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Disentangling demographic co-effects of predation and pollution on population dynamics

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In nature species react to a variety of endogenous and exogenous ecological factors. Understanding the mechanisms by which these factors interact and drive population dynamics is a need for understanding and managing ecosystems. In this study we assess, using laboratory experiments, the effects that the combinations of two exogenous factors exert on the endogenous structure of the population dynamics of a size-structured population of *Daphnia*. One exogenous factor was size-selective predation, which was applied on experimental populations through simulating: 1) selective predation on small prey, 2) selective predation on large prey and 3) non-selective predation. The second exogenous factor was pesticide exposure, applied experimentally in a quasi-continuous regime. Our analysis combined theoretical models and statistical testing of experimental data for analyzing how the density dependence structure of the population dynamics was shifted by the different exogenous factors. Our results showed that pesticide exposure interacted with the mode of predation in determining the endogenous dynamics. Populations exposed to the pesticide and to either selective predation on newborns or selective predation on adults exhibited marked nonlinear effects of pesticide exposure. However, the specific mechanisms behind such nonlinear effects were dependent on the mode of size-selectivity. In populations under non-selective predation the pesticide exposure exerted a weak lateral effect. The ways in which endogenous process and exogenous factors may interact determine population dynamics. Increases in equilibrium density results in higher variance of population fluctuations but do not modify the stability properties of the system, while changes in the maximum growth rate induce changes in the dynamic regimes and stability properties of the population. Future consideration for research includes the consequences of the seasonal variation in the composition and activity of the predator assembly in interaction with the seasonal variation in exposure to agrochemicals on freshwater population dynamics.

Keywords: population dynamics, pesticides, size-selective predation

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Introduction

Along the first half of the 20th century population ecologists were intensively debating for the predominance of exogenous (environmental, Andrewartha and Birch 1954) or endogenous (density dependent, Nicholson 1933) determinants of population dynamics. A main synthesis achieved in ecology was the recognition of the interrelated role of endogenous and exogenous factors in shaping the fate of population size and structure (Royama 1992, Berryman 1999, Turchin 2003). Further, a robust toolbox was developed for their detection as determinant of population dynamics (Royama 1992, Berryman 1999, Turchin 2003, Estay et al. 2014). Despite this, the experimental support for the putative connections between ecological mechanisms and the emerging regulation structure and dynamic has been rare (Benincà et al. 2008). In addition, the utility and predictive capacity of these tools is rarely used for sound ecological problems in which they could provide truly complementary perspectives (Arim et al. 2006).

Detection of feedbacks and interactions among determinants of species dynamics under a scenario of global change is a pressing issue in ecology (Brook et al. 2008). A shift in species composition of communities driven by extinctions and biological invasions, particularly of consumers (May et al. 1995), and a widespread presence of pollutants (Groom et al. 2006) are main components of global change. Understanding the effect of alternative pressures of consumers and pollutants on population feedbacks and its interaction with environmental conditions is essential to understand the mechanisms through which global change could impact communities (Garay-Narváez et al. 2013, 2014). Grazer populations in freshwater ecosystems are continuously subjected to fish or invertebrates predation, which strongly modulates their body size and age distribution due to preferences for larger or smaller individuals respectively (Brooks and Dodson 1965, Williamson 1986, Brandl 1998a, b, Arim et al. 2011, Quintana et al. 2015). In addition, pollutants are released in several freshwater systems with effects determined by the age or size of exposed organisms (Medina et al. 2002, Willis and Ling 2004, Hoang and Klaine 2007). Often, pollutant stress exerts stronger effects on younger or smaller individuals (Bodar et al. 1988, Forget et al. 1998). In general, the size or age dependency of predator and pollutant effects are expected to have consequences on the population dynamics of impacted species (Claessen et al. 2002, de Roos et al. 2003). Further, predation may interact with pollutants because their size-dependent nature determining non-linear effects on impacted populations (Gergs et al. 2013). Considering that several natural populations are being stressed predation and pollutant pressures in a size-dependent way, a better understanding of their dynamic consequences is needed.

Environmental factors by themselves cannot regulate population dynamics (Royama 1992). Indeed, the association between abundance and environmental conditions depends on the density dependent structure (Royama 1992). Understanding the effect of the environment on population dynamics depends on properly account for both endogenous and exogenous factors and their potential interaction (Stenseth et al. 2002, Lima et al. 2008). Exogenous factors could impact the density dependent (endogenous) factors with vertical, lateral or nonlinear changes in their structure (Royama 1992, summarized in Fig. 1). The endogenous structure essentially describes the relationship between the expected growth rate and the population abundance. For example, exogenous factors (e.g. pollutants) may affect survival and reproduction directly, causing what Royama (1992) calls 'vertical' perturbation effects in the relationship between per capita rate of change and population density. Consequently, it is possible to evaluate the effect of climate on the per capita rate of population change independently of population density (Royama 1992). This is the simplest type of perturbation by exogenous factors and it is common to observe in ectotherm organisms such as invertebrates where annual changes in temperature or winter severity affect directly survival and reproductive rates (Estay and Lima 2010, Estay et al. 2012). An alternative scenario occurs with 'lateral' perturbation effects where exogenous factors influences the availability of – or the requirements for – some limiting resource; hence, the effect of the exogenous factor can only be evaluated jointly with the effect of population density (Royama 1992). Therefore, the exogenous factor represents a non-additive force affecting the equilibrium population size (carrying capacity) without altering maximum growth rate. For example, the impressive rainfall-induced changes in resources of small rodents in semi-arid regions of Chile are an excellent example of this type of exogenous perturbation (Lima et al. 2006, Previtali et al. 2009). Finally, 'nonlinear' exogenous effect implies a change in the shape of function relating growth rate with abundance because the effect of the exogenous factor on the intensity of competition via the curvature of the population growth rate function (Royama 1992). A main consequence of this interaction between exogenous and endogenous factors is that a full understanding of the population-level effects of pollutants and predation depends on considering their interaction with endogenous factors.

The present study experimentally analyzes the concomitant effect of size-selective predation and pesticide exposure on the population dynamics of the water flea *Daphnia ambigua*. To this aim the effects of both the endogenous and exogenous factors were evaluated and contrasted among treatments. The interaction between exogenous and endogenous factors determined significant differences in the dynamics and final abundances among predation and pollutant treatments. As a whole, this study highlights the importance of a population dynamics perspective to advance on the mechanisms through which global change impact natural systems.

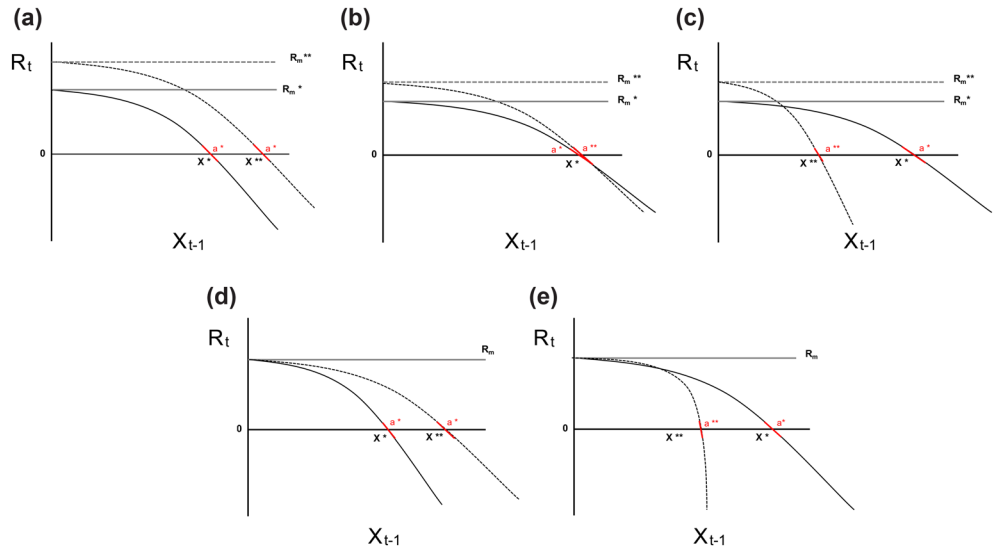

Figure 1. Royama's classification of exogenous perturbations to population dynamics, captured by the R-function. Vertical perturbations (a) shift the maximum reproductive rate and the carrying capacity additively, changing the relative position of the whole R-function along the y-axis in an additive way without changing parameters a and C. Lateral perturbations (b) shift the carrying capacity without altering parameters R_m and a, therefore the R-function shifts along the x-axis in a non-additive manner by changes in the individual requirements or the limiting resource availability (parameter C). Nonlinear perturbations (c–e) could affect together or independently the maximum reproductive rate, the individual interference and the intra-specific competition intensity, shifting the shape of the R-function in a more complex manner. Superscripts * and ** represent two different treatments to be compared.

Methods

Zooplankton and phytoplankton cultures

All experiments were conducted using a clone of *Daphnia ambigua* isolated from Peñuelas reservoir (33°07′S, 71°24′W), Valparaiso, Chile. Laboratory cultures of *Daphnia* were maintained in COMBO medium (Kilham et al. 1998) at 20 ± 1°C under a light:dark cycle of 16:8. The phytoplankton species *Pseudokirchneriella subcapitata* was used as food for *Daphnia* during maintenance and trials. *P. subcapitata* was cultured using Bristol medium (James 1978) in 2-l glass bottles constantly aerated through a 0.2 um filter. Prior to its use as food, the phytoplankton medium was removed through centrifugation at 3500 r.p.m. and resuspended in *Daphnia* medium.

Size-specific individual tolerance of *Daphnia* to methamidophos

The effects of the pesticide methamidophos (O, S-dimethyl phosphoramidothioate) on *Daphnia* were evaluated at individual and population levels. This pesticide belongs to the organophosphorus family and has been used worldwide due to its high efficiency against insect pests in agroindustry (Malato et al. 1999). Methamidophos exerts its harmful effect affecting the central nervous system by inhibiting acetylcholinesterase activity (Hussain 1987). Its effects have been reported in mammals, birds and terrestrial insects. It is also highly toxic for aquatic organisms, with $LC_{50\text{-}96}$ values between 25–51 mg l^{-1} for fish (Tomin 1994).

To compare the magnitude of pesticide effects across different *Daphnia* body sizes, we performed acute toxicity tests on newborn (< 1.2 mm), juvenile (1.2–1.5 mm) and adult (> 1.5 mm) individuals of *D. ambigua*. The experiment was performed modifying the OECD (2004) procedure to determine the 24h-LC_{50} and 48h-LC_{50} for *Daphnia*. Unlike the standard procedure, we fed the individuals during the experiment with 10^6 cell ml^{-1} of *P. subcapitata* in order to reproduce food conditions of the main experiments. Nominal concentrations of methamidophos were 0, 50, 100, 200, 400 and 800 µg l^{-1} (for newborns); 0, 5, 150, 300, 600, 1200 µg l^{-1} (for juveniles) and 0, 90, 180, 360, 720, 1440 µg l^{-1} (for adults). In each assay, five replicates were run per concentration, each one containing four individuals in 40 ml of medium. The LC_{50} values for methamidophos were calculated using probit analysis.

Main experiment

The main experiment was composed of three sequential phases. In phase 1 (transient), the experimental populations were maintained without manipulation during 12 days (determined in previous tests) to allow the stabilization of their abundances. In phase 2 (selective predation), the experimental populations were subjected during nine days to treatments of simulated predation, described below. In phase 3 (selective predation and pesticide exposure), pesticide exposure was applied simultaneously to simulated predation until the end of the experiment.

The experiment was started in phase 1 by disposing, for each of the 24 experimental units, 10 individuals

(4 newborns, 3 juveniles and 3 adults) in a 1-l flask filled with COMBO medium containing 10^6 cel ml^{-1} of *P. subcapitata* as food. During phase 2 and 3 the 24 experimental units were divided into three groups of eight units each. In each of these groups we conducted one of the three different predation treatments, consisting of removing by manual pipetting a given proportion of newborns, juveniles and adults of the *Daphnia* individuals. We defined the different *Daphnia* developmental stages according to their body sizes, as described in the previous section. The size classes were defined after building body growth curves, shown in the supporting information (Supplementary material Appendix 1 Fig. A1). Selective predation on smaller prey size (typical of invertebrate predators) was simulated through removing 30% of newborns. Selective predation on larger prey size (typical of vertebrate predators) was simulated through removing 30% of adults. Unselective predation was simulated through removing 10% of each of the three size classes. These predation treatments were applied every three days simultaneously, the media were renewed and the population densities were assessed to determine the number of individuals to be removed in each treatment. During the first days, when population densities were low, population densities were assessed by censuses. At higher densities, we counted the individuals in three 40 ml aliquots for each flask.

To determine a sublethal pesticide concentration to be used in phase 3, we conducted a chronic bioassay with methamidophos concentrations below the acute 48h-LC_{50} value of newborns, which resulted to be the most sensitive size class. The chosen concentration of pesticide to be used in phase 3 was 80 mg l^{-1}, for being higher than the lowest observable effect concentration (LOEC = 60 mg l^{-1}) determined in the chronic assay, and just high enough to reduce reproduction in about 50% in (Supplementary material Appendix 1 Fig. A3). The experiments were finished after 121 days, when populations reached a stable age structure, tested by the statistical tools described in the following section. In summary, we conducted a factorial experiment of two levels of pesticide exposure (80 and 0 mg l^{-1} of methamidophos), three levels of predation modes (selective to small prey, selective to large prey, unselective), with four replicates in each pesticide/predation combination.

Asymptotic analysis of population structure

Once completed the phase 2, χ^2-tests were performed to evaluate differences in the size structure of populations. Pairwise comparisons were made between the size structure reached in phase 1 and the size structure reached in phase 2 by each replicate within the three predation treatments.

In phase 3, χ^2-tests were performed to contrast the size distribution at time *t* with the final size distribution. The detection – or not – of a significant difference ($p < 0.05$) was related with time by means of a logistic regression. This regression was used to determine the time at which populations in each treatment reached their stable size structure (counts of newborns, juveniles and adults), which indicated the beginning of the asymptotic dynamics of phase 3.

We also tested for differences in the asymptotic size structure between exposed and unexposed populations under each predation treatment. This was done by a permutation test, randomly assigning (10 000 runs) newborns, juveniles and adults between control and exposed treatments. At each run, the difference between size structures between control and exposed treatments was assessed through the χ^2 metrics. If the observed difference between the control and the exposed treatment in the actual data (χ^2_{obs}) exceeded the 95% of the distribution of randomized χ^2 values (χ^2_{p95}), significant differences were accepted at the 5% level.

Finally, one-way (for phase 2) and two-way (for phase 3) ANOVA were used to test differences in mean population densities. Post hoc Tukey-HSD test was used to perform specific comparisons.

Analysis of population dynamics

To determine the endogenous structure of *D. ambigua* population dynamics, we used the Ricker's (1954) equation, which is the exponential form of the discrete logistic model, to model the R-function from our empirical population data (Berryman 1999). R-functions represent the relationship between the realized per capita population growth rates and population abundance, reflecting the processes of individual survival and reproduction (Berryman 1999). This allowed us to model the basic influences of endogenous and exogenous forces on these dynamics.

To model the R-function and the effects on it exerted by pesticide exposure as an exogenous factor, we used the following density-dependent population dynamics model Ricker (1954):

$$N_t = N_{t-1} r_m \exp[-c N_{t-1}^a] \quad (1)$$

where N_t represents the population abundance at time t, r_m represents the maximum finite reproductive rate, c represents the strength of the intra-specific competition which is directly proportional to the individual requirements for limiting resources and inversely related to the abundance of limiting resources. Numerically, c represents the amount of change in $\log(N_t/N_{t-1})$ with the addition of one individual to the population. When $a = 1$ this effect is equal for all abundances, if $a<1$ the effect of each additional individual on population growth rate attenuates with abundance, and the opposite is true when $a>1$. Consequently, parameter a indicates how self-interference is modified as density increases (Royama 1992). By defining Eq. 1 in terms of the R-function and applying log-transformation we obtain:

$$R_t = R_m - \exp[aX_{t-1} + C] \quad (2)$$

where R_t is the realized per capita growth rate $R_t = \log_e(N_t/N_{t-1})$, $R_m = \log_e(r_m)$, a comes from Eq. 1, $C = \log_e(c)$, and $X = \log_e(N)$. Using this equation and solving for the

equilibrium log density ($R_t = 0$), the log transformed carrying capacity of the system can be defined as:

$$X^* = \frac{\log_e(R_m) - C}{a} \quad (3)$$

Royama (1992) classified the responses of R-functions to exogenous forces by introducing three categories of perturbations: vertical, lateral and nonlinear ones (see Fig. 1 for a graphical explanation).

We fitted Eq. 2 to our data of R_t versus X_{t-1} by means of nonlinear regression analyses (Bates and Watts 1988) using the nls library of R (<www.r-project.org>). Both R_t and X_{t-1} values were empirically assessed for each replicate in all treatments by direct counting. For each replicate, R-function parameters were estimated from our abundance data during experimental phase 3. Parameter values were then compared among treatments by paired t-test.

Data deposition

Data available from the Dryad Digital Repository: <http://dx.doi.org/10.5061/dryad.4jt80p4> (Reyes et al. 2018).

Results

Size-specific individual tolerance to methamidophos

Acute toxicity test showed that tolerance to methamidophos, measured through both 24 h and 48 h LC_{50}, increased with age/size (Supplementary material Appendix 1 Fig. A2).

Asymptotic analysis of population size-structure

Population trajectories of *Daphnia ambigua* (all size classes lumped) under the two levels of pesticide exposure and the three size-selective predation modes are shown in Fig. 2. All *D. ambigua* populations exhibited a phase of exponential growth during the first 12 days, reaching a peak density of about 600–700 individuals l^{-1} (Fig. 2). Newborns were the most abundant size class during phase 1, while juveniles and adults did not differ in their densities (Supplementary material Appendix 1 Fig. A4a). In Supplementary material Appendix 1 Fig. A5–7 we show the trajectories for all treatments, disaggregated by age classes.

In phase 2, when simulated size-selective predation was applied, population density decreased and then oscillated around 300 individuals l^{-1} (Fig. 2). One-way ANOVA showed significant differences in asymptotic mean density among predation modes ($F_{2,21} = 16.2$, $p < 0.001$), while Tukey-HSD test showed that selective predation on adults led to higher densities, as compared to the others predation treatments ($p < 0.001$; Supplementary material Appendix 1 Fig. A4b). In this phase, a higher density of the adult class was observed in populations under selective predation on newborns. In contrast, populations under selective predation on adults were dominated by newborns. In populations under non-selective predation, slightly higher abundances of newborns were obtained (Supplementary material Appendix 1 Fig. A4a). The χ^2 analysis showed that size structure reached in populations at phase 1 differed significantly from the size structure reached in phase 2. This difference exerted by predation held for populations under selective predation on newborns ($\chi^2 = 42.56$, $p < 0.0001$), also for populations

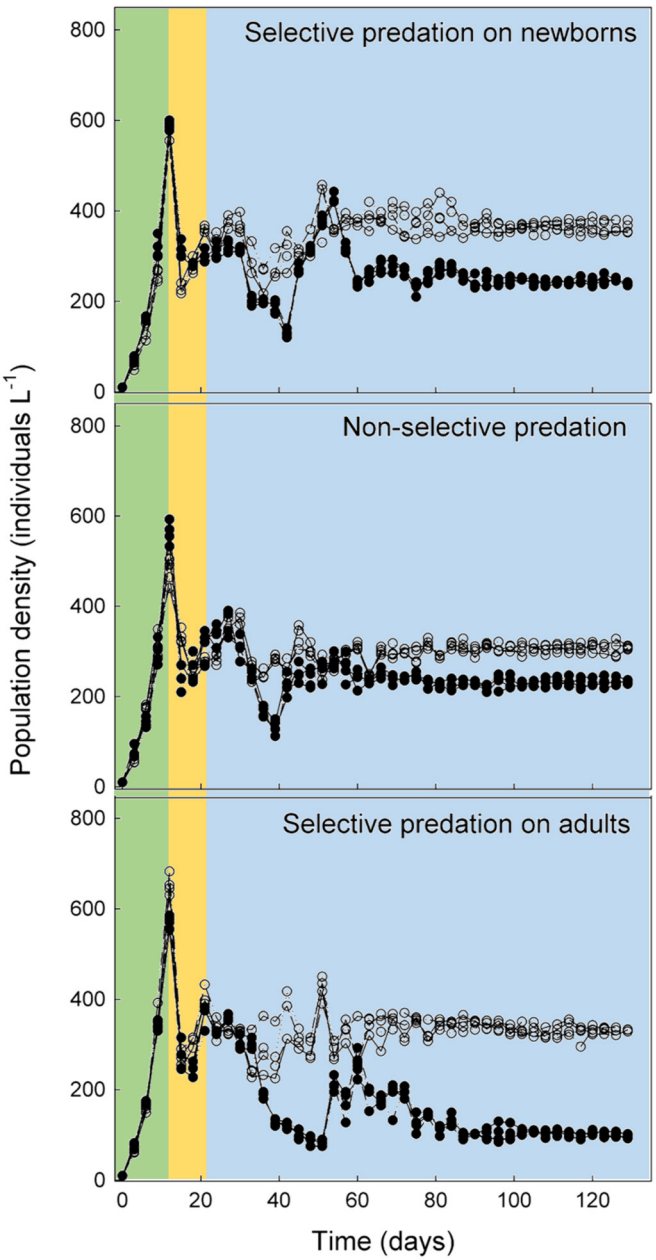

Figure 2. Time series of *D. ambigua* population density under selective predation on newborns (upper panel), non-selective predation (middle panel) and selective predation on adults (lower panel). Open and filled circles represent populations not exposed and exposed, respectively, to the pesticide methamidophos. Colored areas represent phase 1 (growing populations, green shading), phase 2 (simulated predation, orange shading) and phase 3 (simulated predation and pesticide exposure, light blue shading).

subjected to non-selective predation ($\chi^2 = 8.039$, $p < 0.05$) and for selective predation on adults as well ($\chi^2 = 6.73$, $p < 0.05$). Furthermore, significant differences were observed in phase 2 among size structures generated by the different predation treatments ($\chi^2 > 20$, $p < 0.001$ for all pairwise comparisons). Therefore, the three patterns of simulated predation effectively generated different size structures in the experimental populations.

In phase 3, after the addition of methamidophos to the corresponding treatments, two-way ANOVA showed that predation mode ($F_{2,354} = 2238.5$, $p < 0.001$), pesticide exposure ($F_{1,354} = 18076.8$, $p < 0.001$) and their interaction ($F_{2,354} = 1889.1$, $p < 0.001$) affected significantly the asymptotic population density (Fig. 3). Under all predation treatments, the populations exposed to the pesticide exhibited lower population densities (Tukey-HSD test, $p < 0.05$, Fig. 3). During phase 3 all populations showed an initial decrease followed by an increase in densities (Fig. 2). However, populations under selective predation on adults were the most affected relative to control, showing the strongest reduction in asymptotic density as compared to each other predation treatment (Fig. 2). Conversely, populations under non-selective predation exhibited the weakest reduction in total density (Fig. 2).

The permutation analysis showed that pesticide exposure caused a marked shift in size structure of populations under selective predation on adults ($\chi^2_{obs} = 19.04$, $\chi^2_{p95} = 13.10$, $p < 0.05$), reversing the dominance of newborns (Fig. 3c) observed during phase 2 (Supplementary material Appendix 1 Fig. A4), as well as during phase 3 in control treatments (Fig. 3c). Also, our analyses revealed a minor but significant difference in size structure for populations under non-selective predation ($\chi^2 = 6.58$, $\chi^2_{p95} = 4.86$, $p < 0.05$, Fig. 3b). For selective predation on newborns no differences in population structure were driven by pesticide exposure ($\chi^2 = 1.18$, $\chi^2_{p95} = 1.232$, $p > 0.05$, Fig. 2a), relative to unexposed populations. In summary, pesticide exposure exerted particular effects on populations depending on the predation treatment applied. Under predation on, the pesticide strongly reduced population density and markedly shifted the population structure, from a size distribution heavily dominated by newborns to a rather uniform one. Under non-selective predation, both population density and population structure were only slightly affected. Finally, under predation on newborns, population density was reduced but population structure was unaffected. These results indicate a strong interactive effect of pesticide exposure and selective predation mode, on asymptotic population properties.

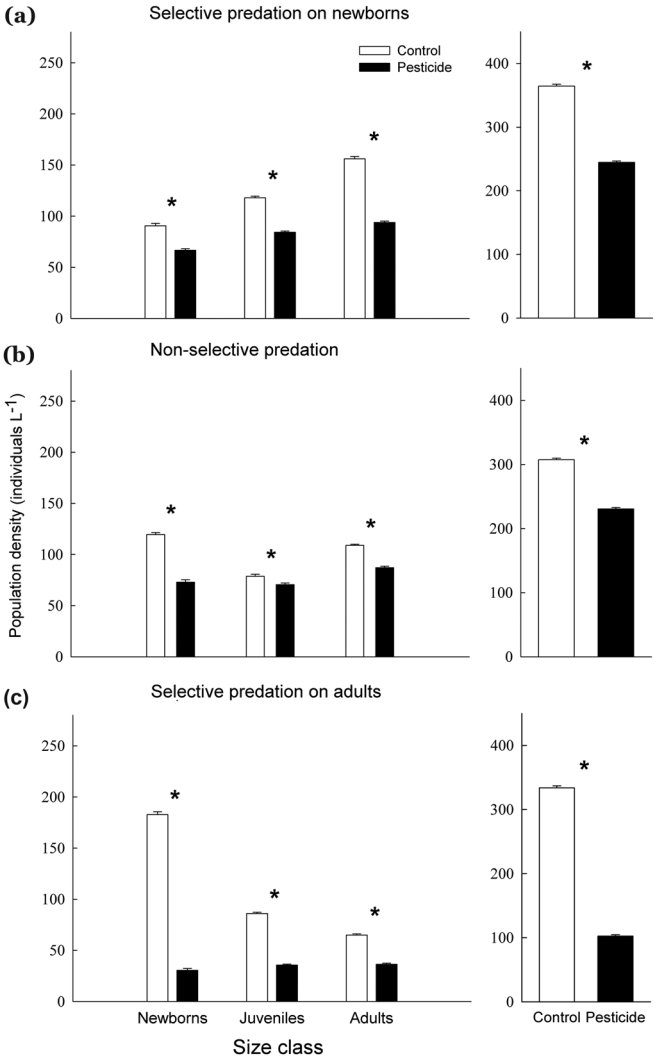

Figure 3. Mean asymptotic density ± 95% CI for each size class (left panels) and total population density (right panels) during phase 3 for populations unexposed (open bars) and exposed (filled bars) to the pesticide, under the three experimental predation modes: (a) selective predation on newborns, (b) non-selective predation and (c) selective predation on adults. Asterisks indicate significant differences among control an exposure treatments (Tukey-HSD test, $p < 0.001$).

Population dynamics

R-functions were constructed by fitting the realized per capita growth rate at time t (R_t) to the logarithm of total abundance at time t-1 ($\log N_{t-1}$). Parameters of each fitted curve were all significant ($p < 0.005$ in all cases) and the pesticide exposure, depending on the predation mode, exerted particular effects on the R-function that we explain below.

In terms of Royama's classification the pesticide exposure exerted nonlinear effects in populations under selective predation on newborns and under selective predation on adults (Fig. 4a, c). However, the mechanisms that led to changes in population dynamics were different for each treatment. In populations under selective predation on newborns, the changes in population dynamics exerted by pesticide exposure were driven by a significant decrease in the maximum reproductive rate R_m ($t = 8.85$, $p < 0.001$, Supplementary material Appendix 1 Fig. A11a), while maintaining without significant differences the values of the parameters representing individual interference a ($t = -1.18$, $p = 0.28$, Supplementary material Appendix 1 Fig. A11b) and

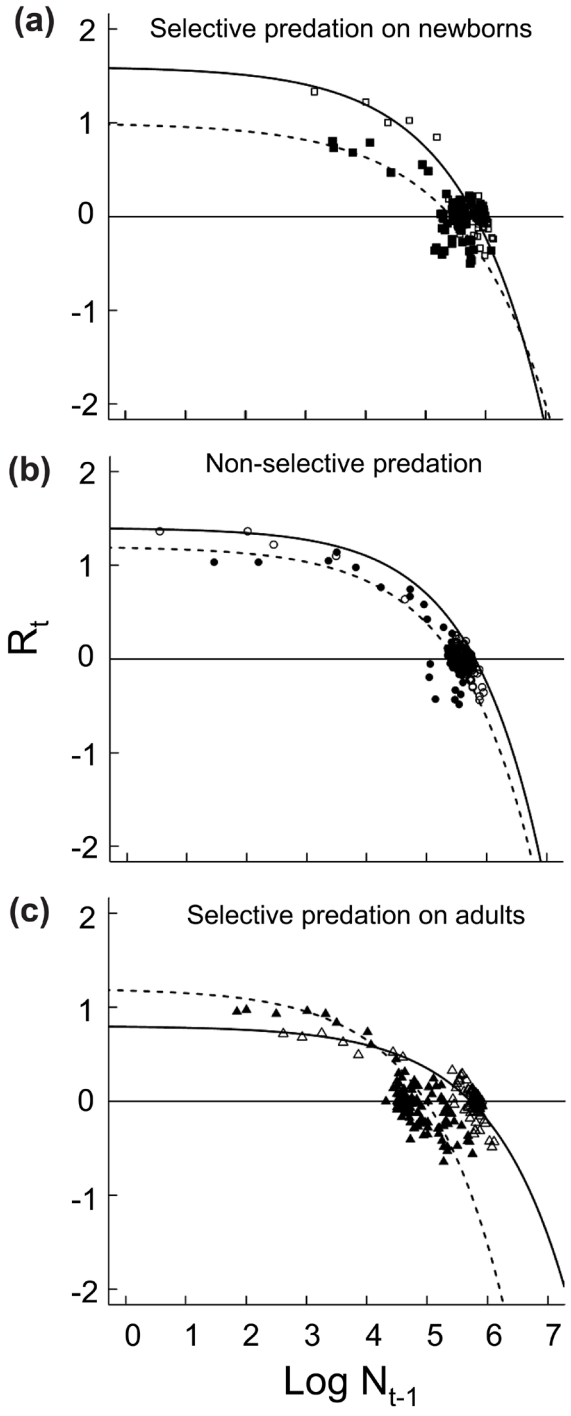

Figure 4. Estimated R-function of *D. ambigua* populations in experimental phase 3, subjected to selective predation on newborns (upper panel), non-selective predation (middle panel) and selective predation on adults (lower panel). Unexposed (open symbols and solid fitted lines) and exposed (closed symbols and dashed fitted lines) populations under each predation mode are shown.

competition intensity C ($t=1.22$, $p=0.27$, Supplementary material Appendix 1 Fig. A11c). According this result we expect a vertical additive effect, but the graphs showed a displacement only in the intercept of the R-function downward along Y-axis (Fig. 4a), which defines a nonlinear effect, suggesting that a and C showed some compensatory changes (Fig. 1e, 4a). Under non-selective predation, populations under pesticide exposure showed a small but statistically significant increase in the intensity of intra-specific competition (a decrease in C) ($t=-9.75$, $p < 0.001$, Supplementary material Appendix 1 Fig. A11c), while values of R_m ($t=0.69$, $p=0.53$, Supplementary material Appendix 1 Fig. A11a) and a ($t=1.50$, $p=0.11$, Supplementary material Appendix 1 Fig. A11b) did not show significant changes. Therefore the R-function plots suggest a weak lateral effect of pesticide exposure (Fig. 4b). Finally, in populations under selective predation on adults, the changes in population dynamics exerted by pesticide exposure were driven by a significant increase in R_m ($t=-8.40$, $p < 0.001$, Supplementary material Appendix 1 Fig. A11a) and a significant decrease in C ($t=-22.86$, $p < 0.001$, Supplementary material Appendix 1 Fig. A11c), maintaining without significant differences the value of parameter a ($t=1.54$, $p=0.17$, Supplementary material Appendix 1 Fig. A11b). These changes determined a displacement in the R-function up on the y-axis and leftward on the x-axis (Fig. 4c), which defines a clear nonlinear effect.

Discussion

In this study, we advanced on the mechanistic connection between changes in size structure by predation, its effect on the vulnerability to pollutants, and its dynamic consequences, explicitly identifying the involved shifts in the regulation structure of populations. Changes in the population size structure resulting from size-selective predation interact with pesticide exposure – which also exerts size-dependent effects – to shape population dynamics of *Daphnia ambigua*. Our results revealed that pesticide exposure exerted profound changes in the structure of populations subjected to size-selective predation either on adults or newborns. We evaluated pesticide effects on population dynamics through its impact on the parameters that govern the shape of the R-function: maximum reproductive rate (R_m), competition intensity (C) and individual interference (a). Our analysis of R-functions revealed that size selective predation led to non-additive effect while non-selective predation led to weak effects on population dynamics.

Our experimental analysis of the effect of predation on zooplankton size structure is congruent with a profuse literature on the topic. In the initial phase of our experiment all populations showed a dominance of smaller individuals, which is characteristic of growing populations under high food availability and weak intraspecific interactions (Hanazato and Hirokawa 2004, Takahashi and Hanazato 2007, Gergs et al. 2013). In phase 2, predation treatments determine that the population structure of *D. ambigua* was shifted to an accumulation of smaller or larger individuals, resembling the natural changes exerted either by fish

(selective predation on adults) or invertebrates (selective predation on newborns) in natural freshwater populations (Zaret 1980, Brett 1992, Gliwicz et al. 2010, Quintana et al. 2015, Viaene et al. 2015). Conversely, the non-selective predation treatment mirrored the pressure exerted by a mixture of vertebrate and invertebrate predators (Hanazato and Yasuno 1989, Arim et al. 2011). The greater effects of predation pressure were observed in populations under selective predation on adults, in which density of newborns predominated likely due to lower intraspecific competition among adults (Glazier 1992, Preuss et al. 2009).

Main effects of pesticides translated into lower population densities, as compared to unexposed populations, regardless of predation treatment (Fig. 2). This negative effect was consistent across replicates and was not damped through time. Stable population densities were reached after about 60 days (predation on newborns), 70 days (non-selective predation) and 80 days (predation on adults) once initiated experimental phase 3, following an initial unstructured impact driven by the pesticide exposure. Moreover, our results advance on the population consequences of the interplay between the well-reported effect of size-dependent predation and the also size-dependent effect of pesticide (Supplementary material Appendix 1 Fig. A2). Stronger negative effects of pesticide on population density were observed when predation focus on adults. These populations were predominantly composed of smaller individuals. This reduction is congruent with our results at the individual level, where smaller individuals were less tolerant to methamidophos and with previous report about a higher sensitivity of smaller sizes cladocerans to pesticide exposure (Klein 2000, Takahashi and Hanazato 2007). The populations under selective predation on newborns did not exhibit the same response, pointing to a different mechanism as compared to the populations under adult predation. In populations with predation on newborns, pesticide exposure decreased the relative representation and abundance of individuals of larger size classes. In size-structured populations with maturation rate larger than recruitment, at equilibrium juveniles may operate as a source of biomass and adults as a sink (de Roos and Persson 2013). Congruently, a lower transition rate from smaller to larger individuals under pollutant exposure (Liess et al. 2006, Liess and Foit 2010) is expected to reduce the relative representation of adults. Further, pollutant effects may be exacerbated under conditions of high crowding, as is the case of newborns and non-selective predation treatments (Hanazato and Hirokawa 2004, Takahashi and Hanazato 2007). However, a recent study carried out with selective predation by invertebrates together with short-term pesticide exposure revealed greater effects on abundance in populations with dominance of smaller individuals, in contrast to populations composed by larger ones (Gergs et al. 2013).

A main difference between the experiment conducted by Gergs et al. (2013) and ours rest in the degree of experimental control of predation mode. In the study of Gergs et al. (2013), larvae of *Notonecta maculata* were added at the beginning the experiment, which were growing and exerting different modes of size-specific predation as long as the experiment progressed. Early in their experiment the selective predation on small daphnids led to a shift in population structure to larger daphnids so that only a small proportion of the prey population was affected by the pesticide. Second, later in their experiment a selective predation on larger daphnids led to a population structure biased towards smaller individuals, thus a larger proportion of the population was affected by pesticide exposure. This contrasts with our experimental design, in which we simulated an even predation pressure on each population, which allowed us to isolate more clearly the effects of different predation modes on pesticide exposed population demography.

Our analysis of the R-function parameters revealed that pesticide-exposed populations subjected to selective predation on newborns decreased their maximum reproductive rate, although it could be found some small compensatory changes in the competition intensity and individual interference coefficients, as compared to control populations. This effect could be due to the use of time and energy by the remaining individuals on repair processes before regaining their reproductive capability (Liess 2002, Beketov and Liess 2005, Foit et al. 2011). In a population mostly structured by adult individuals, pollutants seem to reduce reproductive rates, thus decreasing R_m. The change in equilibrium density was less than the expected from a pure vertical perturbation effect, possibly due to small decreases (non-significant) in the intra-specific competition intensity. In contrast, exposed populations under selective predation on adults showed the opposite effect: an increase in the competition intensity that could be due to higher individual requirements for survival of smaller individuals in a stressed environment. The higher R_m values suggests that despite of pollutants, populations at low densities are capable to reach high growth rates sustained by high reproductive rate, due to resource release after removal of large sized individuals from the population. The noticed increase in R_m at higher pollutant stress can be interpreted as an overcompensation effect in production rate and deserves special consideration. De Roos and Persson (2013) showed that, in structured population, this effect can be expected as a result of a release from intraspecific competition, commonly driven by adult harvesting in circumstances when large-bodied individuals are competitively superior, as shown for *Daphnia* (Gliwicz 1990). In addition, changes in maximum growth rate may emerge from changes in the life history of prey in response to predation, as those related with differences in allocation for growth or reproduction among size classes (Beckerman et al. 2007, Cressler et al. 2010). However, such overcompensation observed in our empirical tests is not fully explained by existing theory. Specifically, our results focus on the dynamic consequences of the interaction between stage dependent predation and physiological conditions determined by pollutants. Previous analyses mainly focused on only one those determinants (Abrams and Rowe 1996, Beckerman et al. 2007, Cressler et al. 2010, de Roos

and Persson 2013). Consequently, the connections between our results and previous models are not straightforward. Secondly, physiological and behavioral hypotheses usually focus on predator cues as determinants of changes in life history and population dynamics (Beckerman et al. 2007, Cressler et al. 2010). Our experiment exclusively considered the lethal effect of predation. Finally, it is not well defined the empirical relationship between overall intraspecific competition intensity, size-specific competition intensity and production of newborns under two opposing size-selective pressures, such as the ones explored here: predation on larger individuals and pollutants with stronger impact on smaller individuals.

We observed that size-selective predation, either on smaller or on large size classes, drove stronger impacts to the population dynamics, as compared to non-selective predation. Populations exposed to pollutant under both size-selective predation treatments showed nonlinear effects in their R-function but differed in the mechanisms by which the endogenous dynamics was altered by the exogenous force. In contrast, populations under non-selective predation exhibited a weak effect of pesticide exposure. The ways in which endogenous process (population feedbacks) and exogenous factors (environmental perturbations) may interact have implications for the resulting population dynamics and for the response of a species to environmental conditions (Johst and Drechsler 2003, Owen-Smith 2011). The evidence suggests that changes in equilibrium density driven only by the parameter C do not modify the stability properties of the system (Royama 1992). On the other hand, changes in the maximum growth rate or in the interference coefficient (a) could induce changes in the dynamic regimes and stability properties of the population (Estay et al. 2012). In our experiments, the mode of predation pressure at which *Daphnia* were subjected determined the type of impact the pesticide exposure exerted on the endogenous population dynamics; however, the observed shifts in parameter estimates led to changes in the magnitude of the reduction of equilibrium population density but not to changes in the stability of the system.

Previous assessments of the interplay between exogenous factors and endogenous dynamics using the Royama's framework had considered a few types of environmental factors such as food fluctuations, weather and predation (Lima and Berryman 2006, Lima et al. 2008, Previtali et al. 2009, Johst et al. 2012). We observed how the interaction between pollution and selective predation modulate the qualitative properties of population dynamics and could help to extend the view of exogenous perturbation by incorporating in the same and simple framework the effects of anthropogenic stressors on population dynamics of freshwater ecosystems. In spite of the main synthesis about the interrelated role of endogenous and exogenous factors in population dynamics (Royama 1992, Berryman 1999, Turchin 2003), few studies have experimentally explored the connection between the density-dependent structure and exogenous forces (Costantino et al. 1997, Benoît et al. 1998, Benincà et al. 2008, Estay et al. 2009). In addition, the robust methodological framework of population dynamics is rarely applied in systems other than population time series from field data (Arim et al. 2006, Gotelli et al. 2017). Our study contributed both in expanding the range of ecological phenomena analyzed with the population dynamics theory and in the experimental detection of the underlying mechanisms connecting exogenous and endogenous determinants of population dynamics.

We focused on some scenarios of predation and pollutant effect over the wide range of effects that has been reported. We considered than predation determines the mortality of 30% of the individuals in the vulnerable size classes. This represents a type 1 functional response since the number of individuals removed is proportional to prey abundance (Turchin 2003). For predatory species on zooplankton that are not limited by prey abundance and/or have not feedbacks involved in the range of prey abundances, this is a plausible scenario (Arim et al. 2010). In addition, trait mediated effect are a main component of species interactions in which direct mortality may be not involved (Schmitz et al. 2004). Conversely, our treatments with pollutant exposure, although at low doses, may express the combined action of lethal as well as nonlethal effects of the toxicant. Decomposing effectively both sources of pollutant effects is experimentally challenging when the responses of interest are at the population level. Therefore, our study focused on the pure lethal effects of predation along with predominantly sublethal effects of pesticide exposure. Mortality effects of pollutants, a wider range of non-lethal effects and the interaction between pollutant and food web structure (Garay-Narváez et al. 2013, 2014), may have different dynamic consequences that should devote future attention. In this context, experimentally exploring the dynamic consequences of alternative functional responses, population feedbacks, and the whole set of predatory and pollutant effects is emerging as a promising area of research. This is particularly relevant in a world progressively more polluted, with changes in species composition and with significant trends in exogenous forces driven by climate change.

We conclude that aquatic populations in natural environments could respond in different ways to pesticide exposure depending on size structure, and ultimately to diversity of the dominant predators. In presence of fish, which are characterized by a selective predation towards larger size classes, *Daphnia* population dynamics could be affected by strongly reducing population densities and shifting size structure, through changes in both the interaction between individuals and the population reproductive rate. On the other hand, a selective predation on smaller prey sizes as the one exerted by invertebrates or a non-selective predation which could be present at low fish densities and presence of invertebrates, promote changes in population dynamics by exerting lower decrease in population densities, through changes in the reproductive rate of populations and in the interaction between individuals, respectively. Our work provides

evidence of how two exogenous factors interact with the endogenous dynamics of populations, while several scientific evidence using Royama's framework evaluates a single exogenous effect on population dynamics. In addition, the results of this study can be used to speculate how the effects of selective predation may emerge in simple models of population dynamics. The classic studies of Abrams and Rowe (1996) and more recently Beckerman et al. (2007) and Cressler et al. (2010) have explored the consequences of selective predation on the life history and behavior of organisms. It will be interesting to connect the potential effects of different types predation risk on optimal age and size at maturity and foraging behavior with Royama's classification of vertical, lateral and non-linear effects. Of further consideration for research is the consequences of the seasonal variation in the composition and activity of the predator assembly in interaction with the seasonal variation in exposure to agrochemicals, and the potential shifts expected for these seasonal trends as a product of climate change. Through a deeper understanding of the interactive effects of natural and anthropogenic stressors we could advance our ability to manage natural systems.

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Supplementary material (available online as Appendix oik-05450 at www.oikosjournal.org/appendix/oik-05450>). Appendix 1.