanesulfonic acid. Epidemiology 24(4):569–576

Frisbee, S.J., A. Shankar, S.S. Knox, K. Steenland, D.A. Savitz, T. Fletcher, and A. Ducatman. 2010. Perfluorooctanoic acid, perfluorooctanesulfonate, and serum lipids in children and adolescents: results from the C8 health project. Archives of Pediatrics and Adolescent Medicine 164:860–869

Fromme, H. et al., Perfluorinated compounds—exposure assessment for the general population in Western countries. Int J Hyg Environ Health, 2009. 212(3):p239-70.

Gallo, V., G. Leonardi, B. Genser, M.J. Lopez-Espinosa, S.J. Frisbee. L. Karlsson, A.M. Ducatman, and T. Fletcher. 2012. Serum perfluorooctanoate (PFOA) and perfluorooctane sulfonate (PFOS) concentrations and liver function biomarkers in a population with elevated PFOA exposure. Environmental Health Perspectives 120:655–660

Grandjean, P., E.W. Andersen, E. Budtz-Jørgensen, F. Nielsen, K. Mølbak, P. Weihe, and C. Heilmann. 2012. Serum vaccine antibody concentrations in children exposed to perfluorinated compounds. Journal of the American Medical Association 307:391–397

Granum, B., L.S. Haug, E. Namork, S.B. Stølevik, C. Thomsen, I.S. Aaberge, H. van Loveren, M. Løvik, and U.C. Nygaard. 2013. Pre-natal exposure to perfluoroalkyl substances may be associated with altered vaccine antibody levels and immune-related health outcomes in early childhood. Journal of Immunotoxicology 10(4):373–379

Halldorsson, T.I., D. Rytter, L.S. Haug, B.H. Bech, I. Danielsen, G. Becher, T.B. Henriksen, and S.F. Olsen. 2012. Prenatal exposure to perfluorooctanoate and risk of overweight at 20 years of age: A prospective cohort study. Environmental Health Perspectives 120:668–673

Hlouskova, V., P. Hradkova, J. Poustka, G. Brambilla, S.P. De Filipps, W. D'Hollander, L. Bervoets, D. Herzker, S. Huber, P. De Voogt, and J. Pulkrabova. 2013. Occurrence of perfluoroalkyl substances (PFASs) in various food items of animal origin collected in four European countries. Food Additives & Contaminants: Part A 30(11):1918–1932

Kato, K. et al., Trends in exposure to polyfluoroalkyl chemicals in the U.S. population: 1999–2008. Environmental Science and Technology, 2011. 45(19): p8037-45.

Knox, S.S., T. Jackson, B. Javins, S.J. Frisbee, A. Shankar, and A.M. Ducatman. 2011. Implications of early menopause in women exposed to perfluorocarbons. Journal of Endocrinology and Metabolism 96:1–7

Lau, C., J.R. Thibodeaux, R.G. Hanson, M.G. Narotsky, J.M. Rogers, A.B. Lindstrom, and M.J. Strynar. 2006. Effects of perfluorooctanoic acid exposure during pregnancy in the mouse. *Toxicological Science* 90:510–518.

Luebker, D.J., M.T. Case, R.G. York, J.A. Moore, K.J. Hansen, and J.L. Butenhoff. 2005. Twogeneration reproduction and cross-foster studies of perfluorooctanesulfonate (PFOS) in rats. Toxicology 215:126–148

Olsen, G.W., J.M. Burris, M.M. Burlew, and J.H. Mandel. 2000. Plasma cholecystokinin and hepatic enzymes, cholesterol and lipoproteins in ammonium perfluorooctanoate production workers. Drug and Chemical Toxicology 23:603–620

Olsen, G.W., J.M. Burris, M.M. Burlew, and J.H. Mandel. 2003. Epidemiologic assessment of worker serum perfluorooctanesulfonate (PFOS) and perfluorooctanoate (PFOA) concentrations and medical surveillance examinations. Journal of Occupational and Environmental Medicine 45:260–270

Olsen, G.W., and L.R. Zobel. 2007. Assessment of lipid, hepatic, and thyroid parameters with serum perfluorooctanoate (PFOA) concentrations in fluorochemical workers. International Archives of Occupational and Environmental Health 81:231–246

Shankar, A., J. Xiao, and A. Ducatman. 2011. Perfluoroalkyl chemicals and chronic kidney disease in US adults. American Journal of Epidemiology 174(8):893–900

Steenland, K., S. Tinker, S. Frisbee, A. Ducatman, and V. Vaccarino. 2009. Association of perfluorooctanoic acid and perfluorooctane sulfonate with serum lipds among adults living near a chemical plant. American Journal of Epidemiology 170:1269–1278

Steenland, K., S. Tinker, A. Shankar, and A. Ducatman. 2010. Association of perfluorooctanoic acid (PFOA) and perfluorooctane sulfonate (PFOS) with uric acid among adults with elevated community exposure to PFOA. Environmental Health Perspectives 118:229–233

Steenland, K., L. Zhao, and A. Winquist. 2015. A cohort incidence study of workers exposed to perfluoroctanoic acid (PFOA). Occupational and Environmental Medicine 0:1–8

USEPA (U.S. Environmental Protection Agency), Exposure Factors Handbook 2011 Edition (Final). USEPA, 2011. PA/600/R-09/052F.

USEPA (U.S. Environmental Protection Agency). 2014. Emerging Contaminants – Perfluorooctane Sulfonate (PFOS) and Perfluorooctanoic Acid (PFOA). U.S. Environmental Protection Agency, Solid Waste and Emergency Response. Washington, DC. Accessed: September 2016. <u>http://nepis.epa.gov/Exe/ZyPDF.cgi/P100LTG6.PDF?Dockey=P100LTG6.PDF</u>

USEPA (U.S. Environmental Protection Agency). 2016a. Health Effects Support Document for Perfluorooctane Sulfonate (PFOS). U.S. EPA, Office of Water. Accessed: September 2016. <u>https://www.epa.gov/sites/production/files/2016-05/documents/pfos_hesd_final_508.pdf</u>

USEPA (U.S. Environmental Protection Agency). 2016b. Drinking Water Health Advisory for Perfluorooctane Sulfonate (PFOS). U.S. EPA, Office of Water. Accessed: September 2016. <u>https://www.epa.gov/sites/production/files/2016-</u>05/documents/pfos health advisory final 508.pdf

USEPA (U.S. Environmental Protection Agency). 2016c. Factsheet: PFOA and PFOS Drinking Water Health Advisories. Accessed: September 2016. <u>https://www.epa.gov/sites/production/files/2016-</u> 06/documents/drinkingwaterhealthadvisories pfoa pfos updated 5.31.16.pdf

USEPA (U.S. Environmental Protection Agency). 2016d. Drinking Water Health Advisory for Perfluorooctanoic Acid (PFOA). U.S. EPA, Office of Water. Accessed: October 2016. <u>https://www.epa.gov/sites/production/files/2016-05/documents/pfoa_health_advisory_final-plain.pdf</u> Van Asselt, E.D., R.P.J.J. Rietra, P.F.A.M. Romkens, and H.J. van der Fels-Klerx. 2011. Perfourooctane sulphonate (PFOS) throughout the food production chain. Food Chemistry 128:1–6

Viberg H, Lee I, Eriksson P. 2013. Adult dose-dependent behavioral and cognitive disturbances after a single neonatal PFHxS dose. Toxicology 304:185-191

Wolf, C.J., S.E. Fenton, J.E. Schmid, A.M. Calafat, Z. Kuklenyik, X.A. Bryant, J. Thibodeaux, K.P. Das, S.S. White, C.S. Lau, and B.D. Abbott. 2007. Developmental toxicity of perfluorooctanoic acid in the CD-1 mouse after cross-foster and restricted gestational exposure. Toxicological Science 95:462–473.

Wu, X.M, et al. Serum concentration perfluorinated compound (PCF) among selected population of children and adults in California. Environmental Research, 2015. 136: p264-73.

REPORT PREPARATION

This Public Health Consultation for the *Oatman Water Company* site, located in the Town of Oatman, Mohave County, Arizona was prepared by the Arizona Department of Health Services under a cooperative agreement with the federal Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with the approved agency methods, policies and procedures existing at the date of publication. Editorial review was completed by the cooperative agreement partner. ATSDR has reviewed this document and concurs with its findings based on the information presented.

Author

Hsini Lin, ScD, MSPH, Toxicologist Environmental Toxicology Program

ADHS Reviewers

Matthew Roach, MPH, Program Manager Environmental Toxicology Program

Brigitte Dufour, Chief Office of Environmental Health

ATSDR Technical Project Officer

Eva McLanahan, Ph.D., REHS/RS Commander, U.S. Public Health Service Division of Community Health Investigations Office of the Director

ATSDR Regional Representatives

Libby Vianu Regional Representative Division of Community Health Investigations Western Branch, Region IX

Ben Gerhardstein, MPH Regional Representative Division of Community Health Investigations Western Branch, Region IX

Jamie Rayman, MPH Health Educator and Community Involvement Specialist Division of Community Health Investigations Western Branch, Region IX

Oatman Water Company

May 19, 2016

Yesterday, the EPA issued new health advisory on two contaminants which are unregulated. These two contaminants are fluorinated organic chemicals. They are referred to as PFOA and PFOS. They are manmade chemicals (not naturally found in nature) and are used in industrial and commercial applications, such as carpeting, coating (Teflon) and fire fighting foam. Tests were done on Oatman Water Co. water in 2014 and these two were found. PFOA was 32 parts per trillion and PFOS was 230 parts per trillion.

The EPA health advisory says:

To provide Americans, including the most sensitive population, with a margin of protection from a lifetime exposure to PFOA and PFOS from drinking water, EPA has established the health advisory levels at 70 parts per trillion. When both PFOA and PFOS are found in drinking water, the combined concentrations of PFOA and PFOS should be compared with this 70 parts per trillion. This health advisory level offers a margin of protection for all Americans throughout their life from adverse health effects resulting from exposure to PFOA and PFOS. Health advisories provide information on contaminants that can cause human health effects. They are non-enforceable and non-regulatory. They provide technical guidance to state agencies and public health officials.

As a precaution, pregnant women, breast feeding mothers and women feeding the babies with formula should not drink or use water that has PFOA and PFOS over the health advisory limit.

The purpose of this notice is to make you aware of this recent EPA announcement. I will be retesting the water for PFOA and PFOS to update the 2014 test results. If this continues, we will work with agency officials to set forth an action plan to resolve this situation.

A copy of this letter will be made available to customers upon request.

Oatman Water Co.

Appendix **B**

Tables

Table 1. The 2014 water sampling results in parts per billion¹ (ppb). The water samples were collected under EPA's UCMR 3. The sampling data were provided to ADHS by ADEQ.

Sampling	PFOA ²	PFOS ³	PFHpA ⁴	PFHxS⁵
Date	(ppb)	(ppb)	(ppb)	(ppb)
04/22/2014	0.030	0.30	0.014	0.73
10/29/2014	0.032	0.23	0.015	0.69

^{1.} 1 ppb = 1,000 parts per trillion (ppt) = 1 micrograms per liter (μ g/L)

^{2.} PFOA: Perfluorooctanoic acid

^{3.} PFOS: Perfluorooctane sulfonate

^{4.} PFHpA: Perfluoroheptanoic acid

^{5.} PFHxS: Perfluorohexane sulfonate

Table 2. Exposure Pathways Analysis

Exposure Pathway Elements						Type of	
Source	Medium	Point of Route of exposure		Potentially exposed population	Time frame	Exposure Pathway	
Contaminated groundwater well*	Public Drinking Water	Residences, tap	Ingestion	Residents	Past	Completed	
					Current	Completed	
					Future	Potential	

*According to the ADEQ Drinking Water Section, OWC believes fire-fighting foam may be the source of PFAS contamination in the groundwater well.

Table 3. Comparison to Screening Value

Chemical	# of samples	Maximum detected concentration (ppb)	со	Health-based mparison value (ppb)	Does it exceed the comparison value?
PFOA + PFOS	2	0.332	0.07 EPA LTHA		Yes
PFHpA	2	0.015	Not available		
PFHxS	2	0.73	Not available		

PFOA: Perfluorooctanoic acid: 0.032 ppb maximum concentration detected PFOS: Perfluorooctane sulfonate: 0.3 ppb maximum concentration detected

PFHpA: Perfluoroheptanoic acid

PFHxS: Perfluorohexane sulfonate

EPA LTHA: US Environmental Protection Agency Lifetime Health Advisory

1 parts per billion (ppb) = 1,000 parts per trillion (ppt) = 1 micrograms per liter (μ g/L)

	Exposure Assumptions			Estimated Exposure Dose (mg/kg/day)					
Age Group	Drinking Water Intake (L/day)		Body Weight (kg)	Maximum PFOA Water Concentration = 0.032 ppb		Maximum PFOS Water Concentration = 0.3 ppb		Hazard Index (HI) for PFOA + PFOS	
	RME	CTE	Mean	RME	CTE	RME	CTE	RME	CTE
Birth to < 1 yr	1.113	0.504	7.8	4.57E-06	2.07E-06	4.28E-05	1.94E-05	2.37	1.07
1 to < 2 yr	0.893	0.308	11.4	2.51E-06	8.65E-07	2.35E-05	8.11E-06	1.30	0.45
2 to < 6 yr	0.977	0.376	17.4	1.80E-06	6.91E-07	1.68E-05	6.48E-06	0.93	0.36
6 to < 11 yr	1.404	0.511	31.8	1.41E-06	5.14E-07	1.32E-05	4.82E-06	0.73	0.27
11 to <16 yr	1.976	0.637	56.8	1.11E-06	3.59E-07	1.04E-05	3.36E-06	0.58	0.19
16 to <21 yr	2.444	0.770	71.6	1.09E-06	3.44E-07	1.02E-05	3.23E-06	0.57	0.18
Adults ≥ 21 yr	3.092	1.227	80	1.24E-06	4.91E-07	1.16E-05	4.60E-06	0.64	0.25
Lactating Women	3.588	1.665	73	1.57E-06	7.30E-07	1.47E-05	6.84E-06	0.82	0.38
Pregnant Women	2.589	0.872	73	1.13E-06	3.82E-07	1.06E-05	3.58E-06	0.59	0.20

Table 4. Estimated exposure doses for community members drinking water from the Oatman WaterCompany

Notes: CTE = central tendency of exposure, L = Liter, mg/kg/day = milligrams of chemical per kilogram of body weight per day, RME = reasonable maximum exposure concentration, 1 parts per billion (ppb) = 1,000 parts per trillion (ppt) = 1 micrograms per liter (μ g/L), PFOA RfD = 0.00002 mg/kg/day, PFOS RfD = 0.00002 mg/kg/day.