

Consequences of size structure in the prey for predator–prey dynamics: the composite functional response

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Summary

1. Current formulations of functional responses assume that the prey is homogeneous and independent of intraspecific processes. Most prey populations consist of different coexisting size classes that often engage in asymmetrical intraspecific interactions, including cannibalism, which can lead to nonlinear interaction effects. This may be important as the size structure with the prey could alter the overall density-dependent predation rates.
2. In a field experiment with damselfly and dragonfly larvae, 16 treatments manipulated the density of a small prey stage, the presence of large conspecific prey and the presence of heterospecific predators.
3. Size structure in the prey (i.e. when both prey stages were present) decreased the impact of the predator on overall prey mortality by 25–48% at mid and high prey densities, possibly due to density-dependent size-structured cannibalism in the prey. The predation rates on small prey stages were determined by the interaction of large prey and predators. Predation rates increased with prey density in the absence of large prey, but predation rates were constant across densities when large conspecifics were present.
4. The functional response for unstructured prey followed a Holling type III model, but the predation rate for size-structured prey was completely different and followed a complex pattern that could not be explained with any standard functional response.
5. Using additional laboratory experiments, a mortality model was developed and parameterized. It showed that the overall prey mortality of size-structured prey can be adequately predicted with a composite functional response model that modelled the individual functional responses of each prey stage separately and accounted for their cannibalistic interaction.
6. Thus, treating a prey population as a homogeneous entity will lead to erroneous predictions in most real-world food webs. However, if we account for the effects of size structure and the intraspecific interactions on functional responses by treating size classes as different functional groups, it is possible to reliably predict the dynamics of size-structured predator–prey systems.

Key-words: density-dependent predation, indirect interactions, intraguild predation, intraspecific predation, population dynamics, predator–prey dynamics, stage structure

Introduction

A central goal in ecology is to understand and predict how the consumption rate of a predator changes with its prey density, because it links the dynamics of the prey and the predator and therefore strongly determines the dynamics of communities. Predation theory has been largely dominated by the use of Holling's (1959) three types of functional responses and by its extensions that included effects of the predator density (Abrams & Ginzburg 2000) to describe this change in *per capita* predation

rates per unit time across prey densities. All these standard functional responses have in common that they assume a homogeneous prey population. However, no population is truly homogeneous. Indeed, most species undergo considerable changes in size during their ontogeny, resulting in the coexistence of different size classes within a prey or predator population, and recent theoretical studies have shown that stage/size structure in the prey population can strongly alter the long-term dynamics of predator–prey systems (Claessen, de Roos & Persson 2000; de Roos, Persson & Thieme 2003; Abrams & Quince 2005; van Kooten, de Roos & Persson 2005; van de Wolfshaar, de Roos & Persson 2006; Rudolf

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2007). Functional responses are strongly determined by the relative body size relationship of a predator and its prey (Streams 1994; Thompson 1975; Hewett 1980; Hirvonen & Ranta 1996; Persson *et al.* 1998; Wahlstrom *et al.* 2000; Aljetlawi, Sparrevik & Leonardsson 2004) and the abundance of alternative prey (Murdoch 1969; Colton 1987; Soluk 1993). Thus, current functional responses that ignore the size structure within a prey species might not adequately describe the mortality patterns in real world predator–prey systems.

Density-dependent predation rates generally depend on two main factors: the handling time and the attack rate, which are both known to be size-dependent (Thompson 1975; Hewett 1980; Spitze 1985; Werner 1988; Bystrom & Garcia-Berthou 1999; Persson *et al.* 2000; Wahlstrom *et al.* 2000; Persson & Bronmark 2002; Aljetlawi *et al.* 2004; Bystrom & Andersson 2005). Functional responses for different size classes of the same prey species may be of the same type and only differ in the specific slopes (Hewett 1980; Spitze 1985; Persson & Greenberg 1990; Wahlstrom *et al.* 2000). However, in some species the functional response types can change from a type II response for large prey to a type III response for the small stage of the same prey (Streams 1994; Hassell, Lawton & Beddington 1977) or vice versa in other species (Eggleston 1990). If the functional responses differ across prey size classes, the size structure within a prey should strongly affect the overall density-dependent mortality of the prey due to predation. Yet, only one study has examined how differences in the size structure in the prey impact the mortality pattern, but the results were mixed and the functional response was specified by the author and not estimated from the data (Spitze 1985). Thus, we still lack the basic understanding of how the overall density-dependent mortality of the prey is affected by the size structure within the prey despite its importance for community dynamics.

One additional factor that has been largely neglected in that context is the impact of intraspecific interactions between the prey stages on the consumption rate of the predator. The interaction of intraspecific processes with predation can lead to interaction effects that can alter the impact of a predator across a prey density gradient and thus alter the realized predation rate for the total prey population. Cannibalism is common in size-structured populations at all trophic levels (Fox 1975; Polis 1981; Woodward & Hildrew 2002; Woodward *et al.* 2005). Because cannibalism is density-dependent (Polis 1981; Van Buskirk 1989; Hopper, Crowley & Kielman 1996; Wagner & Wise 1996; Moksnes 2004) it will alter the density of the available prey for the predator and the observed density-dependent prey mortality is not equal to the predation rate. Furthermore, the presence of cannibals often induces antipredator behaviour in small conspecifics, which can also alter heterospecific predation rates (Persson & Eklov 1995; Sih 1982; Leonardsson 1991; Biro, Post & Parkinson 2003; Rudolf 2006). These interactions of cannibalism and predation could strongly alter the predator induced mortality in size-structure prey populations. However, because previous studies have generally examined the functional responses for unstructured populations, the consequences of the

density-dependent interactions of cannibalism and predation for the density-dependent mortality of the prey are largely unknown.

In natural populations, numerical changes (i.e. changes in total abundance) are generally associated with changes in relative frequency of size classes (i.e. structural changes). While older stages might stay relatively constant or decline due to mortality, small stages strongly increase during reproductive periods. Indeed, most of the density-dependent predation can be expected in the small stages during these periods of high densities. If the density-dependent predation rate of the predator is dependent on the presence of larger stages in the prey, predictions derived from current unstructured functional responses that focus on simple numerical changes (i.e. total prey abundance) might over- or underestimate the observed mortality.

The aim of this study was to examine how the size structure and size-structured interaction (including cannibalism) in the prey alter the density-dependent predation rate of a predator and its impact on the prey density. In particular, the goals of this experiment were: (1) to test if the impact of the predator on the mortality of the prey is altered by the size structure in the prey; (2) to determine how the interaction of cannibalism in the prey and heterospecific predation affects the density-dependent mortality of the prey; and (3) develop a mechanistic model to describe the functional relationship of a predator and its size-structured prey.

Materials and methods

STUDY SPECIES

Larvae of *Anax junius* and *Enallagma aspersum* are generally restricted to fishless ponds (McPeck 1990) where predation by *A. junius* is a major factor that determines the abundance and distribution of *E. aspersum* (McPeck 1990). Owing to the often very high densities of *E. aspersum* (up to *c.* 1500 individuals m⁻²) (McPeck 1990), density-dependent interference is another likely factor that controls the abundance and growth of this species (Pierce, Crowley & Johnson 1985). The long reproductive period of *E. aspersum* and the presence of overwintering larvae result in the coexistence of different size classes in spring due to overlapping cohorts (Paulson & Jenner 1971; Ingram & Jenner 1976). While cannibalism rates can be very high in some *Enallagma* species (Anholt 1994), little is known about the importance of cannibalism in *E. aspersum*. Larvae of both species never leave the water except at metamorphosis.

FIELD EXPERIMENT

This field experiment was designed to determine the impact of size structure and cannibalism on the density-dependent predation of predators and density-dependent mortality of the prey under natural conditions. Larvae of both species were collected from one pond at Mountain Lake Biological Station (MLBS), Giles Co., Virginia during the week before the start of the experiment. The experiment was carried out in cylindrical field enclosures (*d* = 40 cm, *h* = 60) made with mosquito netting (1 mm mesh width) lining the inside of a stable frame of plastic fencing. Each cage was open at the top to allow colonization by aerial invertebrates. The bottom was sealed

and contained a removable plastic dish ($d = 40$ cm) with a 2-cm layer of sifted pond sediment and leaf litter. Four large, rinsed *Isoetes* plants obtained from the same pond were added to each cage representing its approximate natural density. Cages were set up 1 week before the start of the experiment to allow colonization by aquatic invertebrates. At the start of the experiment, cages contained a high density of zooplankton and small insects (e.g. backswimmer, adult and larval beetles, dipteran larvae) and some tadpoles that serve as food for damselfly larvae and to some extent for dragonfly larvae. Filamentous algae grew at the side of the cages providing additional structural complexity. Predatory invertebrates that were large enough to consume small *E. aspersum* were removed before the start of the experiment. Cages were set up at a water depth of 30 cm in a line along the border of the Station pond at MLBS, where both species naturally coexist. The experiment started on 2 June 2007 and ended after 14 days. At the end of this period, the first large prey individuals initiated metamorphosis and emerged from the cage. In the predator treatments, one *A. junius* was added. All *A. junius* were within the F2 instar (~ 4.0 cm body length). The 'large stage' of *E. aspersum* included two instars (F1–F2, 1.34 cm \pm 0.06 SD body length, $n = 23$), and the 'small stage' had a mean body length of 0.91 cm (\pm 0.11 SD, $n = 545$). These size classes reflected the natural size distribution and the density range in the experiment (79 – 557 individuals m^{-2}) represented low to intermediate natural densities (Pierce *et al.* 1985; McPeck 1990). Small individuals were visually divided into four different size classes and individuals within a size class were randomly assigned to density treatments such that the means and the standard deviations of the size were similar across density treatments (for mean and SD of head width and body length: $F_{3,12} < 2.15$, $P > 0.15$).

The experiment consisted of 16 treatments (Table 1) that manipulated the presence or absence of a large prey stage and the presence or absence of a predator along a density gradient of the small prey stage. Different subsets of these treatments were used for three different factorial analyses. The first analysis consisted of a 3 (total prey number (small + large): 30, 50 or 70) \times 2 (large prey stage present or absent) \times 2 (presence/absence of predator) factorial design and examined how the size structure within prey altered the impact of the predator on total prey mortality (small + large stages) across a prey densities gradient. Note that this design prevents changes of the total prey density (small + large prey) with changes in the size structure within the prey population and thus avoids the confounding effects of changing the total prey density. The second analysis consisted of a 3 \times 2 \times 2 factorial design that combined three densities of small prey (10, 30 or 50) with the presence or absence of

large prey and presence or absence of the predator. This subset was used to determine the individual and interaction effects of large prey and predators on the survival of the small prey stage across a gradient of small prey densities. The third analysis consisted of a 4 \times 2 factorial design and examined how changes in the density of small prey (0, 10, 30, 50) and predation (absence or presence of the predator) affect the mortality of the large prey stage. Each treatment was replicated three times and larva and treatments were randomly assigned to a cage. Owing to an error in the set-up, one cage did not receive 30 small larvae, resulting in four replicates of the treatment with 20 large prey and two replicates of the treatment with 30 small + 20 large. During the experiment, a juvenile bullfrog entered one cage and predacious beetle larvae entered two other cages consuming most of the prey. These experimental units were significant outliers (Dixon's Q -test: $P < 0.05$) and were thus removed from the analysis (see Table 1 for details). At the end of the experiment, the number of surviving individuals (including metamorphs) was recorded, and all surviving individuals were photographed and measured using ImageJ (<http://rsb.info.nih.gov/ij/>) to confirm the size class assignment of survivors. The mortality rate (m) of the prey was estimated as $m = [-\ln(N_t/N_0)]/t$, assuming an exponential survival function: $N_t = N_0 e^{-mt}$, with N_t = number of surviving prey at the end of the experiment, N_0 = number of prey at the start of the experiment and t = duration of the experiment (= 14 days).

The set of predator treatments with small prey only and the set of predator treatments with large and small prey were used to determine and compare the functional response for 'unstructured' prey (small prey only) and the density-dependent predation for 'size-structured' prey (small + large), respectively. Note that the size-structured scenario represents the numerical and structural change (i.e. change in the relative abundance of small vs. large prey) that occurs in natural populations (see Introduction). The type of the functional response (i.e. Holling type I, II, III) for structured and unstructured prey was first determined by fitting a polynomial logistic regression model to the observed proportional mortality (N_{consumed}/N_0). These data transformation is the best way to clearly separate all three functional response types (Trexler, McCulloch & Travis 1988). Parameters were estimated using the method of maximum likelihood. The analysis started with a cubic term, and nonsignificant higher-order terms were removed using a lack of fit-test (Trexler *et al.* 1988). The signs of the model coefficient in the final model identified the appropriate functional response (i.e. Type I, II, III) that should then be fitted to the untransformed survival data to estimate the respective model parameters of the mechanistic predation model. Note that this procedure evaluates and compares the fit of the different

Table 1. Experimental design of the field experiment

Unstructured prey			Unstructured prey + predator			Structured prey			Structured prey + predator		
SE	LE	Tot	SE	LE	Tot	SE	LE	Tot	SE	LE	Tot
10	0	10	10*	0	10	0	20†	20	0	20	20
30	0	30	30	0	30	10	20*	30	10*	20	30
50	0	50	50	0	50	30	20*	50	30	20	50
70	0	70	70	0	70	50	20	70	50	20	70

SE and LE indicate the number of individuals in the small stage and the large stage of the prey *E. aspersum*, respectively. Tot = total prey density (SE + LE); unstructured = small prey; structured = small + large prey.

All treatments were replicated three times unless indicated otherwise (see Materials and methods for details).

*Two replicates.

†Four replicates.

functional responses/density-dependent mortality and that it ensures that only the significantly best (i.e. most parsimonious) model is used. The significant lack of fit test indicated that for the unstructured prey a type III response was required, while the functional response for the structured prey could not be described with any standard Holling-type response (see Results). Thus, only the parameters for the unstructured prey model were estimated. To account for the prey depletion during the experiment, Rogers's (1972) 'random predator equation' was used:

$$N_{\text{cons}} = N_0(1 - \exp[(d + aN_0)(T_h N_{\text{cons}} - t)/(1 + cN_0)])$$

(where N_{cons} = consumed prey, N_0 = initial prey, t = total time available, T_h = handling time, a , c , d = constants), which is the integral of Holling's type III response over time. Parameters (d , c) that were not significantly different from zero were subsequently removed from the model until only significant parameters remained (Juliano 2001). Nonlinear least square models in combination with Newton's method were used to solve the implicit equations, fit the models, and estimate parameter values (Juliano 2001). The functional response for 'unstructured prey' was determined using predator treatments where only small prey were present. In the case of the 'size-structured prey' (small + large prey) cannibalism was present (see Results). Thus, to only estimate the functional response of the predator for size-structured prey, the functional response of cannibals had to be removed. To account for the cannibalism rate, I first calculated the average cannibalism rate observed at each density without the predator. To account for change in cannibalism rate due to the consumption of cannibals by predators, I corrected the realized cannibalism rate expected in treatments with predators by multiplying the observed cannibalism rate by the factor k ($= 0.135$), the average proportional reduction of cannibals due to predation. Then, the net predation rate was estimated by subtracting the correct cannibalism induced mortality rate from the total mortality rate observed in the respective density treatments where both cannibals and the predator were present. The estimated predation rate was then used to calculate the expected number of prey that survived and the fraction consumed using the exponential survival model (see above). Note, that this only accounts for the multiplicative interaction effects between cannibalism and predation and the indirect lethal effect due to the consumption of cannibals but not for potential nonlethal indirect interactions that are possible in size-structured systems (Rudolf 2006; Rudolf 2007). Owing to the experimental set-up, the density treatment with 20 individuals was not present in unstructured prey. A comparison of two separate analyses, one that included the treatment with 20 individuals and one that excluded the treatment, showed that the general shape of both functional responses was similar and not altered by including the treatment. Thus, I included this treatment into the model for 'structured prey' to reduce the standard error and thus the accuracy of the model. The slopes of the functional responses for structured and unstructured prey were then compared across the same densities to detect differences in the functional response due to size structure in the prey.

SIZE-SPECIFIC HANDLING TIME ESTIMATION

The handling time of the predator for large *E. aspersum* could not be obtained in the field. Thus, the relative difference in the handling time for large and small *E. aspersum* was estimated in a laboratory experiment. One *A. junius* larva was added to seven boxes (30 cm × 16 cm × 10 cm) filled with pond water. Each box included one large

stick that served as perch for *A. junius*. Three boxes received 30 small prey and four boxes received 15 large prey. The densities were based on previous measurements of consumption rates, and assured that predators were never prey limited while minimizing mortality due to cannibalism in the prey (see results below). The size of both prey stages and the predators were similar to the field experiment, but this limited the number of replicates due to the low number of predators and large prey in the required size class available this late in the season. The total number of surviving prey was determined every 24 h when consumed prey were replaced. The experiment started on 16 June 2006 and replicates were terminated after 14 days or else when a predator moulted. The handling time for each stage was estimated from the average number of consumed prey (N_{cons}) per predator per day after discarding the first day of predation to avoid effects due to initial variation in hunger levels. Assuming that the predators were fully saturated during the experiment and feeding at their maximum rate, the inverse of the N_{cons} will approximate the handling time.

CANNIBALISM EXPERIMENT

I carried out a laboratory experiment to determine if cannibalism occurs in *E. aspersum* and if the probability of cannibalism is dependent on the relative size difference to test if this could be a potential explanation for the increased mortality in the presence of the large prey stage observed in the field experiment. Larvae were divided into three size classes: large (L), medium (M) and small (S), and randomly assigned to one of six respective size-pairs: LL, LM, LS, MM, MS, SS. The large and small size classes were equivalent to the size classes used in the field experiment. Ten pairs of each size were added to a plastic cup with pond water and one plastic stick that served as perch. After 24 h cups were checked for mortality and injured individuals. For the analysis, pairs were assigned to three groups based on the size difference: none (i.e. same size class), medium (one size class difference) and large (two size classes difference). A logistic regression was used to test for size difference effects on the probability of cannibalism or injury. While this method likely overestimates the cannibalism rates observed in the field, it is a commonly used method that provides a good estimate of the relative probability of cannibalism across interacting size classes in the field (Wissinger 1988; Block & Stoks 2004; Rudolf unpubl. data).

Results

PREY MORTALITY

In general, heterospecific predation significantly increased total prey mortality rates and this effect was independent of the total prey density (Table 2). However, the significant interaction effect indicated that the impact of the predator on the total prey mortality rate was dependent on the presence or absence of large cannibalistic prey (Table 2): the presence of large cannibalistic prey reduced the mortality added by the predator (i.e. $(m_{P+L,i} - m_{L,i}) - (m_{P,i} - m_{\text{control},i})(m_{P+L,i} - m_{L,i})$, with P and L = presence of predator and large prey, respectively, i = respective density) on average by 26.4% compared with prey populations without a large prey stage. This pattern was largely driven by the strong decrease in predation rates at mid (48.1%) and high (31.8%) prey densities (Fig. 1a). There was also a significant interaction effect of prey density and the

Source	Numerator and Denominator d.f.	F	P
Total prey mortality			
Total prey density	2, 21	0.12	0.8834
Anax	1, 21	272.38	< 0.0001
Large prey	1, 21	13.64	0.0013
Anax × total prey density	2, 21	0.41	0.6716
Large prey × total prey density	2, 21	5.06	0.0161
Large prey × Anax	1, 21	7.59	0.0118
Large prey × Anax × total prey density	2, 21	2.04	0.1555
Small prey mortality rate			
Small prey density	2, 20	10.40	< 0.001
Anax	1, 20	88.23	< 0.0001
Large prey	1, 20	0.34	0.565
Anax × small density	2, 20	2.95	0.0752
Large prey × small prey density	2, 20	6.34	0.0074
Large prey × Anax	1, 20	0.88	0.359
Large prey × Anax × small prey density	2, 20	6.65	0.006
Large prey mortality rate			
Small prey density	3, 14	1.42	0.5267
Anax	1, 14	65.71	< 0.0001
Anax × small prey density	3, 14	0.94	0.4461

Significant treatment effects are in bold.

Table 2. ANOVA of treatment effects of prey (*E. aspersum*) densities, presence/absence of the large prey stage and presence/absence of the predator *A. junius* (*Anax*) on the total (small + large) mortality rate and the mortality rate of the small prey stage and the large prey stage only; for treatment details see Table 1

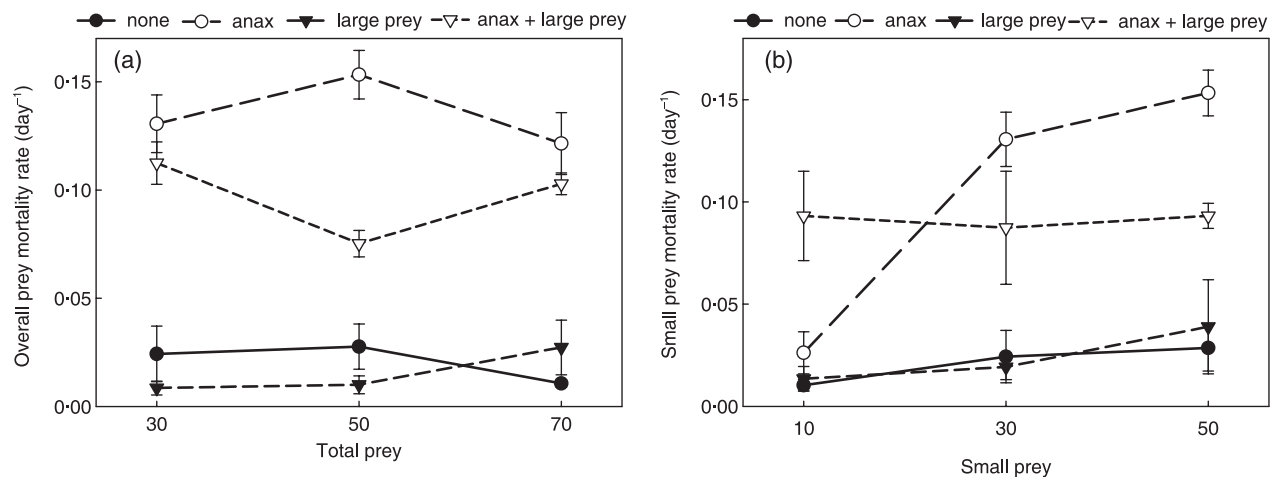


Fig. 1. (a) Overall (small + large) prey mortality rate, and (b) mortality rate of the small stage of the prey *E. aspersum* across prey densities for treatments without predators or a large prey stage (none), with the predator *A. junius* (*anax*), with large conspecifics (large prey) or both predator and large prey. For treatment details see Table 1.

presence of large cannibalistic prey (Table 2, Fig. 1a). A separate regression analysis using all available densities of the respective treatments confirmed that in the absence of predators, the total per capita prey mortality rate remained constant across densities without large cannibalistic prey ($F_{1,10} = 0.01$, $R^2 = 0.01$, $P > 0.90$), but increased significantly with density in the presence of large prey ($F_{1,9} = 7.36$, $R^2 = 0.45$, $P < 0.025$) (Fig. 1a).

The presence of the predator generally increased the mortality of small prey (Table 2, Fig. 1b), but the three-way interaction effect indicated that the magnitude of this effect was dependent on the presence of large cannibalistic prey, and the density of small prey (Table 2, Fig. 1b). A separate regression analysis showed that in predator treatments, the mortality

rate of small prey increased with the density of small prey in the absence of large cannibalistic prey ($F_{1,5} = 26.93$, $R^2 = 0.78$, $P < 0.005$), but the mortality rate was independent of the density of small prey in the presence of large prey ($F_{1,5} = 0.0$, $R^2 = 0$, $P = 1$). While the presence of large prey increased the mortality of small prey at low densities by 78.1%, large prey strongly decreased the impact of the predator by 35.2% and 52.5% at mid and high densities, respectively (Fig. 1b).

The mortality rate of large prey was increased significantly by 83.3% in the presence of the predator (no predator: mortality rate (m) = 0.031 ± 0.011 SD, $n = 11$; with predator: $m = 0.188 \pm 0.055$ SD, $n = 12$), but was independent of the density of small prey and the interaction of predator and prey density (Table 2).

Table 3. Maximum likelihood estimates of parameters in the final polynomial logistic regression models for predation on ‘unstructured prey’ (small prey only), ‘structured prey’ (all treatments with large prey and the treatment with lowest density of small prey only). Note that the ‘structured prey’ model was corrected for the morality due to cannibalism to determine the functional response of the predator only. The final models represent the most parsimonious model estimated using a lack of fit test (see Materials and methods for details). The models are displayed in Fig. 2

Source	Functional response	Parameter	Estimate	SE	χ^2	<i>P</i>
Unstructured prey	Type III	Intercept	-2.2663	0.7244	9.79	< 0.005
		$a(N)$	0.1767	0.0354	24.85	< 0.0001
		$b(N^2)$	-0.0018	0.0004	21.60	< 0.0001
Structured prey	Composite	Intercept	-4.3268	1.2187	12.52	0.0004
		$a(N)$	0.4949	0.1101	20.21	< 0.0001
		$b(N^2)$	-0.0129	0.00286	20.33	< 0.00001
		$c(N^3)$	0.000099	0.000022	20.05	< 0.00001

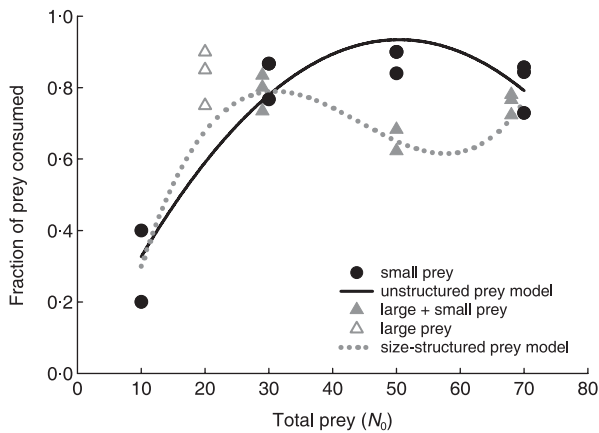


Fig. 2. Observed overall (small + large) fraction of prey consumed (N_{consumed}/N_0) in predator treatments across total prey densities in treatments with only small prey, only large prey and small + large prey. The lines represent the most parsimonious polynomial logistic regression models determined by a lack-of-fit test for ‘unstructured prey’ (small prey only), and ‘size-structured prey’ (small + large prey) (see Table 3 and Materials and methods for details).

FUNCTIONAL RESPONSES

The most parsimonious model with the best fit identified by the lack-of-fit test showed a change from an initial positive to a negative slope in the logistic regression model for the proportional mortality of unstructured prey (Table 3, Fig. 2). This humped shape of the relative morality across a density gradient indicates that in the absence of large prey predators exhibited a type III functional response. The only significant parameters in the final fitted predation model were the attack rate a and the handling time T_h resulting in the final model: $N_{\text{con}} = N_0(1 - \exp(aN_0(T_h N_{\text{con}} - T)))$, with $T_h = 0.1769$ (± 0.0199 SE) and $a = 0.0062$ (± 0.0012 SE). When the prey population was size-structured (i.e. large + small prey), the significant lack-of-fit test indicated that an additional cubic term was required to describe the observed mortality pattern due to change from a significant negative slope at low to medium densities to a significant positive slope at medium to high densities (Table 3, Fig. 2). This pattern at mid–high

densities (i.e. the signs of the corresponding coefficients) was exactly opposite to the hump-shaped mortality pattern in unstructured prey that peaked at mid densities and decreased at high densities (Table 3, Fig. 2). These significant differences in shape (i.e. slopes) of the mortality function clearly indicate that across the same densities the type of the functional response was very different for unstructured and size structure prey. The complex mortality pattern in size-structured prey with the increase in proportional mortality at high densities cannot be described with any Holling-type functional response.

The laboratory handling time experiment showed that predators spent twice as much time handling large prey ($T_{hL} = 0.2$ individuals day⁻¹, ± 0.04 SD) as small prey ($T_{hS} = 0.1$, ± 0.022 SD) (unequal variance *t*-test: $t_{4.752} = -4.2$, $P < 0.01$).

CANNIBALISM

The laboratory experiment showed that the probability of cannibalism was significantly affected by the size difference (Wald’s $\chi^2 = 11.37$, d.f. = 2, $P < 0.005$). The probability of cannibalism was 70% when individuals differed by two size classes and this was significantly different from treatments with no difference in size where cannibalism occurred in only 7% of the pairs (odds ratio = 22.2, $\chi^2 = 4.26$, $P < 0.05$). In the latter, all cannibalism events occurred when both individuals were in the largest size class. When individuals differed by one size class, cannibalism was still low and occurred on average in 15% of the pairs. On average 23% of the individuals were injured, and this was similar across size classes (Wald’s $\chi^2 = 1.30$, d.f. = 2, $P > 0.50$).

Discussion

While there is ample evidence demonstrating the importance of prey size on the functional response, the consequences of size structure and intraspecific interactions within the prey are largely unknown. This study presents the first empirical evidence demonstrating that size structure within the prey can strongly reduce and alter the impact of a predator on the density-dependent predation rate of the prey.

PREDICTING PREDATION RATES ON SIZE-STRUCTURED PREY: THE COMPOSITE FUNCTIONAL RESPONSE

Classical functional responses predict consumption rates solely based on the total number of individuals and ignore the size variation in the prey (Holling 1959; Abrams & Ginzburg 2000). If the size structure within the prey is irrelevant and only the total number of prey is important, then the general shape of the functional response should be the same for structured and unstructured prey. However, the results clearly show that the functional response for the size-structured prey was not even of the same type as the functional response for the unstructured prey and could not be explained with any single classical functional response type. It is important to note that in the size-structured prey, the change in density also resulted in a structural change (*sensu* Rudolf 2006) in the prey population, i.e. the relative frequency of large and small prey changed. However, this represents a typical density change in species with overlapping cohorts, which is common in many vertebrate and invertebrate species with delayed development, iteroparity, or multiple breeding events during one season (bi- or multivoltine). Thus, it can be expected that this discrepancy between the predictions of classical unstructured functional responses and the observed mortality is common for a variety of systems.

The complex mortality pattern across densities observed with size-structured prey suggests that a single functional response is not appropriate for describing the mortality of the prey. Indeed, the results indicate that the functional response for large prey was independent of the density of small prey, suggesting that the predation rate of both stages should be treated separately. Furthermore, large prey also cannibalized small conspecifics especially at high densities, albeit at a relative low rate. Thus, to predict the mortality pattern across a density gradient we also need to account for the intraspecific interactions in the prey. The total number of prey consumed (N_{cons}) is the sum of consumed small (S) and large (L) prey: $N_{\text{cons}} = N_{\text{OS}} \times g(P, N) + N_{\text{OL}} \times f(P, N)$, with $N_{\text{OS}}, N_{\text{OL}}$ indicating the initial density of small and large prey stage, respectively, and g and f the mortality rates (or proportion) of small and large prey, respectively. In the present study, g had two different mortality components: mortality due to predation (h) and mortality due to cannibalism (w). From the experiment we know that the mortality due to predation (h) follows a type III functional response and that the cannibalism rate (w) increases linearly with the initial density of small prey: $w = \beta N_{\text{OS}}$. Thus, after extending the random predator equation, the number of consumed small prey is given by $N_{\text{consS}} = N_{\text{OS}}(1 - \exp(h + w)) = N_{\text{OS}}(1 - \exp(aN_{\text{OS}}(T_{\text{hS}}N_{\text{consS}} + T_{\text{hL}}N_{\text{consL}} - T) + \beta N_{\text{OS}}N_{\text{OL}}))$. Note that this formulation accounts for the time predators spend handling large prey, which is a standard extension of the random predator equation for multiple prey types (Murdoch 1973). However, as cannibals were consumed by predators, we need to correct the cannibalism-induced mortality rate w by the factor k , the average proportional reduction of cannibals due to predation: $w = k\beta N_{\text{OS}}$. As the

mortality of large prey remained constant across densities in the predator treatments, the mortality rate of large prey is: $f(P, N) = (1 - \exp(-\alpha T))$, with α = predation rate on large prey. Thus, when accounting for prey depletion, we can predict the number of consumed prey by:

$$N_{\text{cons}} = N_{\text{OS}}(1 - \exp(aN_{\text{OS}}(T_{\text{hS}}N_{\text{consS}} + T_{\text{hL}}N_{\text{consL}} - T) + k\beta N_{\text{OS}}N_{\text{OL}})) + N_{\text{OL}}(1 - \exp(-\alpha T)).$$

The prey consumed by the predator (N_{Pcons}) is then given by the difference between the total number of consumed prey minus the prey that was cannibalized [$N_{\text{Pcons}} = N_{\text{cons}} - N_{\text{OS}}(1 - \exp(k\beta N_{\text{OS}}N_{\text{OL}}))$] and can be predicted by the 'composite functional response':

$$N_{\text{Pcons}} = N_{\text{OS}}(1 - \exp(aN_{\text{OS}}(T_{\text{hS}}N_{\text{consS}} + T_{\text{hL}}N_{\text{consL}} - T))) - N_{\text{OS}}(1 - \exp(k\beta N_{\text{OS}}N_{\text{OL}})) + N_{\text{OL}}(1 - \exp(-\alpha T))$$

While there is no reason to expect that the estimated handling time in the laboratory corresponds with the field due to the differences in spatial scale and lack of alternative prey, the laboratory experiments give a good estimate on the relative difference in the handling time: $T_{\text{hL}} = 2T_{\text{hS}}$, that can be used to estimate the handling time for large prey in the field. I used the handling time estimated from the field experiment as it is more likely to be realistic, and because the estimate of the attack coefficient is contingent on the corresponding estimate of the handling time. All other model parameters can be obtained from the field experiment (see Results). The full composite functional response model showed a good fit and adequately reproduced the complex wave-shaped pattern of the proportional mortality observed across all densities and size structures in the experiment (Fig. 3). This indicates that the observed complex pattern of the prey mortality with varying size structure can be explained by a composite functional response that accounts for the functional response of both stages separately. Note, that the general shape and the relative differences between curves are more informative than the exact fit of the individual curves to the observed data due to the variation around the estimates for handling time and attack rate. The wave-like shape of the proportional mortality results due to the longer handling time for large prey. In the present study cannibalism was low, which had a relatively small effect on the model and did not alter the general shape (Fig. 3). If cannibalism would be stronger, the impact of cannibalism on the prey mortality will depend on the consumption of the cannibals (Fig. 3) and potential corresponding behavioural indirect interactions (Rudolf, unpublished manuscript). Furthermore, due to predator satiation, the functional response for the large prey will eventually depend on the abundance of small prey at higher densities. While a detailed analysis of the effects of such factors on the model is beyond the scope of this study, future work that examines the dynamics of such composite functional responses in more detail will provide important insight into predator-prey dynamics in size-structured systems.

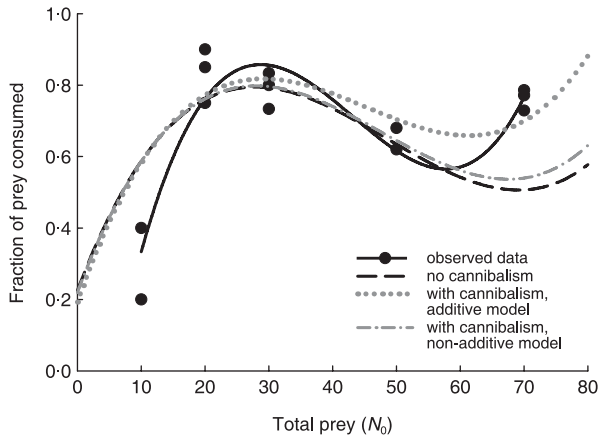


Fig. 3. Observed relative mortality (filled circles) for the ‘size-structured prey’ across total prey densities with the best fit polynomial regression model (black line), and subsets of the fully parameterized mechanistic ‘composite functional response model’ that accounts for the different functional response and relative densities of both small and large prey stage individually (see Discussion for details). The composite functional response model ‘without cannibalism’ (i.e. $\beta = 0$), the model ‘with cannibalism, additive model’ (grey dotted line) accounts for the cannibalism rate observed in the experiment ($\beta = 0.0008$) but does not correct for the reduction in cannibalism rates due to the observed consumption of cannibals (i.e. $k = 1$), the model ‘with cannibalism, nonadditive model’ (grey dashed/dotted line) is similar to the latter, but accounts for the consumption effect of cannibals on the cannibalism rates ($k = 0.135$) and represents the full parameterized composite functional response model. The curves are extended past the actual observations to facilitate comparison between the shapes of the different models. $T_{hs} = 0.1769$, $T_{hl} = 2 \times T_{hs}$, $a = 0.0062$, $\alpha = 0.1423$.

There is ample evidence that the functional response of a prey type is strongly influenced by the density of alternative prey (e.g. Murdoch 1969; Colton 1987; Soluk 1993). The model and empirical results in this study demonstrate that such interactions also occur among different sized cohorts within a species. However, the difference in the latter scenario is that both prey types represent only one species. Thus, if we want to predict the overall density-dependent predation of a prey we need to treat different prey stages separately. This is consistent with previous theoretical work that demonstrated that stage/size structure in the prey can strongly determine the dynamics of predator–prey systems (Claessen *et al.* 2000; de Roos *et al.* 2003; Abrams & Quince 2005; van Kooten *et al.* 2005; van de Wolfshaar *et al.* 2006; Rudolf 2007). This also suggests that previous predictions for community dynamics based on classical functional responses that ignore size/stage structure in the prey might be erroneous for most natural systems.

SIZE-STRUCTURED INTERACTIONS, TOP-DOWN CONTROL AND WEAK FOOD WEB LINKS

Previous empirical studies have shown that the effect of numerical changes in the predator on the mortality of the prey strongly depends on the size structure within the

predator population due to lethal or behavioural interactions between different size classes that often reduce the impact of predators (Murdoch & Sih 1978; Sih 1981; de Roos & Persson 2002; Crumrine 2005; Rudolf 2006; Rudolf 2007, 2008). The present study demonstrates for the first time experimentally that the size structure in the prey also strongly alters the effect of numerical changes in the prey and reduces the density-dependent control of the prey by the predator. This provides further evidence suggesting that size-structured interactions can reduce the interaction strength between interacting species and that this might be irrespective of the specific trophic level. Theoretical work suggests that weak interactions within food webs tend to stabilize community dynamics (Yodzis 1981; Neutel, Heesterbeek & de Ruiter 2002). Thus, size-structured interactions are likely to play an important part for the stability of natural communities, but more studies are needed to test if this effect is consistent across species and ecosystems.

Conclusions

Most ecologists would never attempt to predict the impact of a predator on the prey community by throwing different prey species into a single pool and treat them as one single homogeneous prey. This, however, is readily done when dealing with different size classes of a single species, although it is generally accepted that size strongly determines the shape and type of functional responses. This study demonstrates that size structure and size-structured interactions such as cannibalism within the prey strongly alter predator prey dynamics and the shape and type of functional responses. Thus, treating the prey as one homogeneous entity will lead to erroneous predictions in most real-world populations. However, if we account for the effect of size structure and the intraspecific interactions on functional responses by treating size classes as different functional groups, it is possible to reliably predict the dynamics of communities.

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