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The effects of predation risk on prey stoichiometry: a meta-analysis

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Abstract. A decade ago, the general stress paradigm (GSP) aimed to develop a predictive framework linking predator effects to ecosystem function. The GSP was based on the notion that animals, across taxa, exhibit similar physiological responses to predation risk that divert resources from growth and reproduction (which require N-rich biomolecules [i.e., protein]) to emergency functions (which require C-rich biomolecules [e.g., carbohydrates]). The GSP predicts that stressed prey should have a greater dietary demand for C-rich resources, a higher body C:N ratio, and elevated N excretion. Now, 10 yr later, we aim to revisit the GSP—using quantitative meta-analysis to test the original predictions of the GSP and how (1) predator hunting mode, (2) multiple stressors, and (3) prey dietary shifts affect prey stoichiometric responses to predation risk. Our data set was consistent with previous work showing that predation risk increases prey glucocorticoid levels and metabolic rates and decreases prey growth rates. We found that predation risk tended to decrease the fat, carbohydrate, and protein content of prey bodies; increased the C:P and N:P of prey bodies; but had no effect on the C, N, P, or C:N content of prey bodies. Additionally, we found no effect of predation risk on the N content of prey excretions. Prey responses to predation risk were unaffected by multiple stressors or the prey's ability to shift their diet, but predator hunting mode did affect the nature of prey stoichiometric responses. Specifically, ambush predators decreased prey macronutrient content and suppressed prey growth, and active predators had no effect on prey macronutrient content and a smaller effect on prey growth than ambush predators. The significant effects of predation risk were supported by robust fail-safe numbers, despite the high between-comparison heterogeneity that was found in all analyses. Our findings highlight the need (1) to test the underlying mechanisms and emerging patterns of the GSP in diverse taxa, (2) to explore the mismatch between prey macronutrient content and elemental stoichiometry, and (3) to expand the conceptual framework to include more inducible defenses (e.g., behavioral and morphological) and predator traits.

Key words: general stress paradigm; hunting mode; induced defenses; nonconsumptive effects; phenotypic plasticity; predation risk; predator effects; stoichiometry; stress physiology.

Introduction

Predators regulate the rates of nutrient cycling by consuming prey (consumptive effects [CEs]; Abrams et al. 1996) or by inducing behavioral, morphological, physiological, and life history defensive phenotypes in prey (nonconsumptive effects [NCEs]; Schmitz et al. 1997, Werner and Peacor 2003). The induction of defensive phenotypes can alter prey diets and the nutritional content of prey bodies and waste materials, as well as stunt prey growth and fecundity (McPeek 2004, Trussell et al. 2006, DuRant et al. 2008, Hawlena and Schmitz 2010*a*, *b*). These predator-induced changes in prey physiology can determine the rate of nutrient transfer between prey and their environment by indirectly affecting the quantity and nutritional quality of basal resources (e.g., plant communities) as well as prey bodies and waste materials (Hawlena and Schmitz 2010*a*, *b*, Hawlena et al. 2012).

Despite the compelling contextual linkages and mounting evidence that predation plays a key role in regulating ecosystem processes (see Schmitz 2008, Schmitz et al. 2010, Hawlena et al. 2012, Guariento et al. 2018, Schmitz et al. 2018), ecologists have struggled to develop predictive theories linking predator NCEs to ecosystem function. This is largely because of the need to integrate the organismal approach, which focuses on species, functional traits, and macronutrients, with the ecosystem approach, which takes a holistic view—focusing on the flux of elements through biotic and abiotic components of the environment (Hawlena and Schmitz 2010*a*). To link NCEs to ecosystem processes, Hawlena and Schmitz (2010*a*) introduced the general stress paradigm (GSP). This pioneering framework makes use of prey physiological stress responses to generate testable

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predictions for how predator effects can cascade to alter ecosystem dynamics using elements as the common currency. For simplicity reasons the GSP focused solely on stress physiology and excluded behavioral, morphological, and life history defenses.

Physiological stress responses are evolutionarily conserved—leading to similar stress responses across taxa (Wingfield and Ramenofsky 1999). In general, the physiological stress response involves elevated concentrations of stress hormones (e.g., glucocorticoids [hereafter, CORT] and cateholamines) and heat shock proteins, as well as elevated cardiovascular and respiratory function (Hawlena and Schmitz 2010*a*). Prey stressed by predation is expected to reallocate resources from growth and reproduction (processes requiring N-rich proteins) to emergency functions (processes requiring C-rich carbohydrates and fats). The GSP predicts that prey should compensate for this heightened energetic demand by consuming energy-rich resources, using energy storages (e.g., fats and glycogen), and converting noncarbohydrate substrates (e.g., proteins) into glucose (i.e., gluconeogenesis; Hawlena and Schmitz 2010*a*, *b*). Thus, prey stressed by predation should have reduced protein, fat, and glycogen content, as well as increased labile carbohydrate content (see Table 1).

Stress-induced changes in prey macronutrient content can ultimately be converted to changes in prey elemental composition (i.e., C, N, P) using the principals of ecological stoichiometry (Elser et al. 1996, Sterner 1997; see Table 1). Decreased prey protein content under stress, due to gluconeogenesis, and lower consumption of protein-rich resources, should result in concurrent reductions in prey N content—because protein is a N-rich biomolecule. Similarly, reduction of C-rich storage molecules (e.g., fats) may decrease C content, but this reduction may be restrained by increased consumption of C-rich resources and elevated levels of labile carbohydrates. Consequently, the GSP predicts moderate or no change in prey C content under predation risk. These predictions regarding C and N content should lead to a higher prey C:N ratio under predation risk. The GSP does not provide specific predictions for phosphates (Hawlena and Schmitz 2010*a*), but the allocation of resources from growth and reproduction may lead to lower P content and consequently a higher C:P ratio. Thus, by using ecological stoichiometry, the GSP aimed to develop a predictive framework linking predation risk to the elemental composition of prey bodies and waste materials—which has important consequences for ecosystem-level processes (i.e., nutrient cycling).

Since the introduction of the GSP, attempts have been made to test its predictions empirically in a variety of ecosystems. For example, in an old field ecosystem, Hawlena and Schmitz (2010*b*) found that grasshoppers exposed to ambush spider predators increased their metabolism, consumed diets with higher carbohydrate-to-protein ratios, had greater body C:N ratios, and excreted more N. These findings support the overall predictions of the GSP. However, several empirical tests have opposed the predictions of the GSP and have suggested that predicting predator-induced changes in prey stoichiometry may be more complex. For example, Kirschman et al.

Table 1. Hypotheses for prey stoichiometric response to predation risk based on the original general stress paradigm (GSP) and complementary works.

Stoichiometric response	Prediction (body content)	Mechanism	Source
Macronutrients			
Carbohydrates	Increase	Protein gluconeogenesis generates glucose, fat is immobilized, and prey shift dietary preferences to carbohydrate-rich resources	Hawlena and Schmitz (2010*a*, *b*)
Fats	Decrease	Fats used to fuel elevated respiratory and cardiovascular function	Hawlena and Schmitz (2010*a*), Janssens and Stoks (2013)
Proteins	Decrease	Protein gluconeogenesis reduces proteins and prey shift to low-protein diets	Hawlena and Schmitz (2010*a*)
Elements			
Carbon	Not Explicit	Allocation of energy from fat to carbohydrate may reduce C content	Hawlena and Schmitz (2010*a*)
Nitrogen	Decrease	Reduced protein should be reflected in lower N content	Hawlena and Schmitz (2010*a*), Sterner (1997)
Phosphorous†	Decrease	Reduced growth should decrease RNA:DNA ratio and reduce P content	Janssens et al. (2015), Sterner (1997), Elser et al. (1996)
Elemental ratios			
C:N	Increase	Decreased N should increase ratio	Hawlena and Schmitz (2010*a*, *b*), Sterner (1997)
C:P†	Increase	Decreased P should increase ratio	Hawlena and Schmitz (2010*a*), Elser et al. (1996)
N:P†	No Prediction	N and P should both decrease	Hawlena and Schmitz (2010*a*), Sterner (1997), Elser et al. (1996)

† Represents new additions to the original GSP predictions as written in Hawlena and Schmitz (2010*a*).

(2016) found that there was no difference in body C:N, C:P, %C, and %N between wood frog (*Lithobates sylvaticus*) prey in the presence and absence of larval dragonfly (*Anax* spp.) predation risk. In another study, damselfly larvae (*Enallagma cyathigerum*) exposed to chemical cues derived from dragonfly larvae (*Anax* spp.) predators had higher C:N and C:P than damselfly larvae not exposed to predation risk (Janssens et al. 2015). Janssens et al. (2015) attributed these changes in C:N and C:P to a decrease in C-rich biomolecules (i.e., fat and sugar) and no change in N-rich proteins—opposing the underlying mechanisms of the GSP (Hawlena and Schmitz 2010*a*). These recent empirical studies highlight the need to revisit the GSP in order to address key conceptual gaps in the theory and identify additional sources of variation (e.g., predator hunting mode) that may improve the predictive ability of the framework.

Our goal was to use the growing number of published empirical studies to test the predictions of and mechanisms underlying the GSP. Specifically, we used a quantitative meta-analysis to synthesize data from 27 manuscripts assessing how predation risk affects prey stoichiometry. We targeted manuscripts that quantified how predation risk affects the macronutrient composition (e.g., carbohydrates, proteins, or fat content), elemental content (e.g., carbon and nitrogen), and elemental ratios (e.g., C:N) of prey bodies. Additionally, we sought to understand how (1) predator hunting mode, (2) prey diet shifts, and (3) multiple environmental stressors (e.g., toxicants and warming) alter prey macronutrient content and stoichiometry in response to predation risk. First, we expected that prey exposed to ambush predators would induce stronger stoichiometric responses than prey exposed to active predators, as prey exposed to ambush predators typically induce stronger defensive responses (Schmitz et al. 2004, Preisser et al. 2007). Second, prey able to shift their diets under predation risk were expected to have weaker stoichiometric responses than prey unable to shift their diets, as the former can behaviorally compensate for predator-induced nutritional demands (Hawlena et al. 2011). Third, because various stressors should induce similar physiological stress responses (Hawlena and Schmitz 2010*a*), we hypothesized that prey under simultaneous exposure to different stressors should additively or synergistically augment their stoichiometric response. Understanding how predation risk and context-dependent factors (e.g., predator hunting mode) affect prey stoichiometry will strengthen the GSP and, ultimately, our ability to predict how predators should indirectly affect ecosystem function.

Methods

Literature survey

We surveyed the literature using Web of Science and the following search terms, "predat* AND prey stoichiometry OR prey physiological stress OR prey nutritional geometry OR prey general stress paradigm OR prey physiological plasticity." The search was conducted on 10 June 2019. We used the preferred reporting practices outlined by PRISMA to structure our overall literature search (Moher et al. 2009). Our search identified 435 potential manuscripts. For each potential manuscript, we read the abstract and determined if the study measured prey stoichiometric responses to predation risk (including conspecific alarm cues). This screening yielded 51 manuscripts that we read in full to determine if they were eligible for inclusion in our quantitative meta-analysis (see Fig. 1). Studies were deemed eligible if they induced one or more measurements of prey stoichiometry (i.e., macronutrient or elemental composition) in the presence and absence of predation risk. We targeted studies that measured the effects of predation risk on (1) the macronutrient composition, (2) the elemental composition, and (3) the elemental ratios of a prey's body and waste materials. After reading 51 manuscripts in full, we had to exclude 24 manuscripts because they either failed to address our objectives or were missing data (e.g., sample size; Fig. 1). From the 27 included manuscripts, we also extracted data on the effects of predation risk on prey growth, foraging, assimilation efficiency, metabolic rate, RNA:DNA, and CORT concentrations when possible. We extracted these additional metrics because previous meta-analyses on predator effects have generated robust predictions for how predation risk should affect these prey traits (see Preisser et al. 2005, Preisser and Bolnick 2008). Thus, by comparing the findings from our data set to these pre-existing conclusions (based on larger data sets) we hoped to gain insight into how studies in our small data set compare to the larger body of literature on predator-risk effects.

Data collection

Our final search yielded 234 comparisons (i.e., independent experiments) from 27 manuscripts. From these manuscripts, we collected data on the macronutrient content, elemental content, and elemental ratio of prey's body and waste materials in the presence and absence of predation risk (Appendix S1: Table S1). Additionally, we obtained 105 comparisons of prey growth, foraging, assimilation efficiency, metabolic rate, RNA:DNA, or CORT in the presence and absence of predation risk (Appendix S1: Table S1). We extracted data from tables and text or used Web Plot Digitizer to extract data from figures (Rohatgi 2015). For each relevant comparison, we collected data on the sample size, mean, and variance (standard error or standard deviation). We extracted the sample size, minimum, first quartile, median, third quartile, and maximum values of prey stoichiometry from two manuscripts (Costello and Michel 2013, Kirschman et al. 2016). We then estimated the means and standard deviations for these studies using methods defined in Wan et al. (2014).

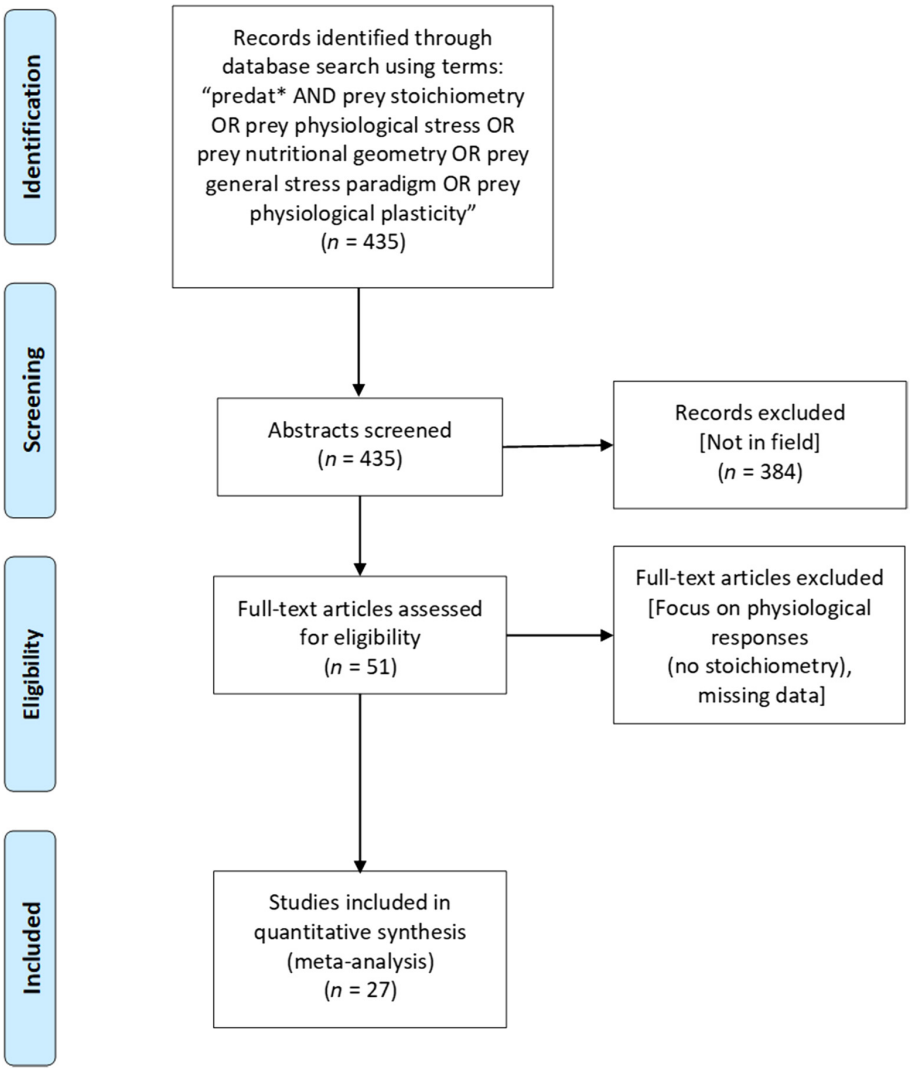

FIG. 1. PRISMA flow chart showing the procedure used to select studies for inclusion in the meta-analysis.

If studies contained multiple relevant independent experiments, we extracted each individual experiment. Where studies measured the macronutrient or elemental composition of multiple prey tissues (see Costello and Michel 2013), we only included data on the composition of the whole organism to prevent nonindependence.

For each extracted comparison, we also recorded (1) if prey were exposed to a second environmental stressor (e.g., food limitation), (2) if prey were able to shift their diets, and (3) the hunting mode of the predator (i.e., ambush [sit-and-wait and sit-and-pursue] or active). We considered prey able to shift their diets if they were provided more than one type of food item during the study. We classified the hunting mode of predators used in each experiment as either active or ambush predators using information presented in the original manuscript, taxonomic databases, and other published literature (Preisser et al. 2007; for classifications see Appendix S2: Table S1).

Meta-analysis of predation-risk effects on prey stoichiometry and physiology

We conducted our meta-analysis using OpenMEE software (Build date: 26 July 2016; Wallace et al. 2017). We used both the Hedges' d (hereafter, d) and the log response ratio (hereafter, LRR) to compare the effects of predation risk (present/absent) on all extracted prey responses (Hedges 1981). We used two measures of effect size (d and LRR) to increase the robustness of our analysis, because d is sensitive to differences in sample standard deviation and LRR can be biased for small samples sizes (Osenberg et al. 1997, Lajeunesse and Forbes 2003). For both effect sizes, a positive effect size

indicates that predation risk increases the response variable, while a negative effect size indicates that predation risk decreases the response variable.

We used separate meta-analyses (random-effect models with a Der Simonian–Laird approach) to determine the overall effect of predation risk on each response variable. To minimize the effects of small sample sizes, we excluded all response variables supported by less than three separate manuscripts and five comparisons. Although the literature suggests that only two separate studies are needed to run a meta-analysis (Valentine et al. 2010), we chose a higher threshold of three separate studies (and five comparisons) because a synthesis of meta-analyses in ecology found that the minimum number of separate studies included in published quantitative syntheses was three (Koricheva and Gurevitch 2014). We used meta-regressions (random-effect models with a restricted maximum-likelihood approach) to assess the impact of predator hunting mode, a second environmental stressor, and prey diet shifts on a prey's stoichiometric response to predation risk for each eligible response variable. We considered response variables eligible for meta-regressions if each subgroup in the analysis (e.g., ambush predators and active predators) was supported by at least three separate manuscripts and five comparisons.

For all meta-analyses and meta-regressions, we tested the heterogeneity of our data set by calculating both Q (total heterogeneity) and I^2 (heterogeneity due to between-comparison variance). We tested for potential publication bias by calculating Kendall's rank correlations (T_b) between effect size and pooled variance within each data set (Begg and Mazumdar 1994). If potential bias was detected (T_b with $P < 0.05$), we used funnel plots to identify outliers for removal visually (Begg and Mazumdar 1994, Palmer 1999). Additionally, we calculated Rosenthal's fail-safe number, N_{fs}, for all significant tests (Rosenthal 1979, Rosenberg 2005). Rosenthal's fail-safe number predicts the number of additional studies with neutral effect sizes (effect size = 0) that would need to be added to the data set to lose significance. We classified fail-safe analyses as robust if they were $>5n + 10$, where n is the number of comparisons for a given response variable (Rosenberg 2005).

Results

Our final data set included comparisons from freshwater (76.1%), terrestrial (20.9%), and marine (3.0%) environments, with a heavy bias towards freshwater ecosystems (Appendix S1: Table S1). Insecta was the most studied prey taxa, comprising 46.9% of included comparisons. Other prey taxa in our data set include Actinopterygii (i.e., ray-finned fish [26.3% of comparisons]), Branchiopods (e.g., *Daphnia* [16.5% of comparisons]), Amphibia (7.1% of comparisons), and Mammalia (3.2% of comparisons; Appendix S1: Table S1).

Impacts of predation risk on stress physiology and growth rate

Of the 27 manuscripts included in our full data set, 22 manuscripts contained 105 total comparisons of the effects of predation risk on broad physiological prey traits (Appendix S1: Table S1). These studies contained data on the effects of predator risk on prey CORT concentrations, foraging, assimilation efficiency, metabolic rate, RNA:DNA, and growth. However, we could not run meta-analyses on assimilation efficiency data because of its limited sample size ($n = 1$ manuscript). We found no evidence of publication bias in our Kendall's rank correlations for all metrics except growth, which showed a significant positive correlation between LRR and pooled sample size ($T_b = 0.44$, $P < 0.001$; Appendix S3: Table S1). Because of this, we consulted the funnel plot for the LRR of growth and removed one outlier from the growth data set for our LRR meta-analysis.

Predation risk increased the CORT and metabolic rate of prey and decreased the RNA:DNA ratio and growth rate of prey (Appendix S4: Fig. S1). Fail-safe calculations for CORT, metabolic rate, and growth rate were robust to publication bias (Appendix S5: Table S1). However, the fail-safe calculation for RNA:DNA ratio was not robust, suggesting that our findings may be susceptible to publication bias (Appendix S5: Table S1). For all models, we observed high total heterogeneity, with >26% of the true heterogeneity being due to between-comparison variation (Appendix S4: Table S1).

Impacts of predation risk on prey macronutrient content

Our search found 19 manuscripts and 123 total comparisons assessing the effects of predation risk on the macronutrient composition of prey bodies (Appendix S1: Table S1). Carbohydrates were the most studied macronutrient, accounting for 51% of all macronutrient studies, and fats and proteins accounted for 31% and 18% of macronutrient studies, respectively.

Predation risk reduced prey's fat content (d: $P = 0.008$; LRR: $P = 0.010$; Appendix S4: Table S1; Fig. 2). This effect is supported by robust fail-safe calculations (Appendix S5: Table S1). We found no strong correlation between effect size and pooled standard deviation for either d or LRR (Appendix S3: Table S1). Both effect-size models contained high total heterogeneity, with ≥72% of the true heterogeneity being due to between-comparison heterogeneity (Appendix S4: Table S1).

The presence of predation risk tended to reduce the carbohydrate content of prey bodies (d: $P = 0.061$; LRR: $P = 0.096$; Fig. 2). Both d and LRR models had high total heterogeneity, with 49 and 58% of the total heterogeneity being attributed to between-comparison variation, respectively (Appendix S4: Table S1). We found no strong correlation between effect size and

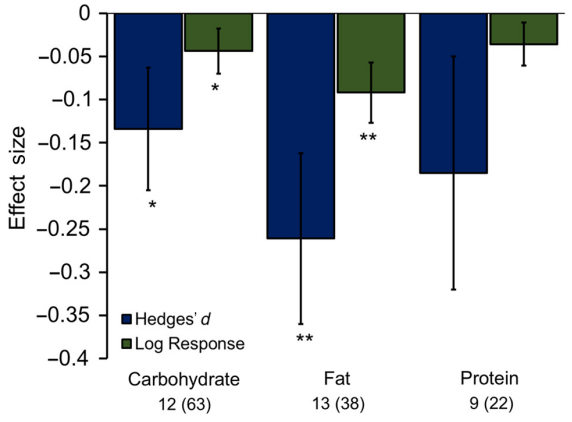

FIG. 2. Mean (±SE) Hedges' *d* and log response ratio (LRR) effect sizes for the effects of predation risk on the fat, carbohydrate, and protein content of prey bodies. Numbers below labels are sample sizes [number of manuscripts (number of comparisons)]. Two asterisks (**) indicates significance at $\alpha \leq 0.05$, a single asterisk (*) indicates significance at $\alpha \leq 0.10$.

pooled standard deviation for either *d* or LRR (Appendix S3: Table S1).

There is a tendency for predation risk to decrease the protein content of prey bodies (*d*: $P = 0.169$; LRR: $P = 0.145$; Appendix S4: Table S1; Fig. 2). Prey-body protein models had high total heterogeneity, with 63–69% of total heterogeneity being attributed to between-comparison variation (Appendix S4: Table S1). We found no evidence of publication bias in our Kendall's rank correlations for proteins (Appendix S3: Table S1).

Impacts of predation risk on prey elemental content

Our search found 14 manuscripts and 68 total comparisons assessing the effects of predation risk on the elemental content of prey bodies, egesta, and excretions (Appendix S1: Table S1). These studies compared (1) the carbon (C), nitrogen (N), phosphorous (P), sodium (Na), chloride (Cl), and potassium (K) content of prey bodies; (2) the C and N content of prey egesta; and (3) the N and P content of prey excretions in the presence and absence of predation risk (Appendix S1: Table S1). However, because of limited sample size, we could not conduct formal meta-analyses on egesta C content ($n = 1$ manuscript), excretion P content ($n = 2$ manuscripts), and body K content ($n = 2$ manuscripts). Additionally, because egesta from mammals (i.e., feces) cannot directly be compared to insect frass, we had to exclude any analysis of predator effects on the N content of prey egesta.

We found no effect of predation risk on the C and N content of prey body tissues (Appendix S4: Table S1; Fig. 3a). There was a slight trend for prey exposed to predation risk to have reduced body P; however, this trend was statistically insignificant (*d*: $P = 0.114$; LRR: $P = 0.153$; Appendix S4: Table S1; Fig. 3a). Prey exposed to predation risk also tended to have increased body Na^+ and Cl^- concentrations, but this trend was only present in the LRR analysis ($P = 0.057$ and 0.067, respectively; Appendix S4: Table S1). Additionally, we found that predation risk had no effect on the N content of prey excretions (Appendix S4: Table S1).

All elemental variables, except body C content, had high total heterogeneity, with between-comparison heterogeneity accounting for 73–99% of the total heterogeneity (Appendix S4: Table S1). We found no evidence of publication bias based on our Kendall's rank correlations (T_b) for any of our elemental composition metrics (Appendix S3: Table S1).

Impacts of predation risk on prey elemental ratios

Our search found nine manuscripts and 43 total comparisons assessing the effects of predation risk on the C:N, C:P, and N:P of prey bodies and the C:N of prey excretions (Appendix S1: Table S1). Prey-body C:N was the most studied response variable, comprising 42% of the included comparisons. Because of a limited sample

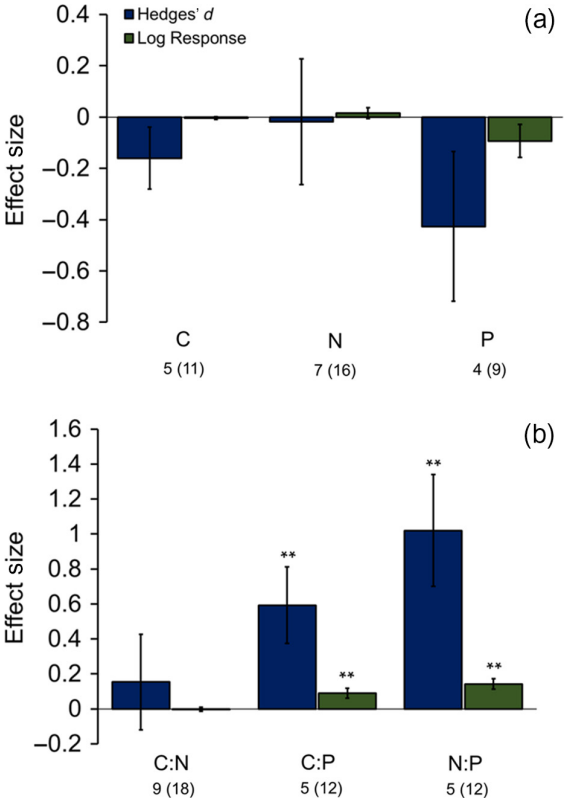

FIG. 3. Mean (±SE) Hedges' *d* and log response ratio (LRR) effect sizes for the impacts of predation risk on (a) the C, N, and P content of prey bodies; (b) C:N, C:P, and N:P ratios of prey bodies. Numbers below labels are sample sizes [number of manuscripts (number of comparisons)]. Two asterisks (**) indicates significance at $\alpha \leq 0.05$, a single asterisk (*) indicates significance at $\alpha \leq 0.10$.

size, we had to exclude analysis of prey excretion C:N (n = 1 manuscript).

Predation risk had no effect on the C:N ratio of prey body tissues (Appendix S4: Table S1; Fig. 3b). However, predation risk increased the C:P and N:P ratios of prey body tissues (C:P, *d*: $P = 0.007$; LRR: $P = 0.002$; N:P, *d*: $P = 0.001$; LRR: $P < 0.001$; Appendix S4: Table S1; Fig. 3b). Fail-safe calculations for prey body C:P and N:P are robust to publication bias (Appendix S5: Table S1).

We observed high total heterogeneity in each of the elemental ratio models, with ≥63% of the true heterogeneity within these models due to between-comparison heterogeneity (Appendix S4: Table S1). Additionally, we found no evidence of publication bias based on our Kendall's rank correlations for any of our elemental ratio metrics (Appendix S3: Table S1).

Impacts of predator hunting mode, prey diet shifts, and multiple stressors

Our data set contained 165 comparisons using active predators (e.g., gray wolves) and 142 comparisons using ambush predators (e.g., blue emperor dragonflies; Appendix S1: Table S1). Because of limited sample sizes, we were only able to test the effects of predator hunting mode on prey N, macronutrient content, and growth.

We found no effect of predator hunting mode on the N content of prey bodies (Appendix S6: Table S1). However, we did find that predator hunting mode affects prey fat content (*d*: $P = 0.021$; LRR: $P < 0.040$) and carbohydrate content (*d*: $P < 0.001$; LRR: $P < 0.001$; Appendix S6: Table S1; Fig. 4a, b). Specifically, we found that ambush predators reduce prey fat (*d*: Estimate = −0.60 ± 0.21 [SE], $P = 0.005$; LRR: estimate = −0.21 ± 0.08 [SE], $P = 0.010$) and carbohydrate content (*d*: Estimate = −0.49 ± 0.10 [SE], $P < 0.001$; LRR: Estimate = −0.19 ± 0.04 [SE], $P < 0.001$), whereas active predators had no effect on prey fat (*d*: Estimate = −0.10 ± 0.11 [SE], $P = 0.339$; LRR: Estimate = −0.04 ± 0.04 [SE], $P = 0.292$) and carbohydrate content (*d*: Estimate = 0.05 ± 0.09 [SE], $P = 0.629$; LRR: Estimate = 0.02 ± 0.04 [SE], $P = 0.553$).

Predator hunting mode tended to affect prey protein content (*d*: $P = 0.075$; LRR: $P = 0.071$) and growth (*d*: $P = 0.420$; LRR: $P = 0.002$; Appendix S6: Table S1; Fig. 4c, d). Ambush predators reduce prey protein content (*d*: Estimate = −0.36 ± 0.18 [SE], $P = 0.045$; LRR: Estimate = −0.06 ± 0.03 (SE), $P = 0.042$), whereas active predators had no effect on prey protein content (*d*: Estimate = 0.13 ± 0.15 [SE], $P = 0.368$; LRR: Estimate = −0.01 ± 0.02 [SE], $P = 0.586$). Ambush and active predators reduced prey growth [Ambush, *d*: Estimate = −1.32 ± 0.35 [SE], $P < 0.001$; LRR:

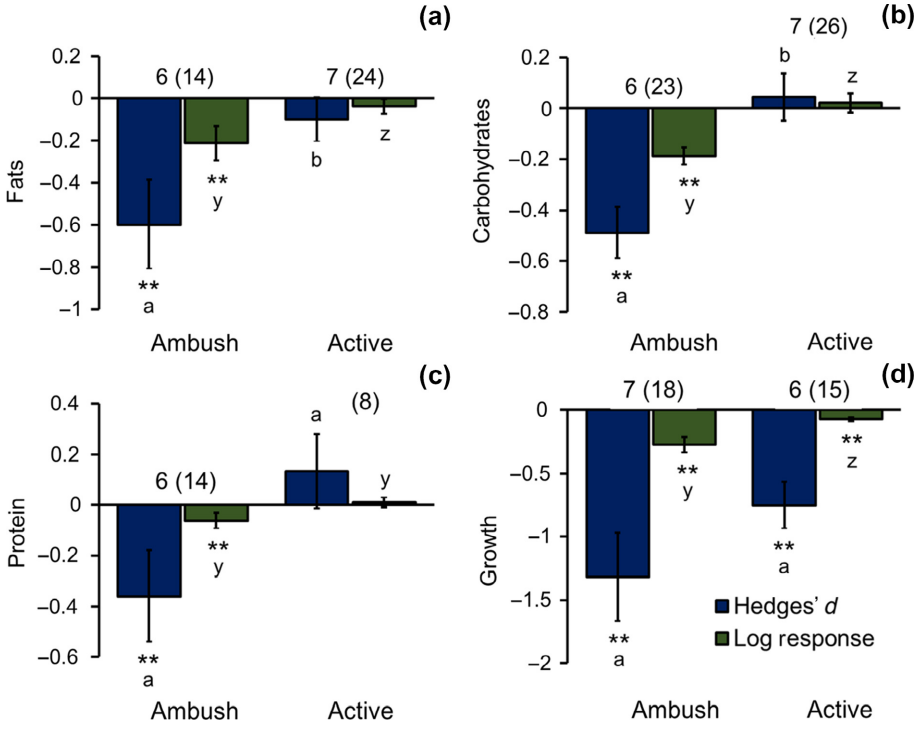

FIG. 4. Mean (±SE) Hedges' *d* and log response ratio (LRR) effect sizes for the effects of predation risk from active and ambush predators on (a) prey fat content, (b) prey carbohydrate content, (c) prey protein content, and (d) prey growth. Numbers next to bars are sample sizes [number of manuscripts (number of comparisons)]. Two asterisks (**) indicates significance at $\alpha \leq 0.05$, a single asterisk (*) indicates significance at $\alpha \leq 0.10$. Different letters indicate significant differences ($\alpha \leq 0.05$) between predator hunting modes by effect size calculation.

Estimate = −0.28 ± 0.06 [SE], $P < 0.001$; Active, *d*: Estimate = −0.75 ± 0.19 [SE], $P < 0.001$; LRR: Estimate = −0.07 ± 0.02 [SE], $P < 0.001$], but prey exposed to ambush predators tended to have lower growth rates than prey exposed to active predators (Appendix S6: Table S1).

Eleven manuscripts within our data set (101 comparisons) exposed prey to predation risk and a second environmental stressor (Appendix S1: Table S1). Warming and food limitation/starvation were the most studied second stressors, representing 29 and 20% of comparisons, respectively. We found no effect of the presence of a second stressor on a prey's stoichiometric response to predation risk (Appendix S7: Table S1).

Most comparisons (78%) in our data set did not allow prey to shift their diets under predation risk (Appendix S1: Table S1). Yet, we had a large enough sample size to test for the effects of prey diet shifts on prey body N, carbohydrates, fats, proteins, and growth. All prey diet shift analyses had very high total heterogeneity, as well as high between-comparison heterogeneity (51–98%), possibly explaining why we found no overall effects of prey diet shifts on prey responses under predation risk (Appendix S8: Table S1).

Discussion

The general stress paradigm (GSP) attempted to use the prey physiological stress response to link predator NCEs and ecosystem processes (e.g., nutrient cycling; Hawlena and Schmitz 2010*a*). The GSP was based on the notion that prey physiological stress responses are highly conserved across taxa and that predator effects can be quantified as changes in prey macronutrient balance. These changes in prey macronutrient content can then be converted to changes in prey elemental content using the principles of ecological stoichiometry—allowing us to link predation risk and ecosystem function using elements as a common currency. Here, we used a meta-analysis of empirical studies to test the predictions of the GSP across ecological systems, with the goal of uncovering additional factors (e.g., predator hunting mode) that may help improve the predictive capabilities of the GSP.

Prey inducing physiological stress responses often exhibit elevated concentrations of stress hormones (e.g., CORT), increased metabolic rates, and decreased growth rates (Wingfield and Ramenofsky 1999, Preisser et al. 2005, DuRant et al. 2008, Preisser and Bolnick 2008, Hawlena and Schmitz 2010*a*, Buchanan et al. 2017). The studies included in our data set support these underlying predictions (see Appendix S4: Table S1). For example, despite large between-comparison variation, we found that prey responds to predation risk by increasing their CORT levels and metabolic rates and decreasing their growth rate (Appendix S4: Fig. S1). However, we did find that the physiological responses quantified by studies depended critically on the taxonomy of the prey (e.g., vertebrate vs. invertebrate) and the ecosystem studied (e.g., marine vs. terrestrial). For instance, all studies in our data set that measured stress-induced biomolecules (e.g., CORT, heat shock proteins) used vertebrate prey, as measuring stress-induced biomolecules in invertebrate prey is a rare practice (but see Adamo 2012). Similarly, studies in marine systems never measured prey metabolic rates or growth rates, whereas at least 4 and 5% of freshwater and terrestrial comparisons, respectively, quantified prey metabolism or growth under predation risk. Because of the methodological variation within our data set, more work needs to be done to verify that these patterns occur across all ecosystems.

The GSP suggests that prey under predation risk should require greater energetic inputs to support their elevated metabolic rates (Hawlena and Schmitz 2010*a*). Prey may meet these energetic demands by increasing their consumption of C-rich foods (i.e., those rich in fats and carbohydrates), mobilizing stored fats and glycogen, or by breaking down noncarbohydrate nutrients (e.g., proteins) into glucose via gluconeogenesis (see Table 1). Thus, prey under predation risk should have lower concentrations of proteins and fats in their bodies than prey under no predation threat (Hawlena and Schmitz 2010*a*). The GSP also suggests that the increased consumption of C-rich foods, mobilization of storage molecules, and protein gluconeogenesis by risk-exposed prey may lead to elevated levels of labile carbohydrates in their bodies (Hawlena and Schmitz 2010*a*). We found that prey exposed to predation risk tended to have lower body fat and protein content than prey not exposed to predation risk—as predicted by the GSP (Fig. 1). However, we found that the carbohydrate content of prey bodies under predation risk tended to decrease (Fig. 1), rather than increase, suggesting that this prediction of the GSP is either incorrect or, due to the short residence time of labile carbohydrates, is dependent on the specific energetic supply and demand of the prey.

All measurements of prey macronutrient content under predation risk exhibited large within- and between-study variation (see Appendix S4: Table S1). For instance, one study in our data set found that the beetle (*Leptinotarsa decemlineata*) increased its fat content and carbohydrate content by 17 and 28%, respectively, when fed only plant leaves under chronic predation risk (Tigreros et al. 2018). However, when these beetles could consume plant leaves and conspecific eggs, their fat content and carbohydrate content decreased by 21 and 4%, respectively, under predation risk (Tigreros et al. 2018). This immense variation within a single manuscript suggests that despite our findings supporting the initial predictions of the GSP (at least for fats and proteins), the GSP fails to account for major sources of variation in prey physiological stress responses such as prey defensive strategy and resource availability.

The theory of ecological stoichiometry suggests that (1) animal C content should be correlated with animal fat, glycogen, and carbohydrate content; (2) animal N content should be tightly associated with animal protein

content (~17% N; Sterner and Elser 2002, Hawlena and Schmitz 2010*a*), and animal P content should be associated with P-rich ribosomal RNA (rRNA) and vertebrate bones. Despite seeing clear declines in prey macronutrient content, we found no effect of predation risk on the body C, N, or C:N of prey bodies (Fig. 3). We also found no change in excretion N content in response to predation risk. This mismatch between prey macronutrient and elemental responses to predation risk may be due to individual studies only measuring a single nutrient type (i.e., macronutrient or elemental). For example, only 7 of 27 included manuscripts quantified both the macronutrient and elemental composition of prey in response to predation risk (see Appendix S1: Table S1). This suggests that the observed mismatch may be due to differences in prey responses in systems where macronutrients vs. elements (or vice versa) were quantified.

Wilder and Jeyasingh (2016) reanalyzed the data provided by Zhang et al. (2016) and found very weak correlations between animal (1) C and carbohydrate content, (2) N and protein content, and (3) lipid : protein and C:N. We observed similar trends in our data set across taxa (Appendix S9: Table S1). These weak correlations may be due to most ecologists focusing on key macromolecules and ignoring other macromolecules that may change in response to predation and shift a preys' elemental balance. For example, most invertebrate exoskeletons are comprised of chitin, which is ~7% N (Liu et al. 2012). Thus, the reduction in prey N associated with predation risk may be due to the reallocation of N to build chitinous structures. This suggests that by not measuring chitin we are likely underestimating prey N content (Dahl and Peckarsky 2002, Rabus et al. 2013). Similarly, glycogen—an important source of energy storage for many animals—may affect the C estimation in prey bodies. Although 31% of the studies in our data set did measure glycogen and glucose separately, most studies only measured glucose or measured the combined concentration of glycogen and glucose (see Stoks et al. 2005). Additionally, there is a clear taxonomic bias in glycogen measurements, as no studies with vertebrate prey measured prey glycogen content. Consistently incorporating additional macromolecules, like glycogen, into the GSP framework, and quantifying these macromolecules in empirical studies, can improve the ability of the GSP to predict prey body composition accurately (see Van Dievel et al. 2016 for further discussion).

Methodological limitations associated with quantifying macromolecules may further exacerbate the mismatch between macronutrients and elements. First, studies vary in the tissues they use for measurements of prey macronutrient content. Most studies in our data set measured macronutrients using a homogenate of the whole prey organism, and others used only a sample of the prey's blood plasma. These methodological differences may impact our interpretation of predation risk effects on macronutrients. For example, when prey are homogenized, the carbohydrate content of prey tends to decrease under predation risk, whereas when prey blood plasma is used, the carbohydrate content of prey increases under predation risk (Appendix S10: Table S1; Appendix S10: Fig. S1). However, these conclusions are confounded by prey taxonomy, because studies using invertebrate prey only used homogenized prey and studies of vertebrate prey only used blood plasma. These taxa-specific inconsistencies in methodology ultimately make generating broad theories difficult, and finding ways to overcome these methodological challenges should be a focus of future work. Second, it is well recognized that common methods used to quantify proteins in animals cannot accurately determine total protein content and instead only offer a rough proxy (Knight and Chambers 2003). Most manuscripts (88%) in our data set used the Bradford method to quantify the total protein content of prey bodies. However, the Bradford method cannot accurately quantify small peptides (<3,000 Da) and free arginine or lysine. The Bradford method also has variable sensitivity to different protein types and can be affected by the presence of detergents and nontarget macromolecules (e.g., lipids and pigments; Kirazov et al. 1993). Further analysis suggests that protein estimations from the Bradford method can also vary in accuracy between species and within species exposed to different experimental conditions (Zaguri et al., *in preparation*). Because the GSP relies on ecological stoichiometry to convert changes in macronutrients to elements, these methodological constraints associated with macronutrient quantification need to be further understood before we can effectively use the GSP to predict how predator NCEs should affect ecosystem dynamics.

We observed high between-comparison variation in prey body C, N, and C:N (Appendix S4: Table S1). This variation suggests that other factors may need to be considered to predict how predators should affect prey elemental stoichiometry accurately. One source of variation could be the additional behavioral, morphological, and life history defenses that prey employ to minimize predation risk, as these defenses may affect prey stoichiometry differently than stress physiology alone. For example, tadpole prey (*Hyla versicolor*) exposed to predatory beetles have slower growth rates, heavier tails, shorter gut tracts, and are less active. These trait changes are accompanied by increased prey body N and C content, but no change in prey C:N (Costello and Michel 2013). Similarly, water fleas (*Daphnia magna*) exposed to size-selective predatory fish mature earlier and produce more offspring—resulting in a higher intrinsic growth rate. The higher investment in growth and fecundity under predation risk leads to *Daphnia* with lower body C:N, as the observed increase in N-rich proteins offset the weak increase in C-rich fats (Zhang et al. 2016). Overall, these studies suggest that a more refined framework—one that considers the suite of defenses a prey induces—is needed to predict accurately how the C, N, and C:N of prey bodies should be affected by predation risk.

Although we found no consistent effects of predation risk on prey C:N, predation risk did increase the C:P and N:P of prey bodies (Fig. 3b) This increase in C:P and N:P may be attributed to reductions in the P content of prey bodies under predation threat (Fig. 3a). However, the two studies that quantified prey P excretion under risk showed that predation exposure either decreased or had no effect on prey P excretion rates (Kirschman et al. 2016, Guariento et al. 2018). In general, our findings support our hypothesis that decreased growth and lower RNA : DNA under predation risk should decrease P and increase the C:P ratio of prey bodies (see Table 2). Overall, our findings suggest that the P content of prey bodies is impacted by predation risk—calling for an expansion of the GSP to integrate the possible effects of predator NCEs on prey P budgets directly.

Our empirical data set also enabled us to explore several factors that may influence how prey physiologically respond to predation risk. Although we found no effect of multiple stressors (e.g., warming) or diet shifts on prey physiological responses, we did find strong effects of predator hunting mode. Predation risk from ambush predators evoked greater reductions in prey growth than risk from active predators (Fig. 4d), in agreement with previous meta-analyses on the effects of predator hunting mode on prey growth (see Preisser et al. 2007). We also found that exposure to cues from ambush predators reduced prey macronutrient content (i.e., carbohydrate, fat, and protein), whereas active predators had no effect on prey macronutrient content (Fig. 3a–c). This effect is likely due to cues from active predators satiating their environment, making it difficult for prey to assess accurately where predators are on the landscape (Schmitz et al. 2004, Preisser et al. 2007). Differences in cue dilution between ambush and active predators may also contribute to the observed pattern. Yet, because most manuscripts (93%) in our analysis standardized predator cues by either caging live predators or manually introducing cues, this is unlikely (Preisser et al. 2007). The impact of predator hunting mode on the magnitude of prey physiological stress responses suggests the need to consider this context-dependent factor when predicting the consequences of predator–prey interactions on ecosystem nutrient dynamics.

Table 2. Comparison of predicted outcomes and findings for all stoichiometric responses of prey body tissues.

Stoichiometric response	Prediction	Meta-analysis Finding	Sample size (no. of studies, no. of comparisons)
Macronutrients			
Carbohydrates	Increase	Decrease	12, 63
Fats	Decrease	Decrease	13, 38
Proteins	Decrease	Slight decrease	9, 22
Elements			
Carbon	No prediction	No change	5, 11
Nitrogen	Decrease	No change	7, 16
Phosphorous	Decrease	No change	4, 9
Elemental ratios			
C: N	Increase	No change	9, 18
C:P	Increase	Increase	5, 12
N:P	No prediction	Increase	5, 12

In summary, the GSP aimed to predict how predation risk affects ecosystem processes (e.g., nutrient cycling) using the following steps: (1) predators cause physiological stress in prey, (2) physiological stress alters the macronutrient balance and resource requirement of prey, (3) changes in macronutrient composition could be translated to changes in elemental content using principles of ecological stoichiometry, and (4) changes in elemental content of prey bodies and waste materials regulate ecosystem processes. Using the accumulated empirical data, we set out to re-evaluate the original predictions of the first three steps of the GSP. Our quantitative meta-analysis found that prey physiological stress responses to predation support the underlying physiological mechanisms of the GSP, and the predictions that stressed prey should have lower fat and protein content (Fig. 1). In contrast to the GSP's predictions, prey under predation risk decreased their body carbohydrate content, possibly reflecting the labile nature of this pool (Fig. 1). We also found that contrary to the GSP there were no changes in prey body C, N, and C:N ratio, and in N content of the prey waste materials. These deviations from the GSP, and the high between-comparison heterogeneity, highlight a need to consider other forms of inducible defenses (e.g., behavioral, morphological, or life history) and the interplay between these responses, as these additional induced defenses may affect a preys' energetic supply and demand in a context-dependent way. Additionally, the inconsistencies between the macromolecule and elemental measurements observed in our data set coincide with recent work identifying major caveats when translating macromolecule content into elemental composition. Moving forward, we urge the field to explore how methodological limitations contribute to this macronutrient–element mismatch and test whether including additional macromolecules in these analyses may help strengthen predicted stoichiometric relationships. Our analysis also uncovered that incorporating predator hunting mode into the GSP may strengthen our predictions regarding how predators may indirectly affect nutrient cycling by altering the quality of nutrients in prey bodies and waste materials.

To date, the GSP has been tested in a limited number of ecosystems and taxa—with a strong bias towards freshwater systems and invertebrate prey (Appendix S1: Table S1). We encourage researchers to expand their investigations of prey physiological stress responses to additional taxa (e.g., Mammalia) and ecosystems (especially marine systems) to allow a more thorough test of the GSP and to help identify additional factors that can modulate predator effects on ecosystem function. Our

work adds to the growing literature highlighting that predation risk can induce physiological stress responses in prey that ultimately alter the macronutrient and elemental stoichiometry of prey bodies and waste materials. The GSP served as a vital step in developing a framework to linked predator NCEs to ecosystem function, but key theoretical and empirical gaps must be filled before we can effectively predict how prey physiological responses to predation risk can affect ecosystem nutrient dynamics.

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Supporting Information

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Data Availability Statement

Data are available on the Dryad Digital Repository: https://doi.org/10.25338/B8D909