Poor condition promotes high-risk behaviours but context-dependency is key: A systematic review and meta-analysis

Short Running Title: Condition effects on risky behaviour

Nicholas P. Moran*[1,2]

Alfredo Sánchez-Tójar[1]

Holger Schielzeth[3]

Klaus Reinhold[1]

1. Evolutionary Biology, Bielefeld University, Bielefeld, Germany

2. Centre for Ocean Life DTU-Aqua, Technical University of Denmark, Lyngby, Denmark

3. Institute of Ecology and Evolution, Friedrich Schiller University Jena, Jena, Germany

Author for Correspondence

Nicholas P. Moran

DTU Aqua, Building 201, Kemitorvet, 2800 Kgs. Lyngby

nicholaspatrickmoran@gmail.com

Telephone: (+45) 31855259

Abstract

Animal behaviour can lead to varying levels of risk, and an individual's physical condition can alter the potential costs and benefits of undertaking risky behaviours. How risk-taking behaviour depends on condition is subject to contrasting hypotheses. The asset protection principle proposes that individuals in better condition should be more risk averse, as they have higher future reproductive potential (i.e. more to lose). Contrastingly, the state-dependent safety hypothesis predicts that high-condition individuals should make riskier choices as they are more likely to survive and maximise the benefits of risky situations. We systematically searched for studies that experimentally manipulated animals' condition (nutritional or energetic), and subsequently measured their risk-taking behaviour. Our meta-analysis quantified condition effects on risk-taking behaviour at both the mean and variance level. We preregistered our methods and hypotheses prior to conducting the study. Phylogenetic multilevel meta-analysis revealed that low-condition individuals showed on average ca. 26% (95% confidence interval: 15% – 38%; n = 126 studies, 1297 effect sizes) greater tendency towards risk than high-condition individuals. Meta-regressions revealed several factors influencing the overall effect, such as the experimental context used to measure risk-taking behaviour, and the life-stage when condition was manipulated. Meta-analysis of variance revealed no clear overall effect of condition on behavioural variance (on average ca. 3% decrease in variance in low- vs high-condition groups; 95% confidence interval: -8% – 3%; n = 119 studies, 1235 effect sizes), however, the experimental context was an important factor influencing the strength and direction of the variance effect. Our comprehensive systematic review and meta-analysis provide insights into the roles of state-dependency and plasticity in intraspecific behavioural variation. While heterogeneity among effect sizes was high, our overall results are consistent with the asset protection principle being relevant in the majority of cases.

Keywords: boldness, exploration, novelty, novel environment, novel object, predation, predator response, animal personality, shoaling, dietary restriction

Contents

I. Introduction

Animals often gamble with their lives, with behavioural decisions frequently involving trade-offs between resource acquisition, reproduction and survival. Many of those decisions have to be made in face of incomplete information or inherent stochasticity in the outcome. Some behaviours are thus inherently 'risky' (defined as involving high outcome variance), and promise large gains, but also the potential of large losses (Barclay, Mishra, & Sparks, 2018). In ecology, the concept of risk is often applied in contexts where the outcome is unpredictable (e.g. responses to novelty, *sensu* boldness; White *et al.*, 2013) or contexts with a high relative likelihood of death (e.g. predator responses; Réale *et al.*, 2007). When to engage in risky behaviours is an important decision in an individual's life, and thus an important research topic in behavioural ecology. State variables, such as individual condition, can modify the costs and benefits of risk taking (Luttbeg & Sih, 2010). State-dependency of behaviour is an important driver of among-individual variation in behavioural traits (Sih *et al.*, 2015; Niemelä & Dingemanse, 2018; Moiron *et al.* 2019), but its specific relationship to risk taking is subject to unresolved competing hypotheses.

Individual condition, defined here as variation in nutritional or energetic state, might affect risk taking in different ways. Animals in high condition might be risk-averse, as these individuals have a lot to lose in terms of future reproductive potential (the 'asset-protection principle'; Clark, 1994; Wolf *et al.*, 2007), whereas individuals in low condition have more to gain in terms of starvation avoidance, improvement of condition, and elevated competitiveness (Luttbeg & Sih, 2010; alternately referred to as the 'needs-based' explanation, Barclay *et al.*, 2018). Contrastingly, the 'state-dependent safety' hypothesis (alternately referred to as the 'ability-based' explanation) predicts that high condition individuals may take greater risks, because they are better able to survive and maximise the benefits of engaging in risky behaviours due to their superior physical and/or cognitive capabilities (Barclay *et al.*, 2018).

Risk taking can depend on the current and/or past condition of an individual, and physical condition in early life may have a disproportionate effect on risk-taking behaviour. For example, individuals may be developmentally primed to engage in risky behaviours when those behaviours were favoured early in life (Zimmer *et al.*, 2017), and poor early-life environments may drive greater risk taking in adults as a way to compensate for their poor start (Krause & Caspers, 2016). Conversely, a favourable developmental environment can result in improved cognitive ability in adulthood (Buchanan, Grindstaff, & Pravosudov, 2013), allowing greater risk taking due to a state-dependent safety effect. The asset-protection and the state-dependent safety hypothesis thus make opposite predictions that might act early and late in life. Theoretical support for the two alternative hypotheses is mixed, and show that the direction of an effect may depend on environmental conditions, such as overall resource availability or acuteness of the risk factor (Luttbeg & Sih, 2010; Engqvist, Cordes, & Reinhold, 2014). Empirical results are similarly mixed, and thus it remains unknown if there are any generally applicable effects of condition on risk-taking behaviour, or the ecological context in which any one hypothesis applies.

Regardless of the hypothesis, condition effects on risk taking are often framed as adaptive responses to variation in an individual's future fitness expectations (as in Clark, 1994; Wolf *et al.*, 2007). The key proposition being that decisions to take risks are related to variation in state, where an individual's state includes all intrinsic and extrinsic factors strategically relevant for their fitness (Wolf & Weissing, 2010). But an individual's state, and therefore their state-dependent behavioural responses may vary due to factors other than their physical condition, such as life-history differences within- or among-species (McNamara & Houston, 1996). For example, differences between male and female reproductive roles can alter their behavioural responses to poor dietary conditions (Han & Dingemanse, 2015). In some cases, males could be more sensitive to condition due to condition-dependent sexual selection, but in other cases, females may be more sensitive to condition since they often bear a disproportionate energetic burden of reproduction (Houslay *et al.*, 2015; English & Uller, 2016). Similarly, interspecific differences in longevity may influence behavioural responses, since long-lived species generally have a larger future reproductive asset and/or more future opportunities to improve their own condition, and thus might be less willing to display risky behaviour (Clark, 1994).

Animal risk-taking behaviour can be measured in different ways reflecting different ecological contexts. Responses to novelty, referred to as boldness-shyness or exploratory behaviour (Réale *et al.*, 2007), involve inherently high outcome variance as the potential benefits and dangers of engaging with novel situations are usually unknown to the individual. Risk taking is also often quantified in assays involving the presence of predators, which emphasize the risk of mortality (Moschilla, Tomkins, & Simmons, 2018). Furthermore, some studies manipulate the outcome variance of foraging-related behaviour directly (Andrews *et al.*, 2018). Studies of risk-taking behaviour across a variety of contexts have shown different responses, for example between predator and novel object experimental setups (Carter *et al.*, 2012), or between emergence into a novel environment and startle responses

(Beckmann & Biro, 2013). As such, we expect condition effects to vary across experimental contexts. For example, state-dependent safety may be more relevant in a predator-response context, such that high-condition individuals are more prone to take risks. Similarly, the effects of starvation avoidance may be more relevant in experimental contexts where food is involved, where low-condition individuals may show increased risk taking.

Thus far, most studies have focused on mean behavioural effects of condition (i.e. higher or lower levels of risk taking). There has, however, been growing interest in individual-level variation in recent years (Westneat, Wright, & Dingemanse, 2015), and new tools to meta-analyze variances alongside means are revealing that meta-variance effects may be both prevalent and often overlooked (Nakagawa *et al.*, 2015). While a recent meta-analysis of variance has shown diet restriction can increase variation in longevity (Senior *et al.*, 2017), another has shown little evidence of environmental stress (including diet restriction) effects on phenotypic behavioural variance (Sánchez-Tójar *et al.*, 2019). Furthermore, case studies have shown increased within-individual behavioural variation in high-condition animals, via an increased capacity to express behavioural plasticity (Royauté & Dochtermann, 2017; Royauté *et al.*, 2019). Conversely, it is conceivable that extremely poor conditions may lead to the expression of cryptic genetic variation, and thus increased variation in state and behaviour among low-condition individuals. However, if a high-risk strategy is the only viable option for acquiring adequate resources in a poor environment, individuals (including low-condition individuals) may converge on a high-risk phenotype (Han & Dingemanse, 2017). Overall, condition-dependent effects on the variance in risky behaviours are likely present, but currently are difficult to predict in direction and magnitude.

We here present a systematic review and meta-analysis of studies that experimentally manipulated individual condition (nutritional or energetic) through dietary treatments, and independently quantified risk-taking behaviour. Specifically we address six questions, which we preregistered previous to the study (see details below):

1. Do condition manipulation treatments have an overall effect on mean risk-taking behaviour? We do not predict a clear non-zero overall effect, but instead expect high heterogeneity among effect sizes resulting from the various contexts in which risk is measured and the multiple mechanisms that may drive condition effects on risk taking.
2. Is the effect of condition on mean risk-taking behaviour context-dependent? We expect low-condition treatment groups to show increased risk-taking behaviour in both foraging and feeding contexts (starvation avoidance effect), but reduced risk-taking behaviour in predator-response contexts (state-dependent safety effect). Across the remaining contexts (e.g. novel environment exploration, novel object response), we predict high-condition treatment groups to show reduced risk-taking behaviour (asset-protection effect).
3. Does condition have differential effects on mean risk-taking behaviour in males and females? We do not predict an overall difference between males and females, due to the high heterogeneity in sex-based ecological differentiation across species. However, sex-specific differences in behaviour are widespread, and thus should be quantified.
4. Does condition at different life stages have differential effects on mean risk-taking behaviour? We expect that early-life treatments will have a greater effect on mean risk-taking behaviour than late-life treatments, as early-life treatments may affect mean risk-taking behaviour through both developmental and state-dependent behavioural plasticity.
5. Does the life-history of a species determine how condition affects risk-taking behaviour? We expect that a species' maximum lifespan, a key life-history measure, will influence the condition effect on risk taking. According to the asset protection principle, longer lived species should be less willing to display risky behaviour (Clark 1994).

6. Does condition affect the amount of total variation in risk-taking behaviour within high- and low-condition treatment groups? We do not predict an overall clear variance effect between high- and low-condition experimental groups, however, as for hypotheses 1 and 2, we predict variance effects to show high heterogeneity and context-dependence.

In addition to the hypotheses above, we conducted the following exploratory (i.e. not preregistered) analyses to test for an effect of: (a) manipulation type, e.g. quantity, quality or starvation treatment; (b) manipulation direction, e.g. restriction, enrichment, or combined; (c) manipulation duration relative to maximum longevity; and (d) whether study subjects were reared in the laboratory or the wild.

II. Methods

(1) Protocol

Study protocols (research questions, a priori hypotheses, search methods and planned analyses) were registered prior to data collection to enhance the objectivity of our analysis and conclusions (see preregistration at https://osf.io/xgrkz/ Moran *et al.*, 2018). Non-preregistered analyses are hereafter labelled as exploratory. This review was conducted following PRISMA reporting guidelines (for PRISMA diagram see Supporting Information S1; Moher *et al.*, 2009).

(2) Systematic review and data collection

Database searches were conducted in *Web of Science* and *Scopus*, with a search query designed to identify studies involving both diet manipulations (e.g. "*nutrition*", "calori*", "bod* condition*") and risk-taking experiments (e.g. "bold*", "risk*", "novel*", "predat*") within animal behaviour and behavioural ecology (e.g. "personalit*", "temperament*", "behavio* type*", "risk taking behavio*"; for full search strategy see Supporting Information S2).

We screened records to find original experimental studies with separate treatment groups subject to manipulated dietary quantity (i.e. partial restriction, complete deprivation or enrichment) or quality (e.g. protein restriction or enrichment) that were then subject to individual behavioural observations in contexts relating to risk (e.g. novel environments, novel object, risk-sensitive foraging, predator response) in independent trials (for inclusion and exclusion decision trees see Supporting Information S1). Our aim was to test for adaptive condition-dependent behavioural responses in non-human animals, therefore we excluded studies using species with compromised genetic diversity and/or evolved adaptive responses (e.g. domesticated animals, laboratory breeds, genetically modified organisms; as per Kelly *et al.*, 2018) as well as studies on humans. Studies manipulating the micronutrient content of diets, or subjecting animals to high fat diets were also excluded as the relationship between these diet manipulations and body condition is not clear and considered beyond the scope of this review. Dietary treatments were considered as 'non-independent' from behavioural measures when (a) the behaviour was measured in the presence of high and low food availability, (b) the dietary treatments (i.e. periods of deprivation) were applied within the novel environment, (c) the dietary treatments were coupled with additional non-dietary factors, or (d) the dietary treatments were applied longitudinally (rather than cross-sectional) to the same individuals. These studies were excluded.

Both the title and abstract screening of 5453 records (post-deduplication), and the full-text screening of 641 published papers were conducted by two authors (NPM 100%, AST 25% at both stages) to ensure reliability. Title and abstract screening was done using Rayyan (Ouzzani *et al.*, 2016), from which 626 references were included for full-text screening. The title and abstract screening resulted in 67/1377 (4.9%) conflicted decisions between observers, confirming high inter-screener agreement. All conflicted decisions were resolved collectively by both screeners. A few additional references that were not captured by our search but instead identified from different sources were also included for full-text screening ('non-systematic' records, n = 15). Data from five such papers were included in the final

231 analysis, therefore we conducted a sensitivity analysis to test the potential effects of these
232 additional five references by re-running the main effects models without these effect sizes
233 (see Supporting Information S3). Full-text screening of 641 papers resulted in 5/160 (3.1%)
234 conflicted decisions (i.e. where one screener included a reference, and the other excluded
235 it), that were resolved collectively by both screeners. Full-text screening identified 147
236 studies meeting the experimental design criteria for inclusion (see https://osf.io/3tphj/ for full-
237 text screening decision database 'CD_FulltextScreeningDatabase.xlsx', and Supporting
238 Information S1 for the PRISMA diagram and the decision tree summarizing the full-text
239 exclusion reasons).
240
241 Data were extracted as comparisons between the *low-condition* groups (i.e. the treatment
242 group for diet restriction treatments, the control group for diet enrichment treatments) and
243 the *high-condition* groups (i.e. the control group for diet restriction treatments, and the
244 treatment group for diet enrichment treatments). Extractions were conducted by NPM with
245 data extracted from figures where necessary using the R package 'metaDigitise' v1.0.0
246 (Pick, Nakagawa, & Noble, 2019). Data required to calculate effect sizes were (a) group
247 means and (b) estimates of uncertainty (standard error, confidence intervals) or variability
248 (standard deviation) in combination with sample sizes (N) for the behavioural variables of
249 interest. Full or partial extraction of relevant data was possible from the published material of
250 118 studies (80.2% of all studies included after full-text screening). To recover missing or
251 partially reported data, corresponding authors of 72 studies were contacted via a
252 standardized author correspondence email, such that 395 (29.6%) of 1334 effect sizes in the
253 full final dataset were obtained via author correspondence. Data from 25% of included
254 papers (37 papers) were re-extracted by an independent observer to ensure data reliability.
255 Of 1420 re-extracted values, errors requiring correction were identified in only 6 values
256 (0.4%) affecting only two effect sizes included in the final analyses.
257
258 *(3) Effect size calculation*

We analysed mean effects using the log response ratio of group means ('lnRR'; Hedges, Gurevitch, & Curtis, 1999), instead of Cohen's D or Hedge's g, as lnRR is less sensitive to heteroscedasticity. Variance effects were analyzed using the log coefficient of variation ratio ('lnCVR'), as this effect size, unlike log ratio of variances ('lnVR'), is less sensitive to potential mean-variance correlations (Nakagawa *et al.*, 2015). Both ratios were calculated using *low condition* over *high condition*, such that a positive effect size represents higher risk taking or larger variance in risk taking in low-condition animals, respectively (effect sizes calculated via R package 'metafor' version v2.1-0, Viechtbauer, 2010). To maintain consistent directionality, effect sizes were reversed for a subset of lnRR effect sizes where lower values reflected higher risk behaviours (e.g. 'latency to emerge from a shelter', 'distance from a predator' etc.). Since lnCVR directionality is independent of the mean, sign reversals were not required. To assess if our choice of effect sizes affected our conclusions, main effects analyses were also run using alternate effect sizes for mean (standardised mean difference with heteroscedasticity correction 'SMDH'; Bonett, 2009), and variance (lnVR; Nakagawa *et al.*, 2015). Conclusions remained robust (see Supporting Information S4 for details).

(4) Data analysis - main effects models

Two multilevel intercept-only meta-analytic models were run for each effect size, testing for a general effect of condition treatments on risk-taking behaviour at a mean and variance level (using the function 'rma.mv' from the R package 'metafor' v2.1-0, Viechtbauer, 2010). Phylogenetic and non-phylogenetic models were run to investigate whether non-independence due to the degree of relatedness between species influenced both the overall effects and their level of uncertainty. Phylogenetic relatedness were estimated based on existing phylogenies and taxonomic information from the Open Tree of Life, and any polytomies were resolved by randomization (Hinchliff *et al.*, 2015; via R package 'rotl' v3.0.7; Michonneau, Brown, & Winter, 2016; for the final phylogenetic tree see Supporting Information S5). Branch lengths were estimated using Grafen's method (Grafen, 1989; via R

package ‘ape’ v5.3; Paradis & Schliep, 2019), and were used to construct a phylogenetic variance-covariance relatedness matrix.

In addition to phylogeny, we included other random effects in our models to account for non-independence due to the use of the same species across studies (SpeciesID), multiple effect sizes taken from the same study (StudyID), and multiple effect sizes taken from the same experimental group of animals within the same behavioural experiment (ExperimentalID). A unit level random effect (EffectID) was also included as a measure of residual heterogeneity. For a subset of effect sizes, an experimental group was compared to multiple treatment groups (i.e. shared-control non-independence). Sampling variances were modeled as variance-covariance matrices that accounted for correlated sampling variances due to the shared group designs, and were constructed following Lajeunesse (2011; for estimation methods see Supporting Information S4).

A subset of studies used a crossed factorial experimental design by applying an additional treatment factor (e.g. diet x temperature treatments; juvenile x adult dietary treatments etc.). To avoid including variance associated with the additional treatment factor in our analysis, we combined groups across the treatment factor that was not of interest to us (e.g. low condition/low temperature and low condition/high temperature). Groups were combined by calculating marginalised means and SDs (following equations for pooled means and SDs from Pick *et al.*, 2019).

For main effects models, we investigated total, residual and random effect specific heterogeneity (i.e. variance among effect sizes) by calculating ‘I^2’ values (Nakagawa & Santos, 2012, via R package v0.0.0.9000 ‘MetaAidR’, Noble, 2019). For main effects models we also estimated absolute heterogeneity ‘Q’, and for moderator models the estimated percentage of heterogeneity explained by the moderators ‘$R^2_{marginal}$’, the residual heterogeneity ‘Q_E’, and moderator specific heterogeneity ‘Q_M’ (via R package ‘metafor’ v2.1-

0, Viechtbauer, 2010). Where applicable, estimates are presented with 95% confidence intervals in square brackets (hereafter simply refer to as 'confidence interval').

(5) Data analysis - hypothesis testing models

All hypotheses were tested using phylogenetic multilevel meta-regression models for both lnRR and lnCVR including random effects as above (for detailed descriptions of all moderators used for hypothesis testing models see Supporting Information S6). To test if effects were context-dependent, we included a categorical moderator, *risk context*, which classified behavioural variables by both the functional context of the experiment (e.g. assays involving predators or predator cues, novel objects, novel environments etc.; Luttbeg & Sih, 2010), and the specific behavioural measurements (e.g. activity levels, areas explored, willingness to feed and forage, shoaling tendencies etc.; for descriptions of all categories see Supporting Information S6). To test for *sex* effects, we calculated effect sizes for males and females separately when possible. Where insufficient data was available to separate sexes, effect sizes were categorised as mixed (i.e. groups including both sexes), or unknown (i.e. no information about the sex of study subjects). To test for an effect of *life-stage* at the time of the treatments, the level of maturity during diet manipulations was categorised as juvenile, adult, or both (i.e. for treatments spanning both periods). If the paper did not present sufficient information to determine the subject's life-stage, this was inferred from the available information (e.g. age, average length, weight etc.). If life-stage could not be reasonably inferred or if groups may have included both juvenile and adult individuals, these were classed together as mixed/unknown. Since treatments in juveniles may have been imposed a longer time before behavioural testing (e.g. early-life diet treatments with adult behavioural testing) relative to adult diet treatments, *Life-stage* models also included the time between treatments and behavioural experiments relative to maximum longevity as a continuous moderator. To assess the role of life-history variation among species, we separately tested for effects of *maximum lifespan* and *ln*(*maximum lifespan*) as continuous moderators. The logarithmic transformation was used because estimates were heavily

biased towards short lifespans. Lifespan estimates were obtained from online databases (AnAge, *genomics.senescence.info*; FishBase, *fishbase.se*, Animal Diversity Web, *animaldiversity.org*; Longevity Records, *demogr.mpg.de/longevityrecords*). If no estimates were available, *ad hoc* searches for lifespan estimates from primary literature were conducted via *Google Scholar*. Where available, sex-specific and wild/captive-specific longevity estimates were used. Continuous moderators were z-transformed to aid interpretation (Schielzeth, 2010).

(6) Data analysis - publication bias tests

Several meta-regression models were used to assess our lnRR dataset for evidence of publication bias (for all included moderators and descriptions see Supporting Information S6). First, the *precision* of each effect was included as a moderator, calculated as the root of the inverse sampling variance (a variant of an Egger's regression based on Nakagawa & Santos, 2012), to test for small-study bias. Next, time-lag bias was tested using the *year of publication* (z-transformed) as a continuous moderator, where a commonly observed trend is a decrease in effect size over time (Jennions & Møler, 2002; Sánchez-Tójar *et al.*, 2018). For both the precision and time-lag models, a limited dataset excluding effect sizes obtained through author correspondence was used so that we were specifically testing for effects of publication bias in published material. Finally, using the full dataset, we tested whether effect sizes were larger in studies with partial or incomplete reporting of results using the categorical moderator: *effect sizes from publication* (i.e. complete, partial or none; where none refers to studies where all effect sizes had to be obtained via author correspondence). In addition, funnel plots were produced using lnRR and precision for a visual assessment of funnel asymmetry (Nakagawa & Santos, 2012; for plots see Supporting Information S7). As there appeared to be some evidence of publication bias, we also calculated fail-safe N to test the robustness of our results (function 'fsn', R package 'metafor' v2.1-0, Viechtbauer, 2010; see Supporting Information S7). Publication bias tests were not conducted for lnCVR, as the overwhelming majority of papers were focused on effects at the mean behavioural

level, with very few testing for effects on behavioural variance, so we did not expect publication bias on lnCVR.

(7) Data analysis - exploratory models

Additional exploratory analyses (i.e. not preregistered) were included to test if differences in the experimental designs of included studies influenced the results (for moderators and descriptions see Supporting Information S6). As we included effect sizes from studies using differing types of diet manipulation, we included the moderator *manipulation type*. This included quantity (where the amount of food ration/food access differed between groups), starvation (where one group was entirely deprived of food for an extended period), quality (where the nutritional content of food differed between groups) or combined (where both quality and quantity was manipulated in the same treatment group). Since our main models compared low- versus high-condition treatment groups regardless of whether diets corresponded to restriction or supplementation treatments, we also explored potential effects of this by including a moderator *manipulation direction*. This moderator categorised treatments as restriction (where low-condition groups were restricted relative to high condition/control groups), supplementation (where high condition groups were enriched relative to low-condition/control groups), and dual (where both the low-condition group was restricted and the high condition group was enriched from standard conditions). To further explore the effects of treatment designs, *relative manipulation duration* was tested as a continuous moderator, and defined as the time that the treatment was applied as a proportion of the maximum lifespan of the species. Finally, an effect of the source of the study subjects was tested using *rearing environment* as a categorical moderator (wild, laboratory, commercial or mixed).

III. Results

(1) Main effects models

Intercept-only models showed a significant positive effect for lnRR, with the mean estimate corresponding to a 26% increase in risk-taking behaviour in low-condition animals compared to high-condition animals (non-phylogenetic method: lnRR = 0.23 [0.14 – 0.32], phylogenetic method: lnRR = 0.23 [0.09 – 0.38]; Table 1, Figure 1). For lnCVR, the overall estimate was small, negative and the confidence intervals overlapped zero substantially (lnCVR = -0.03 [-0.09 – 0.03]; Table 1, Figure 1). As phylogeny failed to resolve any heterogeneity in lnCVR, the estimates from the phylogenetic and non-phylogenetic models were identical.

(2) Hypothesis testing models

The magnitude of the lnRR was influenced by the experimental context, with the *risk context* moderator explaining a large amount of heterogeneity among effect sizes ($R^2_{marginal}$ = 12.03%; Table 2). Although most *risk context*-specific confidence intervals overlapped with zero, all the mean estimates were positive (Table 4). The highest estimates were found for behaviours relating to feeding under predation (lnRR = 0.75 [0.53 – 0.97]), feeding in a novel environment (lnRR = 0.36 [0.20 – 0.52]), and shoaling in a novel environment (lnRR = 0.36 [0.06 – 0.67]; Table 4; Fig 2A). *Risk context* also explained a large amount of heterogeneity in lnCVR ($R^2_{marginal}$ = 10.22%; Table 3), and the confidence intervals of some context-specific effects did not overlap with zero, including refuge use in a novel environment (lnCVR = 0.18 [0.04 – 0.31]), feeding in a novel environment (lnCVR = -0.16 [-0.25 – -0.07]), and, dispersal/migration decisions (lnCVR = -0.49 [-0.86 – -0.11]; Table 5; Fig 2B), showing a reduction in total variance in low- vs. high-condition treatments in those specific risk contexts. *Sex* appeared to have some effect on lnRR (Table 2), but there was no evidence for an effect on lnCVR (Table 3). The lnRR estimates were positive but the confidence intervals slightly overlapped with zero for both females (lnRR = 0.15 [-0.03 – 0.33]) and males (lnRR =0.12 [-0.06 – 0.30]), while effects were strongest for mixed (lnRR = 0.34 [0.06 – 0.61]) and unknown sex groups (lnRR = 0.29 [0.14 – 0.44]; Fig 2C). *Life-stage* also influenced lnRR (Table 2), and less clearly also lnCVR (although this model showed a particularly high $R^2_{marginal}$ = 16.64, Table 3). *Life-stage* specific estimates for lnRR were

lowest and overlapping zero in adult treatments (lnRR =0.12 [-0.06 – 0.30]), and strongest for treatments that spanned both the juvenile and the adult life stage (lnRR = 0.45 [0.17 – 0.73]; Table 4; Fig 2E). *Life-stage* effects on lnCVR showed a negative estimate for juvenile treatments (lnCVR = -0.08 [-0.16 – 0.00]), and a positive effect, i.e. an increase in behavioural variance in low-condition treatments, when treatments spanned both the juvenile and the adult life stage (lnCVR = 0.18 [0.01 – 0.34]; Table 5; Fig 2F). Raw *maximum lifespan* did not appear to influence lnRR (0.00 [-0.08 – 0.09]). However, *ln*(*maximum lifespan*) showed a positive lnRR effect with its confidence intervals only slightly overlapping with zero (0.15 [-0.01 – 0.30]; Table 2, 4), although this moderator did not appear to explain any heterogeneity ($R^2_{marginal}$ = 0.00%; Table 2). Neither *maximum lifespan nor ln*(*maximum lifespan*) appeared to have a clear effect on lnCVR, however, these moderators explained a substantial amount of heterogeneity ($R^2_{marginal}$ = 13.81%, 13.14% respectively; Table 3, 5).

(3) Publication bias tests

Funnel plots showed some potential evidence of asymmetry (for plots and fail-safe N calculations see Supporting Information S7). The estimated effect of *precision* on lnRR was negative and the confidence intervals slightly overlapped with zero (-0.002 [-0.005 – 0.000]; Table 2, 4), while $R^2_{marginal}$ was comparably high (7.81%; Table 2), showing some potential evidence of small-study bias. There was also possible evidence of time-lag bias in published data, with effect sizes appearing to trend slightly downwards over time but the confidence intervals overlapped with zero (-0.05 [-0.14 – 0.05]; Table 2, 4), while $R^2_{marginal}$ was again relatively high (8.18%; Table 2). Last, effects calculated from papers where effect sizes could be partially (lnRR = 0.26 [0.07 – 0.63]) or completely (lnRR = 0.24 [0.09 – 0.40]) calculated from the publicly available material were relatively large (Fig 3), whereas the effect from papers where effect sizes could only be obtained through author correspondence were small and the confidence intervals overlapped with zero (lnRR = 0.10 [-0.16 – 0.35]), however, $R^2_{marginal}$ was zero for this moderator (Table 2). This difference suggests that non-

reported results might be biased towards inconclusive (likely statistically non-significant) results.

(4) Exploratory models

There was limited evidence that diet either *manipulation type* or *manipulation direction* influenced lnRR with all diet types and directional treatments, respectively, showing positive mean estimates, and no heterogeneity explained by either of those moderators ($R^2_{marginal}$ = 0.00; Table 2, 4; Fig 4A, 4C). *Relative manipulation duration*'s effect on lnRR was almost zero too (Table 2, 4). There a small amount of heterogeneity explained by the *rearing environment* of the experimental subjects ($R^2_{marginal}$ = 1.44%; Table 2, 4), with effect sizes from laboratory reared animals being the smallest (lnRR = 0.13 [-0.03 – 0.30]), and effect sizes from wild reared animals being the largest (lnRR = 0.32 [0.16 – 0.48]; Fig 4E).

Both *manipulation type* and *manipulation direction* did not appear to influence lnCVR substantially, whereas *relative manipulation duration* had a small positive effect on behavioural variance (0.05 [0.00 – 0.10]), and explained a substantial amount of heterogeneity ($R^2_{marginal}$ = 16.17%; Table 3, 5; Fig 4B, 4D). There was limited evidence that *rearing environment* influenced lnCVR, with less than 1% of heterogeneity explained by this moderator (Table 3, 5; Fig 4F).

IV. Discussion

Despite our ambiguous expectations based on available contradictory hypotheses, we found a convincing directional effect on mean risk-taking behaviour, where individuals in lower condition are more likely to undertake risk-taking behaviours than individuals in high condition. This condition-dependency may be caused by increased risk aversion in higher-condition individuals due to their greater reproductive expectations (an interpretation most consistent with the asset-protection principle), or by increased risk preference in low-

condition animals due to their elevated danger of starvation (a starvation avoidance mechanism; Luttbeg & Sih, 2010). These adaptive interpretations contrast with a recent meta-analysis showing that riskier behavioural types had higher survival in the wild (Moiron, Laskowski, & Niemelä, 2020), which may highlight a distinction between behavioural variation due to personality trait differences and due to state-dependent effects. Nonetheless, our result is consistent with the idea of a trade-off between the potential benefits of high outcome-variance behaviours (e.g. accessing resources) and the potential costs (e.g. predation or starvation), which animals balance based on their current or past state (Clark, 1994; McNamara & Houston, 1996).

Although our overall effect was relatively strong, there was high heterogeneity in lnRR effect sizes with a large proportion (>20%) related to among-species differences. Variation among species, however, was only minimally related to their shared ancestry, with phylogeny only accounting for a very small proportion of heterogeneity (3%). It would be interesting to know if condition-dependence of risk-taking behaviour also applies to our own species (Wilson *et al.*, 1994; Gosling, 2008), but the large amount of context-specificity might suggest that the effect might vary between contexts. The high heterogeneity among effect sizes is also evident from the wide prediction intervals estimated, and the substantial heterogeneity among studies and experiments. Since theory predicts that state-dependent effects on risk taking vary in strength and direction with factors such as life history traits (Clark, 1994; McNamara & Houston, 1996) and/or local environmental/ecological conditions (Luttbeg & Sih, 2010), such a pattern of variation among species, studies and experiments was to be expected. Critically, given the extent of heterogeneity, our overall positive effect does not preclude the opposite pattern (e.g. a state dependent safety effect) being applicable in certain systems.

Risk context was the most explanatory of lnRR moderators, revealing that the effect of condition in certain contexts was clear and particularly strong, such as those involving

feeding. This is consistent with studies showing that the choice of experiment used to measure risk taking is important to the outcome, and that different risk-taking behaviours can show divergent patterns of individual-level variation (e.g. Carter *et al.*, 2012). The concept of a 'risky' behaviour can be applied to a broad range of circumstances, as shown by the range of behavioural variables included here, and 'risk-taking' can refer to a suite of potentially independent behaviours. A risk context that was particularly strongly affected was shoaling behaviour in a novel environment (and, with less certainty, shoaling when exposed to a predator). Whether decisions to venture from a group can be considered a risk-taking behaviour or boldness trait has been disputed, partly due to overlap with sociability traits (Toms, Echevarria, & Jouandot, 2010), but our findings are consistent with these decisions being related to risk taking as a trade-off between resource acquisition and group safety. Contrastingly, the estimated effect was highly uncertain and close to zero for refuge emergence into a novel environment, a commonly used variable to measure bold-exploratory personalities. Studies have shown refuge emergence to be unrelated to within-species variation in other risk-taking behaviours (e.g. startle responses in *Pomacentrus spp*., Beckmann & Biro, 2013; or novel object tests in *Chlamydogobius eremius*, Moran *et al.*, 2016), such that the relationship between refuge emergence and risk taking remains unclear.

Sex effects on lnRR did not show evidence of male-female differences, with both male- and female-specific effects being relatively small and similar to each other. It has been suggested that different reproductive roles may lead to sex-specific responses to diet variation (Han & Dingemanse, 2015), but there does not appear to be a generalizable direction to this effect. *Life-stage* did show evidence that treatments in juvenile stages had strong and positive effects, while effects in adults were less clear. The *life-stage* and *sex* results may be interrelated in a way that was not originally anticipated, as the strong effect in unknown sex groups may be related to an overrepresentation of juveniles in that category.

Whereas studies where sex was identifiable may have been more likely to involve adult treatments groups, with both sex-specific and adult-specific estimates being smaller.

Our exploratory analyses revealed a few key patterns in condition-dependent behavioural responses, and the suitability of our methodology. Modelling studies have suggested there may be non-linearity in state-dependent phenotypic responses in risk-taking behaviour, due to potential factors such as inconstant correlations between condition and reproductive value (Clark, 1994; McNamara & Houston, 1996; Luttbeg & Sih, 2010). While not directly testing this, evidence of a non-linear effect of condition and risk taking was detected in the analysis of *manipulation direction*. Effects were similar for each group (i.e. reduced vs. standard condition; standard vs. enriched condition, reduced vs. enriched condition), supporting a more constant directional effect of condition on mean risk taking, and suggesting that our methodology of pooling these designs together for analysis was sound. Similarly, the mean effect estimate was positive across all classes of diet treatment analysed (e.g. quality, quantity etc.), such that pooling these experiments was unlikely to influence results. Finally, wild-reared animals did show the largest effect of treatment on mean risk taking (and also a particularly strong negative effect on behavioural variation), suggesting that these animals might be more sensitive to imposed dietary manipulations.

Contrasting with overall mean effects, support for an overall effect of condition on behavioural variation was limited, with only a small, slightly negative and rather uncertain overall lnCVR estimate. This contrasts with the expectation that poor condition may increase phenotypic variability (e.g. by exposing cryptic genetic variation), but agrees with a recent meta-analysis showing that environmental stress does not seem to influence variation in behavioural traits across species (Sánchez-Tójar *et al.*, 2019). Heterogeneity was generally lower in lnCVR models relative to lnRR ones, which is likely because variance effect sizes are generally associated with larger sampling variances (Sánchez-Tójar *et al.*, 2019).

562 Variance meta-analyses are expected to be more data hungry, although this is unlikely to be
563 the cause of the overall weak lnCVR effect found in our study given the large dataset used.
564
565 Variation in behaviour was sensitive to *Risk context*, with variation in both the strength and
566 direction of context-specific effects. In particular, variance in feeding behaviour within novel
567 environments was far lower in low-condition groups, providing some evidence that being
568 highly motivated to feed in this context is an optimum phenotype for individuals in poor
569 energetic state. In contrast, variation in refuge use in a novel environment was higher in low-
570 condition groups, which may be evidence of the opposite (complementary) pattern where
571 high refuge use is a preferred strategy for high condition individuals. *Life-stage* effects on
572 behavioural variation are consistent with recent empirical evidence suggesting that
573 developmental diet is related to phenotypic plasticity and personality development (see
574 examples in Royauté & Dochtermann 2017; Kelleher *et al.* 2019). Buchanan, Grindstaff, &
575 Pravosudov (2013) suggested that poor condition during early life stages may reduce an
576 individual's capacity to express behavioural plasticity. This is potentially consistent with our
577 finding of reduced behavioural variation in groups subject to low-condition treatments as
578 juveniles, while the effect in adults heavily overlapped with zero. We also found that
579 treatments that spanned juvenile and adult life stages (often longer term, chronic diet
580 restriction treatments) had a positive effect on behavioural variation. Similarly, the duration of
581 diet treatments had a positive effect on behavioural variation, consistent with the proposition
582 that extremely poor diet conditions can expose cryptic genetic and phenotypic variation (Han
583 & Dingemanse, 2017). Nonetheless, identifying mechanisms from unpartitioned phenotypic
584 variance remains challenging, as the proposed mechanisms for effects on variability in risk-
585 taking behaviour often apply specifically to among- or within-individual levels (Han &
586 Dingemanse, 2015).
587
588 A pertinent question in behavioural ecology is whether phenotypic variation is primarily within
589 or among individuals (Westneat, Wright, & Dingemanse, 2015). Any effects on the variance

as estimated in our meta-analysis (and more generally in most meta-analysis using lnCVR) may arise from either source. Individuals might become more variable in their behaviour in response to some treatment (or some environmental effect) as a form of behavioural bet-hedging or reduce accuracy of performance (i.e. within-individual level). Alternatively, individuals might differ in their average responses to changes in conditions if they have intrinsically different reaction norms (i.e. among-individual level). Only repeated measurements per individual would help to separate the two variance components. However, this type of data is usually not available in the literature (Niemelä and Dingemanse 2018). Future studies should focus on the relative importance of within- vs. among-individual variance in the variance effects identified in our study.

Considered together, our publication bias analyses suggest there may be some limited influence on the overall results. Time-lag analysis showed that effect sizes might be decreasing over time, while precision analysis showed a small negative effect, both of which can be signs of publication bias toward a positive effect (Jennions & Møler, 2002; Jennions *et al.*, 2013). Moreover, effect sizes obtained from author correspondence where no data could be extracted from published material showed the lowest and most uncertain effect, suggesting preferential publication of positive effects. Intriguingly, publication bias appears to be present even where there are competing hypotheses, with positive effect hypotheses (e.g. the asset protection principle) potentially seemingly preferred. We avoided methods to compensate for bias (e.g. trim and fill) as these can perform poorly in high heterogeneity datasets (Moreno *et al.*, 2009). Instead, we advise caution when interpreting our results, and ecological meta-analyses in general, given the ubiquity publication bias effects in the literature.

V. Conclusions

(I) Evidence of diet and thus condition (or state) effects on risk-taking behaviour in the literature seems clear, as low-condition individuals appear willing to take greater risks across a range of contexts relating to predators and novelty.

(II) While condition-dependency appears to have broad relevance across the animal kingdom, the strength and certainty of this effect may be somewhat overstated due to publication bias and large heterogeneity among effect sizes.

(III) Furthermore, the effect is strongly context-dependent, at both the mean and the variance level, suggesting that the specific ecological (and experimental) factors of any context must be considered when studying risk-taking behaviour.

(IV) Overall, there appears to be complex and nuanced effects of diet and condition on behavioural variance warranting further empirical study. Future research should focus on separating among- and within-individual variance effects of individual condition.

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VII. Authorship

NPM: Conceptualization, Investigation, Methodology, Data curation, Formal analysis, Project administration, Software, Validation, Visualization, Writing - original draft, Writing - review & editing. AST: Conceptualization, Investigation, Methodology, Data collection, Software,

Validation, Writing - review & editing. HS: Conceptualization, Funding acquisition, Writing - review & editing. KR: Conceptualization, Funding acquisition, Supervision, Writing - review & editing.

VIII. Data Accessibility

All data and code used (including data processing, preparation, analysis and presentation) are available at the Open Science Framework (https://osf.io/3tphj/, doi: 10.17605/OSF.IO/3TPHJ).

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789

Tables

Table 1: Main effects models estimates, with random effect specific heterogeneity estimates (I^2) expressed as percentages, and *Q*-test for absolute heterogeneity among effect sizes (*Q*). Square brackets represent 95% confidence intervals. Round brackets represent 95% prediction intervals, i.e. the range in which 95% of future or unknown effects are likely to fall. Positive lnRR and lnCVR effects represent higher either risk taking or variance in risk taking in low-condition animals, respectively.

Effect size	*k*	Mean effect	$I^2_{Experiment\ ID}$ (%)	$I^2_{Study\ ID}$ (%)	$I^2_{Species\ ID}$ (%)	$I^2_{Phylogeny}$ (%)	$I^2_{Effect\ ID}$ (%)	I^2_{Total} (%)	*Q*
lnRR (non-phylo)	1297	0.23 [0.14, 0.32] (-0.90, 1.36)	20.3 [17.1 - 23.5]	7.9 [6.1 - 9.8]	23.2 [18.6 - 28.3]	-	45.9 [42.1 - 49.8]	98.0 [97.8 - 98.1]	25864.30 p < 0.0001
lnRR (phylo)	1297	0.23 [0.09, 0.38] (-0.91, 1.37)	19.9 [17.0 - 23.0]	7.9 [6.0 - 9.8]	21.7 [17.1- 26.7]	3.4 [2.5 - 4.4]	45.3 [41.7 - 49.2]	98.0 [97.9 - 98.2]	25864.30 p < 0.0001
lnCVR (non-phylo)	1235	-0.03 [-0.09,0.03] (-0.78, 0.72)	11.6 [9.8 - 13.5]	21.6 [17.5 - 26.1]	0.0 [0.0 - 0.0]	-	28.0 [25.9 - 30.2]	61.2 [58.8 - 63.6]	2543.32 p < 0.0001
lnCVR (phylo)	1235	-0.03 [-0.09,0.03] (-0.78, 0.72)	11.5 [9.7 - 13.5]	21.6 [17.3 - 26.0]	0.0 [0.0 - 0.0]	0.0 [0.0 - 0.0]	28.1 [25.9 - 30.2]	61.1 [58.8 - 63.6]	2543.32 p < 0.0001

Table 2: Hypothesis testing, publication bias and exploratory moderators for lnRR, with *Q*-test for residual heterogeneity (Q_E), moderator explained heterogeneity (Q_M), and the estimated percentage of heterogeneity explained by the moderators ($R^2_{marginal}$). Note, where $R^2_{marginal}$ estimates were negative, the value was set to zero. Numbers preceding hypotheses refer to the a priori hypotheses as laid out in the introduction.

Hypothesis (model)	Effect size	*k*	Moderator(s)	Q_E (residual)	Q_M (moderator)	$R^2_{marginal}$ (%)
Hyp. 2. Context-dependency of risk (*rr.Full.h2*)	lnRR	1297	*RiskContext*	14657.13 p < 0.0001	79.42 *** p < 0.0001	12.03
Hyp. 3. Sex difference in risk taking (*rr.Full.h3*)	lnRR	1297	*Sex*	24006.28 p < 0.0001	15.92 ** p = 0.0031	0.53
Hyp. 4. Effects across life stages (*rr.Full.h4*)	lnRR	1214	*ManipLifeStage* + *RelativeTimeFromTreatment.C*	16753.8 p < 0.0001	21.2 *** p = 0.0007	0.00
Hyp. 5(i). Life-history effects (*rr.Full.h5.i*)	lnRR	1214	*MaxLongevity.C*	23933.71 p < 0.0001	0.00 p = 0.9651	0.00
Hyp. 5(ii). Life-history effects (*rr.Full.h5.ii*)	lnRR	1214	*lnMaxLongevity.C*	22654.52 p < 0.0001	3.46 p = 0.0628	0.00
Publication bias 1 (*rr.Full.pub1*)	lnRR	908	*Precision*	13245.28 p < 0.0001	2.81 p = 0.0938	7.81
Publication bias 2 (*rr.Full.pub2*)	lnRR	908	*Year.C*	21211.43 p < 0.0001	0.97 p = 0.3254	8.18
Publication bias 3 (*rr.Full.pub1*)	lnRR	1297	*EffectSizesFromPublication*	23269.07 p < 0.0001	11.43 * p = 0.0096	0.00
Exp a. Effect of manipulation type (*rr.Full.exp.a*)	lnRR	1297	*ManipType*	22616.48 p < 0.0001	8.24 p = 0.0833	0.00
Exp b. Effect of manipulation direction (*rr.Full.exp.b*)	lnRR	1297	*ManipDirection*	20399.67 p < 0.0001	10.26 * p = 0.0165	0.00
Exp c. Effect of manipulation duration (*rr.Full.exp.c*)	lnRR	1214	*RelativeManipDuration.C*	24024.39 p < 0.0001	0.06 p = 0.8007	0.00
Exp d. Effect of rearing environment (*rr.Full.exp.d*)	lnRR	1297	*WildLabRear*	22799.97 p < 0.0001	16.57 ** p = 0.0023	1.44

Table 3: Hypothesis testing, publication bias and exploratory moderators for lnCVR, with *Q*-test for residual heterogeneity (Q_E), moderator explained heterogeneity (Q_M), and the estimated percentage of heterogeneity explained by the moderators ($R^2_{marginal}$). Note, where $R^2_{marginal}$ estimates were negative, the value was set to zero.

Hypothesis (model)	Effect size	*k*	Moderator(s)	Q_E (residual)	Q_M (moderator)	$R^2_{marginal}$ (%)
Hyp. 2. Context-dependency of risk (*cvr.Full.h2*)	lnCVR	1235	*RiskContext*	2450.98 p < 0.0001	38.4 *** p = 0.0002	10.22
Hyp. 3. Sex difference in risk taking (*cvr.Full.h3*)	lnCVR	1235	*Sex*	2520.5 p < 0.0001	5.9 p = 0.2066	2.44
Hyp. 4. Effects across life stages (*cvr.Full.h4*)	lnCVR	1153	*ManipLifeStage* + *RelativeTimeFromTreatment.C*	2158.2 p < 0.0001	9.5 p = 0.0908	16.64
Hyp. 5(1). Life-history effects (*cvr.Full.h5.i*)	lnCVR	1153	*MaxLongevity.C,*	2185.53 p < 0.0001	1.41 p = 0.2348	13.81
Hyp. 5(ii). Life-history effects (*cvr.Full.h5.ii*)	lnCVR	1153	*lnMaxLongevity.C*	2187.91 p < 0.0001	0.34 p = 0.5615	13.14
Exp a. Effect of manipulation type (*cvr.Full.exp.a*)	lnCVR	1235	*ManipType*	2535.9 p < 0.0001	3.1 p = 0.5406	0.00
Exp b. Effect of manipulation direction (*cvr.Full.exp.b*)	lnCVR	1235	*ManipDirection*	2541.4 p < 0.0001	2.23 p = 0.5256	0.00
Exp c. Effect of manipulation duration (*cvr.Full.exp.c*)	lnCVR	1153	*RelativeManipDuration.C*	2182.57 p < 0.0001	4.59 * p = 0.0322	16.17
Exp d. Effect of rearing environment (*cvr.Full.exp.d*)	lnCVR	1235	*WildLabRear*	2514.93 p < 0.0001	4.6 p = 0.3312	0.86

Table 4: Parameter estimates for lnRR hypothesis testing, publication bias, and exploratory models, with 95% confidence intervals. *k* shows the number of effect sizes, and n_{study} shows the number of studies. Bold estimates correspond to confidence intervals that do not overlap zero. Note that models with categorical moderators were run as no-intercept models for ease of interpretation.

Hypothesis (model)	Moderator(s)	Level	*k*	n_{study}	Estimate
Hyp. 2. Context-dependency of risk (*rr.Full.h2*)	*RiskContext*	*novelenvironment_activity*	248	46	0.09 [-0.06, 0.25]
		novelenvironment_exploration	153	33	0.11 [-0.05, 0.28]
		novelenvironment_feeding	331	37	**0.36 [0.20, 0.52]**
		novelenvironment_lightdarktest	26	6	0.20 [-0.11, 0.52]
		novelenvironment_refugeemergence	39	7	0.03 [-0.23, 0.30]
		novelenvironment_refugeuse	75	16	**0.22 [0.03, 0.42]**
		novelenvironment_shoaling	29	5	**0.36 [0.06, 0.67]**
		novelobject_response	92	11	0.18 [-0.04, 0.41]
		predation_feeding	81	14	**0.75 [0.53, 0.97]**
		predation_response	172	34	**0.19 [0.02, 0.36]**
		predation_shoaling	20	4	0.28 [-0.04, 0.61]
		dispersalmigration	15	6	0.03 [-0.38, 0.45]
		other	16	5	0.23 [-0.16, 0.61]
Hyp. 3. Sex difference in risk taking (*rr.Full.h3*)	*Sex*	*female*	421	39	0.15 [-0.03, 0.33]
		male	291	37	0.12 [-0.06, 0.30]
		mixed	120	14	**0.34 [0.06, 0.61]**
		unknown	465	61	**0.29 [0.13, 0.44]**
Hyp. 4. Effects across life stages (*rr.Full.h4*)	*ManipLifeStage*	*adult*	423	48	0.12 [-0.06, 0.30]
		both	179	8	**0.45 [0.17, 0.73]**
		juvenile	601	66	**0.30 [0.14, 0.46]**
		unknown/mixed	94	11	**0.40 [0.11, 0.69]**
	RelativeTimeFromTreatment.C	*(covariate)*	-	-	0.01 [-0.03, 0.06]
Hyp. 5(i). Life-history effects (*rr.Full.h5.i*)	*MaxLongevity.C*	*intercept*	-	-	**0.26 [0.15, 0.36]**
		(covariate)	-	-	0.00 [-0.08, 0.09]
Hyp. 5(ii). Life-history effects (*rr.Full.h5.ii*)	*lnMaxLongevity.C*	*intercept*	-	-	**0.22 [0.02, 0.43]**
		(covariate)	-	-	0.15 [-0.01, 0.30]
Publication bias 1 (*rr.Full.pub1*)	*Precision*	*intercept*	-	-	**0.28 [0.08, 0.49]**
		(covariate)	-	-	0.00 [-0.01, 0.00]
Publication bias 2 (*rr.Full.pub2*)	*Year.C*	*intercept*	-	-	**0.26 [0.07, 0.44]**
		(covariate)	-	-	-0.05 [-0.14, 0.05]
Publication bias 3 (*rr.Full.pub1*)	*EffectSizesFromPublication*	*no*	130	13	0.10 [-0.16, 0.35]
		partial	360	31	**0.26 [0.07, 0.45]**
		yes	807	82	**0.24 [0.09, 0.40]**
Exp a. Effect of manipulation type (*rr.Full.exp.a*)	*ManipType*	*combined*	24	4	0.27 [-0.08, 0.62]
		quality	248	18	**0.35 [0.07, 0.63]**
		quantity	390	50	**0.30 [0.07, 0.53]**
		starvation	635	59	0.19 [-0.04, 0.41]
Exp b. Effect of manipulation direction (*rr.Full.exp.b*)	*ManipDirection*	*dual*	60	7	0.30 [-0.06, 0.66]
		restrict	1170	112	**0.23 [0.09, 0.38]**
		supplement	67	9	0.20 [-0.04, 0.44]
Exp c. Effect of manipulation duration (*rr.Full.exp.c*)	*RelativeManipDuration.C*	*intercept*	-	-	**0.25 [0.16, 0.35]**
		(covariate)	-	-	-0.01 [-0.07, 0.05]
Exp d. Effect of rearing environment (*rr.Full.exp.d*)	*WildLabRear*	*commercial*	139	12	0.25 [-0.02, 0.52]
		lab	711	58	0.13 [-0.03, 0.3]
		mixed	15	1	0.21 [-0.5, 0.93]
		wild	432	57	**0.32 [0.16, 0.48]**

847 **Table 5:** Parameter estimates for lnCVR hypothesis testing, and exploratory models, with
848 95% confidence intervals. *k* shows the number of effect sizes, and n_{study} shows the number
849 of studies. Bold estimates correspond to confidence intervals that do not overlap zero. Note
850 that models with categorical moderators were run as no-intercept models for ease of
851 interpretation.

Hypothesis (model)	Moderator(s)	Level	*k*	n_{study}	Estimate
Hyp. 2. Context-dependency of risk (*cvr.Full.h2*)	*RiskContext*	*novelenvironment_activity*	248	46	0.02 [-0.06, 0.11]
		novelenvironment_exploration	153	33	-0.05 [-0.15, 0.05]
		novelenvironment_feeding	312	34	**-0.16 [-0.25, -0.07]**
		novelenvironment_lightdarktest	24	5	-0.09 [-0.35, 0.16]
		novelenvironment_refugeemergence	39	7	0.04 [-0.18, 0.25]
		novelenvironment_refugeuse	75	16	**0.18 [0.04, 0.31]**
		novelenvironment_shoaling	29	5	0.01 [-0.25, 0.26]
		novelobject_response	88	10	-0.08 [-0.24, 0.08]
		predation_feeding	61	13	-0.01 [-0.21, 0.18]
		predation_response	167	33	0.02 [-0.08, 0.13]
		predation_shoaling	20	4	0.01 [-0.24, 0.26]
		dispersalmigration	13	5	**-0.49 [-0.86, -0.11]**
		other	6	3	**0.59 [0.16, 1.02]**
Hyp. 3. Sex difference in risk taking (*cvr.Full.h3*)	*Sex*	*female*	401	38	0.05 [-0.05, 0.15]
		male	276	37	0.03 [-0.08, 0.14]
		mixed	117	13	-0.09 [-0.28, 0.09]
		unknown	441	56	-0.08 [-0.17, 0.00]
Hyp. 4. Effects across life stages (*cvr.Full.h4*)	*ManipLifeStage*	*adult*	402	45	0.00 [-0.10, 0.09]
		both	116	7	**0.18 [0.01, 0.34]**
		juvenile	578	63	**-0.08 [-0.16, 0.00]**
		unknown/mixed	89	11	-0.02 [-0.21, 0.16]
	RelativeTimeFromTreatment.C	*(covariate)*	-	-	0.02 [-0.02, 0.05]
Hyp. 5(i). Life-history effects (*cvr.Full.h5.i*)	*MaxLongevity.C*	*intercept*	-	-	-0.03 [-0.09, 0.03]
		(covariate)	-	-	-0.03 [-0.08, 0.02]
Hyp. 5(ii). Life-history effects (*cvr.Full.h5.ii*)	*lnMaxLongevity.C*	*intercept*	-	-	-0.03 [-0.09, 0.03]
		(covariate)	-	-	-0.02 [-0.09, 0.05]
Exp a. Effect of manipulation type (*cvr.Full.exp.a*)	*ManipType*	*combined*	24	4	0.07 [-0.21, 0.35]
		quality	246	18	0.05 [-0.09, 0.18]
		quantity	363	48	-0.07 [-0.16, 0.03]
		starvation	602	54	-0.04 [-0.12, 0.05]
Exp b. Effect of manipulation direction (*cvr.Full.exp.b*)	*ManipDirection*	*dual*	60	7	0.11 [-0.14, 0.35]
		restrict	1116	106	-0.04 [-0.10, 0.03]
		supplement	59	8	-0.06 [-0.27, 0.14]
Exp c. Effect of manipulation duration (*cvr.Full.exp.c*)	*RelativeManipDuration.C*	*intercept*	-	-	-0.03 [-0.08, 0.03]
		(covariate)	-	-	**0.05 [0.00, 0.10]**
Exp d. Effect of rearing environment (*cvr.Full.exp.d*)	*WildLabRear*	*commercial*	127	11	-0.02 [-0.21, 0.17]
		lab	679	54	0.02 [-0.06, 0.11]
		mixed	15	1	0.10 [-0.41, 0.62]
		wild	414	55	**-0.09 [-0.18, 0.00]**

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Figure Legends

Fig. 1 Higher mean risk taking in low-condition compare to high-condition animals, but similar behavioural variation between them. Phylogenetic (black circles) and non-phylogenetic (white circles) meta-analytic means for lnRR and lnCVR with 95% confidence intervals. The number of effect sizes used in each model is *k*.

Fig. 2 Category-specific estimates for lnRR and lnCVR meta-regression models testing the effect of (A, B) the experimental context for risk-taking behaviour; (C,D) sex of study subjects; and (E,F) life-stage of study subjects during the diet manipulation treatments. lnRR effects are presented on the left (A, C, D) and lnCVR on the right (B, D, F). The areas of the blue shaded circles are proportional to the number of effect sizes *k* used, and bars represent 95% confidence intervals. A positive effect shows higher risk taking or higher variance in risk taking in low-condition animals, respectively.

Fig. 3 Category-specific estimates based on the degree that lnRR effect sizes could be extracted from published material. Fully reported effect sizes are from papers where all effect sizes could be extracted from published material, partially reported effect sizes are from papers where some effect sizes could be extracted but additional effect sizes could be obtained from authors (therefore includes effect sizes from published material and author correspondence), and not reported effect sizes are those that could only be calculated from data obtained through author correspondence. The areas of the green shaded circles are proportional to the number of effect sizes *k* used, and bars represent 95% confidence intervals. A positive effect shows higher risk taking and higher variance in risk taking in low-condition animals.

Fig 4 Category-specific estimates for lnRR and lnCVR meta-regression models for effect of (A, B) the type of diet manipulation; (C, D) the direction of the diet manipulation; and (E, F)

881 the rearing environment of the experimental subjects. lnRR effects are presented on the left
882 (A, C, D) frames and lnCVR on the right (B, D, F). The areas of the orange shaded circles
883 are proportional to the number of effect sizes *k* used, and bars represent 95% confidence
884 intervals. A positive effect shows higher risk taking and higher variance in risk taking in low-
885 condition animals, respectively.
886

887 **Fig. 1**

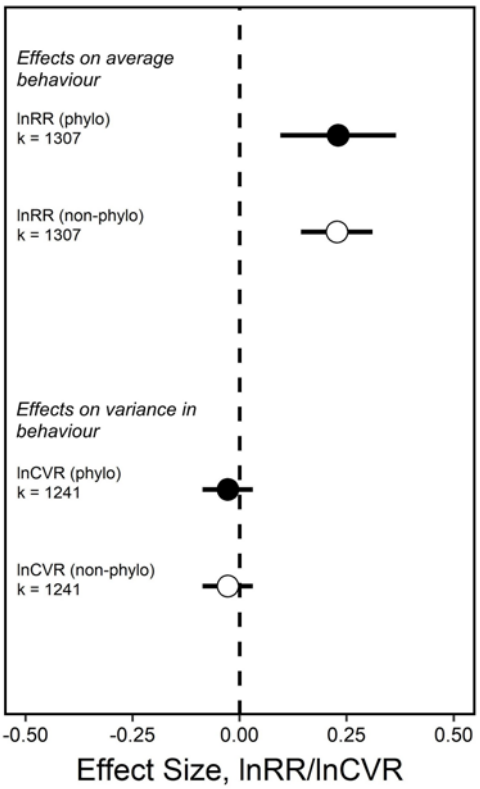

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Fig 2

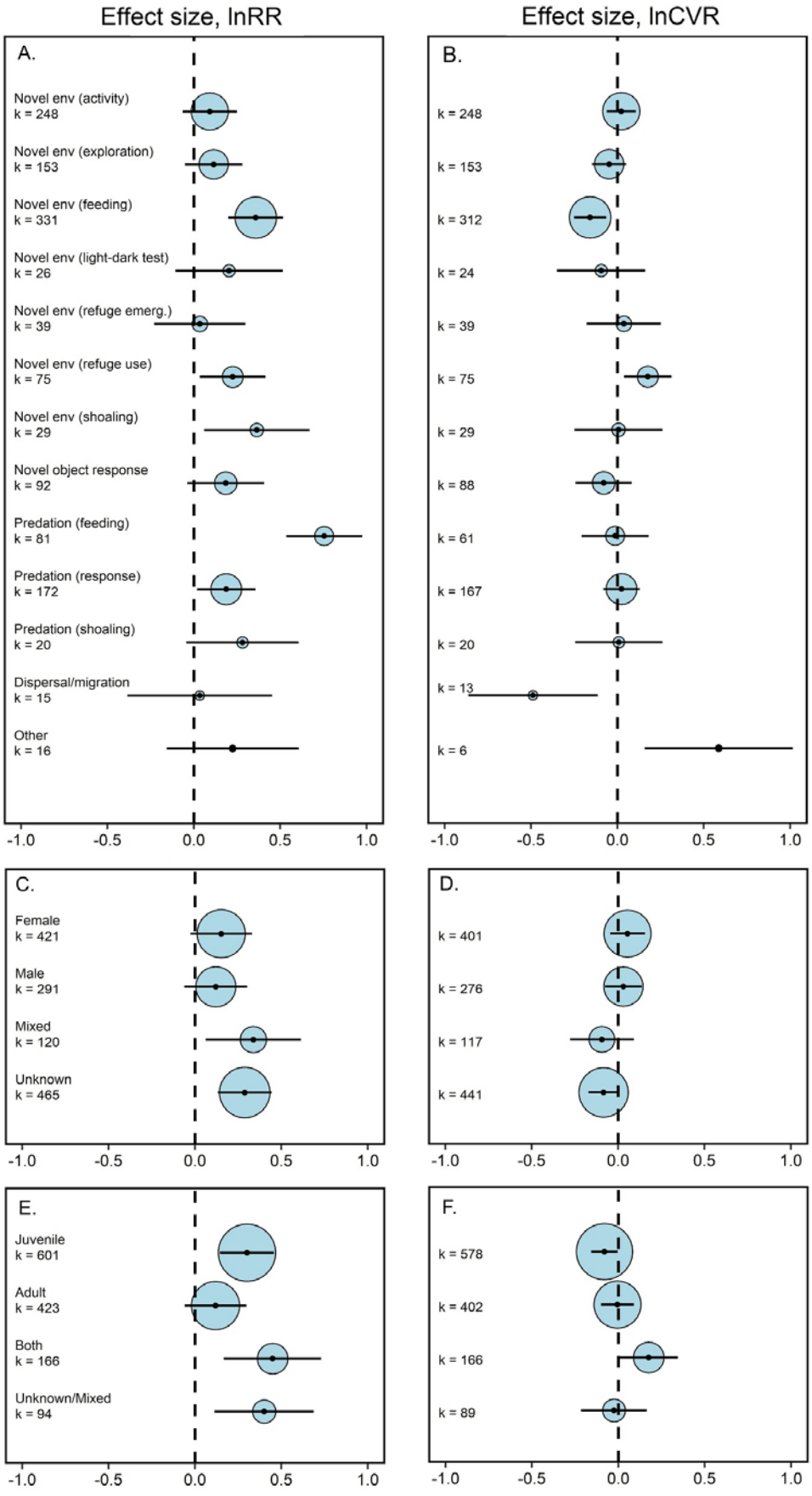

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Fig. 3

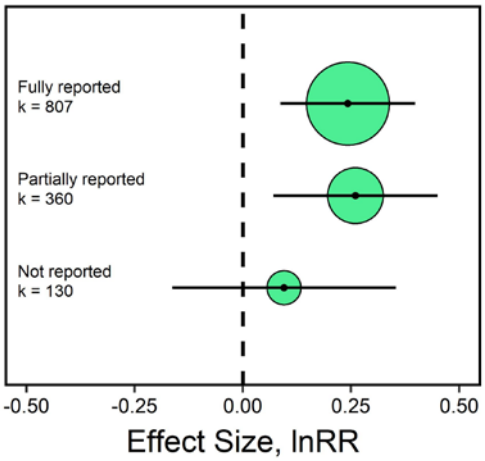
Fully reported
k = 807
Partially reported
k = 360
Not reported
k = 130
-0.50
-0.25
0.00
0.25
0.50
Effect Size, lnRR

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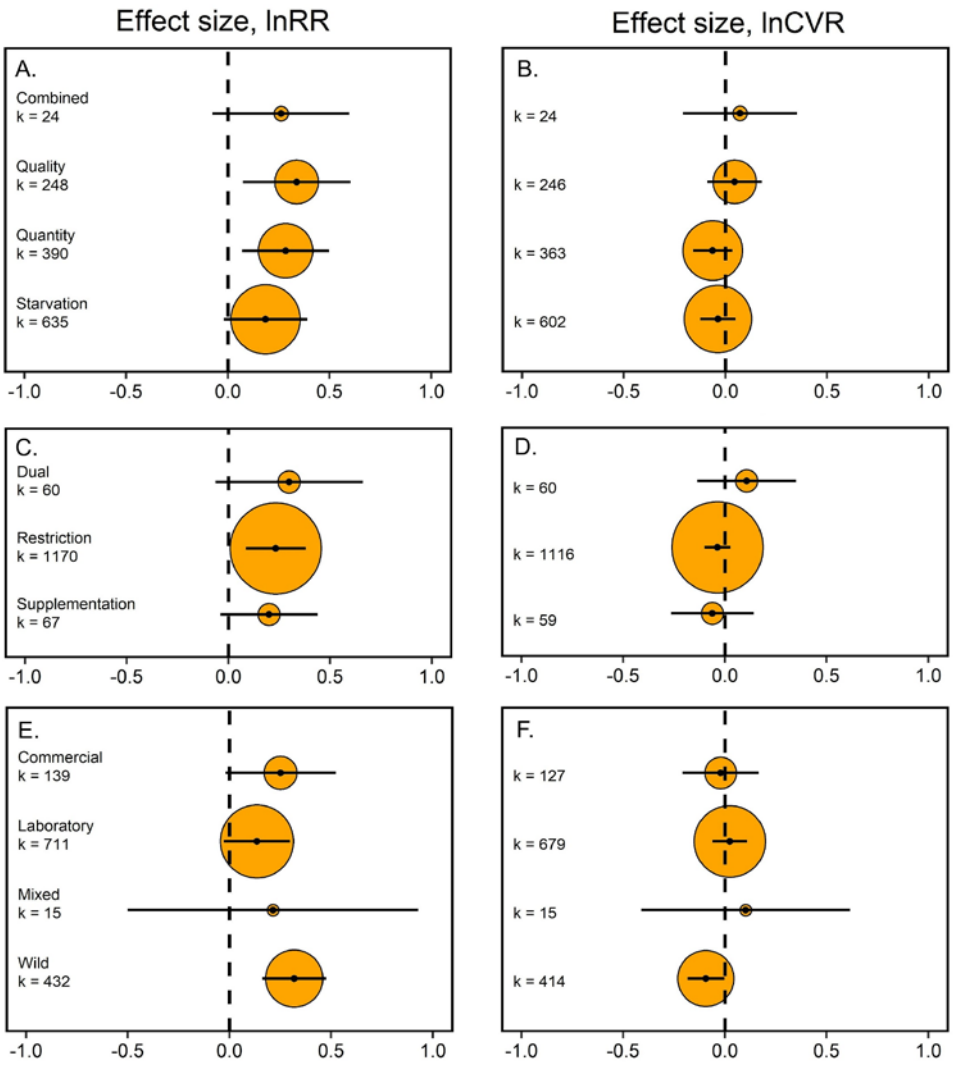
Fig 4.



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