Ultra-low Esfenvalerate Concentrations Increase Biomass and Reduce Competitiveness of *Daphnia magna* Populations

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Abstract

Aquatic communities are frequently exposed to pesticides at sublethal concentrations, known to affect fitness parameters like feeding, reproduction and population growth. Beside adverse effects, also beneficial responses to toxicants at low concentrations may occur. Such positive effects, however, are assumed to generate trade-offs. To identify such trade-offs, we quantified the population level effects on *Daphnia magna* during population carrying capacity in laboratory nanocosms after exposure to a single pulse of the pyrethroid insecticide esfenvalerate, including ultra-low concentrations $\leq {}^{1}/_{30}$ EC_{50}. Population abundance and biomass were monitored 3 times per week for 3 months with a non-invasive image detection technique. High concentrations $\geq {}^{1}/_{10}$ EC_{50} led to reduced fitness endpoints. In contrast, ultra-low concentrations in the range of 0.01 µg/L significantly increased the population abundance and biomass up to 200 % during 2 months after exposure. Already in the first five days after exposure to 0.01 µg/L and 0.03 µg/L esfenvalerate, population biomass increased by 0.1 mg/day while staying constant in the controls. We hypothesize that the reduction of individual competitiveness and consequent reduction of *intra*specific competition is the associated trade-off of the observed hormetic increase of population biomass.

Keywords: sublethal population effects, risk assessment, system-stress (SyS), multiple stress, high-frequency monitoring, hormesis

Abbreviations

ADaM – Aachener Daphnien Medium, DMSO – dimethyl sulfoxide, GC-MS/MS – gas chromatography–tandem mass spectrometry, PDI – posterior density interval

1. Introduction

Current environmental risk assessment (ERA) aims to safeguard species and populations in the environment by combining exposure and effect assessment. The concentrations at which adverse effects of pesticides occur in toxicity tests are reduced with assessment factors to the extent that populations in the field should also be protected. Field-studies, however, show that the ecological status of most streams with an agricultural catchment are affected by pesticides also below regulatory acceptable concentrations (Liess et al. 2021). Similarly, the effects of low-dosed neonicotinoids have been underestimated by the same risk assessment that is based on lethal effects and short-term observations (Rundlöf et al. 2015; Cressey 2017). Therefore, the identification of truly protective thresholds for pesticide applications needs to assess the population effect mechanisms of low pesticide concentrations.

With the ban of most neonicotinoids in Europe and decisions pending in the US in 2024, pyrethroid insecticides emerge as the most common alternative in agricultural applications (Jactel et al. 2019). In the aquatic environment, pyrethroids generally occur as short pulses in the water phase. This is due to their fast dissipation from the water column; in stream waters, only 3% of pyrethroids are bioavailable (dissolved in water and bound to dissolved organic matter) while 97% are bound to suspended solids (Lu et al. 2019). Nevertheless, maximum concentrations only 1–2 orders of magnitude below the acute EC_{50} (*D. magna*) were detected in surface waters (Rösch et al. 2019).

At such concentrations, stimulatory effects of pyrethroids are reported (Gottardi et al. 2017; Margus et al. 2019; Shang et al. 2021; Wolz et al. 2021) and discussed in the framework of hormesis theory (Townsend and Luckey 1960; Stebbing 1982; Calabrese and Baldwin 2003; Liess et al. 2019; Agathokleous et al. 2022). In contrast to linear no threshold or threshold models, hormesis assumes that dose–effect relationships are bi-phasic. This means that low levels of a toxicant have stimulatory effects and high concentrations have adverse effects. It is hypothesized that positive, hormetic effects have associated negative trade-offs, predicting that net population growth cannot be positive (Forbes 2000), because of limitations in available energy (Calow and Sibly 1990). These trade-offs, however, are poorly understood until today (Agathokleous et al. 2021) and it is yet unknown, which effects hormesis may have on population dynamics.

The aim of this work is therefore to investigate the consequences of low-dosed pulsed pyrethroid exposures on the multi-generational development of aquatic populations. For this, we exposed *D. magna* populations close to their carrying capacity with the pyrethroid insecticide esfenvalerate. We tested concentrations of $^1/_{10}$, $^1/_{30}$ and $^1/_{100}$ of the EC_{50}, assumed to provoke sublethal and hormetic responses and contrasted them with concentrations provoking lethal population responses. The effects on population biomass and abundance were monitored with a non-invasive image detection technique 3 times per week over a total of 89 days.

2. Material and Methods:

Experiment Design

The nanocosm experiment consisted of 40 *D. magna* populations, which were initialized from 15 neonates (age < 24 h) each. After 4 weeks of development, populations were assigned to 8 exposure groups (control, 0.01, 0.032, 0.1, 0.32, 1.0, 3.16, 10.0 µg/L esfenvalerate). This assignment of nanocosms to one of the exposure groups was based on the pre-exposure population density to achieve a balanced treatment design. The populations were subsequently exposed to a single pulse of the pyrethroid insecticide esfenvalerate at the respective concentration. Following this, the experiment was continuously monitored for another 9 weeks. Throughout the entire duration of the experiment, populations were constantly fed with 10.8 mg carbon per week and monitored with an image analyzing system 3 times per week. In general, the experimental design followed works of Liess et al. (2006) and Foit et al. (2012).

Test Systems

Each experimental unit consisted of a 5.5 L glass beaker (Harzkristall, Derenburg, Germany), filled with 500 g of washed and aquarium sand of 1–2 mm diameter. The sediment layer served as a habitat for microorganisms to facilitate self-purification of the systems. Aachener Daphnien Medium (ADaM) (Klüttgen et al. 1994) was used as the test medium for the experiment. Throughout the duration of the experiment, the medium was not exchanged and kept at a constant volume of 4.5 L by replenishing the beakers with doubly distilled water on a weekly basis. The amount of water loss from evaporation was kept to a minimum by covering the systems with glass plates. A 2 cm cut at the edge of the glass plates provided access to the nanocosms. Sufficient oxygen saturation was ensured by aerating the test systems with glass tubes, which were connected to air pumps *Osaga Air Compressor LK-35* (Fish farm Schierhölter, Glandorf, Germany) by silicon tubing. Aeration was turned on 3 times per day for 15 minutes each. The populations were fed three times per week with a diet of ground stinging nettle (*Folia urticae*), ground dog food (Organic dog biscuits, Yarrah Organic Petfood BV, Harderwijk, Netherlands) and batch cultured green algae (*Scenedesmus subspicatus*). The exact preparation of the feeding suspensions is detailed in SI2. Throughout the experiment, the organisms received 3.6 mg C per feeding. Only in the first 10 days, the amount was doubled to promote growth of microbial communities in the system.

Environmental Conditions

The experimental units were exposed to a 16–8 h day–night cycle and were positioned with the water surface approximately 20 cm below 70 W fluorescent cool-white lighting tubes. Throughout the whole duration of the experiment, room temperature was controlled to be 20 ± 1 °C. Due to the heat input from the lighting, the temperature of the systems was raised by approximately 1 °C, resulting in

nanocosms temperatures of 21 ± 1 °C. Acidity of the systems reached a stable pH of 7.9 ± 0.1 within the first week. Conductivity was kept constant by replenishing water at a value of approximately 1080 ± 17 µS/cm. In previously conducted experiments of the same type, nutrient levels did not diverge between treatments and were in bounds where no effects on aquatic organisms are expected (Liess et al. 2006). This was asserted once, 4 weeks after exposure: NO_3^{2-}, 0.3 ± 0.1 mg/L; NO_2^-, 0.02 ± 0.003 mg/L; PO_4^{3-} < 0.15 mg/L.

Exposure to Chemicals and Chemical Analysis

In order to study the effects of pyrethroids on *D. magna*, esfenvalerate (CAS 66230-04-4) was selected as a representative for the group of type-II pyrethroid insecticides. Esfenvalerate is approved for use on agricultural sites in the EU until 2023 and more type-II pyrethroids continue to be allowed until the end of the decade (https://ec.europa.eu/food/plant/pesticides/eu-pesticides-database/active-substances/?event=search.as). The hydrophobic insecticide esfenvalerate needed to be dissolved to ensure bioavailability in aquatic environments; we used dimethyl sulfoxide (DMSO) for this purpose at concentrations of 0.01% and 0.02% v/v. In order to maximize the number of replicates per treatment, no solvent controls were run in this study. However, we assessed the influence of DMSO on offspring and survival of *D. magna*, by running chronic exposure tests on a range of DMSO concentrations according to OECD (2012). These tests were run at low feeding conditions (0.016 mg C/individual/day) and high feeding conditions (0.16 mg C/individual/day) to estimate the effects on organisms under food limitation, comparable to conditions in the nanocosms. DMSO concentrations below 1% v/v, 2 orders of magnitude higher than employed DMSO concentration in this study, had no effect on the survival and the cumulative offspring of *D. magna* until 21 days post exposure (Fig. S1, S2), regardless if the organisms were cultured under high or low feeding conditions. The solvent concentrations used in this study (Table S1) are 2 orders of magnitude below this effect threshold. Also, no effects on the movement of *D.* were observed below 0.1 % v/v in a different study (Huang et al. 2018).

The stock solutions were prepared by serial dilutions in DMSO at the day of exposure and measured once for each test concentration in 1L volumes of ADaM, spiked parallel to the experimental units. Individual nanocosms were spiked with corresponding stock solutions, except for control treatments, which were not treated with DMSO. Chemical analysis of the test solutions was performed by Wessling GmbH, Landsberg OT, Oppin, Germany, using a Thermo Fisher Scientific TSQ 8000 Evo Triple Quadrupole GC-MS/MS. Measured concentrations as well as used DMSO concentrations are shown in Table S1 for each treatment and are well in line with the nominal concentrations. Therefore, we report the results in relation to the nominal concentrations of esfenvalerate.

Monitoring, Image Analysis and Calculations

D. magna populations were monitored 3 times per week for a total period of 13 weeks by a non-invasive image detection method described in Liess et al. (2006) and improved by Foit et al. (2012). In this method a series of three photographs is taken of each system, and in an algorithm the grayscale difference of those pictures is taken. Consequently, the resulting images contains only moving objects, which in the case of single species systems are exclusively *D. magna*. Pixels are then counted to estimate size and biomass of the organisms. From the 3 resulting difference-images, the picture with the highest count of *D. magna* is selected. The exact procedure is detailed in Foit et al. (2012). In this experiment, we used the camera model *Canon PowerShot G12* (Tokyo, Japan) with settings provided in Table S2.

The length L of the organisms was calculated using the formula $L = \sqrt{n_{pixel}/25.5}$, which is empirically calibrated to a fixed resolution to which the images are downscaled before calculation. From this, organism biomass W was estimated with the empirical relationship $W = 1.5 \times 1e^{-8} \times L^{2.84}$ (Dumont HJ. et al. 1975).

Population pre-exposure growth rates were calculated by $(BM_{t=0} - BM_{t=-5})/\Delta t$, while post-exposure biomass growth rates were calculated by $(BM_{t=+5} - BM_{t=0})/\Delta t$, with Δt indicating the time interval, BM indicating the biomass in mg and $t = 0$ resembling the time point of exposure to esfenvalerate. Division by $\Delta t = 5\ days$, resulted in the biomass growth rates in mg/day. The 5–day interval was chosen for calculation of population growth rates, because it represented a long-enough time frame to show early effects and was symmetrically available on both sides of the time of exposure.

Statistics

During the 4-week pre-exposure period, 9 systems did not successfully develop. Systems were considered unsuccessful, when the average population abundance during the pre-exposure period stagnated and was lower than the initial abundance of 15 neonates. In contrast, populations in valid systems grew exponentially with an average of 98 neonates throughout the period of exponential growth; the lowest pre-contamination average of successful systems was 50 neonates. Consequently, we removed unsuccessful systems from further investigations. Because of the prior balancing of the treatment groups according to population density, this change equally reduced the number of replicates per treatment from 5 to 4, except for the highest (10 µg/L) treatment, for which 3 valid replicates remained.

We used a bayesian model of a random walk to estimate the treatment trend μ_k dominating the population time series Y_i of all observations i belonging to treatment k, which follows a random normal distribution around the trend at each time point t with a treatment specific variation of σ_k. The trend is estimated by a gaussian random walk *grw* with the size of each step-innovation following a half-normal distribution with a standard deviation of 1. It is offset by a treatment specific intercept,

which follows a half-normal distribution with a standard deviation of 2. Before model calculation, the response variable was centered and scaled and back transformed afterwards to calculate effect sizes.

$$Y_{i:i \in k,\ t} \sim Normal(\mu_{k,t}, \sigma_k)$$

$$\mu_{k,\,t} = grw_{k,\,t} + intercept_k$$

$$grw_{k,\ t+1} \sim Normal(grw_{k,t}, innovation_k)$$

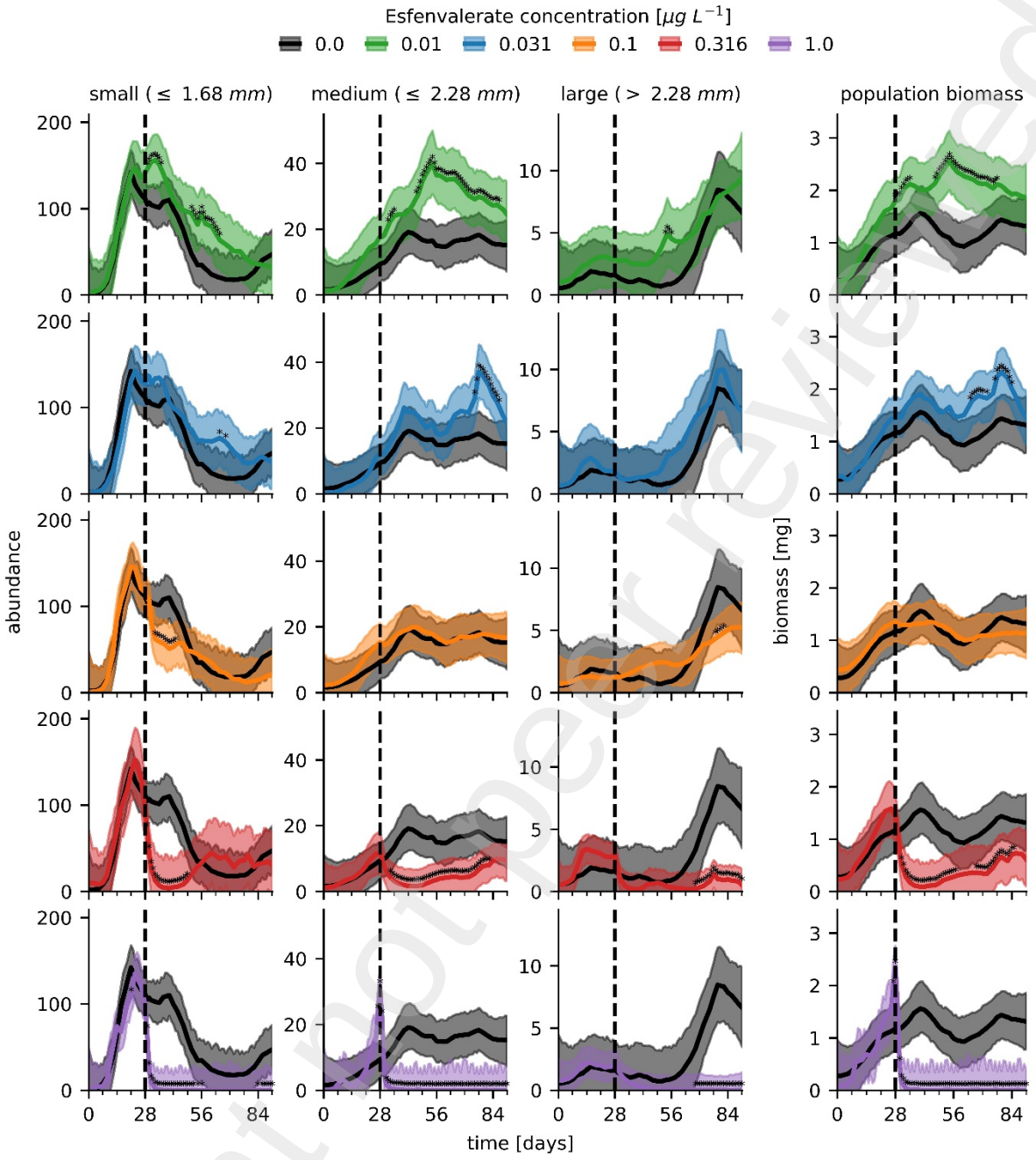
$$innovation_k \sim HalfNormal(sd = 1)$$

$$intercept_k \sim HalfNormal(sd = 2)$$

$$\sigma_k \sim HalfNormal(sd = 1)$$

We concluded that the trend of a treatments significantly differed from the control treatment, if the lower bound of the 95% posterior density interval (PDI) of the difference distribution was greater than zero. This method is an application of bayesian null hypothesis testing (Kruschke 2013). The *pymc* package (Salvatier et al. 2016) was used for computation of the bayesian models. Further data analysis was done with *python* and statistical tests were computed with the package *statsmodels* (Seabold and Perktold 2010). Instrumental for the handling of high dimensional datasets was the package *xarray* (Hoyer and Hamman 2017).

3. Results



184

185 Figure 1. Time-series of abundance and biomass of *D. magna* populations. Each row corresponds to a
186 tested esfenvalerate concentration with increasing level from top to bottom. Solid lines indicate the
187 Bayesian estimates of the mean trend of the abundance (columns 1–3) and biomass (column 4) of
188 experimental replicates over time. Shaded areas indicate the uncertainty of the trend with 95%-PDI
189 intervals. Stars (*) indicate significant deviations from the control trend (black), which is compared to
190 all treatments. The dashed line indicates the timepoint at which the experimental systems were
191 exposed to esfenvalerate.

192 The results displayed in Fig. 1 show *D. magna* population time-series over the entire three-month
193 period of experimentation. We also included the 95%-PDI to indicate trend uncertainty due to

experimental variation. We revealed that trends were highly similar in the exponential growth phase, before the exposure to esfenvalerate, after which time-series of all treatments significantly diverged in terms of abundance and biomass from control populations. The contamination occurred approximately one week after the initial maximum was reached for the smallest individuals, two weeks before the medium sized individuals and about 7 weeks for the large individuals. Related to the population biomass, contamination occurred close to the control carrying capacity (Fig. 1).

Effects of Low Doses

In the first week after the contamination, especially the smallest size class of the populations exposed to 0.01 µg/L esfenvalerate ($^1/_{100}$ EC_{50}) showed a significantly increased abundance compared to pre-contamination values (+ 7%), while exposure to 0.031 µg/L only led to an insignificantly increased abundance of small organisms (+ 5%). At the same time abundance of the smallest size class in control treatments decreased by 10 %. The increased population growth of the lowest esfenvalerate treatment is also reflected in the significantly increased population biomass during this period. In contrast, the medium and largest size class was generally not significantly increased by both lowest concentrations in the first week after the contamination. In weeks 2–3 after contamination, the population dynamic of low-esfenvalerate treatments showed similar trends as the control treatments related to the abundance of all size classes and also for the biomass (Fig. 1).

Beginning with week 3 after contamination, systems exposed to 0.01 µg/L esfenvalerate again significantly diverged from control treatments both in terms of abundance and biomass in all size classes until the end of week 7 after contamination: small organisms peaked in week 3–7 (+ 160 % of control levels), medium-sized organisms peaked in week 3–5 (+ 130 %) and large organisms peaked in week 3–4 (+ 370 %). The total biomass was about twice that of the control during weeks 3–7 after contamination. Exposure to 0.031 µg/L Esfenvalerate resulted in similar, but milder and later divergences from control trajectories: small organisms peaked in weeks 5–6 after contamination (+ 200 % of control levels), medium-sized organisms peaked in weeks 6–8 (+ 80 %), and large organisms were not significantly more abundant in this treatment than in the control. The population biomass was significantly increased in weeks 5–8 after exposure (+ 60 %).

Fig. 2 displays the individual biomass trajectories of the various treatments compared to control trajectories. The figure indicates that 2 out of 4 control populations went through an episode of low population density in weeks 3–7 after exposure. Near the end of the experiment, the onset of a new population growth cycle can be observed in these systems. Episodes of low population density before the onset of a new population growth cycle were not observed in any of the systems exposed to low concentrations of esfenvalerate.

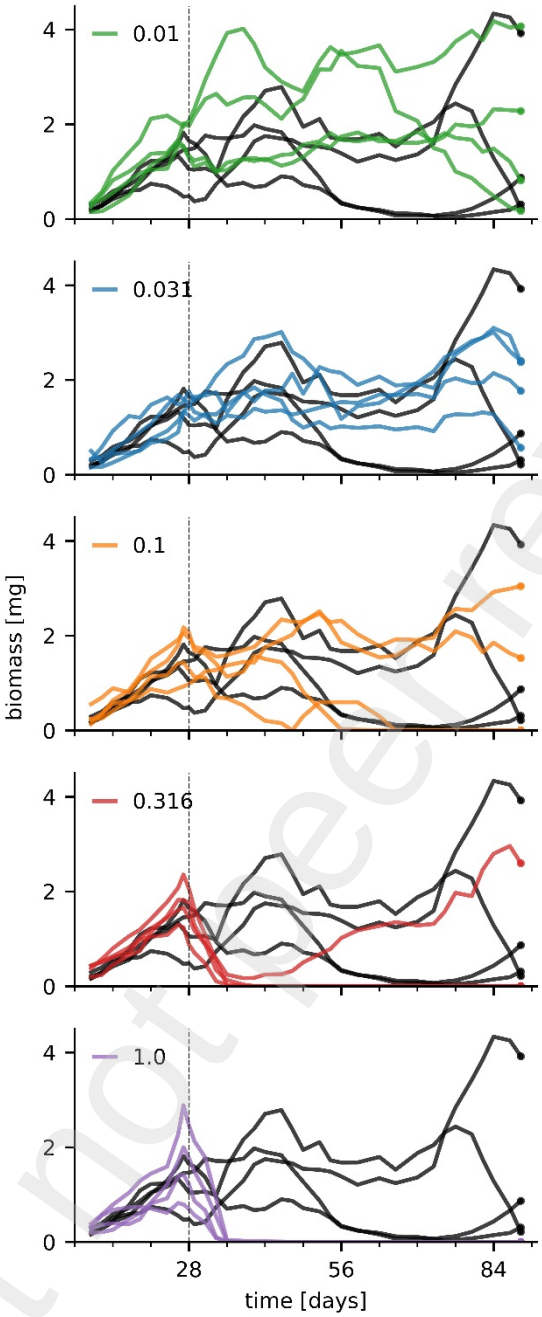


Figure 2. Individual trajectories of population biomass of the control treatment (black) and esfenvalerate treatments (colored lines). Trajectories were smoothed with a moving average (10-day backwards window) to focus on the trend of the individual timeseries.

Effects of Moderate and High Doses

Concentrations of 0.1 µg/L ($^1/_{10}$ EC_{50}) and above resulted in lethal effects on *D. magna*. In the first week after exposure, those concentrations induced a significant decrease of small sized organisms: 0.1 µg/L esfenvalerate resulted in significant reduction of 60%, 0.31 µg/L resulted in 90% decrease and 1 µg/L and above eliminated all small organisms. Such a concentration response relationship can also be

observed in the bigger size classes. Concentrations of 1µg/L esfenvalerate and above eliminated also the larger size classes of the populations entirely without any chance of recovery.

Moderate doses of 0.1 µg/L (1 order of magnitude below Esfenvalerate EC_{50} on *D. magna*) appear to only slightly affect population trends of the medium and large size classes (Fig. 1). However, closer inspection of the individual trajectories (Fig. 2), reveals that contrary to the control populations, 50% of *Daphnia* populations exposed to 0.1 µg/L esfenvalerate collapsed entirely, while others recovered to control population levels. Such a complete population collapse did not occur in control populations. An exposure to 0.316 µg/L esfenvalerate resulted in stronger responses compared to 0.1 µg/L treatments – only one population recovered from the pesticide effects (Fig. 2). This is also reflected in significant decreases of abundance and biomass trends from the control treatment in all size classes. Fig. 1 shows that the size classes of this treatment recover one after the other and have still lasting effects at the end of the experiment.

Short-Term Effects Relates to Esfenvalerate Toxicity and Population Density

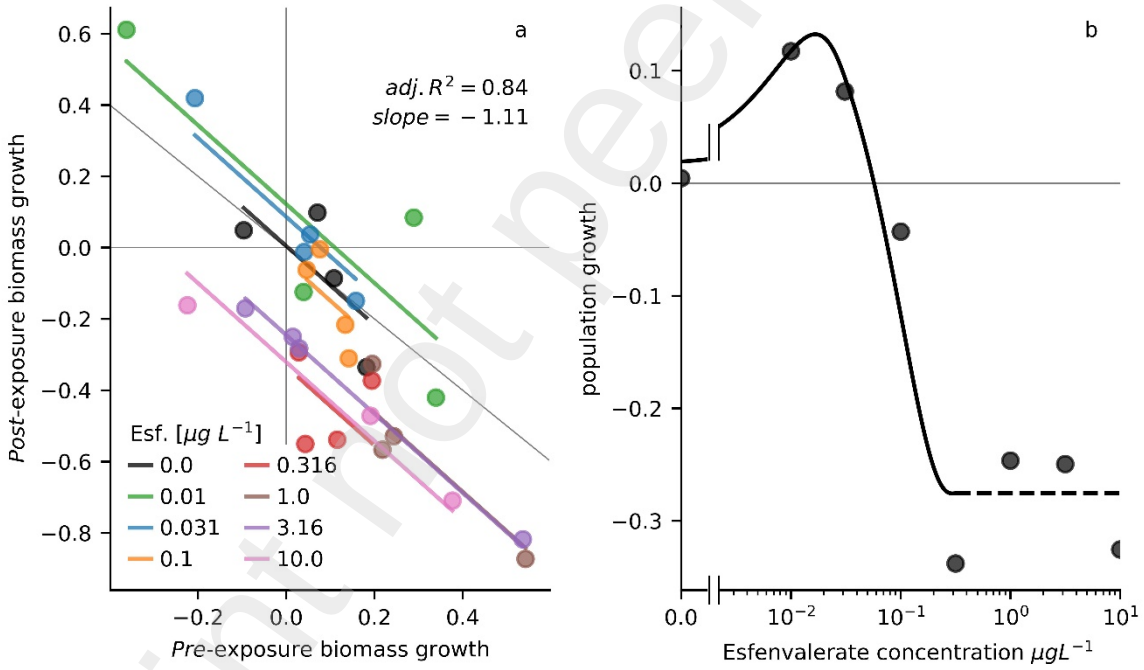


Figure 3. Post-exposure biomass growth rate related to the pre-exposure biomass growth rate and esfenvalerate effects. a) Relationship between 5-day *pre*-exposure and 5-day *post*-exposure growth rates of population biomass. The colored lines represent the regression line, offset by the respective effects of esfenvalerate exposure. The diagonal thin line indicates the 1:1 relation. b) Esfenvalerate effect on 5-day *post*-exposure growth rates, assembled in a dose-response curve. The line through the points is based on a cubic spline calculation. The dashed-extension of the spline indicates that the maximum effect was reached and a sensible fit of the cubic spline was no longer possible at these concentrations. The results of the linear regression model are presented in Table S3.

Figure 3 shows that population biomass growth after the contamination is strongly dependent on biomass growth before contamination (slope = –1.1, $p < 0.001$). This suggests that the population was approaching carrying capacity, regulated by density dependent processes. However, in the treatments exposed to 0.01 µg/L and 0.0316 µg/L esfenvalerate, we observe a slightly increased population growth by +0.12 mg/day and +0.08 mg/day, respectively, although not significant (Fig. 3a, b). On the contrary, control populations did not diverge from the effect of pre-exposure growth rates (+0.005 mg/day post-exposure growth rate) and concentrations of 0.316 µg/L esfenvalerate and above lead to significantly decreased post exposure growth rates (avg. = –0.29 mg/day, $p < 0.02$). In fact, the comparison to a model without the effects of the concentration (Table S4) shows that the concentration explains an additional 24% of the variation in post-exposure growth rates. The resulting hormetic response (Fig. 3b) was also present in the long-term increase of abundance and biomass increase of the two lowest concentrations and decrease in the higher concentrations (Fig. 1).

4. Discussion

We observe significantly increased population abundance and biomass in *D. magna* populations exposed to concentrations of 0.01 and 0.031 µg/L esfenvalerate until 7 weeks after pulse-exposure. Stimulating effects of low toxicant concentrations have been reported for the first time by Schulz (1888) related to increased CO_2 production of yeast populations exposed to various toxicants. Similar, positive effects of toxicants well below acute mortality have been observed in a variety of other studies (Stebbing 1998; Christopher Cutler et al. 2009; Calabrese 2010; Cutler 2013; Carvalho et al. 2020; Wang et al. 2021). Also, for pyrethroids, positive effects of low concentrations on various endpoints have been observed (Liess 2002; Beketov and Liess 2005; Bjergager et al. 2012; Gottardi et al. 2017; Shang et al. 2021; Wolz et al. 2021).

Stimulating effects are assumed to come at price of energetic trade-offs, which would lead to net neutral population growth rates (Calow and Sibly 1990; Forbes 2000). We show that, contrary to this assumption, the resulting net population growth was positive, suggesting that hormetic trade-offs may not be detectable in conventional population responses such as density and biomass. Accordingly, Liess et al. (2019) hypothesized that in the absence of external pesticide stress, an internal stress within the individual occurs – the "system stress". This concept can be translated to *intra*specific competition at the population level. The presence of low toxicant stress reduces the "system stress". With this approach the positive effect of low toxicant concentrations was successfully modelled (Liess et al. 2020). Here we hypothesize that, on the population level, *intra*specific competition is reduced by pesticide contamination level at low, sublethal concentrations.

Under conditions of *intra*specific competition, exposure to esfenvalerate also induced a short-term hormetic response in biomass growth rates of *D. magna* populations (Fig. 3b), which corresponded to the long-term hormetic responses observed in terms of population abundance and biomass at the 2

lowest concentrations for as long as 7 weeks after contamination (Fig. 1). This duration coincides with a full life-span of *D. magna* suggesting a long-term reduction of competition by the pyrethroid during a whole generation. For control populations, in contrast, the high *intra*specific competition during this period led to strong intrinsic stress, which was reflected in the low population density in 2 out of 4 populations. Such an oscillating population dynamic of non-contaminated populations based on *intra*specific competition has been observed since long in laboratory populations after reaching a peak-density (Halbach 1970; Preuss et al. 2009). In this study, none of the populations exposed to low concentrations of esfenvalerate (0.01, 0.03 µg/L esfenvalerate) went through such a low-density period (Fig. 2). This indicates that exposure to low concentrations of esfenvalerate persistently reduced *intra*specific competition, which allowed the short-term increase of population growth to take hold and resulted in an increased population biomass during most of the life span of *D. magna*. Only 8 weeks after contamination – at the end of the life span of *D. magna* – population density and biomass approached control levels again. Therefore, we conclude that the reduction of individual competitiveness is the likely trade-off of hormesis in population growth rates of D. magna exposed to sublethal concentrations of pyrethroids.

Our study also shows that single pulses of pyrethroids are a relevant threat to aquatic organisms even far below acute concentrations. This is extending the assumption of Yang et al. (2006) and Lu et al. (2019), who argued that the toxicity of pyrethroids is overestimated when using total chemical concentration, due to high sorption to sediments and low bioavailability as a consequence. Despite quick dissipation of esfenvalerate, initial pulses occur and result in long-term detrimental effects in populations as shown in this work. In this work's experimental setup – which mimicked a natural system with sediments and suspended organic material – a single pulse of $^{1}/_{10}$ of the esfenvalerate EC_{50}, resulted in the collapse of 50% of the population; and, as argued before, even much lower concentrations affected the population dynamic of *D. magna*. While we acknowledge the low bioavailability of pyrethroids after reaching chemical equilibrium, effects on the level of individual occur after ultra-low pulses (Liess 2002; Beketov and Liess 2005), and our study demonstrates that pyrethroid pulses even have long-term effects on the population level. Considering the demonstrated occurrence of such pyrethroid pulses (Rösch et al. 2019) and the likely increase of pyrethroid-usage in the future (Jactel et al. 2019), we argue that effects of transient pyrethroid pulses should be taken into account in aquatic risk assessment.

Finally, it could be argued that low concentrations of pyrethroid insecticides are acceptable as they lead to increased stability of the exposed systems. However, we do not see this as a valid argument, as the presumed long-term reduction in competitive strength under the condition of *inter*specific competition with another species could have a negative effect on population development.

Conclusion

Concentrations below $^{1}/_{30}$ of the acute EC_{50} significantly increased the population abundance and biomass over several weeks after exposure to the pyrethroid insecticide esfenvalerate. This provides evidence that responses to low-dosed toxicants may reduce long-term intraspecific competition due to a reduced competitive strength of individuals. We, therefore, suggest to apply long-term non-invasive population monitoring to reveal subtle but relevant effects on the performance of individuals within populations. Further, we suggest to perform multi-species experiments to assess population performance under the influence of *inter*specific competition and ultra-low exposure to pesticides.

Supplementary information

The supplementary data to this article is available online at [doi].

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CRediT author statement

Florian Schunck: Investigation, Data curation, Formal analysis, Visualization, Writing – Original draft. **Matthias Liess:** Conceptualization, Investigation – Guiding analytical cognition process, Writing.

Data Availability

Data will be made available on request.

Declaration of competing interest

The authors declare no competing financial interest.

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