Maternal effects are no match for stressful conditions: a test of the maternal match hypothesis in a common zooplankter

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Summary

1. Anticipatory parental effects modulate population responses to environmental conditions and so are predicted to play a large role in the responses of organisms to global change.

2. In response to one such aspect of global change, the eutrophication of freshwaters and associated blooms of the toxin-producing cyanobacteria species *Microcystis aeruginosa*, the rotifer *Brachionus calyciflorus* produces larger offspring.

3. We hypothesized that rotifers, with the short generational times, exposed to highly predictable cyanobacteria bloom conditions, may adaptively increase offspring investment and offspring fitness (i.e., the maternal match hypothesis).

4. We explicitly tested the consequences of this differential investment by rearing offspring produced by rotifers reared under *Microcystis* and the nontoxigenic green alga

Chlamydomonas, in a full factorial design, where offspring were raised under the maternal diet or the opposite food source.

5. We measured age-specific fecundity, survival, and population growth rates under these conditions and found that maternal exposure to *Microcystis* decreased offspring survival and fecundity, regardless of offspring diet. Population growth rates, tested using aster models, differed significantly among maternal and neonate diets, but there was no significant interaction between the two factors.

6. Our evidence thus leads us to reject the maternal match hypothesis in this case of rotifer-toxigenic algal bloom interactions and provides further support that toxigenic algal blooms may have extensive effects on grazer populations in ways that are not evaluated using traditional, single-generation experimental methods.

Key-words *Brachionus calyciflorus*, harmful algal blooms, life history, maternal match hypothesis, optimal allocation, rotifers, anticipatory parental effects

Introduction

Anticipatory parental effects modulate population responses to environmental conditions through control on offspring phenotypic plasticity (Marshall & Uller 2007) and so are predicted to play a large role in the responses of organisms to global change (Meylan, Miles & Clobert 2012). One way in which the parental environment may give rise to phenotypic plasticity in offspring is

through differential provisioning of offspring (Marshall & Uller 2007). For example, resource availability (here defined as food quality and quantity) during the parental generation may affect the tradeoff in the number and size of offspring produced (Smith & Fretwell 1974). However, an important component of such anticipatory parental effects is the ability of the parent to predict the environment that the offspring will encounter (Burgess & Marshall 2014). Anticipatory provisioning of offspring, including tradeoffs in offspring number and size, should only be possible when the offspring's environment is predictable by the parent, that is, when the environment is sufficiently stable that parent and offspring experience similar selective pressures, or when there are reliable cues of impending environmental change.

Such anticipatory provisioning of offspring may vary according to the optimal egg size theory (OEST). As resource availability declines, and thus offspring survival risk increases, the per capita investment by mothers should increase, leading to larger offspring, which comes at a cost of a decline in the number of offspring produced (Krist 2010). This theory has been tested in a broad variety of organisms, and support has been found in diverse taxa including collared flycatchers, *Ficedula albicollis* (Krist & Munclinger 2015); Atlantic salmon, *Salmo salar* (Rollinson & Hutchings 2013); and invertebrates like the crustacean zooplankter *Daphnia pulex* (Li & Jiang 2014) and the rotifer *Brachionus calyciflorus* (Beyer & Hambright 2016). However, support for the OEST is not universal (e.g., Brett 1993; Kirk 1997), leading to the questions of when and under which conditions the OEST operates.

Optimal provisioning of offspring will be particularly important under conditions of food scarcity or nutritional insufficiencies, when offspring fitness may be more dependent on

resources acquired through parental investment than resources acquired through feeding on those scarce resources. For example, young rotifers hatched from larger eggs are more resistant to starvation than rotifers hatched from small eggs (Kirk 1997). The relative importance of parental effects is context dependent because under poor conditions, offspring survival and reproduction will be more tightly linked to parental investment than in good conditions. Thus, the external environment of the offspring will mediate the expression of parental effects. We would thus predict that the realization of parental effects as well as the affected life history traits depend on the quality of the environment, as well as the predictability of the environment. In a test of context-dependent maternal effects in soil mites, Plaistow et al. (2006) showed that effect of egg size differed between low-food and high-food conditions. Variation in egg size only affected tradeoffs in age and size at maturity under low-food conditions.

One caveat of context-dependent parental effects is that this strategy of differential investment in offspring that will encounter good or poor environments assumes that parents can identify their offspring's environment. In unpredictable environments, (i.e., those lacking either correlation of parental and offspring selective pressures or reliable cues of impending changes in selective pressures), anticipatory environmental effects cannot evolve (Burgess & Marshall 2014). The maternal match hypothesis (MMH, described in Sheriff & Love 2013) was established as a framework to predict how maternally-derived stress would affect the phenotype of her offspring when imperfect information and changing environments lead to phenotypic mismatches between the offspring and the environment. Under the MMH, offspring born to mothers in stressful or poor environments are predicted to be more fit when raised in similarly stressful or poor environments compared with offspring born to mothers in good environments (Sheriff & Love

2013). The MMH suggests that, under stressful conditions, mothers will produce offspring that are more fit for the same stressful conditions. However, the phenotype of these offspring may be maladaptive under benign conditions. Thus, to fully test the MMH, offspring produced by mothers reared in benign and stressful conditions must be raised under stressful conditions, and then evolutionarily meaningful response variables (e.g., lifetime fitness) must be monitored. Simultaneous empirical tests of the maternal match hypothesis and the optimal egg size theory are not well developed, yet herbivorous zooplankton provide an ideal system for these tests because they have short lifespans that can allow for the measurement of lifetime fitness, can be maintained as asexual (clonal) lineages (allowing for the isolation of phenotypic changes from genotypic changes), and are regularly exposed to natural variations in food availability.

Within aquatic systems, primary consumers like zooplankton are exposed to frequent variations in food quality and quantity. Rapid changes in phytoplankton composition, including blooms of harmful algae, represent remarkable changes in resource availability that may have dramatic effects on consumers (Sunda et al., 2006). Blooms of cyanobacteria act to change the nutritional quality (Lürling 2003) and toxicity (Tillmanns *et al.* 2008) of the resources available to freshwater grazers, and may have dramatic effects on the life histories of these grazers. Thus, we predicted that anticipatory parental effects in response to changes in resources should be well developed in freshwater grazers, as long as conditions are sufficiently stable that mother and offspring are likely to experience the same food quality and quantity. In one such asexually-reproducing freshwater zooplankton taxon, the rotifer *Synchaeta pectinata*, research has demonstrated that maternal diet affects investment in offspring (Stelzer 2001) and that investment has an effect on offspring fitness (Walz 1995). Additionally, the life-extending

effects of caloric restriction can be passed to offspring in the rotifer *Brachionus plicatilis* (Kaneko *et al.* 2010).

Using the asexually-reproducing rotifer *Brachionus calyciflorus* (Fig. 1), we set out to test the consequences of differential offspring investment produced after maternal exposure to resources that differed in quality. We performed an experiment to test the OEST and MMH in *Brachionus calyciflorus* using a factorial design of maternal and offspring exposure. The first generation of rotifers was raised with either toxigenic cyanobacteria (*Microcystis aeruginosa*) or good-quality food (*Chlamydomonas* sp.) and their offspring were either maintained on the same treatment as their mothers or switched to the other treatment. These two foods are known to produce differential resource investment, with fewer, larger offspring produced by rotifers exposed to *Microcystis* compared with those exposed to *Chlamydomonas,* if mothers were initially reared under high amounts of *Chlamydomonas* for the first three days of life (Beyer & Hambright 2016). We measured age-specific survival and fecundity of the four groups of second-generation rotifers and calculated the population growth rate and total offspring production, two measures of fitness which are predicted to change under the OEST and MMH. OEST would be supported if, under the poor food conditions of *Microcystis*, mothers produce fewer, larger offspring and these large offspring are more fit. However, MMH would be supported if offspring produced in *Microcystis* have higher fitness than those produced in *Chlamydomonas*, but only when reared in *Microcystis*. Additionally, we tested whether environmental conditions were likely to remain constant between maternal and offspring lifespans (environmental predictability), by monitoring algal biomass during a cyanobacterial bloom in a subtropical reservoir and testing for autocorrelation in chlorophyll and phycocyanin measurements.

Materials and methods

<u>Study organisms and culture conditions</u>

We used a clonal strain of the rotifer *Brachionus calyciflorus* (Pallas), that was started from a single resting egg isolated from the subtropical reservoir, Lake Texoma, OK-TX, USA that has regular blooms of cyanobacteria. As is typical of rotifers, this strain reproduces by cyclic parthenogenesis, but we only considered females reproducing asexually in this experiment because earlier experiments have shown that the switch to sexual reproduction is mediated by rotifer population density (Gilbert 2004) and not by variation in resource quality or quantity (Beyer & Hambright 2016). Under our high-quality food conditions (see below for details), the median survival for this rotifer species is five days (Beyer & Hambright 2016), and the average generation time is 3.1 days. Total development time of amictic (asexual) female embryos of *Brachionus calyciflorus* averages between 36 and 46 hours, depending on clonal identity (Yin et al. 2016). Thus, there is a very high degree of overlap between generations; mothers exist in nearly the exact same time and conditions as their daughters, and granddaughters.

All algae and rotifers were cultured in COMBO medium, which is optimized for both algal and zooplankton growth (Kilham *et al.* 1998). Cultures were maintained at 20°C on a 12-h-dark:12-h-light regime. We compared the effects of unicellular *Microcystis aeruginosa* (Kützing) (UTEX LB 2385, UTEX Culture Collection of Algae, Austin, TX, USA) with *Chlamydomonas* sp. (Ehrenberg) (Connecticut Valley Biological Supply, Southampton, MA, USA). Under our conditions, high population growth rates of *Brachionus calyciflorus* are obtained on a diet of

Chlamydomonas but not *Microcystis*. Additionally, this strain of *Microcystis* produces

microcystins under our culture conditions (see Beyer & Hambright 2016 for further details).

<u>Experimental test of maternal diet on offspring fitness</u>

To test for the effects of maternal diet on offspring survival and reproduction, we raised

populations of rotifers at densities below which sex is induced. These populations were fed either

Microcystis (4×10^5 cells ml^{-1}) or *Chlamydomonas* (4×10^5 cells ml^{-1}) for three days. To test how

likely rotifers would be to experience phytoplankton communities dominated by a single species

(and thus, verify the validity of our experimental design), we carried out an analysis of

cyanobacterial abundance in 1153 lakes across the contiguous U.S. that were part of the National

Lakes Assessment (see Appendix S2 in Supporting Information). Of these lakes, 9% had over

90% of phytoplankton biomass comprised of cyanobacteria. In Oklahoma alone, 9% of the 53

sampled lakes had phytoplankton communities with cyanobacteria representing more than 98%

of the biomass. Thus, we used algae treatments of 100% cyanobacteria or green algae for our

experiment because these treatments were within the range of evolutionarily experienced values

that rotifers in Oklahoma would experience. Additionally, pure cultures eliminate the risk of

selective feeding by rotifers changing diet compositions relative to treatments. The densities of

algae used for these treatments were selected to optimize population growth rates under each

treatment, based on earlier population-based experiments using adult and juvenile rotifers (Beyer

& Hambright, 2016).

After three days, we collected neonates from the two treatments and transferred them to

individual 2-mL wells of a 24-well plate (Grenier Bio-One, Monroe, NC, USA) with either the

parental diet or the opposite food (six individuals raised in each factorial combination of

maternal and offspring diet, random assignment of offspring to treatments using random number generator). Sample sizes were chosen based on sizes that were sufficient to measure effects of diet on population growth rates in previous experiments (Beyer & Hambright, 2016). Each day, offspring were transferred to fresh food, and we measured survival and the number offspring produced.

Observational test of environmental predictability

For Lake Texoma, the source of our *Brachionus* clone, we only have fortnightly data for cyanobacterial abundances (as phycocyanin concentrations). These data are insufficient for quantifying autocorrelation and environmental predictability. Therefore, we collected daily data from a nearby lake with similar cyanobacterial dynamics, which allowed us to test for autocorrelation in environmental conditions at the scale of rotifer life history, as described above, by monitoring the subtropical reservoir, Lake Thunderbird, OK, which has similar blooms of cyanobacteria to the lake from which our focal rotifer clone was isolated (see Appendix S1 in Supporting Information). For 46 days (21 June 2016–5 August 2016), we measured the concentrations of chlorophyll, a proxy for total phytoplankton biomass, and phycocyanin, the primary photopigment of cyanobacteria, daily at two locations, Sailboat (Latitude: 35.23029, Longitude: –97.23627) and Fisherman's Point (35.22913, –97.24625). We used the concentration of phycocyanin as a proxy for cyanobacterial dominance, and thus, food quality that would be experienced by *Brachionus calyciflorus*. We quantified chlorophyll from integrated depth samples from the photic zone using acetone extraction (Arar & Collins 1997) which was read on a Turner Trilogy fluorometer (San Jose, CA, USA). Phycocyanin was

measured in the field at 0.5 m intervals (later averaged across the photic zone) using a Hydrolab sonde (H2O Submersible Water Quality Data Transmitter, OTT Hydromet, Kempten, Germany).

<u>Statistical analysis</u>

All statistical tests were carried out in R (R Core Development Team, version 3.2.1, www.r-project.org). Following methods similar to those of Burgess and Marshall (2011), we constructed correlograms in R to assess the autocorrelation of chlorophyll and phycocyanin concentrations at time lags ranging from zero to sixteen days. We carried out the autocorrelation calculations using raw data because the measurements of chlorophyll and phycocyanin were collected at the time when *Brachionus calyciflorus* would be reproductive in a subtropical lake, and we were interested in the day to day changes experienced by these rotifers. We then compared the range of time lags of significant autocorrelation to the life cycle of *Brachionus calyciflorus* to address the question of whether the resource quantity and quality experienced by offspring is predictable by mothers.

Differences in survival among treatments were compared using Cox proportional hazards regression models (survival, Version 2.37-4). Assumptions of the Cox proportional hazards regression model were tested using the cox.zph function and examination of log-transformed survival curves, and we found that the proportional hazards assumption was met ($p > 0.05$ for all terms in model). We tested for differences in total number of offspring using a permutation-based ANOVA to account for the differences in dispersion observed between treatments (lmPerm, Version 1.1.2).

Fitness for each combination of maternal and offspring diets was quantified as the population growth rate (λ), which was calculated as the dominant eigenvalue of the corresponding Leslie matrix (Caswell, 2001). Confidence intervals were constructed using bootstrap resampling (2000 iterations). In assessing the relative contributions of maternal and offspring diet to fitness, we compared the utility of two approaches. For the first approach, we used randomization testing to test for significance of the contributions of maternal and offspring diet to the population growth rate of the offspring (Caswell 2001). Additionally, we modeled individual lifetime fitness using aster models (Shaw *et al.* 2008) (aster, version 0.8-31). Aster models are a parametric approach to modeling life history data that take into account the natural distributions of data (e.g., Poisson distributions for the number of offspring) and the interdependencies of the data (e.g., reproduction at age five is dependent on survival up until age five). Our model integrated both age specific survival and fecundity data. Survival to each age (S_x) was modeled using a Bernoulli distribution. As a result of the experimental treatments, there were too many zero values in the number of offspring produced at age x (B_x) to fit a Poisson distribution, so instead, reproduction was modeled as a two-step process. For each age, we modeled the success of reproduction as a variable that took two values (Bernoulli distribution, 0 or 1) with 1 indicating that the rotifer produced offspring at that age. Then, for those rotifers that produced at least one offspring, the number of offspring produced at that age was modeled using a zero-truncated Poisson distribution (B_x). Using these aster models, we tested for the effect of maternal and offspring diet on the population growth rate, and calculated standard errors for those estimates. We then compared the conclusions drawn from aster modeling with those drawn from randomization testing to test the effectiveness of these two approaches that are commonly used in individual-based life history testing.

Results

<u>Survival and Reproduction</u>

We found that maternal diet had a strong influence on survival and reproduction of offspring (Figs 1, 2). The offspring of rotifers fed the poor quality food, *Microcystis* had lower survival than those produced by rotifers fed the high quality food, *Chlamydomonas* (Cox proportional hazards model, Wald $z = 2.50$, $p = 0.012$). There was no detectable effect of offspring diet on survival (Wald $z = 1.02$, $p = 0.310$). Exposure to *Microcystis*, in both first and second-generation diets, severely reduced reproduction in the second-generation rotifers (Permutation-based ANOVA; $p = 0.007$, $p = 0.004$). None of the rotifers raised in *Microcystis* with maternal diets of *Microcystis* produced any offspring (Fig. 2).

<u>Fitness</u>

Using randomization testing, we found that lifetime fitness was significantly affected by maternal diet ($p = 0.017$, Table 1). Within those rotifers with high quality maternal diet of *Chlamydomonas*, there was no significant effect of offspring diet on fitness ($p = 0.116$). Within those rotifers with poor quality maternal diet of *Microcystis*, there was no effect of offspring diet on fitness ($p = 0.423$). There was no interactive effect between maternal and offspring diet on fitness ($p = 0.351$). Using aster modeling, we found that lifetime fitness was significantly affected by maternal diet and offspring diet, but the interaction between the two had no effect (Table 1).

When we made a direct comparison of parametric (aster model) and nonparametric methods (randomization testing) in estimating population growth rates and effects of treatments on population growth rates, we found some similarities and some differences in drawn inferences (Table 1). Estimates of population growth rates were nearly equivalent for three of the four treatments (Tables 2, 3), but the population growth rate of rotifers where both the neonate and mother were raised in *Microcystis* was over-estimated by the aster model. This is an unusual case where absolutely no offspring were produced by any of the second-generation individuals raised in *Microcystis,* and the variation in number of offspring produced was zero (Fig. 2). Both parametric and nonparametric methods detected a significant effect of maternal diet on offspring fitness (Table 1). Neither approach supported an interactive effect of maternal and offspring diets on offspring fitness. However, using the parametric approach (aster model), we found support for a significant effect of offspring diet on offspring fitness (Table 1), which was not detected using the nonparametric approach (randomization testing).

Environmental Predictability

Using both measurements of phytoplankton biomass, we found significant predictability of phytoplankton at timespans of up to eight to ten days. Results were comparable between our two sites. Phycocyanin measurements predicted future phycocyanin measurements up to nine days away (Fig. 3A-B). Chlorophyll measurements predicted future chlorophyll measurements up to ten days later (Fig. 3C-D).

Discussion

OEST makes the prediction that under lower resource availability (*Microcystis*) the produced eggs and offspring will be larger than under high resource availability (*Chlamydomonas*). We found support for this in earlier work (Beyer & Hambright 2016). As a subset of anticipatory parental effects, the maternal match hypothesis makes the further prediction that offspring produced by *Microcystis*-reared mothers will be more fit than those produced by *Chlamydomonas*-reared mothers when raised with *Microcystis*. That is, the stressed mothers will produce offspring that are more fit under stressful conditions. We found evidence against the MMH within this experiment. In comparing offspring raised with low quality food (*Microcystis*), we found that offspring produced by mothers fed *Microcystis* had lower fitness when fed *Microcystis* (measured as estimated population growth rate and as total offspring produced) than those produced by mothers fed *Chlamydomonas*. That is, the offspring produced by stressed mothers fared worse in stressful situations than the offspring produced by unstressed mothers, even though they were larger. It is clear, then, that offspring size cannot be used as a proxy for fitness in *Brachionus calyciflorus*, and instead total lifetime fitness must be directly measured in order to understand the fitness consequences of environmental stressors and to validly test the OEST and MMH.

Anticipatory parental effects can only be adaptive and evolve where there is predictability of the environment experienced by mother and offspring. We explicitly tested for environmental predictability by measuring two proxies for phytoplankton biomass during a bloom of cyanobacteria in a subtropical reservoir similar to the origin of our rotifer clone. We found that, during the generation time (3.1 days) and lifespan (50% of rotifers died within 5 days) of *Brachionus* grown under high food availability and summer temperatures, algal biomass and

quality, with significant temporal autocorrelations up to 8-10 days, was highly predictable. Thus, based on this criterion, we would predict that anticipatory maternal effects would likely be adaptive and could potentially evolve. In describing the MMH, Sheriff and Love (2013) lay out the additional conditions under which the MMH is expected to operate. Any life history or ecological traits that increase the likelihood of a match between the maternal and offspring environments will increase the adaptive potential of maternally derived stress responses. For example, r-selected organisms (those that are fast maturing and short lived) should have greater responses to maternally derived stress than k-selected organisms, because offspring are more likely to exist within the same ecological conditions that were stressful to the maternal generation. With an average generation time of 3.1 days and a total development time of only 36 to 46 hours, rotifers certainly satisfy both of these criteria. Additionally, organisms with stronger philopatry and low dispersal, such as in our case with planktonic rotifers without active dispersal, should have stronger adaptive responses to maternally derived stress. Based on these criteria, as well as our analysis of the temporal autocorrelation of algal biomass (significant autocorrelation up to 10 days), we would predict that anticipatory maternal effects would be adaptive in *Brachionus calyciflorus*, because these rotifers have short lifetimes, reproduce early in life, and do not actively disperse, leading to overlapping generations, yet, surprisingly, the data did not support our predictions.

In considering the relationship between offspring size and fitness, our results, that larger offspring had lower fitness, are counterintuitive when considered within the traditional framework of the OEST. Although we did not directly measure offspring size in this experiment, we have done so under similar experimental conditions, and found that diet has a strong effect on

offspring size (Beyer & Hambright 2016). That these offspring were less fit, even though they have presumably larger energy reserves for somatic maintenance and reproduction, warrants speculation. While the majority of studies show that larger offspring are more fit (Walz 1995; Kirk 1997), not all research supports this expected relationship between offspring size and fitness. Multiple mechanisms (including lower boundary on egg volume, Guinnee et al., 2007; physiological constraints, Stelzer, 2002; and feeding behavior, Garbutt and Little, 2014) have been invoked to explain this paradoxical empirical result in freshwater zooplankton. In a test of the maternal match hypothesis in *Daphnia magna*, Guinnee et al. (2007) took larger offspring produced by individuals fed low amounts of food and reared them in either low or high food environments. These larger offspring produced by resource-limited mothers had lower fitness in both environments, compared with offspring born to mothers fed high amounts of food, because they took longer to reproduce and did not have a survival advantage. In this case, larger eggs were not adaptive (as OEST would predict), but instead the explanation for this pattern was that there was a lower boundary on egg volume, or that there is a minimum viable size for eggs, and mothers do not produce eggs smaller than this (Guinnee *et al.* 2007). In the rotifer, *Macrotrachela quadricornifera*, Santo et al. (2001) found that larger egg sizes decreased developmental time and time to reproduction, but did not confer benefits under stressful conditions. In the rotifer *Synchaeta pectinata*, Stelzer (2002) found that although low temperatures produce larger eggs and larger offspring, these offspring were not more fit than those smaller offspring produced at warmer temperatures, when tested under colder temperatures. When tested under warmer temperatures, the smaller offspring were found to have higher fitness. In *Daphnia magna*, smaller offspring produced at higher food concentrations have paradoxically higher fitness when compared with larger offspring produced at low food

concentrations (Garbutt & Little 2014). This difference may be due to the feeding behavior of the offspring, because the maternal environment was found to affect feeding rates of offspring, independent of the environment in which the offspring live.

These four examples highlight the many factors governing the relationship between egg size and fitness, and illustrate mechanisms that could produce the counterintuitive pattern of larger eggs having lower fitness. The design of our experiment does not allow us to disentangle the factors leading to the cumulative decreased fitness under the *Microcystis* feeding regime, but many of the causes described above could be playing a role. As both nutrition and toxicity contribute to the negative effects of *Microcystis aeruginosa* on *Brachionus calyciflorus* (Zhao et al. 2014), we suggest that this offspring size-fitness relationship could be produced by constraints on the ability of rotifers to upregulate detoxifying enzymes, by inability to compensate for the nutritional inadequacy of *Microcystis aeruginosa*, or by interaction of these two mechanisms. Both of these mechanisms would fall under the umbrella of transmissive maternal effects, whereby negative conditions experienced during the mother's lifespan may have negative effects on offspring, often through adverse effects on nutritional provisioning (Marshall & Uller 2007).

Although maternal effects have been tested in rotifers relative to resting egg production (Gilbert & Schröder 2007), morphology (Gilbert & McPeek 2013), lifespan (Gribble & Mark Welch 2013), and many other conditions, the contribution of maternal effects to responses to harmful algal blooms including cyanobacteria has not been measured. However, in *Daphnia*, maternal effects play a strong role in population responses to cyanobacteria exposure. Depending on the species and clonal identity, and likely depending on experimental design, exposure of mothers to

cyanobacteria may increase (Gustafsson, Rengefors & Hansson 2005; Ortiz-Rodríguez, Dao & Wiegand 2012) or decrease (Dao, Do-Hong & Wiegand 2010) offspring fitness under similar conditions of cyanobacteria exposure. Maternal exposure of *Daphnia magna* to toxins from *Microcystis* sp. was found to decrease offspring survival, even when offspring were switched to control media (Dao *et al.* 2010). When comparing neonates fed *Microcystis*, those neonates produced by mothers fed a non-toxic algae (*Rhodomonas*) had higher fitness than those produced by mothers fed *Microcystis* (Brett 1993). That is, maternal exposure to *Microcystis* was harmful to offspring raised in *Microcystis*, rather than beneficial, as was later shown by (Gustafsson *et al.* 2005). The results of Brett (1993), where offspring from mothers fed *Microcystis* were less fit, contrary to the predictions of the maternal match hypothesis, square with our results quite well, possibly because in both studies, pure cultures were used rather than the mixed diets employed by Gustafsson and colleagues (2005). It could be that the mixed diets employed by Gustafsson et al. (2005) allow rotifers to compensate for nutritional inadequacies of the poor food making apparent only effects of toxins, as opposed to a pure diet approach, where rotifers are subjected to simultaneous differences in nutrition and toxicity, with both factors influencing offspring fitness and transgenerational effects.

We set out to test the maternal match hypothesis in an asexually reproducing invertebrate subject to natural and frequent changes in resource quality and availability. In an earlier experiment, we found that mothers exposed to poor food quality produced fewer and larger offspring, contingent upon early exposure to high amounts of high quality food, thus supporting the OEST. Based on the maternal match hypothesis, we predicted that if larger offspring were produced under poor food quality (as shown in Beyer and Hambright 2016), they would have higher fitness if they

were raised in the same food quality, assuming predictability of offspring environment by maternal experiences. Our evidence leads us to reject the maternal match hypothesis under these conditions and provides further support that toxigenic algal blooms may have more extensive effects on grazer populations through transmissive maternal effects than can be evaluated using traditional experimental methods, which use only single generations of rotifers, because the effects of exposure to toxigenic algae propagate and intensify across generations.

Acknowledgements

Data Accessibility

Data will be made available on Dryad upon publication, and R scripts will be uploaded as online supporting information.

Authors' Contributions

JEB and KDH conceived the ideas and designed methodology; JEB collected the data; JEB analysed the data; JEB led the writing of the manuscript. All authors contributed critically to the drafts and gave final approval for publication.

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Supporting Information

Additional supporting information may be found in the online version of this article.

Appendix S1 Comparison of phycocyanin between Lakes Texoma and Thunderbird

Figure S1 Comparison of phycocyanin between Lakes Texoma and Thunderbird

Appendix S2 Cyanobacterial Dominance across Lakes and Reservoirs in the United States

Figure S2 Distribution of cyanobacterial dominance values

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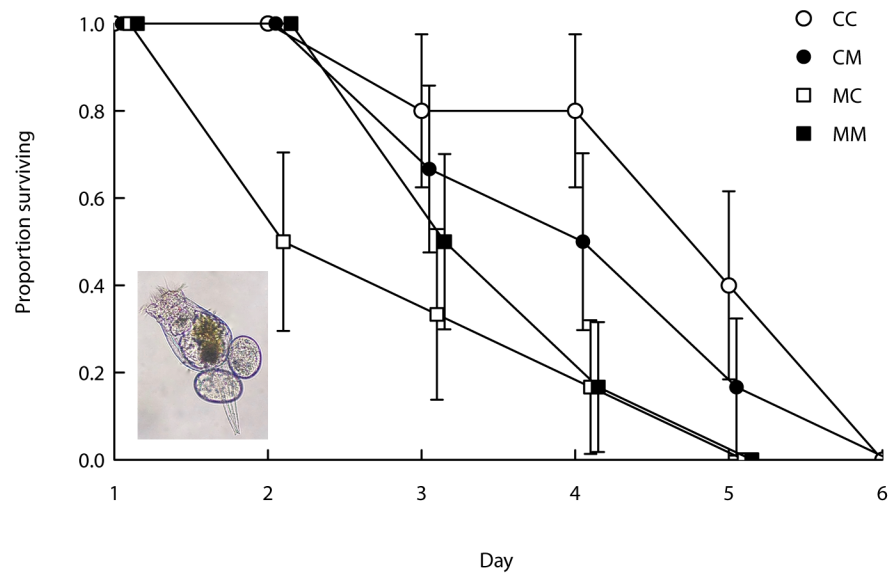
Figure legends

Figure 1: Comparison of survival of the second generation of rotifers subjected to four treatments. Points represent mean proportion surviving ± 1 s.d. (calculated from 2000 bootstrap replicates). For treatments, the first letter indicates the maternal diet and the second letter indicates the offspring diet. C represents *Chlamydomonas*, the non-toxigenic green alga, and M represents *Microcystis*, the toxigenic cyanobacterium. Inset photo shows *Brachionus calyciflorus* adult female carrying two eggs.

Figure 2: Comparison of fecundity among the four treatments. Individual data points are overlaid as small, open shapes. Median values for each treatment are represented by solid shapes, and lines indicate the norm of reaction.
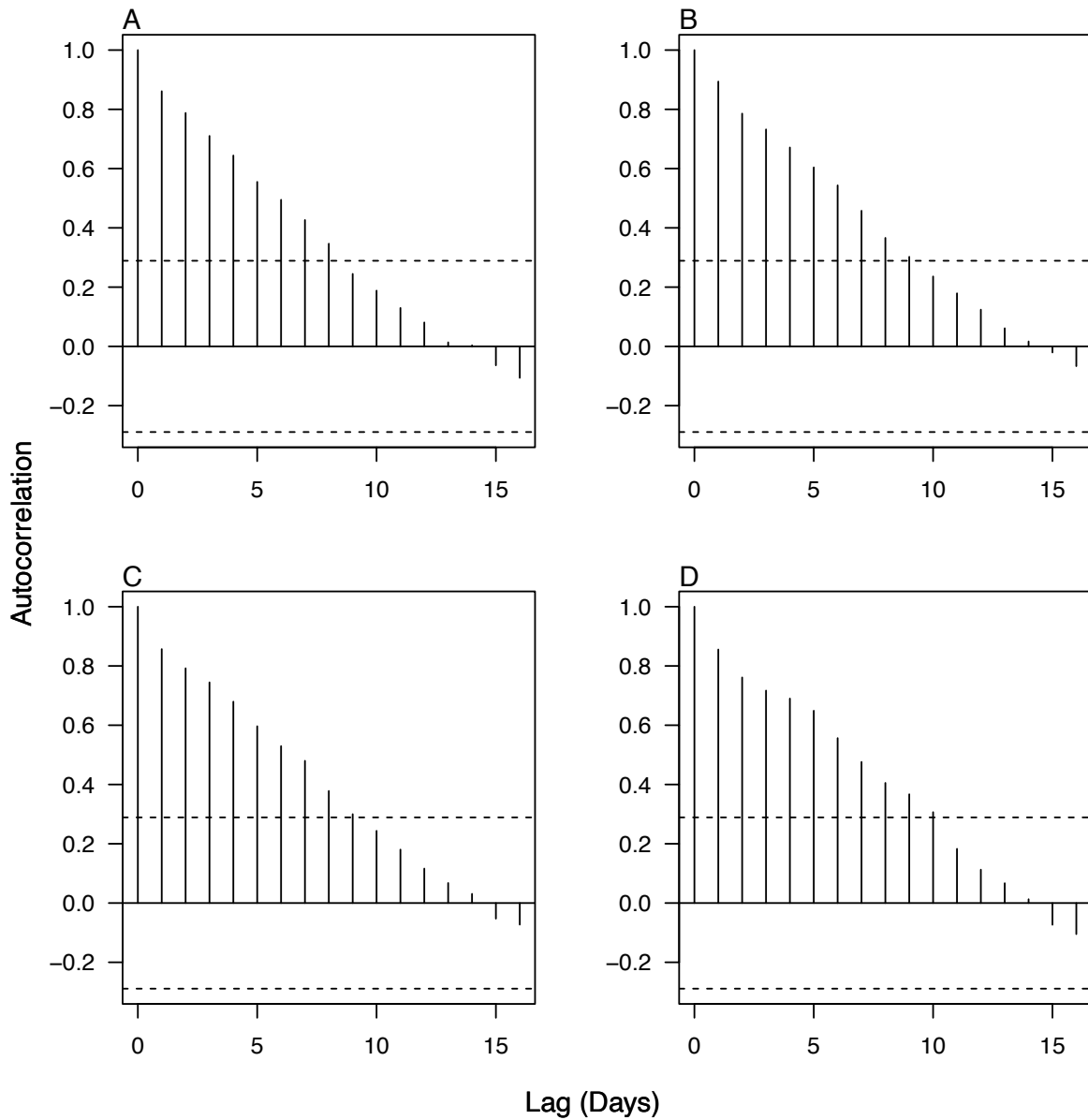
Figure 3: Predictability of phytoplankton biomass measured in subtropical Lake Thunderbird,
OK. Correlogram shows autocorrelation of measured (A) phycocyanin at Fisherman's Point, (B)
phycocyanin at Sailboat, (C) chlorophyll at Fisherman's Point, and (D) chlorophyll at Sailboat.
Dashed lines represent 95% confidence intervals for a series with autocorrelation of zero.

Tables

Table 1: Summary of results from aster model comparison testing the effects of maternal diet, offspring diet, and their interaction on lifetime individual fitness. The interaction was tested relative to the full model, and the individual effects of maternal diet and offspring diet were tested relative to a model including only the main effects and not interactions. Results of randomization testing are shown, for comparison, in the far-right column.

Term	Model degrees of freedom	Test degrees of freedom	Deviance	p (aster model)	p (randomization testing)
Full Model	13				
Maternal × offspring diet	12	1	0.7496	0.387	0.351
Main effects only	12				
Maternal diet	11	1	12.89	< 0.001	0.017
Offspring diet	11	1	5.845	0.012	*Chlamydomonas* 0.116 *Microcystis* 0.423

Table 2: Population growth rates and standard errors estimated from aster models for each combination of maternal and offspring diet.

Maternal diet	Offspring diet	Aster λ	Standard Error
Chlamydomonas	*Chlamydomonas*	1.29	0.10
Chlamydomonas	*Microcystis*	0.97	0.27
Microcystis	*Chlamydomonas*	0.79	0.60
Microcystis	*Microcystis*	0.58	2.31

Table 3: Population growth rates and standard errors estimated with bootstrap resampling for each combination of maternal and offspring diet.

Maternal diet	Offspring diet	Leslie matrix λ	Standard Error
Chlamydomonas	*Chlamydomonas*	1.53	0.41
Chlamydomonas	*Microcystis*	0.62	0.18
Microcystis	*Chlamydomonas*	0.36	0.18
Microcystis	*Microcystis*	0.00	0