A macro-ecological approach to predators' functional response

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Abstract

Predation often deviates from the law of mass action: many micro- and meso-scale experiments have shown that consumption saturates with resource abundance, and decreases due to interference between consumers. But does this observation hold at macro-ecological scales, spanning many species and orders of magnitude in biomass? If so, what are its consequences for large-scale ecological patterns and dynamics?

We perform a meta-analysis of predator-prey pairs of mammals, birds and reptiles, and show that predation losses appear to increase, not as the product of predator and prey densities following the Lotka-Volterra (mass action) model, but rather as the square root of that product. This suggests a phenomenological power-law expression of the effective cross-ecosystem functional response. We consider the possibility that the same power-law holds dynamically within an ecosystem, and explore its consequences in a simple food chain model. The empirical exponents fall close to the boundary between regimes of donor and consumer limitation. Exponents on this boundary are singular in multiple ways. First, they maximize predator abundance and some stability metrics. Second, they create proportionality relations between biomass and productivity, both within and between trophic levels. These intuitive relations do not hold in general in mass action models, but are widely observed empirically.

These results provide evidence of mechanisms limiting predation across multiple ecological scales. Some of this evidence was previously associated with donor control, but we show that it supports a wider range of possibilities, including forms of consumer control. Limiting consumption counter-intuitively allows larger populations. It is worthwhile to reconsider whether the observed functional response arises from microscopic mechanisms and constraints, or could hint at selective pressure at the population level.

1 Introduction

Dynamical food web models must summarize, at the population level, the complexity of predator and prey behavior, physiology and ecology, often aggregating over both space and time. Since Lindeman[1], all models of trophic dynamics have shared the same fundamental structure, i.e. a balance of energy gains and losses from predation and from non-trophic processes. Ecologists have typically circumvented the hidden complexity and specificity of a given ecosystem by summarizing it in a simple function, the predator functional response, which describes how fluxes between trophic levels (predation) depend on stocks at each level (abundances). The most basic assumption is *mass action* – encounter probability scaling as the product of predator and prey density – but natural settings often deviate from this baseline[2]. The expression of the functional response has been the topic of extensive empirical and theoretical inquiries, and even of significant debates[3;4]. But there is no reason to expect that a single form of functional response, whether mechanistic or phenomenological, will be appropriate to model all ecological processes[5]. We may have to derive different expressions for

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different questions and scales, from short-term laboratory experiments[6], through multi-generation predator-prey cycles[7], to macro-scale food web properties such as biomass pyramids and stability[8].

Here we propose a new, empirically-motivated and phenomenological formulation of the functional response at large spatio-temporal scales. By contrast with mechanistic theories[9–12], our aim is not to ascertain the most realistic expression of predation rates, but to find a simple expression that can mimick observed rates across multiple scales, and then to understand its consequences for other food chain properties.

We first perform a meta-analysis of predation rates observed in the field for higher vertebrates, covering a wide range of study durations and areas. In the existing literature, most measurements of the functional response come from feeding experiments, restricted to time scales far shorter than the predator's generation time[10;13]. While experimental measurements are valuable for mechanistic models of specific populations, they may be misleading when extrapolating to macro-scale dynamics. For the latter, we need a phenomenological expression that exhibits some form of scale invariance, so that it may hold across different levels of aggregation. We propose a power-law expression similar to the Cobb-Douglas production function in economics[14]. We then find best-fit exponents suggesting a square root dependence of predation on both predator and prey density. These exponents are starkly lower than those expected from the simple mass action (Lotka-Volterra) model, suggesting that hidden mechanisms strongly limit predation. This phenomenological trend likely arises, in part, from aggregation over spatially and temporally heterogeneous distributions of predator and prey indivdiuals.

We then use a simple food chain model to explore the potential ecological consequences that this cross-ecosystem trend may have, provided that it also holds dynamically within one ecosystem. A first unexpected consequence is the emergence of consistent scaling relationships between predator and prey densities, and between production and biomass within each trophic level. Such scaling laws are widely found in data and postulated in models of ecosystem functioning[15;16]. They are notably used in ecosystem management models (fisheries, forestry, etc)[17;18]. But these relationships do not emerge spontaneously from arbitrary dynamical models, and only prevail under particular ecological conditions[8]. Our fitted exponents recover these simple proportionality rules that are usually associated with donor control.

The empirical scaling exponents also occupy a special position in our model predictions, as they correspond to a maximum in predator abundance and some metrics of stability. While our sublinear model suggests that predation frequency is much lower than in a classic Lotka-Volterra model, it also leads to larger standing predator populations. This apparent paradox is closely related to the hydra effect[19]. While this term originally applies to situations where increasing individual predator mortality leads to larger population size, it can be extended to other situations, such as ours, where predators that seem individually less fit than their competitors (e.g. consume less prey) may become more abundant in the long term[20].

Our work suggests that the prevalence of consumptive saturation and interference across scales, the empirical robustness of various allometric relationships, and the hydra effect (with its maximization of predator abundance) are all tied into the same puzzle of macro-scale trophic dynamics.

2 Methods

2.1 Dataset

We compiled data from 32 observational studies (details in Appendix S4) that reported or allowed us to infer kill rates k_{12} (number of prey killed per predator per year) in the field, as well as prey density N_1 and predator density N_2. For each species i, we also collected typical body mass measurements w_i (in kg) and mass-specific metabolic rate measurements m_i (in W/kg) from the AnAge database[21] and two studies by White and Seymour[22] and Makarieva et al.[23], as these traits were rarely reported for the populations studied in the field[24]. Whenever multiple values where found, we used the average values for each species.

The final dataset includes 46 predator-prey pairs including 26 predator species and 32 prey species. Predator species belong to classes Mammalia, Aves and Reptilia, while prey species belong to classes Mammalia, Actinopterygii, Aves and Malacostraca.

For each predator-prey pair, we computed the rate of biomass loss in the prey population due to predation (in $kg/km^2/year$) as

$$C = k_{12}w_1N_2 \tag{1}$$

and population biomass densities $B_1 = w_1N_1$ and $B_2 = w_2N_2$ in kg/km^2.

2.2   Food chain model

We investigate a simple food chain model of arbitrary length, where we follow the biomass density B_i of trophic level i as it changes through time,

$$\frac{dB_i(t)}{dt} = - \underbrace{L_i(t)}_{\text{internal losses}} + \underbrace{P_i(t)}_{\text{production}} - \underbrace{C_i(t)}_{\text{predation losses}} \tag{2}$$

where L_i and P_i represent internal losses and biomass production at level i, while C_i represents losses from predation at level $i + 1$. Production at level 1 arises from autotrophic growth

$$P_1(t) = g_1 B_1(t) \tag{3}$$

while at higher levels, we assume

$$P_{i+1}(t) = \varepsilon C_i(t) \tag{4}$$

where ε (taken here to be constant for simplicity[1]) is the conversion rate between the biomass lost through predation at level i and produced at level $i + 1$. Internal losses can arise from individual metabolic costs and mortality, μ_i, and from self-regulation or density-dependent processes such as competition and pathogens, D_i,

$$L_i(t) = \mu_i B_i(t) + D_i B_i^2(t). \tag{5}$$

Alternatively, we later consider the possibility of a power-law expression $L_i(t) = l_i B_i^{\delta}(t)$ which can interpolate between these two types of losses.

In the following, we only consider stationary properties of the dynamics (2), and thus drop the time-dependence of all quantities, which are assumed to take their equilibrium values.

2.3   Functional response

Throughout this study, we assume a power-law dependence of predation losses (see discussion in Box 1)

$$C_i \equiv C(B_i, B_{i+1}) = A B_i^{\beta} B_{i+1}^{\gamma} \tag{6}$$

with $\beta, \gamma \in [0, 1]$, and A a constant which contains the attack rate, i.e. the basic frequency of encounters for a pair of predator and prey individuals.

In this expression, $\beta = \gamma = 1$ recovers the classic mass action (Lotka-Volterra) model. An exponent $\beta < 1$ is our counterpart to the well-studied phenomenon of *prey saturation*: predation increases sublinearly with prey density, meaning that the ability of predators to catch or use each prey decreases with their availability[2]. On the other hand, $\gamma < 1$ indicates *predator interference*: larger predator densities lead to less consumption per capita[13].

These two predation-limiting phenomena are commonly reported and have been studied in various mathematical models, but these other classic functional responses are not appropriate for fitting data

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[1]The conversion rate ε is known to vary, but its range is limited compared to other quantities which can span orders of magnitude, see discussion in Barbier and Loreau[8].

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Box 1: Dimensional analysis and functional response

The difficulty of building a phenomenological cross-scale functional response is that it should account for both:

- between-systems parameter differences (e.g. different study areas, spatial structures, species traits),
- the within-system range of dynamical variables B_i that can lead to saturation and interference if they cross system-dependent thresholds.

We first appeal to dimensional analysis [27]. From (2) we see that predation losses C_i have the dimensions of biomass density over time, e.g. kg/km^2/year. A classic nondimensionalization choice [28] is to express these losses using predator density and mass-specific metabolic rate (see below for other options)

$$f_i = \frac{C_i}{m_{i+1}B_{i+1}} \tag{8}$$

where f_i is the normalized functional response, incorporating all biological mechanisms. Since f_i has no dimension, it can only depend on dimensionless quantities. If we introduce no other parameters than densities and metabolic rates, we can only construct two dimensionless ratios π_1 and π_2, so that f_i must take the form

$$f_i = f\left(\pi_1 = \frac{m_i}{m_{i+1}}, \pi_2 = \frac{B_i}{B_{i+1}}\right) \tag{9}$$

with some arbitrary function f. The choice $f(\pi_1, \pi_2) = 1$ leads to consumer dependence ($C = m_{i+1}B_{i+1}$), while $f(\pi_1, \pi_2) = \pi_1\pi_2$ leads to donor dependence ($C_i = m_iB_i$). More generally, the expression (9) is strongly reminiscent of ratio-dependent functional response [29], but it does not posit a specific functional form.

To deviate from this ratio-dependent expression, we must use additional parameters to construct other dimensionless quantities. The expressions (6) and (7) in the main text imply the form

$$f_i = f\left(\frac{B_1}{B_{1,\min}}, \frac{B_2}{B_{2,\min}}\right) \tag{10}$$

where $B_{i,\min}$ is a threshold for nonlinearity which may be system-dependent (see discussion in Appendix S1). These thresholds can be derived from other parameters that characterize each system, such as movement range $\mathcal{R}_i$ and body mass w_i, which could be combined into a density scale $w_i/\mathcal{R}$ above which interference may happen, see e.g. Pawar et al. [10]. Likewise, other biological rates can intervene if metabolic rate cannot be used as a universal "clock" [30]. These ratios can capture important differences between systems, and a number of heuristics and mathematical theories exist to guide their choice [31]: for instance, it is often useful to find ratios that are significantly larger than 1 in some systems and smaller than 1 in others, as this can indicate different processes that dominate in each case.

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compiled from different systems and scales. The standard saturating (Michaelis-Menten or Holling Type 2) expression

$$C(B_i, B_{i+1}) = \frac{AB_iB_{i+1}}{1 + HB_i},$$

with H/A traditionally defined as the handling time [2], and its extension with both saturation H and interference I (the DeAngelis-Beddington model [25;26])

$$C(B_i, B_{i+1}) = \frac{AB_iB_{i+1}}{1 + HB_i + IB_{i+1}} \tag{7}$$

are applicable to short-term feeding experiments where we observe consumption as a function of abundances for the same pair of species. In our dataset, H and I are expected to depend on species traits [10], but also on the spatial structure and area of each study site. We discuss in Results the difficulties associated with using this expression, which may produce similar predictions as (6), but only for carefully tuned H and I, and in a fully-saturated regime ($HB_i + IB_{i+1} \gg 1$) where this model does not seem meaningful as a mechanistic account of predation.

2.4 Statistical analysis

We seek to identify the empirical relationship $C(B_1, B_2)$ between predation losses and the biomasses of predator and prey. Throughout most of the text, we choose to express this relationship in terms

of biomass density B_i rather than population density N_i as we expect the ratio of predator and prey biomasses to be more meaningful than the ratio of their numbers (which can vary dramatically with their size ratio). We nevertheless include results based on N_i below, as well as results based on $E_i = m_i B_i$ the "energy" density [32], in order to find which variables lead to the best predictions, i.e. the lowest amount of residual variation in predation intensity.

As the colinearity between B_1 and B_2 (Fig. 1) may obscure their respective contributions to $C(B_1, B_2)$, we perform a commonality analysis, which is a statistical test based on variance partitioning. We use the function *regr* of the R package *yhat* to check the existence of suppression i.e. the distorsion of regression coefficients due to the colinearity between predictors [33]. This test shows the absence of suppression, allowing us to directly interpret the respective contributions of B_1 and B_2 to $C(B_1, B_2)$ below.

3 Results

3.1 Meta-analysis of kill rates

Expression y	$\dfrac{C}{B_1}$	$\dfrac{C}{\sqrt{B_1 B_2}}$	$\dfrac{C}{B_2}$	$\dfrac{C}{N_1}$	$\dfrac{C}{\sqrt{N_1 N_2}}$	$\dfrac{C}{N_2}$	$\dfrac{C}{E_1}$	$\dfrac{C}{\sqrt{E_1 E_2}}$	$\dfrac{C}{E_2}$	$\dfrac{C}{B_1^\beta B_2^\gamma}$
$std(\log_{10} y)$	0.90	**0.77**	0.97	1.6	1.5	1.5	0.93	**0.77**	0.9	**0.74**

Table 1: Residual standard deviation for each considered expression of yearly kill density against biomass density B_i, population density N_i, and "energy" density $E_i = m_i B_i$. See Fig. 1 for a visualization of the residual variation with B_1/B_2 for the first three expressions. For the rightmost expression, we use the best-fit exponents $\beta = 0.47$, $\gamma = 0.30$, and see that this contributes only a small reduction of residual variation compared with the square root expression (second column).

The classic Lotka-Volterra (mass action) model suggests a bilinear relationship between the amount of biomass lost per year and per unit area to predation (C) and the biomass density of prey (B_1) and predator (B_2),

$$C(B_1, B_2) \sim B_1 B_2 \tag{11}$$

which is a special case of both the power-law model (6) and the DeAngelis-Beddington model (7).

We first focus on the power-law model, for which we find the best-fit exponents $\beta = 0.47 \pm 0.17$ and $\gamma = 0.30 \pm 0.19$. The limited extent of our dataset implies large uncertainty on the exact exponents, but they are starkly sublinear. In the following, we will consider various arguments for thinking that these exponents might approximately satisfy the relationship $\beta + \gamma \approx 1$, which would imply a ratio dependent form

$$C(B_1, B_2) \sim B_1^\beta B_2^{1-\beta} = B_2 \left(\frac{B_1}{B_2} \right)^\beta \tag{12}$$

Due to the colinearity between B_1 and B_2 ($R = 0.56$, Fig. 1b), we might expect any model of this form to provide a comparable fit, ranging from $\beta = 1$ (donor dependent) to $\beta = 0$ (consumer dependent). Nevertheless, our colinearity analysis strongly indicates $\beta \approx \frac{1}{2}$ as the most likely value. This is supported by the fact that C/B_1 and C/B_2 both exhibit a residual variation in B_1/B_2 ($p < 10^{-6}$, $R = 0.61$ and $R = -0.52$ respectively, Fig. 1c) whereas our best fit exhibits no residual variation ($p = 0.4$, $R = -0.08$), suggesting that neither strict donor nor consumer dependence prevails in our data.

As discussed in Box 1, we could also expect predation loss C to depend on metabolic rates m_i and m_{i+1}. Fitting a power-law expression with the structure (9)

$$C \sim m_2 B_2 \left(\frac{m_1}{m_2} \right)^\nu \left(\frac{B_1}{B_2} \right)^\beta \tag{13}$$
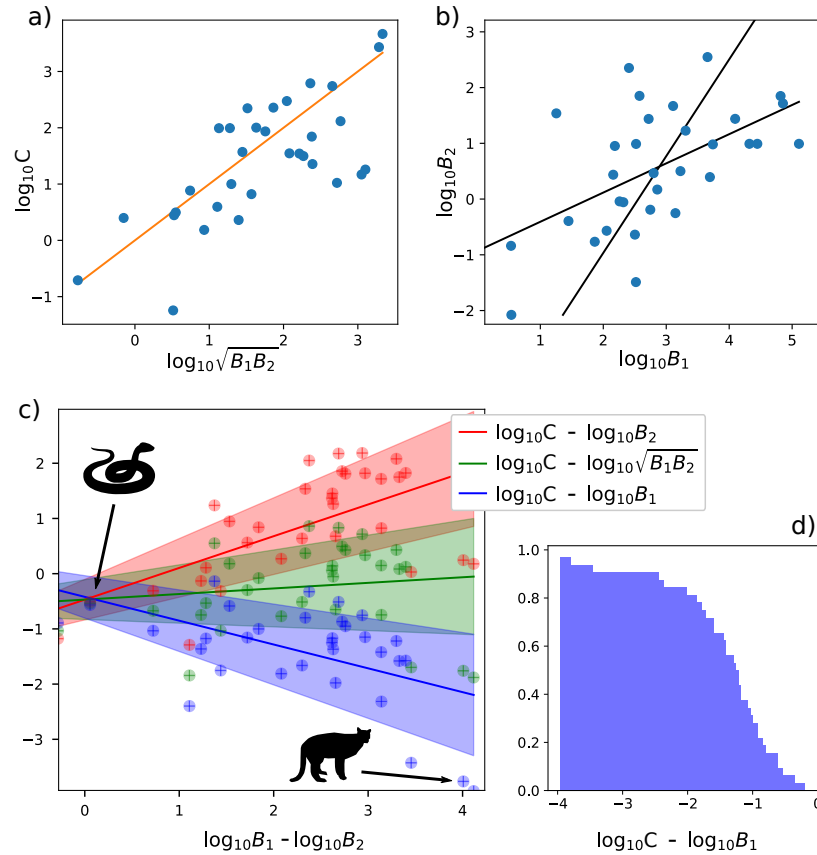
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Figure 1: Empirical scaling of biomass and predation. Each point represents one study in our meta-analysis. **(a)** Yearly predation losses C against square root product of prey and predator biomass densities $\sqrt{B_1 B_2}$ in log scale. The solid line represents the 1:1 relationship. **(b)** Colinearity of prey and predator biomass densities B_1 and B_2 in log scale ($R = 0.61$). Solid lines show power-law fits taking each species biomass as the dependent variable: $B_2 \sim B_1^{0.52}$ and $B_1 \sim B_2^{0.51}$. **(c)** Residual variation with $\log_{10} B_1/B_2$ when assuming strict donor dependence ($C \sim B_1$), our proposed square-root law ($C \sim \sqrt{B_1 B_2}$), or strict consumer dependence ($C \sim B_2$). Residues around the square-root law shows no trend ($p = 0.4$, $R = 0.08$), and have minimal variance (see Table 1). **(d)** Anti-cumulative distribution $P(\log_{10}(C/B_1) > x)$ of the fraction C/B_1 of standing biomass consumed per year. More than 10% of prey biomass is consumed yearly in 30% of systems.

we find exponents $\nu = 0.42 \pm 0.3$ and $\beta = 0.53 \pm 0.17$, meaning that this approximates a square-root relationship in "energy" densities [32]

$$C(m_1, m_2, B_1, B_2) \sim \sqrt{E_1 E_2}, \qquad E_i = m_i B_i. \tag{14}$$

We can repeat our analysis using population densities N_i rather than biomass densities B_i, but find significantly worse fits. We show in Table 1 the residual standard deviation for these various options, and find that our model (15) gives the lowest residues, although the difference with (14) is minimal.

3.2   Comparison to a classic functional response

The DeAngelis-Beddington (DAB) model (7), or Type 2 with predator interference, can be fit to our data. While it may be useful as a mechanistic model for a single system [5], it is unsatisfactory in our macro-ecological approach.

We can either take handling time H and predator interference I as constant parameters across systems, or try to construct them as functions of other known parameters for each system (e.g. body sizes or metabolic rates). We show in Appendix S4 that we were unable to identify any expression that allows a meaningful fit: we always find that almost all predator-prey pairs are in the saturated part of the function, i.e. $HB_1 + IB_2 \gg 1$, meaning that we recover a simple ratio-dependent expression rather than a true saturating function.

Furthermore, we show that the colinearity $B_1 \sim B_2$ can make it difficult to differentiate between an additive law, $HB_1 + IB_2$, and a multiplicative law with exponents adding up to one, $B_1^{1-\beta} B_2^\beta$, in the denominator of the functional response. This has been put forward as an important criticism of the Cobb-Douglas production function [34] which resembles our power-law model (6). But we find here that prey and predator are equally limiting. This requires a fine-tuning of the ratio of handling time to interference, H/I, so that HB_1 and IB_2 are comparable in magnitude. This is found in our best fit of the DAB model (7) but is difficult to justify mechanisticially, whereas the same reality may be more clearly and simply encapsulated by $\beta \approx 0.5$.

We conclude that Occam's razor cuts in favor of the power-law expression in our data. A parsimonious model approximating a square-root relationship (Fig. 1a)

$$C(B_1, B_2) \approx A_{12} \sqrt{B_1 B_2}. \tag{15}$$

appears, given its simplicity, to be a good phenomenological approximation that holds across scales and species. We now discuss its potential theoretical implications.

3.3   Consequences for population sizes

We consider two trophic levels, an show predator density B_2^* in Fig. 2a as a function of both exponents β and γ, and observe two distinct regimes.

These regimes can be understood by first assuming that self-regulation can be neglected at both levels. In that case, our dynamical model (2) with the power-law functional response (6) has the equilibrium solution (see Appendix S1)

$$B_1^* = \left(\frac{g_1^{1-\gamma} (\mu_2/\varepsilon)^\gamma}{A_{12}} \right)^{\frac{1}{\beta+\gamma-1}}$$

$$B_2^* = \left(\frac{g_1^\beta (\mu_2/\varepsilon)^{1-\beta}}{A_{12}} \right)^{\frac{1}{\beta+\gamma-1}} \tag{16}$$

where we see that $\beta + \gamma = 1$ leads to a singularity, meaning that self-regulation cannot be neglected for these exponents.

The first regime is found when $\beta + \gamma > 1$, and we call it the *flux-limited* regime (see Box 2 and Fig. 2b). In this case, the solution (16) is a good approximation at low self-regulation (small D_i).

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Box 2: Definitions

We introduce two sets of terms to distinguish:

- what sort of *dependence* is assumed for predation losses C

  donor-dependent predation A model where $C(B_1, B_2) = C(B_1)$

  consumer-dependent predation A model where $C(B_1, B_2) = C(B_2)$

  ratio-dependent predation A model where $C(B_1, B_2) = C(B_1/B_2)$

- which of predation or non-trophic losses is the *limiting* factor for abundances:

  flux-limited regime A solution of our model where prey stocks are strongly depleted, so consumers compete over the flux of prey production. If we increase individual consumption (attack rate and exponents), fewer predators coexist at equilibrium.

  stock-limited regime A solution of our model where prey levels are mainly set by their own self-regulation, so that equilibrium predator abundance increases with attack rate and exponents.

These definitions should be distinguished from terms of *control* (e.g. donor control, bottom-up and top-down control), which are often used in more complex statements about the dynamics, e.g. how the system will respond to a perturbation, see discussion in Barbier and Loreau[8]. While these notions have strong overlaps (e.g. the stock-limited regime only allows bottom-up control), we employ a distinct terminology throughout this study to avoid possible confusions surrounding these terms.

We notice that this solution has two counter-intuitive properties: predator biomass B_2 decreases with attack rate A_{12} and exponents β and γ, and increases with predator mortality μ_2 (if there is saturation, $\beta < 1$). This is because prey stocks are brought to low levels, so predators simply divide between themselves the flux of prey production: the more each predator consumes per capita, the fewer predators can coexist. As we discuss below, this inverse relationship between individual fitness (high attack rate, low mortality) and population abundance has been demonstrated in various models and called the "hydra effect"[19]. I

If $\beta + \gamma < 1$, the solution without self-regulation (16) becomes unstable. In the stable equilibrium, the biomass of prey will be mainly determined by their self-regulation i.e. $B_1^* \approx g_1/D_1$, leading to what we call the *stock-limited* regime (Box 2 and Fig. 2c). In that case, predator biomass

$$B_2^* = \left(\frac{\varepsilon A_{12}}{\mu_2}\right)^{\frac{1}{1-\gamma}} \left(\frac{g_1}{D_1}\right)^{\frac{\beta}{1-\gamma}} \tag{17}$$

increases with attack rate and with exponents β and γ. The fact that the previous equilibrium (16) is unstable reveals a type of Allee effect: if initial prey levels are below a threshsold, predators will consume all the prey (consumption will decrease too slowly to adjust to the falling resoruce level) then collapse.

We show in Fig. 2 that predator abundance is maximized at the boundary between these two regimes. This boundary lies close to the line $\beta + \gamma = 1$, where predation losses scale as

$$C(B_1, B_2) \sim B_1^\beta B_2^{1-\beta}$$

which is close to the exponents suggested by our meta-analysis. We however note that the regime boundary moves away from this line as we decrease attack rates A_{12} and increase self-regulation D_i at both levels. For sufficiently high self-regulation, even Lotka-Volterra dynamics can enter the stock-limited regime, which we interpreted as bottom-up control in previous work[8].

The overarching pattern is that the predator density first increases with attack rate A_{12} and exponents β and γ, when predation losses are still negligible for the prey, up to the regime boundary described above. Past this point, faster consumption leads to significant resource depletion and a drop in predator population[15], as competition increases more than growth.
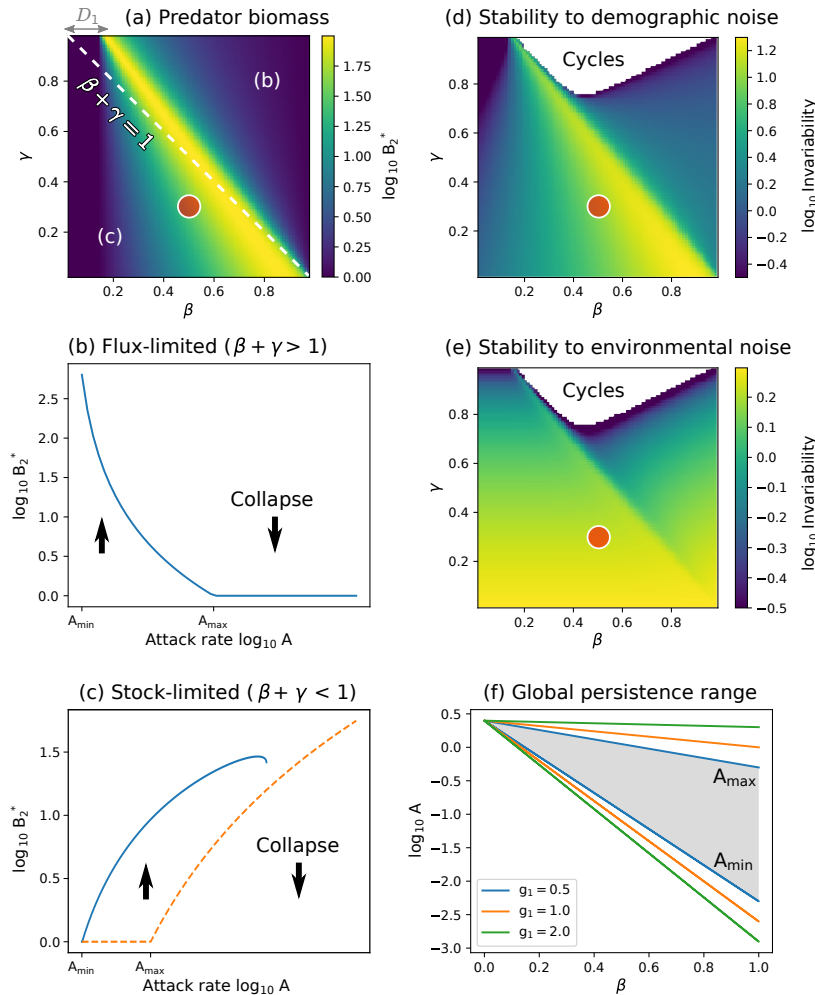
Figure 2: Equilibrium predator abundance and persistence in the dynamical predator-prey model ($D_i = 10^{-3}$ at both levels, $g_1 = 1$, $\mu_2 = 2$, $\varepsilon = 0.8$). The red dot indicates the empirically supported exponents $\beta \approx 0.5$, $\gamma \approx 0.3$. **(a)** Predator biomass density B_2^* (in log scale) varies with exponents β and γ in the power-law functional response (6). It is maximized on a boundary (lightest color on the graph) separating two different regimes (b and c). This boundary is close to the dashed line $\beta + \gamma = 1$, but deviates toward larger β due to prey self-regulation (the deviation increases with D_1). **(b)** For $\beta + \gamma > 1$, the resource is mainly limited by predation, and predator population decreases with attack rate, going extinct ($B_2 < 1$) at $A_{\max}$. **(c)** For $\beta + \gamma < 1$, predator biomass (solid line) increases with attack rate, since the resource is mainly limited by its own self-regulation. The predator survives only if $A > A_{\min}$, but high attack rates $A > A_{\max}$ give rise to an unstable equilibrium (dashed line) under which populations can go extinct. The stable equilibrium disappears at higher A. (f) For the predator to persist in both regimes and for all initial conditions, we must have $A_{\min} < A < A_{\max}$ (shaded region). This interval expands with increasing β (less saturation) and basal growth g_1. **(d,e)** Predator invariability (1/coefficient of variation) around equilibrium (see e.g. Barbier and Loreau[8]; Arnoldi et al.[35]) in response to (d) demographic fluctuations and (e) environmental perturbations affecting the predator. For large γ and intermediate β, the equilibrum is unstable and replaced by limit cycles. This area is left blank as invariability cannot be computed analytically, but it remains well-defined in simulations.

3.4   Consequences for stability

We also observe how functional response exponents affect the stability of predator populations. Fig. 1(d,e) represents the invariability (inverse of the coefficient of variation over time) of predator abundance, computed in two scenarios: demographic stochasticity, where fluctuations (arising from birth and death processes) are proportional to $\sqrt{B_2}$, and environmental perturbations which affect the predators proportionally to their abundance B_2, see Arnoldi et al. [35]. Other scenarios and more stability metrics (including asymptotic resilience) are represented in Fig. S1 in Supporting Information, and show qualitatively similar patterns.

   We make three main observations. First, the optimal parameter values for stability depend on the perturbation scenario (e.g. demographic or environmental noise) and the choice of stability metric, as having a larger abundance leads to higher stability in some metrics [35]. It is therefore important to know which scenario and metric are relevant for empirical dynamics, and different ecosystems may possibly have different optima. Second, all the cases studied here display a ridge of increased predator stability at the maximum of its abundance, close to the transition line $\beta + \gamma = 1$, and a drop in stability right after this ridge. Third, while abundance depends almost symmetrically on β and γ, and is maximized close to the transition between regimes, stability favors systems closer to donor-dependence. Moving toward larger β along the line $\beta + \gamma = 1$ reduces the likelihood of cycles and widens the region of parameters with high stability both to demographic and environmental noise (Fig. 2d and e). More generally, stability to environmental perturbations can be improved by having lower γ than what would maximize abundance (i.e. going toward the bottom of Fig. 2e).

3.5   Scaling of biomass and production across levels

Previous literature has reported scaling laws between biomass and production within one trophic level, and between biomasses at different trophic levels [16]. While these studies generally focus on cross-ecosystem scaling laws, we consider here the laws that would arise within an ecosystem as we vary our model parameters (we thus assume that there are no other latent variables that might create different laws within and between ecosystems).

   We show in Appendix S3 that our food chain model at equilibrium can give rise to a scaling of production with biomass

$$P_i \sim B_i^{\delta} \tag{18}$$

and the scaling of predator biomass with prey biomass

$$B_{i+1} \sim B_i^{\alpha}. \tag{19}$$

As discussed in Barbier and Loreau [8], neither of these laws can arise for more than two levels in a classic Lotka-Volterra model without self-regulation ($\beta = \gamma = 1$, $D_i = 0$).

   There are only two ways in which these scaling laws can hold consistently across multiple trophic levels: either they are imposed by an extreme type of functional response, or they originate from an equilibrium balance between trophic processes and other losses. We now show that only the second possibility is compatible with our data.

   The first possibility requires predation to be strictly donor-dependent or consumer-dependent ($\gamma = 0$ or $\beta = 0$). The functional response then completely determines the scaling between production and biomass,

$$P_i \sim B_i^{\beta} \qquad \text{or} \qquad P_i \sim B_{i-1}^{\gamma} \tag{20}$$

In this case, we can ignore internal losses, i.e. set $L_i = 0$ in (2), and find

$$P_{i+1} = \varepsilon P_i \qquad \Rightarrow \qquad B_{i+1} \sim B_i \tag{21}$$

which entails a strict proportionality between predator and prey biomass (exponent $\alpha = 1$). This has been widely discussed by proponents of the ratio-dependent functional response [29], and the existence of such proportionality laws was put forward as evidence of donor control. But since we find nonzero β and γ, this possibility is excluded by our data.

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The second possibility arises in our model from the power-law functional response (6). If we assume that internal losses remain comparable to trophic gains and losses, $L_i \sim P_i$, these internal losses will then determine the scaling between production and biomass. Indeed, if they scale with B_i with some exponent δ, we find

$$L_i \sim B_i^{\delta} \quad \text{and} \quad L_i \sim P_i \qquad \Rightarrow \qquad P_i \sim B_i^{\delta}. \tag{22}$$

For instance, we have $\delta = 1$ for individual mortality, and $\delta = 2$ when a self-regulating process, such as intra-specific competition or pathogens, is the main contributor to internal losses. We show in Appendix S3 that that a relationship emerges between the exponents defined here and the functional response exponents

$$\alpha(\delta - \gamma) = \beta. \tag{23}$$

Commonly assumed scaling exponents for biomass and production are $\alpha = \delta = 1$, i.e. simple proportionality between predator and prey biomass, and between production and biomass at each level. These relations are used in various mass-balanced models[36] or theory on biomass pyramids[17]. They entail $\beta = 1 - \gamma$, meaning that the relationship between predation losses and biomasses would be

$$C(B_1, B_2) \sim B_1^{\beta} B_2^{1-\beta} = B_2 \left(\frac{B_1}{B_2} \right)^{\beta}$$

for which we found some empirical support.

4 Discussion

The predator functional response, describing how predation rates depend on predator and prey abundances, is a crucial component of trophic ecology that has a considerable impact on predator-prey dynamics[2]. Long-standing debates have opposed advocates of various forms of functional response, such as prey-dependent or ratio-dependent expressions[3,4]. The current consensus seems to be that there is no correct universal expression; rather, different functional responses are appropriate in different settings, and they should be derived mechanistically for each studied ecosystem[4,5].

From a macro-ecological point of view, however, what matters is how the functional response scales up across several orders of magnitude in abundance, body size and area. Experiments typically measure predation rates for a given pair of species, and how they respond to varying the number of prey and predator individuals, on a short time scale and in an enclosed space. Here, we used kill rates recorded in the field to estimate how predation rates over long times (e.g. a year) vary between species (mainly terrestrial vertebrates) and across much larger spatial scales.

Our empirical analysis points to a phenomenological power-law, which differs from commonly-studied functional responses. We discuss below whether this cross-ecosystem trend could be an artefact that does not reflect the dynamical dependence between predation rates and species densities.

If this law does hold dynamically within each ecosystem, it may have striking theoretical implications. The empirical exponents fall close to the line $\beta + \gamma = 1$, for which our dynamical food chain model makes two predictions: predator abundance is maximized, and simple allometric scaling laws appear between different ecosystem functions and between trophic levels. We may then ask whether these two important macroscopic properties arise accidentally from a functional response shaped by other constraints, or whether the functional response is in fact selected for its dynamical consequences.

4.1 Is the empirical functional response a statistical artefact?

Our results may appear to invite an objection: predators cannot consume more than all the prey. Therefore, our findings might not reflect any dynamically-relevant functional response, but a simple effect of aggregating very diverse data sources in which predation losses C are always bounded

by prey density B_1, giving the illusion of a donor-dependent or similar relationship. But we first note that, as shown in Fig. 1d, yearly predation losses rarely constitute a large fraction of standing biomass, meaning that this objection, even if true, should not apply to most of the data.

We must further discuss the validity of this objection, by remarking that predation losses are in principle limited not by standing biomass, but by production,

$$C_i \lesssim P_i$$

which can be much larger than standing biomass. For instance, a Lotka-Volterra model with a classic biomass pyramid $B_{i+1} \sim B_i$ would predict the predation and production of all consumers to scale *quadratically* with biomass, as $B_{i+1}B_i \sim B_i^2$. In other words, more and more productive systems would exhibit more and more predation, but populations would only increase as the square root of these fluxes, and prey density B_1 would not represent a bound on predation.

Whether losses are truly bounded by prey biomass depends on aspects of ecosystems and of data that are obscured by aggregation and by the continuous-time description (2). For instance, it is plausible to have $C_i > B_i$ (where C_i is measured over a year) if predators mainly consume individuals aged less than a year – many juveniles are produced and quickly consumed, without appearing in the standing population whenever it is measured. If, however, predators target individuals of age one and above, these individuals must have appeared in last year's census and we expect $C_i < B_i$ (with a year's delay that we ignore under equilibrium assumptions).

The studies and the life histories of species considered here, although limited to terrestrial carnivory, span a rather wide range of time scales. Hence, a supralinear scaling of production and losses with biomass is not implausible. Analogous relationships have been proposed in economic systems, with empirical exponents ranging from 1.16 (economic growth as a function of city size[37]) to 1.6 (stock trading rate as a function of number of shares[38]). We could also invoke empirical facts that have been interpreted as evidence for the Allee effect[39], since this effect assumes that per-capita growth rate increases with density, and hence, that production P_i is supralinear with biomass B_i. We saw in Fig. 1d that $C_i < B_i$ does hold in our data, but there is no clear mechanism enforcing this bound here.

While a broader analysis across taxonomic groups, timescales and life histories would be needed to test for possible artefacts such as those suggested above, our provisional conclusion is that this empirical scaling of predation with densities is indeed meaningful.

4.2 Is this functional response a consequence of other constraints?

Nonlinear functional responses, with saturation and interference, are generally assumed to be imposed by a broad range of microscopic or mechanistic factors (physiology, behavior, spatial structure, individual trade-offs between competition and consumption, etc). In various ways, these factors limit the space or time available for encounters, leading to less predation than under mass action.

The most immediate candidate mechanism to explain a pattern that holds across ecosystems and scales is spatial structure. For instance, local prey refuges have been proposed as a classic explanation for a donor-dependent[40] or DeAngelis-Beddington response[41]. More generally, spatial structure has been shown to lead to power-law-like functional responses in agent-based or spatially explicit simulations[5;42]. But this seems to allow a wide range of possible exponents, and therefore, it does not provide an unequivocal explanation for our observations.

A functional response $C \sim \sqrt{B_1 B_2}$ ($\beta, \gamma \approx \frac{1}{2}$), similar to our measurements, could be imposed by spatial structure in two dimensions, if prey and predators occupy almost mutually exclusive regions and only meet at the boundaries (given that the periphery of a region scales like the square root of its area). This could happen due to local prey depletion by predators, and it is a plausible explanation, although it requires very strong negative correlations in space[43], and would predict a different scaling in three-dimensional settings[10], which remains to be tested.

We cannot conclude, based on our data alone, that spatial heterogeneity is the main driver of our empirical functional response. Spatially explicit data may be necessary to test this hypothesis. If

spatial structure does not impose an exponent, but allows a range of possibilities, it could provide the means through which predators and prey organize to achieve a functional response that is selected by other processes[44;45].

4.3   Can this functional response emerge from selection?

The functional response and exponents found here are close to those which theoretically maximizes predator abundance and some metrics of stability. Yet this comes at an apparent cost to the individual predator, which consumes less here than under a classic Lotka-Volterra (mass action) scenario. We recall the so-called hydra effect[19], a counter-intuitive but common phenomenon in resource-based dynamics: decreasing the fitness of individual consumers, e.g. increasing their mortality or reducing attack rates, can lead to larger populations in the long run, when this reduces competition more than it reduces growth. We showed that a similar effect applies here to consumptive saturation and interference, as represented by the power-law exponents β and γ in the functional response. Maximal predator abundance is reached when exponents follow the relationship $\beta + \gamma = 1$, which holds approximately in data.

This resonates with long-standing debates in ecology and evolution, in particular the group selection controversy[46] and its accounts of selection for population abundance[47] or persistence[48]. Should we conclude here that consumption rates are selected to optimize population-level properties? And would this optimization require population-level selection, or can it arise strictly at the individual level? We now show that these are valid possibilities in our setting.

A first possibility is that our proposed functional response is outcompeted at the individual level, but selected at the population level, due to some positive consequences of having larger abundances. Extending our model to include simple adaptive dynamics of attack rate and scaling exponents, we indeed find that this functional response is dominated by other strategies (Appendix S2). We observe maladaptive evolution: mutants with ever faster consumption will outcompete and replace residents, reaching ever lower equilibrium abundances, up until the point where the predators go extinct. A classic solution is to invoke competition between groups in a spatial setting[46;47], as a larger group can send out more propagules and will often disperse faster[49]. These intuitions pervade a large literature on the evolution of "prudent" or "restrained" predation strategies through group selection, e.g. the milker-killer dilemma[44;45;50;51].

A less considered possibility is that our functional response is favored even at the individual level, as a result of direct competitive interactions. The dominance of faster consumers relies on the assumption that competition takes place only through resource consumption. In the presence of other competitive interactions that are not tied to resource levels, a larger standing population can resist invasion both by mutants and by other species. We suggest in Appendix S2 that, for the resident population to be evolutionarily stable, this non-consumptive competition must induce higher mortality in individuals that *search* for more resources, regardless of their success. This feature seems plausible for territorial behavior and aggression as widely displayed by higher vertebrates[2], but could also be induced e.g. by allelopathy in other organisms. Under these conditions, slow consumers can avoid being overtaken by fast consumers, even within a single population.

As a further perspective, all our arguments so far have taken the viewpoint of the predator's strategy and abundance, despite the fact that our empirical results suggests a roughly equal role of predator and prey density in limiting predation. Our best-fit exponents situate real systems close to the limit between a regime where prey suffer from predation, and a regime where they are mainly self-regulated. It is thus possible that prey strategies, or a balance between prey and predator selective pressures, are at play.

We conclude that selection on the functional reponse exponents to maximize population size can easily arise in variations of our model, provided that we include non-consumptive processes

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[2] As an illustration, lions are both outliers in our dataset, since they display lower consumption rates than the general trend (see Appendix S5), and are a major cause of mortality among other carnivores[52]. These two features together may indicate a counter-intuitive predator strategy, which involves both consuming less prey and spending more time in non-consumptive competition, leading to and benefitting from population maximization.

(from dispersal to aggression). Some of these explanations involve competition between populations, while others take place within a single population, but all favor strategies that maximize predator abundance.

4.4 Can this functional response explain other power-law relations between ecosystem functions?

Relations between production and biomass, and between predator and prey densities are widely assumed and observed to take linear or power-law forms [16;17]. Yet, we have shown here and in previous work [8] that such relations do not emerge universally from arbitrary dynamical models. They require particular ecological conditions, where internal losses L_i within trophic level i are comparable to, or larger than, predation losses C_i (both in their magnitude and in their density-dependence). For instance, in a Lotka-Volterra model, strong self-regulation within a trophic level is required to observe linear relations between functions and between levels, creating classic biomass and energy pyramids [8]. When internal losses are only weakly density-dependent, our proposed functional response with exponents $\beta + \gamma = 1$ recovers the same linear relations and pyramids.

A recent meta-analysis by Hatton et al. [16] found sublinear scalings of production with biomass (with exponent α) and of predator density with prey density (with exponent δ), following $\alpha \approx \delta \approx \frac{3}{4}$, reminiscent of metabolic allometry. Assuming that this cross-ecosystem law also holds dynamically within one system, so that our model results apply, this requires strong interference and saturation, $\beta + \gamma < 1$. Inserting our empirical best-fit exponents ($\beta \approx 0.47 \pm 0.17$, $\gamma \approx 0.30 \pm 0.19$) gives a predicted scaling $\alpha, \delta \approx 0.85 \pm 0.17$ which is compatible with either a linear relationship or the findings of Hatton et al.'s meta-analysis.

In summary, the existence of well-defined scaling laws across trophic levels, between ecosystem functions such as biomass and production, is less self-evident than it may appear. We suggest here that some empirically-supported laws might arise from an underlying functional response, and would thus be secondary consequences of the mechanisms that determine this functional response.

4.5 Conclusions

We have shown that the intensity of predation across a range of spatial scales and species can be modeled as a power-law function of both consumer and resource densities, that deviates strongly from the mass action (Lotka-Volterra) assumption. This empirical investigation should be extended to other taxonomic groups, and confronted to experimental evidence, to pave the way for a deeper understanding of the emergent functional response at macro-ecological scales.

This phenomenological power-law model can recover classic donor control as a special case, but our empirical estimation suggests that predator and prey are roughly equally limiting, leading to an unusual square root expression. This model is reminiscent of the Cobb-Douglas production function in economics [14], which similarly arises in a macroscopic context, and is also disputed as either a salient empirical fact or a simple consequence of colinearity and aggregation [34]. Nevertheless, the main observation in both fields is that two factors – labor and capital, or consumers and resources – are co-limiting, so that doubling the output requires doubling each of the two inputs.

Scaling laws measured across ecosystems do not always hold dynamically within a single ecosystem [53]: other latent variables could differ between ecosystems, altering the relationship between predation and abundances that we would observe locally. Future work should test whether our proposed functional response truly applies to the dynamics of an ecosystem. If it does, we have shown that our empirical exponents have important implications for various features of trophic systems: in particular, they are close to maximizing predator population size and some measures of stability. This could reflect an universal selection pressure acting upon consumption rates. We notably discussed the possibility that non-consumptive competition (e.g. territorial exclusion or allelopathy) would prevent the usual dominance of smaller populations of faster consumers, and offer an explanation for the evolution of strategies that limit predation to maximize predator abundance.

We hope that future work can address two fundamental questions: (1) whether the functional response that we observe stems purely from aggregation or from biologically meaningful phenomena, and (2) whether some universal principle, such as selection for larger populations, could explain this functional response and, through it, the emergence of other widespread macro-ecological scaling laws.

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Author contributions

All authors contributed to the design of the study and the writing of the manuscript. MB analyzed the theoretical model. LW designed the meta-analysis and assembled the data. LW and MB jointly analyzed the data.

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