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Aquatic Effects of a Localized Oil Spill on Lake Conway, AR and Its Tributaries

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Running title: Localized Oil Spill on Lake Conway, AR and Tributaries

Abstract

Oil spills, no matter where they occur, elicit environmental concern and avoiding these disasters should be a priority. Old pipelines that are not regularly maintained and carry large amounts of crude oil long distances are of particular concern. One such pipeline is the 65 year-old Pegasus pipeline owned by ExxonMobil. On March 29, 2013, 795,000 L of Wabasca Heavy Canadian crude oil spilled into a neighborhood of Mayflower, Arkansas, when the Pegasus pipeline ruptured. This spill led to the evacuation of many homes in the surrounding neighborhood. Drainage ditches in the affected neighborhood drained oil into a nearby cove of Lake Conway. This lake is popular for recreational fishing, thus concerns were raised not only about the potential effects of the oil spill on area residents, but also the lake and its biological communities. Ultimately, this project assessed the effect of the oil spill in water and sediment samples on freshwater test organisms. Samples were collected at 6 sites in the affected neighborhood and in Lake Conway. Chronic Whole Effluent Toxicity (WET) tests were performed on water samples using *Pimephales promelas* and *Ceriodaphnia dubia*. Acute sediment toxicity tests were performed using *Chironomus dilutus*. These tests measured sub-lethal toxicity in at least one of the sampled sites, indicating that further investigation of environmental after-effects is warranted.

Introduction

On March 29, 2013, a 6.71 meter rupture occurred in the 65 year-old Pegasus pipeline running through Mayflower, Arkansas, spilling 795,000 L of Wabasca Heavy Canadian Crude oil into a nearby neighborhood (Gallucci 2013b). This led to the evacuation of many homes and many complaints of sickness ranging from nausea to bronchitis. Spilled oil also reached a cove of nearby Lake Conway, a 2,700 hectare recreational

fishing lake (Gallucci 2013a). Remediation began immediately after the spill and concluded with the affected cove being completely dredged. This action included removal by vacuuming the oil and contaminated water, excavation of contaminated vegetation and soil, and blocking the flow of water from the cove to the main body of the lake (Hardy 2013).

The extension of hook cracks was responsible for the rupture in the Pegasus pipeline (Douglas 2013). These cracks are common in old pipelines, however, the cracks in the Pegasus pipeline probably grew because of high pressure swings due to the type of oil the pipeline was carrying. At the time of the rupture, the Pegasus pipeline was carrying Wabasca Heavy Canadian crude oil, a form of diluted bitumen or dilbit, which is heavy and possibly made the pressure swings harder to push through the pipeline (Douglas 2013). Dilbit also could have contributed to the increase of hydrogen atoms moving to the fragile hook cracks of the pipeline. This type of crude oil contains the second-highest sulfur content of 29 types of Canadian crude oil (Douglas 2013). When hydrogen sulfide decomposes, it releases hydrogen atoms which move to fragile seams in pipelines and increases stress.

Dilbit not only causes harm inside pipelines, but also poses a great risk to humans and the environment due mainly to its harmful chemical makeup. The United States Environmental Protection Agency (USEPA) and United States Coast Guard (USCG) rank petroleum-based oil on a scale from 1-5. Group 1 includes gasoline or kerosene, having a density of less than 0.8, while group 5 includes crudes having a density greater than 1 (POLARIS 2013). Dilbit can be found in group 2, having a density of 0.85-0.95, higher than gas oil and light crudes (POLARIS 2013). The greater the density of the oil, the more likely it is to sink into the water column or sediment, increasing the chance of harm done to surrounding organisms.

Total petroleum hydrocarbons (TPH) are a mixture of several hundred chemicals that are found in crude oil (ATSDR 1999). Instead of focusing on each

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individual chemical, TPHs compile all of these chemicals, including hexane, toluene, xylenes, and naphthalene (ATSDR 1999). TPH exposure could cause nervous system issues such as headaches and dizziness (ATSDR 1999). In aquatic environments, TPH can sink to the bottom or float and may remain in soil for long periods of time (ATSDR 1999).

Dilbit is composed of benzene, polycyclic aromatic hydrocarbons (PAHs), and several heavy metals such as vanadium and arsenic (Swift et al. 2011). PAHs are cause for concern due to their environmental persistence and recalcitrant nature in water (USEPA 2008). In humans, acute exposure to benzene and PAHs have been shown to cause respiratory, gastrointestinal, and neurological problems, while long term exposure has been known to cause cancer (Swift et al. 2011). Heavy metals, such as vanadium and arsenic, are not biodegradable, accumulate in the environment, and are hazardous to humans and wildlife (Swift et al. 2011). Based on these possible effects, a dilbit spill should not be taken lightly, which is why action occurred immediately to remediate the effects of the spill.

ExxonMobil and the Arkansas Department of Environmental Quality (ADEQ) collected daily water and air samples in the days following the spill (ADEQ 2013). Sediment samples were collected at a later date, allowing time for any remaining chemicals to settle. Samples were analyzed extensively for the presence of a variety of chemicals commonly associated with oil spills as mentioned previously, including arsenic (As), chromium (Cr), lead (Pb), vanadium (Vd), and PAHs including benzo(a)anthracene, benzo(a)pyrene, and pyrene. However, no whole effluent toxicity (WET) or sediment toxicity tests were performed to determine the potential threat to resident organisms (ADEQ 2013). Therefore, the research in this study by ASU Ecotoxicology Research Facility (ERF) included WET and sediment toxicity testing to determine if there was any measured toxicity that could possibly be linked to the spill.

Aquatic organisms used in this study include *Ceriodaphnia dubia*, *Pimephales promelas*, and *Chironomus dilutus*, exposed to water and sediment respectively. All of these organisms are regularly used in toxicity testing for many reasons. They are easily cultured in the laboratory (ASU ERF), sensitive to many different pollutants, and are generally available throughout the year (USEPA 2002). The fact that these organisms are susceptible to a variety of pollutants makes them very suitable for use in toxicity testing.

All of these chemicals are to some extent toxic to aquatic organisms. Benzo(a)anthracene is the most toxic of the three PAHs with a lethal concentration at 50 percent (LC₅₀) of 10 µg/L when exposed to *Daphnia pulex* (a standard aquatic test organism) for four days (USEPA, 2014). Pyrene, is the next toxic of the three PAHs with an LC₅₀ of 135.8 µg/L and an effective concentration at 50 percent (EC₅₀) for growth of 72.7 µg/L when exposed to *Daphnia magna* (USEPA 2014). While still toxic, benzo(a)pyrene has the least toxicity of the three PAHs with a LC₅₀ of 250 µg/L when exposed to *D. pulex*.

While PAHs are more toxic overall to aquatic test organisms compared to the other chemicals, the metals that were analyzed in this study are also harmful to aquatic organisms at high concentrations. Toxic effects of metals vary between species such as *D. magna* and *Hyallela azteca* (aquatic sediment organism). The range of toxicity of the metals when *D. magna* were exposed to them for a 48-h acute test are as follows (greatest to lowest toxicity): Cr (22 µg/L), Vd (1550 µg/L), As (3800 µg/L) and Pb (4400 µg/L). The ranges of toxicity for the metals when they are exposed to *H. azteca* for a 7-d acute test are somewhat different: Pb (20 µg/L), As (426 µg/L), Vd (1251 µg/L) and Cr (>3150 µg/L) (USEPA 2014). Due to the potential toxicity of these chemicals, extensive remediation should take place after spills of this nature occur.

The purpose of this project was to perform bioassays on *Pimephales promelas*, *Ceriodaphnia dubia*, and *Chironomus dilutus* to determine if there was any measurable toxicity in the areas closest to location of the spill. Bioassays were performed using water and sediment samples from six sites in and around Lake Conway. Aquatic and sediment toxicity testing utilizes surrogate organisms with known toxic endpoints to assess the impact of the oil spill on the surrounding environment. This process will determine if the remediation protocols enacted were appropriate and/or sufficient to minimize environmental impacts.

If toxic chemicals are measured in water or sediment then aquatic organisms are also predicted to have greater mortality, slower growth, and decreased reproductive output. Therefore, the hypothesis of this project is that the areas proximal to the location of the oil spill will have greater measured toxicity than areas farther away. The results of the toxicity tests were compared to the results of the chemical analyses performed by ADEQ and ExxonMobil at sites in close geographical proximity to those sampled for toxicity testing.

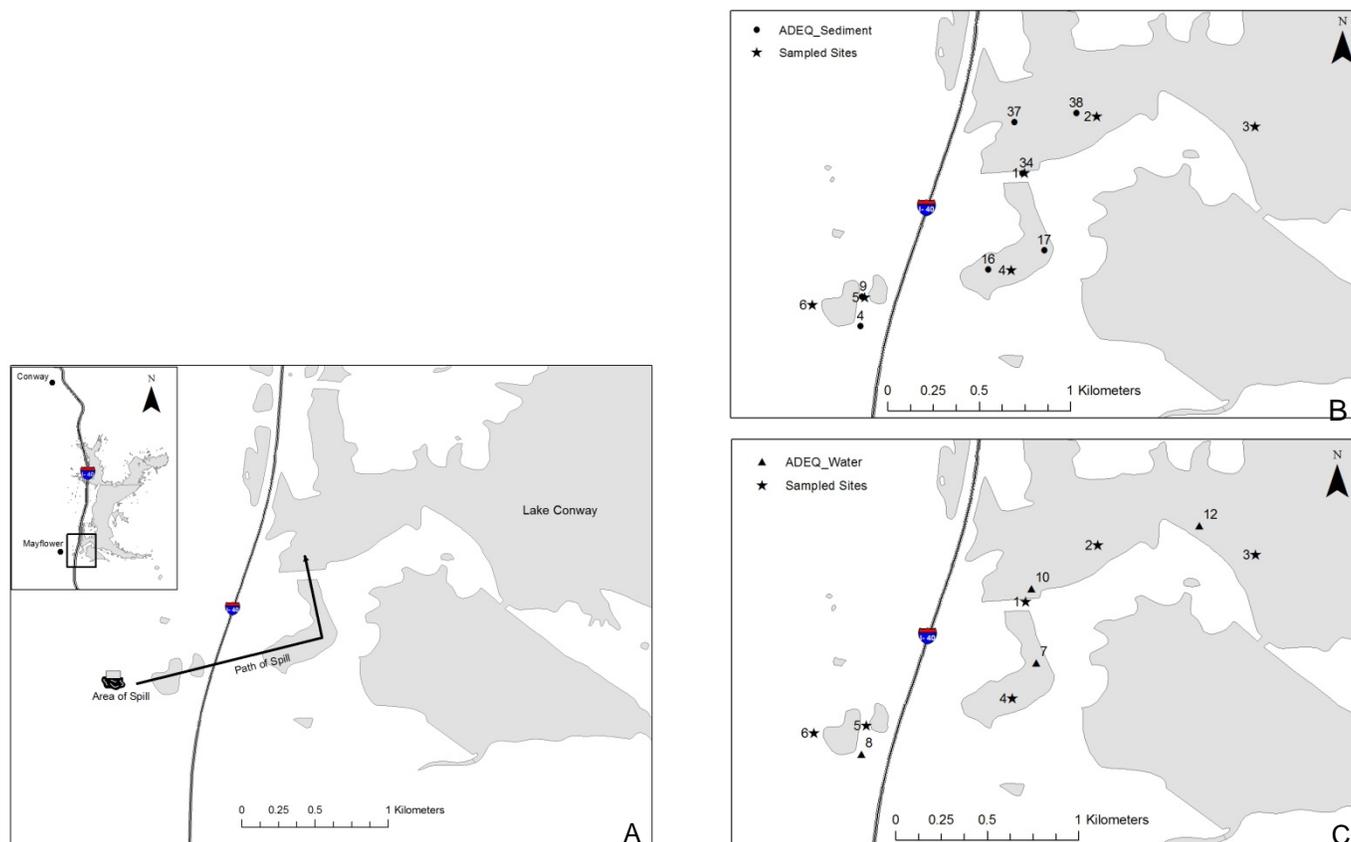


Figure 1. (A) Depicts area of spill in Mayflower, AR including location and pathway of spill from housing division through cove of Lake Conway. (B) Map depicting sites sampled by ASU with corresponding ADEQ sediment sampling sites. (C) Map depicting sites sampled by ASU with corresponding ADEQ water sampling sites.

Methods and Materials

Water and sediment samples were collected from 6 sites near the affected area, as well as in Lake Conway corresponding to sites sampled by ExxonMobil and Arcadis. ASU sampled for water and sediment on June 7, 2013 and again on September 11, 2013. Sites 1, 2, and 3 were located in Lake Conway and were accessed by boat: Site 1 was inside one of the barrier booms (also the location of water entry from the cove), Site 2 was in the main channel, and Site 3 was outside of the main channel of the lake and served as the lake control site. The other sites were located out of the main body of the lake: Site 4 was located in the cove of Lake Conway (the area where water, sediment and vegetation were removed for remediation), Site 5 was a ditch collecting water from the neighborhood of the oil spill and lastly, Site 6 was located in a drainage ditch immediately upstream from the affected cove (Figure 1).

The results obtained from the WET and sediment toxicity testing were compared to that of analytical testing done on water and sediment samples by

ExxonMobil and Arcadis (ADEQ 2013). Water samples were collected approximately every day for an extended period of time following the oil spill by ExxonMobil and Arcadis. Therefore, the first sampling date for this study (6/7/2013) can accurately be compared to that of the work done by these organizations. However, there were no samples collected by the agencies on the second sampling date so the closest date was used for comparison. The sediment sampling done by ExxonMobil and Arcadis was not performed until July and August following the spill, serving as the only data to compare with the sediment bioassay results in this study. The sites that were sampled in this study were correlated as closely as possible to the sites sampled by ExxonMobil and Arcadis in order to best compare the data measured for each study. The data provided by ExxonMobil and Arcadis was compared to the data measured in this study by relating the amount of certain chemicals in the water and sediment to that of toxicity measured in the aquatic organisms.

Several chemicals were tested by the agencies;

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however, those that do not occur naturally in water and sediment, such as the amount of TPHs and PAHs were specifically chosen to correlate the two research studies. The chemicals that were chosen to compare to the data measured in this study are listed in Tables 2 and 3.

Water samples for our study were collected from the water column and stored in 10-L containers (USEPA 2002). The sediment samples were taken from the top 2-3 centimeters at each site and were stored in plastic Ziploc bags (USEPA 2000). The samples were put on ice and taken back to the ERF at ASU for WET and sediment toxicity testing.

Chronic (7-day) WET testing followed the United States Environmental Protection Agency (USEPA 2002) protocol. Synthetic, moderately-hard water prepared in the ERF (according to EPA standards) was used as the control for each test. WET tests were conducted with *P. promelas* (measuring survival and growth) and *C. dubia* (survival and reproduction). Following the USEPA protocol, 5 replications of 8 fish (40 fish per beaker) and 10 replications of 1 *C. dubia* were used for each WET test (USEPA 2002). Acute sediment toxicity testing was conducted using *C. dilutes* (survival and growth), also following the USEPA method (USEPA 2000). Acute testing consists of using 6 replicates of 10 chironomids, following USEPA protocol (2000). Black River sediment was used as the control, as it has been determined to be suitable for use in reference sediment toxicity testing for the Arkansas Delta ecoregion (Moore et al. 1996).

The results from all bioassays were analyzed using ToxCalc Version 5.0 (Dunnett's ANOVA, $\alpha=0.05$). The results from the water and sediment bioassays were then compared to that of ExxonMobil and Arcadis' analytical data for aqueous and sediment samples most closely corresponding with sampling sites in this study (Figure 1, Table 4).

Results

Neither survival nor growths were significantly different from controls in *P. promelas* for either sampling date at any sampling sites. However, a significant decrease in *C. dubia* reproduction was measured in water collected in June at the cove site (Site 4). Additionally, significant decreases in *C. dilutes* growth were measured in sediment collected at Site 2 and 4 (June collection) and site 2 and 5 (September collection) (Table 1). ASU did not measure the chemical composition of collected water and sediment samples, however, the toxicity measurements obtained through WET testing was compared to analytical measurements done by ExxonMobil and Arcadis. Therefore, Table 1 includes the toxicity measurements from bioassays performed by ASU, while Tables 2 and 3 include the chemical composition data for sediment and water published by ADEQ that is comparable to the toxicity measurements performed in this study for the corresponding sites.

Table 1. Results from aqueous and sediment toxicity tests; *C. dubia* and *P. promelas* were exposed to water samples while *C. dilutes* were exposed to sediment both collected in June and September 2013. Endpoints include: % survival and reproduction (+/-SD) for *C. dubia* and % survival and growth (+/-SD) for *P. promelas* and *C. dilutes*. *=indicates significant difference from the control at $\alpha=0.05$.

ToxCalc Results from Aquatic Organisms Exposed to Mayflower Water/Sediment						
Sampling Sites	<i>C. dubia</i> (June 2013)		<i>P. promelas</i> (June 2013)		<i>C. dilutes</i> (July 2013)	
	Survival (%)	Reproduction	Survival (%)	Growth (mg)	Survival (%)	Growth (mg)
Site 1	100±0.0	33.7±3.97	98±0.06	0.20±0.01	70±0.25	2.88±1.01
Site 2	100±0.0	32.3±3.47	98±0.06	0.21±0.02	95±0.09	2.18±0.20*
Site 3	100±0.0	30.6±1.58	100±0.0	0.22±0.01	87±0.14	2.10±0.31
Site 4	100±0.0	22.4±4.74*	100±0.0	0.18±0.02	78±0.23	1.89±0.49*
Site 5	100±0.0	29±4.24	98±0.06	0.21±0.01	80±0.15	2.18±0.24
Site 6	100±0.0	29±3.18	93±0.10	0.21±0.02	86±0.14	3.07±0.76

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Table 1 cont'd

Sampling Sites	<i>C. dubia</i> (Sept. 2013)		<i>P. promelas</i> (Sept. 2013)		<i>C. dilutus</i> (Sept. 2013)	
	Survival (%)	Reproduction	Survival (%)	Growth (mg)	Survival (%)	Growth (mg)
Site 1	80±0.19	25.6±8.80	98±0.06	0.41±0.05	80±0.16	2.82±0.5
Site 2	100±0.0	24.1±8.31	95±0.07	0.40±0.02	95±0.12	2.25±0.38*
Site 3	100±0.0	25.5±4.95	100±0.0	0.38±0.02	78±0.22	2.69±0.48
Site 4	90±0.15	24.9±5.95	98±0.06	0.37±0.01	87±0.16	2.97±0.40
Site 5	no water	no water	no water	no water	58±0.28	2.04±0.34*
Site 6	100±0.0	16.1±3.14	95±0.07	0.38±0.05	57±0.30	3.13±0.48

Table 2. Selected chemicals in water samples collected by ExxonMobil/Arcadis (Figure 1C). N.D. = chemical was not detected; J = compound was positively identified, however, the associated numerical value is an estimated concentration only.

ExxonMobil, Arcadis Mayflower/Lake Conway Water Sampling Results						
Sites	7		8		10	
Date	6/7/2013	9/24/2013	6/7/2013	9/24/2013	9/24/2013	9/24/2013
Depth (m)	0.15-0.31	0.15-0.31	Surface	0.46-0.61	0.46-0.61	0.46-0.61
Acetone (µg/l)	4.3 ^J	7.4	3.3 ^J	3.5 ^J	N.D.	N.D.
Benzo(a)anthracene (µg/l)	0.22	N.D.	N.D.	N.D.	N.D.	N.D.
Benzo(a)pyrene (µg/l)	0.22	N.D.	N.D.	N.D.	N.D.	N.D.
Pyrene (µg/l)	0.65	N.D.	0.011 ^J	N.D.	N.D.	N.D.
Arsenic (mg/l)	0.0108 ^J	N.D.	N.D.	N.D.	N.D.	N.D.
Chromium (mg/l)	0.0167	N.D.	0.0024 ^J	N.D.	N.D.	N.D.
Lead (mg/l)	0.0306	N.D.	N.D.	N.D.	N.D.	N.D.
Vanadium (mg/l)	0.0243	N.D.	0.0024 ^J	N.D.	N.D.	N.D.

Table 3. Selected chemicals in sediment samples of Lake Conway/Mayflower collected by ExxonMobil & Arcadis (Figure 1A) correspond to those in this study; dates above are the only available for sediment samples. *Site 16, 17 (Lake Conway cove) and 37, 38 (Lake Conway) were averaged since each were close to site 4 and 2 respectively. All were collected at 0-0.15 m. J = compound was positively identified; however, the associated numerical value is an estimated concentration only. TPH = Total Petroleum Hydrocarbons.

ExxonMobil & Arcadis Lake Conway/Mayflower Sediment Samples						
Sites (Figure 1)	4		9		16, 17	
Dates	8/15/2013	7/31/2013	8/2/2013	8/5/2013	7/27/2013	7/28/2013
Acetone (µg/kg)	62 ^J	33 ^J	23	40.5	78	120
Benzo(a)anthracene(µg/kg)	29.8	9.46	0.33	8.7	17	34.3
Benzo(a)pyrene (µg/kg)	47.9	10.9	0.14	29	14.4	30.9
Pyrene (µg/kg)	131	0.745	24.5	51.9	33.6	68.5
Arsenic (mg/kg)	11.5 ^J	3.6	4.6	4.5	5.8	8.8
Chromium (mg/kg)	21.2 ^J	18.0 ^J	13.3	17.55	28.4	30.5 ^J
Lead (mg/kg)	18.7	18	12	19	40.8	38.8 ^J
Vanadium (mg/kg)	23.1 ^J	23.4	21.6	28.1	46.7	48.8
TPH (mg/kg)	2277	300	51	995.5	558	689

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Discussion

The toxicity that was measured in this study for sediment showed a decrease in growth for *C. dilutus* in sites 2 and 4 in June and sites 2 and 5 in September, while toxicity was measured in the water from site 4 in June with a decrease in reproduction for *C. dubia* (Table 1). Site 2 was located inside the body of Lake Conway where there was reportedly no oil contamination. However, sites 4 and 5 were closer to the location of the spill. Site 4 was located in the cove of Lake Conway which was contaminated by the spill. Site 5 was close to the housing division in which the pipeline burst, located in a drainage ditch under railroad tracks.

All of the chosen chemicals tested by ExxonMobil and Arcadis were detected for each of the corresponding sites measured for toxicity in this study (Table 2). For example, the levels of TPH in sediment were the greatest for the sites sampled in this study in which toxicity was measured. While lower than the TPH levels, the level of the three different types of PAHs in these three sites sediment sites were also the greatest. According to the results compiled by ADEQ, the levels of PAHs and TPHs from the spill were great enough to cause the toxicity measured at these sites in our study.

Summary of Water Sample Data

As previously mentioned, WET testing toxicity in this study was only measured in site 4 or the cove of Lake Conway, showing a decrease in the reproduction of *C. dubia* as compared to the control. When this was compared to the results from ExxonMobil and Arcadis for this specific date in the corresponding cove site, it can be seen that all of the selected chemicals were detected. This correlation could possibly be a reason for the toxicity measured in the aqueous samples from site 4 (Table 2). When either *D. Magna* or *D. pulex* (as discussed in above introduction) were exposed to these chemicals in toxicity testing, the endpoints were greater for each of the chemicals than the measured value by ExxonMobil and ADEQ (Table 2). For example, the toxic endpoint of *D. pulex* when exposed to benzo(a)anthracene is greater than the measured value (LC50 10 µg/L, *D. pulex*; measured value 0.22 µg/L) (Table 2). Since *D. pulex* are larger than *C. dubia* used in this study, they are more tolerant and thus the smaller, more sensitive *C. dubia* will be sensitive to levels measured at this site (Bossuyt and Janssen 2004). While the site in the cove that was sampled by ExxonMobil and Arcadis was closer to the

main body of the lake and was not in the same exact location of the site measured in this study, it can be inferred that if these chemicals were detected farther away from the point of the spill they might possibly of higher concentration closer to point of the spill, such as the location of site 4 (Figure 1).

Summary of Sediment Sample Data

The toxic results measured from the sediment were possibly due to the increased levels of TPHs and PAHs found in corresponding ADEQ sites. Toxicity was measured in the sediment from sites 2 and 4 for the June sampling date; *C. dilutus* exposed to this sediment showed a decrease in growth as compared to the control sediment used. *C. dilutus* exposed to sediment from sites 2 and 5 for the September sampling date also showed a decrease in growth. When compared to the data from ExxonMobil and Arcadis sites 37/38, 16/17, and 4 (corresponding respectively with sites 2, 4 and 5 from this study) have the greatest measured TPH levels as well as considerable PAHs such as benzo(a)anthracene, benzo(a)pyrene, and pyrene (Table 2). Therefore, there was a consistent sublethal effect detected in site 2 between the two sampling dates, as well as toxicity measured in sites 4 and 5 which were in close proximity to each other.

Even though the sampling dates were not the same as those done by ExxonMobil and Arcadis, it can be inferred that the chemicals from oil spills leach, as they remain in the sediment for extended periods especially concerning this type of crude oil. For example, approximately four million liters of heavy crude oil or dilbit leaked into the Kalamazoo River in 2010 and remnants still remain in the floodplains, riverbanks and sediment of the river (Brooks 2014). An Environmental Working Group study on the Mayflower oil spill states that chemicals from crude oil, especially dilbit can remain in sediment for at least three years as this is when it was determined the Kalamazoo River would need to be dredged (Sharp et al. 2013). Therefore the settling of heavy chemicals from the crude oil is most likely the reason for the measured sediment toxicity at those sites.

Conclusions

Toxicity was measured in organisms exposed to water and sediment contaminated by oil. There were three sites in which toxicity was measured, one was very close to the point of the spill (site 5 drainage ditch), the other in the cove of Lake Conway (as far as oil reportedly reached) and lastly inside the main body

of the lake. Water and sediment toxicity was measured in *C. dubia* (reduced reproduction) and *C. dilutus* (reduced growth), however, *P. promelas* showed neither a decrease in survival or growth. Previous research has shown that invertebrates, such as *C. dubia*, are more sensitive to contaminants than vertebrates (Bossuyt and Janssen, 2004). Also, the prediction that was made stating that the sites close to the point of the oil spill would be more likely to measure toxicity was also inconclusive. While toxicity was measured in site 4 (*C. dubia*) and sites 4 and 5 (*C. dilutus*) which were close to the point of the spill, there was also toxicity measured in site 2 (*C. dilutus*) inside the lake, perhaps due to the natural flow of the water and the accumulation of heavier constituents of the crude oil into the sediment.

Even though daily water samples were taken, sediment sampling done by ExxonMobil and Arcadis did not begin until July 27, 2013 (ADEQ 2013). The toxicity in sediment from site 2 was measured although the oil reportedly did not reach the main body of the lake (Figure 1A-B). Exxon deployed 1097 m (3600 ft) of containment (or hard) booms between point of the oil spill and Lake Conway (Duke 2013). This type of boom not only contains buoyant material that keeps it afloat and prevents oil from leaking, but also contains a skirt below the surface extending to the bottom which is designed to prevent oil from escaping underneath (NOAA Office of Response and Restoration 2015). This prevents the oil from spreading and provides easy removal. It is interesting that toxic results as well as measured constituents of the crude oil were measured at this site, indicating that the boom was not completely effective in preventing movement of contaminants into the lake. The water and sediment toxicity results can be compared and correlated between the two sampling dates in that there was no toxicity measured for each in site 4 for September. However, there is no analytical data to compare the sediment toxicity results of the September sampling date in site 5. The reduced growth of *C. dilutus* for site 5 in September indicates some residual chemical remained several months after the spill. This is of concern due to the proximity to the neighborhood and the recalcitrant nature of some of the chemicals present in the crude oil.

The analysis reported in this experiment on the effects of the Mayflower oil spill highlight the importance of preventing similar occurrences from happening. Oil spills, especially those occurring from underground pipelines carrying heavy crude oil can cause extensive damage. The Mayflower oil spill

leaked a significant portion of heavy crude into the surrounding neighborhood, reaching the cove of Lake Conway. This not only affected the individuals living in the neighborhood, but also disturbed the water running into storm drains and ditches, ultimately leading to the cove of Lake Conway. The type of oil in the Pegasus pipeline in Mayflower contained many contaminants that are toxic to aquatic organisms. Appropriate maintenance of the pipeline infrastructure could have protected the individuals living close to the pipelines as well as Lake Conway. Maintenance of pipelines can prevent leaks especially if the pipelines are going to be carrying heavy crude, such as dilbit, which causes pressure swings, damaging old pipelines. Although Exxon Mobile was mostly effective in cleaning up the initial spill and the containment booms prevented most of the oil from reaching the main body of the lake, toxic effects were visible. The toxicity measured in aqueous organisms inside the lake at site 2 confirmed some contaminant movement beyond the booms. Continual sampling of the affected area, including the use of bioassays would increase the understanding of post-spill effects on the environment.

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