Prymnesium parvum toxic blooms: Allelopathy, predation and aquatic population conservation
Abstract

Golden alga, *Prymnesium parvum*, has become a rising concern in terms of conservation. This is due to the toxic blooms they create that kill populations of fish and phytoplankton. *P. parvum* produce prymnesin toxins, which cause the blooms to be so dangerous. Previous research has claimed that prymnesins lyse cells and toxify other cells, neurons and fish. Specifically, the toxins have been shown to cause ion leakage due to the damage done to cell membranes. Further investigation has provided understanding into the correlation *P. parvum* toxin has with the acclimation of inorganic phosphorus and nitrogen at the genomic level. Of the 23 genes already identified in *P. parvum*, three genes associated with phosphorus transport indicate phosphorus deficiencies. Nutrient limitations have aided in the characterization of compounds within *P. parvum* toxins. Specifically, seven fatty acids in addition to a hydroxamic acid have been identified among all the toxic compounds that are ichthyotoxic and cytotoxic. Further research has delved deeper into the types of prymnesin toxins. Specifically, polyketides prymnesin-1 and prymnesin-2 were identified via liquid chromatography and mass spectrometry. *P. parvum* toxins have been identified to play a role in micro-predation that is different from allelopathy. Factors that alter the levels of toxicity in *P. parvum* have been under study as well. Sunlight has proven to reduce the levels of toxicity in cells with increased exposure in terms of time and magnitude. This leads to the idea that fish population survival depends on where the toxic blooms occur. Fish populations in the Colorado River and the Brazos River in Texas have shown evidence of fish population decline due to toxic *P. parvum* blooms. However, the location of the river correlates with aquatic life survival. Specific conservation acts have used ammonium successfully to reduce toxic *P. parvum* blooms. Others have attempted to add inorganic nitrogen and phosphorus plus cornseed meal to the blooms, but this attempt caused the increase in pH levels. Thus far, research has made great strides in understanding *P. parvum* and prymnesins at
the genomic, molecular, and organism level, which in turn has aided in understanding of how to control the toxic blooms.

**Introduction**

*Prymnesium parvum* is an alga species belonging to the division Haptophyta, which is in the class Prymnesiophyceae (Graneli et al. 260). This alga is unicellular and is primarily immobile (Graneli et al. 261). *P. parvum* has two flagella used for movement and a haptonema used to attach to surfaces (Graneli et al. 261). The haptonema has also been thought to secrete chemicals (“Prymnesins: Toxic” 679). *P. parvum* live all over the world with the exception of Antarctica (Graneli et al. 260). They are mixotrophic and release chemicals into the surrounding waters, which produces a slew of toxic events (Graneli et al. 260) Internal concentrations of inorganic nutrients nitrogen, phosphorus, and carbon vary depending on if the surrounding environment is nutrient replete or deficient (Graneli et al. 261). It is typically observed that under nutrient deficient conditions, toxic algal blooms that appear golden and result in massive fish and gilled-organism mortalities occur (Graneli et al. 260). These toxic blooms are caused by chemicals that are “hemolytic to other organisms” and “inhibit growth or kill competing phytoplankton species” (Graneli et al. 261). Harmful algal blooms occur most frequently in “cooler waters located in the subtropical and temperate zones”, although events have occurred in “mainland fresh water reservoirs” (Manning and Claire 679). The blooms generally occur 5 m from the surface (VanLandeghem 582) and an average toxic bloom produced by *P. parvum* is about 1000000 cells/mL water (Remmel and Hambright 126). In addition to organism mortalities, these blooms are creating eutrophication and excessive demands on the supply of freshwater (VanLandeghem 582).
In this review, new information obtained within the last five years has been reported in addition to embellishment from a previous similar review published in 2011. The previous review talked specifically about the prymnesin toxins released by *P. parvum*. This review adds new, relevant information regarding the alga toxins as well as ways to mediate toxic *Prymnesium parvum* bloom events.

Discussion

**PREVIOUS REVIEW ON PRYMNESIN TOXINS**

A complete set of the prymnesin toxins contributing to harmful toxic blooms has yet to be established due to the variety of techniques used to extract the metabolites (“Prymnesins: Toxic” 681). As of 2010, 15 active metabolites have been extracted containing “glycolipids, galactolipids, proteolipids and lipid-carbohydrate compounds” (“Prymnesins: Toxic” 681). Prymnesin-1 and prymnesin-2 have been previously identified as metabolites of the toxic blooms that cause ichthyotoxicity and hemolytic behavior (“Prymnesins: Toxic” 682). Additionally, it is known that polyketides have similar synthesis processes as fatty acids (“Prymnesins: Toxic” 683). However, the “structural elucidation” of these compounds (“Prymnesins: Toxic” 682) and mechanisms of prymnesin toxicity have yet to be reported (“Prymnesins: Toxic” 684).

Some modes of prymnesin toxicity were identified as of 2010. These include those with “saponin-like properties” such as “cytotoxicity, neurotoxicity, hemolysis, and ichthyotoxicity” (“Prymnesins: Toxic” 686). Cytotoxicity is known to produce cell swelling and cell lysis (“Prymnesins: Toxic” 686). The process of neurotoxicity has been reported with actions at the myoneural junction (“Prymnesins: Toxic” 686). Specifically, prymnesins act on the processes of calcium influx and neurotransmitter release (“Prymnesins: Toxic” 686). Most studies have
shown effects on gill-breathing organisms, or ichthyotoxicity and well as hemolysis on mammalian cells ("Prymnesins: Toxic" 687).

Outside factors that have been established or have been initially investigated in terms of prymnesin toxin presence and Prymnesin cell growth include “nutrient availability, salinity, light, temperature, [and] pH” ("Prymnesins: Toxic" 687). Specifically, harmful toxic blooms are known to increase in toxicity with phosphorus limitation and carbon addition ("Prymnesins: Toxic" 688). Salinity has been highly studied; results have shown that increased levels of salt increases toxicity levels ("Prymnesins: Toxic" 689). Information regarding light and temperature on prymnesins has been inconsistent since 2010 ("Prymnesins: Toxic" 690). pH has been frequently studied; results have shown that certain pH ranges effect the levels of prymnesin toxicity, with ichthyotoxicity optimal at pH 7.0, but maxing at pH 9.0 ("Prymnesins: Toxic" 690). Hemolysis, on the other hand, occurs optimally at pH 5.0 with toxic effects decreasing with alkalinity ("Prymnesins: Toxic" 690).

As of 2010, prymnesins have been known to be exotoxins, but how they enter the environment is unknown ("Prymnesins: Toxic" 692). Prymnesin toxins are also known to deter grazers and effect other “algae and certain bacteria” ("Prymnesins: Toxic" 692). Other evidence suggests that prymnesins are allelochemicals, but this is not defendant ("Prymnesins: Toxic" 693).

TRANSCRIPTOME EVIDENCE

New transcriptome activities have been detected as of 2012. Beszeteri et al compared P. parvum sequences to other haptophyta species’ libraries, isochrysis glbana, Chrysochromulina polylepis, Pavlova lutheri, Emiliania huxleyi (6), and used microarray to determine active transcripts associated with nutrient-dependent release of prymnesin toxins (11). They found 1526
genes in common, 1695 functions only present “in *P. parvum* and *E. huxleyi*” and seven orthologies shared among the toxic species, *P. parvum* and *C. polylepis* (Beszeteri et al. 6). Additionally, Beszeteri et al. found that many functions encoded in genes are designed for defense mechanisms (6). 14 genes are “involved in the uptake, transport and storage of nitrogen” with six functioning for phosphorus (7). Growth rates were measured during two exponential time points and in stationary phase when nutrients were plentiful, when nitrogen was depleted, and when phosphorus was depleted. The resulting concentration of *P. parvum* cells was greatest with cells in nutrient replete conditions and least with nitrogen-depleted cells (Beszeteri et al. 8). The nitrogen-deprived cells also had the lowest pH at 8.2 (Beszeteri et al 8). This suggests the likelihood of increased ichthyotoxicity potential. In regards to extracellular toxicity, it decreased with increased densities of cells (Beszeteri et al. 9). On the other hand, Beszeteri et al. showed that intracellular toxicity dramatically increased when phosphorus was limited (9). Their research indicates that three known phosphate transporters are upregulated along with “phosphate-repressible phosphate permease” within phosphorous depleted cells (Beszeteri et al. 11). Interestingly, “genes encoded by the arsenic detoxification operon in bacteria” were identified and upregulated with phosphorus depletion in *P. parvum* cells indicating that gene expression is altered when phosphorus is in low supply (Beszeteri et al. 12). On the other hand, nitrogen depleted cells did not show a significant difference in allelopathy compared with nitrogen rich cells. Beszeteri et al. found a significant decrease in “expression of cytochrome and light-harvesting related genes”, however, they believe this could merely be due to the necessity for nitrogen in the creation of chlorophyll and other proteins during photosynthesis (12). Evidence of increased prey consumption was observed when inorganic nutrients were limited (Beszeteri et al. 12). This has led to the prediction that some unknown genes identified correlate with
heterotrophic nutrition by *P. parvum* (Beszeteri et al. 13). Various genes were expressed at different times in *P. parvum*’s growth indicating that the type of environment effects *P. parvum* at the genome level with higher toxicity and gene activity in nutrient-depleted environments, especially those where phosphorus is limited.

**TOXIC FATTY ACID AMIDES**

As of 2012 as well, a new toxic compound has been identified from *P. parvum*. Bertin et al. chemically identified the toxins made by *P. parvum* when the alga was under nitrogen and phosphorus limited environments collected both in the lab and from a harmful alga bloom in Lake Wichita, TX (112) as well as in an “Iowa koi fish pond” (114). The identified toxins were subject to mammalian cells to determine cytotoxicity. Cells were obtained in “late exponential growth phase”, compounds were extracted and identified using HPLC-MS, and “two- or three-day-old red drum larvae fish were used to assess the toxicity on the fish mortality (Bertin et al. 112). 400 ppm of the fractions killed all fish after four hours, 100 ppm killed all fish after six hours, and 20 ppm was not toxic after 24 hours (Bertin et al. 113). Bertin et al. identified oleamide, elaidamide, linoleamide, myristamide, palmitamide, erucamide, stearamide, and linoleyl hydroxamic acid as compounds resonating in the toxin fractions (113). Each ion fragment had the characteristic of primary fatty acid amides at m/z 59 (Bertin et al. 114). Additionally, retention times and mass spectral patterns matched standards (Bertin et al. 114). Oleamide has been previously identified as a sleep-inducer by inactivating gap junctions (Bertin et al. 115). Elaidamide is an enzyme inhibitor and linoleamide induces sleep by affecting calcium channels and potassium currents (Bertin et al. 115). Linoleyl hydroxamic acid inhibits metabolic enzymes necessary for fatty acid utilization (Bertin et al. 115). Neither known prymnesins, prymnesin-1 nor prymnesin-2, nor unsaturated fatty acids were identified (Bertin et al. 114).
Each compound was toxic to mammalian cells alone and toxic in combination to the red drum fish (Bertin et al. 114). Bertin et al. mentioned that divalent metals increase *P. parvum* toxicity, as does high mineral content (Bertin et al. 115). This has been the first study to identify fatty acids as a toxic compound of *P. parvum* (Bertin et al. 114). Further investigation is needed to understand more about the lethality *P. parvum* creates in harmful algal blooms.

**POLYKETIDES**

As of 2013, previously identified polyketides, prymnesin-1 and prymnesin-2, were again detected and metabolically fingerprinted. Results regarding the quantification of prymnesin toxicity has been previously difficult to measure due to the lack of available standards and quantification methods (“Isolation of polyketides” 189). Manning and Claire attempted to ameliorate this issue. They extracted prymnesin-1 and prymnesin-2 via whole-cell and solid-phase extraction methods and subsequently used TLC and LC/MS for detection and identification of them, respectively (“Isolation of polyketides” 190). LC/MS identified prymnesin-1 and prymnesin-2 which has retention times of 68 and 71 min, respectively (“Isolation of polyketides” 192). Prymnesin-2 was slightly less polar than prymnesin-1, which had “two additional sugar residues” (“Isolation of polyketides” 192). Separation of the crude extracts revealed “doubly and triply charged molecules” (“Isolation of polyketides” 193). Polyketide prymnesins presented as protonated ions (“Isolation of polyketides” 193) could explain why prymnesin-1 and prymnesin-2 have not been identified following their first detection (“Isolation of polyketides” 193). Specifically, eight ions were “assigned for prym-1 and prym-2, including their homology form prym aglycone” (“Isolation of polyketides” 193). Purification via SPE revealed 1132.97 m/z represented the intact form of prym-1 (“Isolation of polyketides” 193). This purification was necessary to determine the absolute structure of
prymnesin-2 (“Isolation of polyketides” 193). The most common anylate found in *P. parvum* was the “protonated aglycone form of polyketide prymnesins…which had a similar backbone structure” as prymnesin-1 and prymnesin-2 (“Isolation of polyketides” 194). In order for standards to be made for these compounds, Manning and Claire found that about 25 mg of prym-1 and prym-2 can be isolated from 60 µg of prymnesin polyketides in culture and/or 30 ml of water (“Isolation of polyketides” 194). Different from previous work by Igarshi et al. who identified prymnesin-1 and prymnesin-2 initially, all ions detected were doubly charged (“Isolation of polyketides” 194). In agreement with Igarshi et al., however, the ratio of prym-1 to prym-2 is 2:3 (“Isolation of polyketides” 194).

**CONTACT TOXICITY**

As of 2012, a micropredation contact toxicity mode has been identified. According to Remmel and Hambright, in a typical *P. parvum* bloom, the nearest neighbor is about 200 µm away and an organism is not going to remain still next to the cell releasing toxins (126). Therefore, the only selective advantage of releasing toxins is if *P. parvum* keeps contact or proximity with another organism (Remmel and Hambright 126). Remmel and Hambright showed that *P. parvum* use toxins via contact rather than previously reported studies that claim the toxins are exotoxins (Remmel and Hambright 126). They believe the exotoxins were merely due to stress caused by handling, abnormal high *P. parvum* densities in lab, or from old cultures (Remmel and Hambright 126). In their study, Remmel and Hambright used “10- to 14-day-old fathead minnow” fish obtained from the Colorado River in Texas and exposed them to live *Prymnesium* cells as well as *Prymnesium* cell-free filtrates (Remmel and Hambright 127). Experimental conditions used nutrient-replete cultures, fluorescent lighting and pH of 8-8.5 (Remmel and Hambright 130). They inserted a membrane with a pore size of 3µm, which
separated the fish and the cells only; the fish and the free toxins were not separated (Remmel and Hambright 127). Results revealed that fish death was due to the contact of the fish and the toxins on the same side of the membrane (Remmel and Hambright 129). Additionally toxins were able to pass through the membrane, which led to death of fish on both sides of the membrane (Remmel and Hambright 129). Different filtration processes were used, which caused different concentrations of toxins to be available for fish contact. This filtration resulted in varying amounts of fish mortalities, with higher concentrations of toxins killing more fish (Remmel and Hambright 130). Specifically, gentle vacuum filtering reduced toxicity to half of normal cell toxicity and gravity filtration completely removed the toxins (Remmel and Hambright 130). Remmel and Hambright agree that “Prymnesium toxicity is cellular based” (Remmel and Hambright 130). They also admit that further research is needed to verify their general results (Remmel and Hambright 130).

Remmel and Hambright claim an alternative explanation for fish kills, with support from their study, by saying that the gills are the area of susceptibility because of the large surface area available for exposure by Prymnesium cells (Remmel and Hambright 130). In terms of the Prymnesium view, the affected organism is a source of nutrition (Remmel and Hambright 130). Various species in the genus Prymnesium have various routes of prey capture suggesting that direct toxin contact may be a method used by P. parvum alone. However, whether this is a way of predation or not is still unknown (Remmel and Hambright 130). Further knowledge must be obtained to provide insight into understanding if the toxins have direct or indirect selective advantages for the algae.
EFFECTS OF SUNLIGHT ON TOXICITY

In 2011, the effects sunlight had on the “magnitude and duration of aquatic toxicity” was not well-known (James et al. 265). Previous studies have reported increased growth rates of \textit{P. parvum}, but there was no change in the magnitude of toxicity (James et al. 266). It has also been reported that toxin production is not light dependent, but extended fluorescent light exposure either reduces or inactivates toxins (James et al. 266). However, other previous studies have suggested that “light intensity may influence the stability and fate of toxins released by \textit{P. parvum}” (James et al. 266). Until this particular study in 2011, only one published study was reported that looked at which factors affect the environment where toxins have already been released (James et al. 266). The other study did not look at the effects of natural light (James et al. 266). Therefore, this study did.

James et al. exposed fish from “Texas inland waters” to conditions similar to those where blooms occur (266); James et al. used fish mortality as a representation of the effects of sunlight on the lytic behavior of \textit{P. parvum} toxins (James et al. 266). Specifically, they exposed fish to varying levels of sunlight intensity or to sunlight for varying durations (James et al. 266). \textit{P. parvum} were exposed to full light, partial light, and no sunlight to test magnitude effects and to either full or no sunlight for varying lengths of time to test durational-exposure effects (James et al. 266). Additionally, they used nutrient-limited conditions because it has been known that toxicity is greatest during these conditions (James et al. 267). Results revealed that toxicity was greatest with no light compared to no fish toxicity under “full or partial sunlight for 8 hours” (James et al. 269). Similar to the latter, fish toxicity was “completely ameliorated after just 2 hours of exposure to full sunlight” (James et al. 269). These results suggest that “magnitude and duration of \textit{P. parvum}” can be greatly reduced, if not eliminated, due to “photodegradation”
James et al. claim that different chemical bonding and structures can cause compounds to be more susceptible to photodegradation” depending on the corresponding “light energy” coming in (James et al. 270). Indirect photolysis can also occur if “photons are absorbed by dissolved organic matter” which “facilitate degradation” of the toxins (James et al. 270). It is important to take note of the half-life of photolysis (James et al. 270) to understand more about the degradation process. This study shows that the photons are causing the reduced acute toxicity rather and not other factors such as temperature (James et al. 270). It is also important to note that photodegradation can be effected by the site and by depth of water (James et al. 271). This information can be useful when attempting to manage the toxic P. parvum blooms within inland waters (James et al. 271).

**P. parvum IN THE COLORADO RIVER AND BRAZOS RIVER BASINS**

In Texas, *P. parvum* has affected “state-run fish hatcheries, private ponds, numerous river stretches, and large reservoirs” with an overall mortality of 34 million fish and huge economic losses to adjacent communities (VanLandeghem et al. 582). Prior to the study by VanLandeghem et al., quantitative information regarding the impacts of blooms on fish populations, fish population’s recovery ability, and *P. parvum* effects among different basins has been limited (582). This study in 2013 found that there are two common disturbance patterns of harmful blooms on fish populations: pulsed and sustained/press (VanLandeghem et al. 582). Pulsed patterns are observed when an immediate disturbance to populations occur after a bloom event, but populations recovery is rapid (VanLandeghem et al. 582). Sustained patterns result in unrecoverable fish populations (VanLandeghem et al. 583). Ichthyotoxic blooms, in general, can be detrimental to fish populations long-term, but the varying deleterious effects differs
depending on location of the river basin as well as the species of fish encountering the blooms (VanLandeghem et al. 587).

In Brazos reservoir, fish populations do not experience a long-term decline in number, but in the Colorado, fish abundance and population size structure endured a sustained decline (VanLandeghem et al. 587). Long-term effects may be due to repeated exposure of blooms to fish populations (VanLandeghem et al. 589). Patterns of P. parvum toxin effects vary with the fish populations due to a few possible reasons. One, fish that can move and have high fecundity may have the ability to recover faster from the blooms (VanLandeghem et al. 589). Specifically, in the Colorado, low water levels may prevent fish from dispersing, therefore, causing them to rely on reproductive success for survival (VanLandeghem et al. 589). Unfortunately, blooms might occur during fish spawning, which would alter their ability to recover (VanLandeghem et al. 589). Refuge may be obtainable, however, by fish residing in the deep waters prior to spawning (VanLandeghem et al. 589). The Brazos River can be a better place for this than the Colorado River can (VanLandeghem et al. 589).

Stocking the Colorado with fish such as the Catfish does not prevent the long-term decline of fish populations (VanLandeghem et al. 589). Some Brazos reservoirs, Whitney and Granbury, have sustained fish populations without stocking even through repeated toxic bloom events (VanLandeghem et al. 590). Among the fish populations in the Colorado that have declined, the Largemouth Bass population has declined the least, with more smaller fish remaining (VanLandeghem et al. 591). Explanation for this could be that fish that consume these fish have been killed by the blooms (VanLandeghem et al. 591). In the Brazos, only Blue Catfish “exhibited a sustained decline” (VanLandeghem et al. 591). In the Colorado, White Crappies populations increased, however, explanations as to why vary (VanLandeghem et al. 592).
Regardless of the possible reasons, White Crappie may be a species to keep in mind when managing reservoirs by replacing a weaker fish population with them, closer to where blooms occur due to their possible resistance (VanLandeghem et al. 592). The most commonly reported fish kills, the Dead Gizzard, has not shown long-term effects of the blooms (VanLandeghem et al. 592). This suggests that “ecological and physiological characteristics” aid in the ability of populations to recover (VanLandeghem et al. 592). These varying effects can be attributed, in part, to hydrology between various rivers (VanLandeghem et al. 592). Overall, fish populations’ ability to recover from blooms and improving water quality are aspects that should be considered in terms of managing the blooms (VanLandeghem et al. 593).

**AMMONIUM EFFECTS ON SUPPRESSING TOXIC BLOOMS**

Mesocosm experiments in Lake Granbury, part of the Brazos River, in 2013, tested the effects of ammonium in the pre-bloom and in the bloom decline of *P. parvum* (Grover et al. 4275). In both stages, addition of high concentrations of ammonium can “reduce the abundance and toxicity of *P. parvum*” (Grover et al. 4282). Specifically, concentrations must exceed natural levels (Grover et al. 4282). Higher doses appeared to be dependent of temperature and pH, suggesting that ammonium is deprotonated to make ammonia, which “is toxic to *P. parvum*” (Grover et al. 4282). Specifically, ammonia appears to create similar effects as *P. parvum* toxins do including induction of “osmotic stress, swelling, and cell lysis” (Grover et al. 4282). Ammonia levels must remain in the 8-15µM range to prevent the ammonia from killing the fish (Grover et al. 4282). Caution must be taken to prevent negatively affecting other organism populations. Grover et al. found that ammonium addition acted as a fertilizer and mostly affected the phytoplankton community (4282). Additionally, ammonium addition to enclosures would be
more beneficial for future bloom control due to the mixing of waters and sediment uptake in coves (Grover et al. 4282).

**pH CONTROL**

Further information was gathered in 2011 regarding effects of *P. parvum* toxic blooms and nutrient accumulation. Addition of inorganic nitrogen and phosphorus increased cell densities of *P. parvum* initially and subsequently reduced the ichthyotoxicity created by *P. parvum* (Kurten et al. 148). Previous research has revealed that targeted and time-limited reduction can be used for bloom mitigation, however, Kurten et al. showed that consistent fertilization is necessary to maintain the reduction in toxicity (149). Interestingly, chlorophyll *a* increased as cell densities decreased suggesting further that fertilization reduces toxic substances (Kurten et al. 149). Unfortunately, addition of increased concentrations of inorganic nitrogen and phosphorus raised pH levels (Kurten et al. 150). An increased pH level to 9.2 lowered toxicity, but fish still had a high mortality rate (Kurten et al. 150). pH values varied depending on the time of day (Kurten et al. 144). Moreover, pH was a “function of the amount of inorganic fertilizer added to ponds” (Kurten et al. 144), in addition to carbon dioxide removal by other organisms (Kurten et al. 150). Addition of cottonseed meal was added to try to lower the pH to appropriate levels, however, this was not successful (Kurten et al. 150). Kurten et al. found that cottonseed meal enriched nutrients in tested ponds, but concentrations were unable to control the toxic environment to fish populations (Kurten et al. 148). A previous study done by Boyd had success of lowering pH by the use of aluminum sulfate (Kurten et al. 150). Therefore, indication of nutrient concentrations in various waters can be useful when using aluminum sulfate or other pH-lowering compounds to reduce fish population mortality (Kurten et al. 150).
Conclusion

New information regarding *P. parvum* and its associative toxic compounds causing algal blooms are arising. Transcriptome level molecules have currently been found that are similar to previously identified defense transcripts. Additionally, toxins appear to be released most frequently when *P. parvum* endures stress, in particular, stress related to nutrient depletion. This suggests *P. parvum* produces toxins that have been selectively advantageous to the alga. Another metabolite has been identified within the prymnesin toxins: fatty acid amides. The exact mechanism for these amides is yet to be determined. The newly identified contact-toxicity action of prymnesin toxins indicates that this mode of action may be how *P. parvum* actually utilizes its toxins. On the other hand, this could also be an additional way the toxins cause damage. Further investigation is necessary to understand this new method and its selective advantage to *P. parvum*. In regards to harmful algal bloom mitigation, natural sunlight has a direct, negative effect on prymnesin toxins and an indirect, positive effect on the surrounding fish populations. *P. parvum* toxic blooms have showed varying effects among fish populations in the Texas reservoirs, with most populations declining. Ammonium reduces toxicity of blooms if added in appropriate concentrations. Cottonseed meal does not reduce pH levels caused by the addition of extra nutrients. The current information can be used for further investigation into the toxins that *Prymnesium parvum* releases to create toxic blooms and how to mitigate them for the sake of declining gill-breathing populations.
Works Cited


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