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RESEARCH ARTICLE

# Experimental evidence of rapid heritable adaptation in the absence of initial standing genetic variation

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## Abstract

1. The success of genetically depauperate populations in the face of environmental change is contrary to the expectation that high genetic diversity is required for rapid adaptation. Alternative pathways such as environmentally induced genetic modifications and non-genetic heritable phenotypes have been proposed mechanisms for heritable adaptation within an ecologically relevant time frame. However, experimental evidence is currently lacking to establish if, and to what extent, these sources of phenotypic variation can produce a response.
2. To test if adaptation can rapidly occur in the absence of initial standing genetic variation and recombination in small populations, we (a) exposed replicate monoclonal populations of the microzooplankton *Brachionus calyciflorus* to a culturing regime that selected for phenotypic variants with elevated population growth with either high or low phosphorus food for a period of 55 days and (b) examined population level response in two fully factorial common garden experiments at day 15 and 35 of the exposure experiment.
3. Within six generations, we observed heritable local adaptation to nutrient limitation. More specifically, populations with a history of exposure to low P food exhibited higher population growth rates under low P food conditions than populations with a high P exposure history. However, the capacity for such a response was found to vary among clones.
4. Our study finds that although standing genetic variation is considered essential for rapid heritable adaptation, the rapid emergence of de novo genetic variation or alternative sources of phenotypic variation could aid in the establishment and persistence of low-diversity populations.

KEYWORDS

*Brachionus calyciflorus*, ecological epigenetics, ecological stoichiometry, intraspecific variation, mutation, non-genetic inheritance, phosphorus, transposable elements

## 1 | INTRODUCTION

Environmental fluctuations present a fundamental challenge to organisms as changing conditions may result in a mismatch between the environment and organismal phenotypes. One way in which populations may persist and thrive is through their capacity to adapt their phenotypes on a time frame relevant to environmental variation. The ability to adapt has become acutely important during the current period of anthropogenic environmental change in which climate events have become more extreme and frequent (Collins et al., 2013), and habitats are increasingly disturbed (Fischer & Lindenmayer, 2007; Schweiger et al., 2010; Smith et al., 1999). Adaptive trait shifts within populations are typically considered to occur in two different ways. First, at the individual level, phenotypic plasticity allows organisms to modify their phenotype in response to an environmental trigger (Bradshaw, 1965). Second, at the population level, selection may act upon standing genetic (i.e. DNA sequence) variation to shift mean trait values within the population over several generations (Barrett & Schluter, 2008). Recently, both environmentally induced genetic modifications (Danchin, Pocheville, Rey, et al., 2019; Rey et al., 2016) and non-genetic heritable phenotypes (Bonduriansky & Day, 2018; Jablonka et al., 1995) have been proposed as additional pathways for rapid adaptation. However, experimental evidence is currently lacking to establish if, and to what extent, these sources of phenotypic variation can produce an adaptive heritable response in populations within a time frame relevant for rapid environmental change (Hu & Barrett, 2017; Marin et al., 2020; Perez & Lehner, 2019).

In the last 25 years, selection on standing genetic variation has been firmly established as the dominant process through which populations may adapt within ecologically relevant time-scales (Barrett & Schluter, 2008; Bolnick et al., 2011). This process requires high genetic diversity for natural selection to act on. However, natural processes such as range expansions (Eckert et al., 2008; Excoffier et al., 2009) and colonization events within metapopulations (De Meester et al., 2002) have the potential to generate low-diversity populations (So et al., 2015). Additionally, anthropogenic habitat deterioration (Lino et al., 2019; Mather et al., 2015; Schlaepfer et al., 2018) and the introduction of invasive species (Allendorf & Lundquist, 2003) also generate small, isolated populations vulnerable to drift and genetic erosion. Some populations with low levels of standing genetic variation, nevertheless, show a remarkable ability to maintain performance in the face of environmental change (Rollins et al., 2013). Loss of genetic diversity can be especially pronounced in clonal species where single individuals are able to establish populations, yet such populations have been shown to be remarkably successful. This is exemplified by the introduction of a single genotype of *Daphnia pulex* to Lake Naivasha in Kenya during the 1920s which is now found in water bodies along a 5,500 km continental transect (Mergeay et al., 2006). Similar successful invasion events from a small number of genotypes have also been observed in mollusk (Gomes et al., 2016), plant (Hollingsworth & Bailey, 2000; Shi et al., 2018) and fish species (Golani et al., 2007).

The success of populations with low standing genetic diversity has sparked great interest in the possible mechanisms that generate adaptive heritable phenotypic variation from a single genotype. While de novo mutations have the potential to drive adaptive responses (e.g. Barrick & Lenski, 2013; Chan et al., 2010; Hartley et al., 2006; Hoekstra et al., 2006), they have long been considered far too infrequent to be relevant on ecological time-scales, especially when population sizes are small (Hermisson & Pennings, 2005). However, recent evidence suggests that novel and stressful conditions may result in the rapid generation of genetic variation (Danchin, Pocheville, Rey, et al., 2019). Point mutations and transposable element activity have both been observed to be positively associated with epigenetic features (e.g. methylation of specific genomic sites) that often occur in response to environmental factors (e.g. Makova & Hardison, 2015; Miousse et al., 2015; Yi & Goodisman, 2021). As some variants may be beneficial, mutations associated with epigenetic marks and transposable elements have been suggested to be important for adaptation to environmental change (Danchin, Pocheville, Rey, et al., 2019; Pimpinelli & Piacentini, 2020; Rey et al., 2016; Stapley et al., 2015). Heritable phenotypic variation may also be produced by mechanisms other than changes in DNA sequences. Non-genetic inheritance (NGI) allows environmentally induced phenotypes to be passed between generations via the transmission of factors (e.g. nutrients, methylation patterns, small RNAs or microbiomes) that modify phenotypes but not DNA sequences (Bonduriansky & Day, 2018; Macke et al., 2017). Given the capacity of NGI to generate heritable phenotypic variation, it has been proposed to play an important role in adaptation (e.g. Danchin et al., 2011; Day & Bonduriansky, 2011; Jablonka et al., 1995; Jablonka & Raz, 2009; O'Dea et al., 2016). Sources of de novo phenotypic variation, such as environmentally induced mutations and NGI, are expected to be especially important for adaptation in organisms with low action radius or limited dispersal ability as they cannot move to more favourable environments and have a high chance of being exposed to an environment similar to that of the parents (Beery & Francis, 2011; Galloway & Etterson, 2007; Mirouze & Paszkowski, 2011). These mechanisms would also be expected to promote adaptation in clonal organisms (Verhoeven & Preite, 2014) and populations with low genetic diversity (Rapp & Wendel, 2005; Vogt, 2017) such as invasive species (Marin et al., 2020; Pérez et al., 2006; Prentis et al., 2008).

The capacity to produce environmentally induced heritable variation can be considered an adaptive trait that allows genotypes to persist when conditions fluctuate on a temporal scale greater than that of a single generation (O'Dea et al., 2016). An important question is whether there is genetic variation for this capacity within populations, as its extent may determine their evolutionary potential to respond to changes in the frequency and intensity of environmental fluctuation. Previous studies have documented that genotypes may differ in both the magnitude and direction of de novo phenotypic responses to the same stressor (e.g. Asselman et al., 2015; Gillis & Walsh, 2019; Herman & Sultan, 2016), although a dedicated effort to document intraspecific variation is thus far largely lacking.

Currently, there are very few empirical examples of rapid adaptive responses in animals generated by novel heritable phenotypic variation, despite evidence of the mechanistic potential (Hu & Barrett, 2017, but see Webster et al., 2018). Previous studies have investigated the capacity of monoclonal populations to generate adaptive de novo heritable responses to environmental stressors in pea aphids (Sentis et al., 2018) and green algae (Kronholm et al., 2017). Together, these studies have shown that populations without standing genetic variation can exhibit heritable phenotypic changes in plasticity (Sentis et al., 2018) and that non-genetic mechanisms, such as epimutations, can drive adaptive shifts in phenotypes (Kronholm et al., 2017).

The goal of this study was to experimentally test if adaptation can occur in the absence of initial standing genetic variation and recombination in small populations within an ecologically relevant time frame. To investigate this question, we conducted a two-part experiment using two genotypes of the microzooplankton *Brachionus calyciflorus*. In the first part of the experiment (further referred to as the 'exposure experiment'), we exposed replicate monoclonal populations to a culturing regime selecting for phenotypic variants with elevated population growth rates for at least 20 generations with either high (HP) or low (LP) phosphorus food. LP food has a C:P content that deviates strongly from the requirements for growth in *B. calyciflorus*, and thus represents low quality food in comparison to HP food (Sterner & Hessen, 1994; Zhou & Declerck, 2019). In the second part of the experiment, we performed two full factorial common garden experiments at two successive time points, using individuals isolated from the exposure experiment. Using population growth rate as a fitness measure we aimed to evaluate (i) if and to what extent an adaptive heritable response has occurred, (ii) if clones differ in response strength and (iii) if response strength increases with the duration of exposure to the divergent environmental conditions. We test the hypotheses that an adaptive heritable response occurs within the exposed populations consistent with local adaptation to the food sources; however, genotypes may differ in the magnitude of their response. Additionally, as several generations may be required to elicit an NGI response, or for an adaptive de novo variant to spread through the population, we predict the adaptive response to be greater in the second compared to the first common garden experiment.

## 2 | MATERIALS AND METHODS

### 2.1 | Model system

The freshwater microzooplankton *B. calyciflorus* is an excellent metazoan system to study the role of de novo heritable phenotypic variation because as a cyclical parthenogenetic species it can produce asexual offspring via apomictic parthenogenesis. As such, in asexual populations, recombination can be excluded as a mechanism responsible for the generation of heritable phenotypic variation. When monogonont rotifers reproduce asexually, the oocytes undergo a single maturation with the production of one polar body, and there is no indication for synapsis of homologous chromosomes (Birky & Gilbert, 1971). Thus, under apomixis, *B. calyciflorus* prevents recombination by inhibiting meiosis I and can asexually produce offspring that are genetically identical to itself. While ameiotic crossovers are possible (Omilian et al., 2006), they are rarely observed on time frames relevant to experimental work in other apomictically reproducing zooplankton (Dukić et al., 2019). *Brachionus calyciflorus* is also able to reproduce sexually, but this can be avoided by maintaining experimental populations at low densities (Stelzer & Snell, 2003). When sexual reproduction does occur, it results in the production of diapausing embryos that can easily be distinguished from parthenogenetic eggs (Figure S1). This aspect of the life history of *B. calyciflorus* offers the additional benefit that genetically distinct lines can easily be produced and compared. Although previous investigations of de novo heritable variation have taken advantage of apomictic reproduction in other zooplankton species (e.g. Gillis & Walsh, 2019; Kielland et al., 2017; Walsh et al., 2015), the short generation time of *B. calyciflorus* (Declerck & Papakostas, 2017; Stelzer, 2017) provides the added advantage that many generations can be studied over a relatively short period.

### 2.2 | Maintenance of algae and zooplankton cultures

We maintained cultures of the green algae *Chlamydomonas reinhardtii* as a food source for rotifer cultures in 2L chemostats at 23 ± 1°C with a dilution rate of 0.33 $day^{-1}$ using WC medium (Kilham et al., 1998). By varying medium phosphate concentration and light intensity we created two stoichiometric food quality treatments. High phosphorus algae (HP, molar C:P 102.3 ± 1.4 *SE*) were cultured in media with 65 μmol/L P under ≈40 μmol quanta $m^{-2}$ $s^{-1}$ of continuous light, while low phosphorus algae (LP, molar C:P 690 ± 10.9 *SE*) were cultured in media with 15 μmol/L P and under ≈120 μmol quanta $m^{-2}$ $s^{-1}$ of continuous light. To ensure that phosphorus was not available to the rotifers directly from the media we centrifuged the algae collected from the chemostats and re-suspended the algae pellet in nutrient-free WC media at the desired concentration. Carbon concentration of the suspension was estimated by converting biovolume (Multisizer 3 Coulter Counter, Beckman Coulter) to carbon content (Appendix S1). After dilution, a vitamin mixture (Kilham et al., 1998) was added to the algae suspension at a concentration of 1 ml/L.

For this study we used two genetically distinct clone lines, Clone-7 and Clone-128, belonging to the species *B. calyciflorus* s.s. of the *B. calyciflorus* species complex (Michaloudi et al., 2018). We established clone lines by hatching diapausing eggs that had been produced in the laboratory (Appendix S2). During the period between hatching and initiation of the exposure experiment, we maintained clone lines in asexually reproducing stock cultures. We maintained stock cultures at a density of 10 rotifer individuals/ml with ~1,000 μmol/L C algal suspension comprised of nutrient replete *C. reinhardtii* and nutrient-free WC media (Kilham et al., 1998) at room temperature under a 16:8 (light:dark) light cycle. To keep the genetic identity of the clone line intact we performed daily inspections of batch cultures and removed any sexually produced diapausing embryos which are large (>100 μm) and visible to the naked eye (Appendix S3).

## 2.3 | Part one: Exposure experiment

We initiated the exposure experiment 8 weeks after the founding individual of each genotype was hatched. We estimate this period to correspond with approximately 29–34 generations (Appendix S4), each of which would be comprised of less than 50 individuals. For each genotype, we generated five replicate populations for both food quality treatments (HP and LP) by haphazardly allocating 10 individuals from stock cultures to wells of tissue culture plates with 8 ml of the designated algal suspension at a concentration of 1,000 μmol/L C. The entire design of the exposure experiment thus consisted of 20 populations (i.e. 2 clones × 2 food qualities × 5 replicates).

During the exposure experiment, we maintained populations in culturing conditions that allowed for density independent growth. To achieve these conditions, we restarted the populations every 24 hr by transferring 10 individuals to a new culturing vessel with fresh algal suspension at satiating concentrations (1,000 μmol/L C). Selection of these individuals was done in a haphazard fashion except we avoided females bearing sexually produced diapausing embryos. If fewer than 10 rotifers were available, we transferred all individuals without diapausing embryos. By transferring a subset of the populations daily, we created conditions that favoured phenotypes with high reproductive output, as offspring of such individuals would be more abundant in the population and thus more likely to be transferred. Additionally, we prevented sexually produced individuals from entering the populations as diapausing embryos require at least 10 hr to be produced, followed by an additional 72 hr to hatch (Stelzer, 2017). Following the daily transfer, we counted the number of remaining individuals in each population to calculate the population growth rate realized during that time interval.

The exposure experiment lasted 55 days. As the time to first reproduction and rate of egg production differ with resource P content (Zhou et al., 2018), the length of the exposure experiment represents approximately 32 generations in HP conditions and 20 generations in LP conditions (Appendix S4 for details). Throughout the experiment, populations were incubated at 25 ± 1°C, in constant darkness and at a rotation rate of 50 rpm to prevent algae from settling. Plate position in the incubator was randomized daily to prevent any possible location effects.

## 2.4 | Part two: Common garden experiments

Using the populations from the exposure experiment, we conducted two common garden experiments to test for a persistent signal of adaptation to stoichiometric food quality, and to determine if these responses changed with the duration of exposure. The first experiment began on day 15, and the second on day 39, of the exposure experiment and lasted for 20 and 15 days respectively. For both common garden experiments we cultured individuals from each population from the exposure experiment in both HP and LP algal suspensions and measured population growth rate to determine performance. Each common garden experiment consisted of a fully crossed experimental design (i.e. 2 food quality treatments × 2 clones × 2 exposure histories × 5 exposure populations = 40 common garden units). Each common garden unit was initiated by allocating 10 haphazardly chosen individuals from the respective exposure population. We cultured common garden units in 8 ml of algal suspension with a concentration of 1,000 μmol/L C at 25 ± 1°C and in constant darkness. As in the exposure experiment, at the end of each 24-hr time interval, we counted all rotifers and transferred 10 of them to new algal suspensions. However, we did not select against females with sexual eggs to measure the true population growth rate (i.e. including the demographic cost of sexual reproduction; Montero-Pau et al., 2014).

## 2.5 | Data analysis

For both the exposure and common garden experiments, population growth rate was calculated on a daily basis as $(\ln N_t - \ln N_0)/t$, where $N_0$ and $N_t$ represent the population size at the start and end of each 24-hr period, and t the duration of the period in days. Population growth rates were ln-transformed prior to analysis to reduce variance in the residuals between groups.

We wanted to determine if any change in population growth rate had occurred within the populations during the exposure experiment. Furthermore, if a change had occurred, we aimed to detect whether it was immediate (i.e. increased/decreased during the first or second generation followed by a saturation of the response) or if the change occurred gradually throughout the experiment. As populations consist of multiple individuals, an immediate change would suggest that the majority of individuals were expressing the same phenotype. Such a population level response may indicate the induction of a plastic phenotype in each generation (Kronholm et al., 2017; Sentis et al., 2018), the NGI of an induced phenotype (e.g. Danchin et al., 2011; Heard & Martienssen, 2014; Richards, 2006; Wang et al., 2017), or possibly a combination of both processes. Alternatively, if population growth rate changed gradually it may be due to a tendency of the genotype to accumulate NGI factors across consecutive generations, such as a progressive increase in regulatory small RNAs (Houri-Ze'evi et al., 2016). A gradual change could also reflect the gradual increase in the frequency of a successful mutation (i.e. change in DNA sequence) or epimutation (i.e. change in epigenetic state) over successive generations (O'Dea et al., 2016). To distinguish between these two scenarios, for each population from the exposure experiment we constructed both a piecewise (immediate change followed by stability) and linear (gradual change) regression model for population growth over time and identified the model of best fit using the Akaike information criterion (i.e. model performance is assumed to differ when $\Delta AIC > 2$; (Burnham & Anderson, 2004). If the piecewise regression was found to be the best model, we used the Davies test to determine whether there was a significant difference in slopes before and after the breakpoint. As there was no evidence that breakpoint models performed better than linear models (Table S1), we explored linear trends in growth rate over time using linear mixed effects models (LMMs) for each clone by resource quality combination separately. In these four analyses, we treated time as a fixed effect and the population ID from the exposure experiment as a random effect. For the LMM analysis, the first day

of the exposure experiment was excluded to avoid any influence from previous culturing conditions.

Using an LMM, we tested the response of population growth rate to food exposure history (HP- vs. LP-exposed populations) and food quality treatments (HP vs. LP) of the clones at two time points since the start of exposure (common garden one and two). In this analysis, we used 'food exposure history', 'common garden diet', 'common garden experiment' and 'clone' as fixed effects. Individual populations (i.e. the 20 experimental units) of the exposure experiment were specified as a random effect. In *B. calyciflorus* generation time is longer in LP compared to HP food (Zhou et al., 2018). To standardize across diet treatments and common garden experiments, we only used population growth rate data of the fourth to sixth generations, corresponding with days 6–11 and 9–17 in HP and LP food respectively. By excluding the first three generations we aimed at eliminating the influence of maternal (Zhou & Declerck, 2020) and simultaneous exposure (Danchin, Pocheville, Rey, et al., 2019) effects. As we are interested in genotype specific fitness response patterns concordant with local adaptation, we used a priori contrasts to compare populations with HP- and LP-exposure histories for each genotype in each food treatment across common garden experiments.

All statistical analyses including model selection, a priori contrasts, and post-hoc tests were performed in the R software environment 4.0.2 (R Core Team, 2020). We performed LMM with the LME4 package (Bates et al., 2015) and piecewise regression using the package SEGMENTED (Muggeo, 2008). We obtained statistical significances from type III sums of squares using the CAR package (Fox & Weisberg, 2019) and we used Kenward–Roger degrees of freedom for the LMM. A priori contrasts were performed with the package EMMEANS (Lenth et al., 2019).

## 3 | RESULTS

### 3.1 | Part one: Exposure experiment

The piecewise regression model performed better than the linear model in only two of the 20 populations from the exposure experiment and in these cases, we found no significant differences between the slope before and after the breakpoint (Table S1). In both HP and LP exposure treatments the population growth rate of Clone-7 increased significantly over the course of the experiment (Figure 1; Table 1), by a factor of 46% and 34% in the HP and LP treatments respectively. In contrast, no significant slope was found for Clone-128.

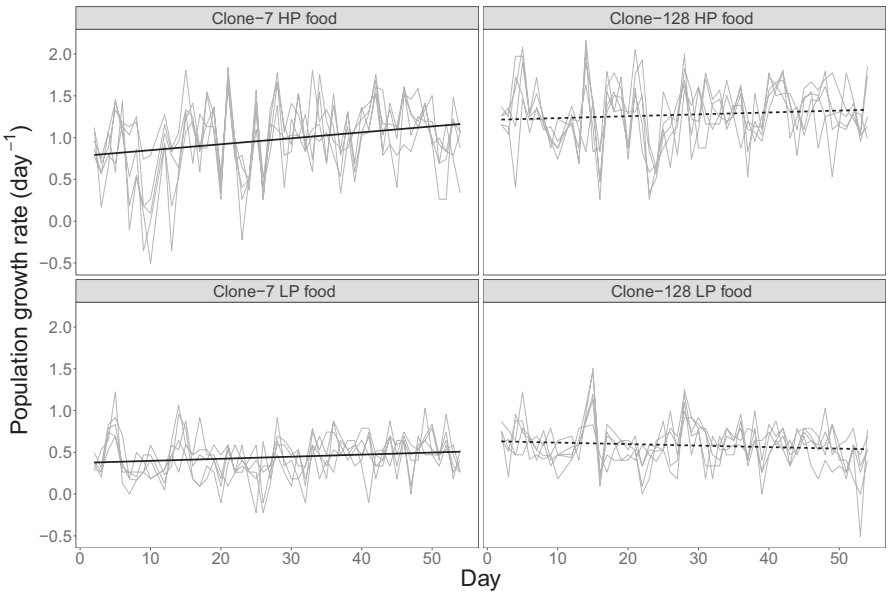


**FIGURE 1** Population growth rate over the course of the exposure experiment for the five replicate populations (grey lines) of Clone-7 and Clone-128 cultured with either a high (HP) or low phosphorus (LP) diet. A solid black line represents a significant change in growth rate over time, non-significant slopes are represented by a dashed black line (Table 1)

**TABLE 1** Summary of the linear mixed effects models describing population growth rate changes through time for each genotype (Clone-7 or Clone-128) in each treatment (HP or LP) of the exposure experiment (see also Figure 1). The effect of experimental unit was specified as a random effect. Bold *p*-values are significant

| Clone | Diet | Slope | SS | MS | Num *df* | Den *df* | *F* value | *p* |
|---|---|---|---|---|---|---|---|---|
| Clone-7 | HP | 7.12E−03 | 3.065 | 3.065 | 1 | 254.0 | 17.83 | **<0.001** |
| Clone-7 | LP | 2.48E−03 | 0.374 | 0.374 | 1 | 254.0 | 7.06 | **0.008** |
| Clone-128 | HP | 2.25E−03 | 0.307 | 0.307 | 1 | 254.0 | 2.54 | 0.112 |
| Clone-128 | LP | −1.80E−03 | 0.193 | 0.193 | 1 | 249.0[a] | 3.36 | 0.068 |

Abbreviations: Den *df*, denominator degrees of freedom; MS, mean squares; Num *df*, numerator degrees of freedom; *p*, *p*-level; SS, sum of squares.

[a]Data were not collected for all replicate populations of this treatment on day 14 of the experiment due to human error.

### 3.2 | Part two: Common garden experiment

The linear mixed model suggested two significant three-way interactions. The effect of exposure history on population growth rate differed between the HP and LP common garden diets, and this interaction differed between the first and second common garden experiment (Figure 2; Table 2: Diet * EH * CG). Furthermore, the interaction between exposure history and common garden diet also tended to differ between clones albeit marginally significantly (Figure 2; Table 2: Diet * EH * Clone).

The planned contrasts allowed us to investigate in more detail the effect of exposure history for the two genotypes in each food quality treatment (i.e. HP or LP) across the two common garden experiments. In the low phosphorus common garden treatment, Clone-7 populations with an LP exposure history had significantly higher population growth rates than populations with an HP exposure history (a priori contrast, LPexp vs. HPexp Clone-7 in LP: $p = 0.030$). However, we found no significant effect of exposure history on the population growth rate of these populations in the high phosphorus common garden treatment (a priori contrast, LPexp vs. HPexp Clone-7 in HP: $p = 0.597$). For Clone-128 there were no differences between populations with contrasting exposure histories in either the high or low phosphorus common garden treatment (a priori contrast, LPexp vs. HPexp Clone-128 in HP: $p = 1.0$; LPexp vs. HPexp Clone-128 in LP: $p = 1.0$). Thus, both three-way interactions seem to have been driven by the difference in response between LP and HP exposed populations of Clone-7 to LP food.

The effect of exposure history, as well as common garden food treatment, differed between the first and second common garden experiment (Figure 2; Table 2: EH * CG, Diet * CG). In both common garden experiments, population growth rate of the two clones was always substantially higher in the HP treatment compared to the LP treatment. Furthermore, population growth rate of Clone-128 was

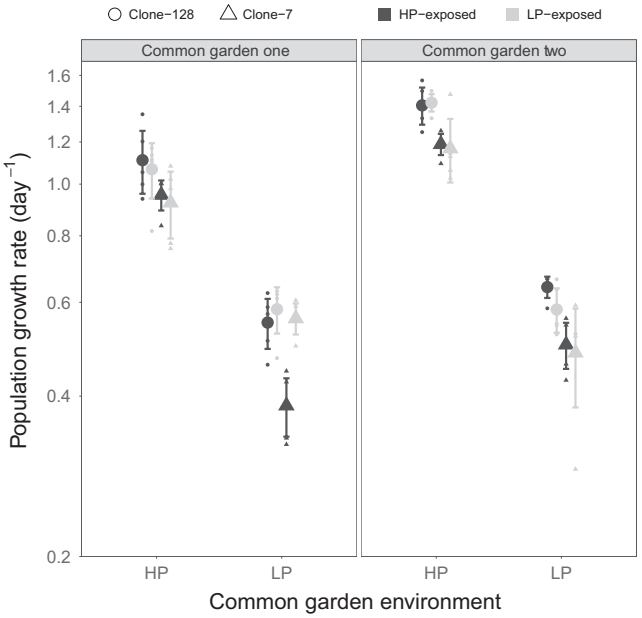


**FIGURE 2** Population growth rate for HP- and LP-exposed populations of Clone-7 and Clone-128 during the first and second common garden experiment with high (HP) and low phosphorus (LP) diets. We present the mean population growth rate over the fourth to sixth generation for each experimental unit (small symbols) as well as the grand mean ±2 standard errors ($n = 5$) for each clone × exposure environment combination (large symbols)

**TABLE 2** Summary of linear mixed effects analyses for the log-transformed population growth rate (see also Figure 2). The effects of common garden diet (Diet: HP or LP), exposure history (EH: HP-exposed or LP-exposed), genotype (Clone: Clone-7 or Clone-128) and common garden experiment (CG: CG1 or CG2) are presented as the fixed components of the model. The effect of experimental unit was used as a random component of the model. Bold *p*-values are significant

| Effect | SS | MS | Num *df* | Den *df* | *F* value | *p* |
|---|---|---|---|---|---|---|
| **Diet** | 2.24E+00 | 2.24E+00 | 1 | 48 | 894.897 | **<0.001** |
| Exposure history (EH) | 1.06E−03 | 1.06E−03 | 1 | 16 | 0.422 | 0.525 |
| **Clone** | 6.71E−02 | 6.71E−02 | 1 | 16 | 26.840 | **<0.001** |
| **Common garden experiment (CG)** | 9.15E−02 | 9.15E−02 | 1 | 48 | 36.576 | **<0.001** |
| Diet * EH | 8.29E−03 | 8.29E−03 | 1 | 48 | 3.316 | 0.075 |
| Diet * Clone | 2.30E−03 | 2.30E−03 | 1 | 48 | 0.921 | 0.342 |
| EH * Clone | 3.04E−03 | 3.04E−03 | 1 | 16 | 1.217 | 0.286 |
| **Diet * CG** | 3.25E−02 | 3.25E−02 | 1 | 48 | 13.009 | **0.001** |
| **EH * CG** | 1.65E−02 | 1.65E−02 | 1 | 48 | 6.611 | **0.013** |
| Clone * CG | 1.25E−03 | 1.25E−03 | 1 | 48 | 0.501 | 0.482 |
| **Diet * EH * Clone** | 9.96E−03 | 9.96E−03 | 1 | 48 | 3.983 | **0.052** |
| **Diet * EH * CG** | 2.61E−02 | 2.61E−02 | 1 | 48 | 10.454 | **0.002** |
| Diet * Clone * CG | 1.00E−05 | 1.00E−05 | 1 | 48 | 0.006 | 0.939 |
| EH * Clone * CG | 5.97E−03 | 5.97E−03 | 1 | 48 | 2.389 | 0.129 |
| Diet * EH * Clone * CG | 3.51E−03 | 3.51E−03 | 1 | 48 | 1.405 | 0.242 |

Abbreviations: Den *df*, denominator degrees of freedom; MS, mean squares; Num *df*, numerator degrees of freedom; *p*, *p*-level; Sum Sq, sum of squares.

always greater than Clone-7 (Figure 2; Table 2). Overall, population growth rates were significantly greater in the second compared to the first common garden experiment (Figure 2; Table 2).

## 4 | DISCUSSION

In the absence of initial standing genetic variation, our results demonstrate a rapid (six generations) heritable adaptive response to nutrient limitation by parthenogenetically reproducing monoclonal zooplankton populations. However, the capacity for a heritable shift in population growth rate differed between the two genotypes studied. Populations of Clone-7 displayed a gradual increase in population growth rate over the course of the exposure experiment consistent with local adaptation, and populations with an LP exposure history outperformed populations with an HP exposure history in the low phosphorus common garden treatment. In contrast, Clone-128 displayed stable population growth rates during the exposure experiment and there were no differences between populations with contrasting resource exposure histories in the common garden experiment. Contrary to prior studies (Sentis et al., 2018), our common garden experiments conducted at two successive time points provided no evidence that the length of exposure to the culturing conditions strengthened patterns of adaptation. The findings of our study contribute to a burgeoning set of empirical evidence that the generation of novel heritable variation is a significant contributor to adaptation to environmental change at short time-scales. This serves to further our understanding of how populations successfully establish and persist in changing environments despite little or no genetic diversity.

### 4.1 | Rapid heritable adaptation in response to exposure to low and high phosphorus resources

Our study provides empirical evidence for rapid heritable adaptation in a monoclonal population, challenging the conventional perspective that substantial initial standing genetic variation is necessary for such adaptation (Barrett & Schluter, 2008). In the low phosphorus treatment of the first common garden experiment, Clone-7 populations with an LP exposure history had higher population growth rates than populations with an HP exposure history. This pattern is partially consistent with the 'home versus away' criterion for local adaptation (Kawecki & Ebert, 2004) and is difficult to explain by intra-generational plasticity and anticipatory maternal effects (Marshall and Uller, 2007) because (i) differences in growth rate between populations with contrasting exposure histories lasted longer than four generations in the common garden low phosphorus treatment, and (ii) the maternal effect transmitted from low phosphorus mothers is known from previous research to be in the opposite direction from what we observed (Zhou & Declerck, 2020). A second striking result of our study that points at rapid adaptation, is the increase in population growth rate that occurred in Clone-7 populations over the course of the exposure experiment. This response suggests gradual adaption to the culturing conditions and contrasts with rapid phenotypic changes generated by both intra-generational and anticipatory maternal effects in response to stressful environments. As the founding individual of each clone line was hatched 8 weeks before the initiation of the exposure experiment, we cannot entirely discount the possibility that heritable phenotypic variants originated during batch culturing. Under these circumstances, the additional phenotypic variation could have accelerated the adaptive response we observed during the exposure experiment. However, the period during which this variation could have emerged was no longer than 71 days, and therefore well within an ecologically relevant time-scale for the study system.

Direct evidence of multigenerational exposure to a stressful environment producing heritable adaptation within initially genetically uniform populations is currently limited. In response to artificial disruptive selection during four or more generations, several studies have observed significant phenotypic differentiation among parthenogenetic lines of aphids or water fleas (*Daphnia*) that originated from a single foundress (Andrade & Roitberg, 1995; Bunting & Van Emden, 1980; Gorokhova et al., 2002; Wilhoit & Mittler, 1991). So far, using a single clone of the apomictically reproducing pea aphid *Acyrthosiphon pisum*, Sentis et al., (2018) were the only group to report a heritable response to natural selection. Our study provides support for their findings by demonstrating rapid heritable adaptation in a monoclonal population of a non-arthropod system. The results of our study thus indicate that the production of environmentally induced heritable phenotypes may be important for adaptation in a wide range of organisms. Furthermore, we observed a heritable adaptive response in less than half the number of generations than previous natural selection studies (Sentis et al., 2018). The rapid phenotypic change in response to varying environmental conditions observed in our study could likely have wide-ranging consequences on the eco-evolutionary dynamics of a system.

The underlying mechanism of the adaptation observed in this study is unknown. Advances in molecular genetic techniques have recently provided evidence that point mutation rates in multicellular organisms (e.g. Flynn et al., 2017; Ho et al., 2020) and ameiotic recombination in parthenogenetic organisms (Flynn et al., 2017; Omilian et al., 2006; Xu et al., 2011) may be significantly greater than previously assumed. Rates of DNA sequence altering events have been estimated to be as great as $10^{-5}$ per site per generation (Flynn et al., 2017). However, as the majority of these mutations are either negative or neutral, the estimated time frame for a beneficial mutation to emerge and go to fixation within a population still requires hundreds to thousands of generations (Danchin, Pocheville, Rey, et al., 2019). Thus, given that the observed phenotypic differences developed rapidly in very small replicate populations within a limited number of generations it is possible that the observed heritable phenotypic modifications were the result of a mechanism other than spontaneous mutations (Hermisson &

Pennings, 2005). One candidate mechanism that may have produced the observed adaptive response is an environmentally induced NGI factor that is transmitted for multiple generations even after the removal of the inducing environment. Transgenerationally stable epimutations or small RNAs have this capacity in some systems (e.g. Danchin, Pocheville, Huneman, 2019; Rechavi et al., 2014; Remy, 2010; Wang et al., 2017). Alternatively, a mechanism that generates rapid genetic variation upon stress exposure, such as transposable element proliferation, could be responsible (e.g. Miousse et al., 2015; Rebollo et al., 2010; Rey et al., 2016; Schrader & Schmitz, 2019). A speculative explanation for the observed gradual linear increase in the growth rate of Clone-7 in both exposure treatments is the accumulation of a non-genetic adaptive factor (e.g. small RNA) over consecutive generations of stressor exposure (Houri-Ze'evi et al., 2016). Further investigations are required to elucidate the molecular mechanism responsible for the observed adaptive response.

Traits that enable adaptation to one environment may come with costs in a different environment (Levins, 1968). However, we did not observe any difference in the relative performance of Clone-7 populations with contrasting exposure histories in the high phosphorus common garden treatment. This may indicate that traits favoured in LP conditions have low physiological costs. Alternatively, the benign nature of the HP common garden environment may have masked subtle trade-offs. Intriguingly, a similar pattern in performance has been observed in several other studies that have used common garden experiments to compare the fitness of LP- and HP-adapted populations (Declerck et al., 2015; Frisch et al., 2014; Lemmen et al., 2020).

## 4.2 | Clonal differences

The two clones used in this study consistently displayed different responses in both parts of the experiment. Previous work has shown that in a variety of organisms, genotypes differ in both their rates of DNA mutation (Ho et al., 2019; Ness et al., 2015) as well as in their ability to produce non-genetic heritable phenotypes (Leung et al., 2016; Menezes et al., 2018; Seudre et al., 2020). As such, we expected to observe differences between clones, however, the magnitude was larger than anticipated. The contrasting responses between clones imply the capacity to produce heritable phenotypes from a single genome is an inclusively heritable trait (Danchin & Wagner, 2010) suggesting that it could thus be subject to selection. This variation may reflect differences in the environmental stability of their source populations. Within-generation plasticity has been shown to be favoured when environmental change occurs either within, or between, adjacent generations (Botero et al., 2015). In contrast, intergenerational NGI of environmentally induced phenotypes is predicted to be favoured in environments that remain stable for several generations but fluctuate over longer periods, so that parental conditions have predictive value regarding those that will be experienced by the offspring (e.g. Herman et al., 2014; Jablonka & Raz, 2009; Klironomos et al., 2013; Lachmann & Jablonka, 1996; Rando & Verstrepen, 2007). This prediction was supported by Walsh et al., (2016) who found that in response to predation cues *Daphnia* from lakes with stable predation regimes displayed a small within generation shift in phenotype but a large intergenerational response. In contrast, *Daphnia* from lakes where predation varied within growing seasons displayed the opposite response. While environments that theoretically favour NGI appear to be common (Colicchio & Herman, 2020), empirical evidence connecting the temporal dynamics of environmental conditions to variation in the capacity of NGI in either laboratory or natural populations is limited. In our study, the two clones originated from different populations, and this may potentially have caused their contrasting responses to the same environmental stressor.

## 4.3 | Effect of exposure history with time

Differences in population growth rates between populations with contrasting exposure histories decreased rather than increased with the number of exposed generations (i.e. from the first to the second common garden). This was contrary to our expectations, as a previous study with genetically uniform populations demonstrated a positive relationship between the number of generations exposed to a stressful environment and the magnitude of the stress-induced phenotypic response (Sentis et al., 2018). As we compared between two time points only, instead of a series of observations, our power to detect directional changes in performance through time was limited. Furthermore, as the first and second common gardens were only differentiated by 10 generations, the time frame of this study may have been too brief to observe the anticipated increased difference between populations with LP- and HP-exposure histories. Instead, we observed that differences between populations with contrasting exposure histories decreased between the first and the second common garden experiment. Currently, we are unable to explain this trend.

## 4.4 | Ecological relevance

Low genetic diversity within populations frequently occurs and can be the result of both natural (Haileselasie et al., 2018; Louette et al., 2007; So et al., 2015) and anthropogenic processes (Roman & Darling, 2007; Schlaepfer et al., 2018). However, the long-term success of these populations is not well understood, given the potential for mismatch between phenotypes and the environment (Pérez et al., 2006). Our observation of rapid, heritable adaptation in the absence of initial genetic variation, may provide an explanation of how low-diversity populations may expand and persist in a variable environment. For example, the production of heritable phenotypes in small genetically isolated populations may allow them to persist by tracking environmental changes in a process akin to evolutionary rescue (O'Dea et al., 2016). Such mechanisms

may be particularly important in asexually reproducing organisms with the capacity for long distance dispersal, as they are likely to encounter environments that differ from where they originate. In such circumstances, the ability to produce offspring with a novel, potentially adaptive heritable phenotype would provide long-term fitness benefits during the colonization of empty patches due to priority effects (De Meester et al., 2002). These mechanisms may also play a role in the successful establishment of clonal invasive species (Gomes et al., 2016; Shi et al., 2018). However, the benefits of de novo heritable phenotypic variation are not restricted to clonal organisms, and they have also been speculated to enhance the adaptive capacity of sexually reproducing species (Rollins et al., 2015). Numerous field studies have found correlations between non-genetic variation and environmental divergence in both invasive (Schrey et al., 2012; Sheldon et al., 2018) and natural populations (Wogan et al., 2020). It has yet to be determined if such environmentally driven non-genetic differentiation is heritable, however, these studies hint at a possible role of NGI in adaptation and warrant further investigation. Given the unprecedented rate and magnitude of current environmental change, it is important that we understand all possible mechanisms that organisms may use to persist and adapt. We believe the production of de novo heritable phenotypic variation may play an underappreciated adaptive role, especially in populations with low genetic diversity.

Predicting the establishment success of potentially invasive populations is of great ecological and economic importance in an increasingly interconnected world (Kolar & Lodge, 2001). Genetically depauperate populations have resulted in both failed (e.g. Hairston et al., 1999) and incredibly successful invasions (e.g. Mergeay et al., 2006). However, it is generally unknown why some invasions succeed while others do not (Zenni & Nuñez, 2013). Our study demonstrates differences in the capacity of genotypes to produce de novo heritable variation. Such differences may contribute to the establishment success of populations, and accounting for this trait may improve our ability to predict the invasiveness of different native populations. For example, populations that display the capacity to produce a diversity of de novo heritable phenotypes could be classified as 'high invasion risk', and stricter measures for decontamination may be put in place for transport vectors originating from these populations (Fournier et al., 2019).

## 5 | CONCLUSIONS

Despite frequent speculation of the ecological and evolutionary importance of mechanisms for generating heritable phenotypic variation (Day & Bonduriansky, 2011; Jablonka & Raz, 2009; O'Dea et al., 2016) there is limited empirical evidence of adaptive responses, especially in animal populations (Hu & Barrett, 2017; Perez & Lehner, 2019). Our study provides a clear example of rapid heritable local adaptation in response to exposure to a stressful environment based on de novo heritable variation in an animal population. Although further investigations are necessary to determine the underlying mechanism, our findings impact how we think about the adaptive capacity of genetically depauperate populations in the face of a rapidly changing environment. As seen in previous studies (Menezes et al., 2018), the capacity to produce heritable phenotypic variation was genotype dependent, and furthermore, was only observed in populations with a low phosphorus exposure history. A better understanding of the mechanisms responsible for producing phenotypes is needed to provide insight into the observed genotypic differences, and their relevance in ecological processes. Thus far, all experimental investigations of adaptation in the absence of genetic variation have been performed either in microbes or in short-lived invertebrates (i.e. *D. pulex*, *A. pisum* and *B. calyciflorus*). To determine whether the adaptive response observed in this study is pertinent in other taxa, further investigations are needed with a wider diversity of organisms. Although standing genetic variation is considered essential for rapid heritable adaptation, we find that alternative sources of phenotypic variation may also play a role and could aid in the establishment and persistence of low-diversity populations.

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### CONFLICT OF INTEREST

The authors have no conflict of interest to declare.

### AUTHORS' CONTRIBUTIONS

K.J.F.V. and S.A.J.D. conceived of this experiment; K.D.L. analysed the data; K.D.L. and S.A.J.D. wrote the first draft of the manuscript. All authors contributed substantially to revisions.

### DATA AVAILABILITY STATEMENT

Data available from the Dryad Digital Repository https://doi.org/10.5061/dryad.qrfj6q5h5 (Lemmen et al., 2021).

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