# Poor nutritional condition promotes high-risk behaviours: A systematic review and meta-analysis

**Short Running Title:** Condition effects on risky behaviour

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# 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). The state-dependent safety hypothesis proposes that high-condition individuals that are more likely to survive and maximise the benefits of risky situations may make apparently riskier choices, as their individual risk is in fact lower. We systematically searched for studies that experimentally manipulated animals' nutritional or energetic condition through diet treatments, and subsequently measured risk-taking behaviour in contexts relating to predation, novelty, exploration. 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 the lower nutritional condition individuals showed on average ca. 26% greater tendency towards risk than high-condition individuals (95% confidence interval: 15% – 38%; n = 126 studies, 1297 effect sizes). 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 results show that poor nutritional state on average increases risk-taking in ecological contexts involving predation, novelty and exploration.

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

# Contents



# I. Introduction

Animals often must 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). The concept of risk may be applied broadly in animal ecology (e.g. participation in aggressive contests, reproductive investment decisions etc.), and is often used 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, considered here as variation in nutritional or energetic state, can lead to differences in morphological, behavioural and cognitive traits among individuals (Borcherding & Magnhagen, 2008; Buchanan, Grindstaff, & Pravosudov, 2013; Han & Dingemanse, 2015), which can subsequently 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'; Ludwig & Rowe, 1990; Clark, 1994), whereas individuals in low condition have more to gain in terms of improved condition, elevated competitiveness, and starvation avoidance, particularly when an individual is relatively close to their starvation threshold (Dall & Johnstone, 2002; Luttbeg & Sih, 2010; also known as the 'needs-based' explanation, Barclay *et al.*, 2018). Contrastingly, the 'state-dependent safety' hypothesis (also known as the 'ability-based' explanation) predicts that individuals may appear to take greater risks where they are better able to survive and maximise the benefits of engaging in risky behaviours, as they individually experience a lower level of risk (Barclay *et al.*, 2018). State-dependent safety might apply if improved condition allows greater investment in physical and/or cognitive capabilities (e.g. increased vigour and/or ability to evade or defend against predation) that reduce the level of risk for the individual (as in Temple, 1987).

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 nutritional environment during

development in particular can increase investment in traits that improve future survival and fitness, such as cognitive ability (Buchanan *et al*., 2013). This might allow greater risk taking if those traits provide an advantage in certain risky contexts by altering effective risk levels, if for example high-condition individuals are better protected/less vulnerable than low-condition individuals in the same situation. Theoretical support for any one directional state-effect on risk-taking is mixed, and show that the outcome 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). State-dependent responses due to nutritional condition may have interactive effects with other state variables, such as life history-differences within- or among-species (McNamara & Houston, 1996). For example, sex is a form of state variation involving differences in reproductive roles, which may alter male and female 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).

A subset of ecological contexts where variation in risk-taking behaviour can apply are those involving trade-offs between resource acquisition and (implied or direct) predation risk, which are often used in connection with the concept of 'boldness'. For example, responses to novelty involve inherently high outcome variance, as the potential benefits and dangers of novel situations are unknown to the individual. Furthermore, greater activity or exploration increases the likelihood of both finding new resources or habitat patches, and encountering predators (Réale *et al.*, 2007, Wohlfahrt et al., 2007). Risk taking is therefore often quantified in assays involving the presence of predators directly or via predation cues, 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, if high-condition individuals are less vulnerable to predation. Similarly, the effects of starvation avoidance may be more relevant in experimental contexts where potential food rewards are explicit, 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

149 an increased capacity to express behavioural plasticity (Royauté & Dochtermann, 2017;
150 Royauté *et al.*, 2019). Conversely, it is conceivable that extremely poor conditions may lead
151 to the expression of cryptic genetic variation, and thus increased variation in state and
152 behaviour among low-condition individuals. However, if a high-risk strategy is the only viable
153 option for acquiring adequate resources in a poor environment, individuals (including low-
154 condition individuals) may converge on a high-risk phenotype (Han & Dingemanse, 2017).
155 Overall, condition-dependent effects on the variance in risky behaviours are likely present,
156 but currently are difficult to predict in direction and magnitude.
157
158 We here present a systematic review and meta-analysis of studies that experimentally
159 manipulated individual nutritional or energetic condition through diet quality or quantity
160 treatments, and independently quantified risk-taking behaviours such as exploration, and
161 predation and novelty responses. Specifically, we address six questions, which we
162 preregistered previous to the study (see details below):
163 1. Do nutritional condition manipulation treatments have an overall effect on mean risk-
164 taking behaviour? We do not predict a clear non-zero overall effect, but instead
165 expect high heterogeneity among effect sizes resulting from the various contexts in
166 which risk is measured and the multiple mechanisms that may drive condition effects
167 on risk taking.
168 2. Is the effect of nutritional condition on mean risk-taking behaviour context-
169 dependent? We expect low-condition treatment groups to show increased risk-taking
170 behaviour in both foraging and feeding contexts (starvation avoidance effect), but
171 reduced risk-taking behaviour in predator-response contexts (state-dependent safety
172 effect). Across the remaining contexts (e.g. novel environment exploration, novel
173 object response), we predict high-condition treatment groups to show reduced risk-
174 taking behaviour (asset-protection effect).
175 3. Does nutritional condition have differential effects on mean risk-taking behaviour in
176 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 nutritional 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 nutritional 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 nutritional 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 that manipulated the condition of animals in independent treatment groups through their diet, via both dietary quantity (i.e. partial restriction, complete deprivation or enrichment) or quality treatments (e.g. protein restriction or enrichment), and including both short term and longer term manipulations up to extended periods of weeks-months. Then we screened for studies that then subjected those animals to 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 excluded as 'non-independent': where the behaviour was measured in the presence of high and low food availability, or dietary treatments such as periods of deprivation were applied within the novel environment (i.e. non-independence of treatments from the behavioural assay); where the dietary treatments were coupled with additional non-dietary factors (non-independence of the diet factor within treatments; e.g. temperature); or, the dietary treatments were applied longitudinally (within individuals) rather than cross-sectionally (i.e. non-independence between high and low treatments).

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 analysis, therefore we conducted a sensitivity analysis to test the potential effects of these additional five references by re-running the main effects models without these effect sizes, and results remain very similar (see Supporting Information S3). Full-text screening of 641 papers resulted in 5/160 (3.1%) conflicted decisions (i.e. where one screener included a reference, and the other excluded it), that were resolved collectively by both screeners. Full-text screening identified 147 studies meeting the experimental design criteria for inclusion (see https://osf.io/3tphj/ for full-text screening decision database 'CD_FulltextScreeningDatabase.xlsx', and Supporting Information S1 for the PRISMA diagram and the decision tree summarizing the full-text exclusion reasons).

Data were extracted as comparisons between the *low-condition* groups (i.e. the treatment group for diet restriction treatments, the control group for diet enrichment treatments) and the *high-condition* groups (i.e. the control group for diet restriction treatments, and the treatment group for diet enrichment treatments). Extractions were conducted by NPM with data extracted from figures where necessary using the R package 'metaDigitise' v1.0.0 (Pick, Nakagawa, & Noble, 2019). Data required to calculate effect sizes were (a) group means and (b) estimates of uncertainty (standard error, confidence intervals) or variability (standard deviation) in combination with sample sizes (N) for the behavioural variables of interest. Full or partial extraction of relevant data was possible from the published material of 118 studies (80.2% of all studies included after full-text screening). To recover missing or partially reported data, corresponding authors of 72 studies were contacted via a standardized author correspondence email, such that 395 (29.6%) of 1334 effect sizes in the full final dataset were obtained via author correspondence. Data from 25% of included papers (37 papers) were re-extracted by an independent observer to ensure data reliability. Of 1420 re-extracted values, errors requiring correction were identified in only 6 values (0.4%) affecting only two effect sizes included in the final analyses.

*(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 relative heterogeneity by calculating '$I^2$' values (Nakagawa & Santos, 2012, via R package v0.0.0.9000 'MetaAidR', Noble, 2019), and estimated absolute heterogeneity '$Q$'. For moderator models, we calculated the percentage of heterogeneity explained by the inclusion of moderators '$R^2_{marginal}$' (i.e. as the estimated percentage decrease in heterogeneity between the moderator model and the non-moderator model), 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).

First, we included a categorical moderator (‘*RiskContext*’) to test if effects were context-dependent by classifying 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). Second, a categorical moderator (‘*Sex*’) tested for differences between male and female experimental groups. Effect sizes were calculated separately for males and females where sufficient data was available, otherwise effect sizes were categorized as mixed (i.e. groups including both sexes), or unknown (i.e. no information about the sex of study subjects). Third, a categorical moderator (‘*ManipLifeStage*’) tested for an effect of life-stage at the time of the treatments, with the level of maturity during diet manipulations categorised as juvenile, adult, both (i.e. for treatments spanning both periods), or unknown/mixed. 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 condition treatment(s) and behavioural experiments relative to the species maximum longevity as a continuous moderator (‘*RelativeTimeFromTreatment.C’)*. Finally, to assess the role of life-history variation among species, we separately tested for effects of maximum lifespan (‘*MaxLongevity.C’)* and the natural logarithm of maximum lifespan (‘*lnMaxLongevity.C’)* as continuous moderators. Log transformed lifespan was used to better captures the variability in lifespan between species, as estimates for included species 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 square root of the inverse sampling variance ('*Precision'*, 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 as a continuous moderator ('*Year.C*'), 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 used a categorical moderator to test whether effect sizes were larger in studies with partial or incomplete reporting of results ('*EffectSizesFromPublication',* 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 of both lnRR and lnCVR (for moderators and descriptions see Supporting Information S6).

We tested a categorical moderator based on the differing types of diet manipulation included in our analysis ('*ManipType*'). 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 categorical moderator ('*ManipDirection* '). This 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 explore how the duration of diet treatments influenced the outcome, a continuous moderator ('*RelativeManipDuration.C*') was defined as the time that the treatment was applied as a proportion of the maximum lifespan of the species. Finally, the influence of the source of the study subjects was tested using via a categorical moderator ('*WildLabRear*', 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 *RiskContext* moderator explaining a large amount of heterogeneity among effect sizes ($R^2_{marginal}$ = 12.03%; Table 2). Although most 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). The 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). Untransformed maximum lifespan did not appear to influence lnRR (0.00 [-0.08 – 0.09]). However, log-transformed 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 lifespan estimate 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 either the type or direction of diet manipulation 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). The effect of the duration of diet treatments 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 the type and direction of diet manipulation did not appear to influence lnCVR substantially, whereas the duration of diet treatments 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 expectations, we found a convincing directional effect on mean risk-taking behaviour, where individuals subject to low condition dietary treatments are more likely to show high-risk behaviour in a range of contexts involving predation and novelty. This condition-dependency may be caused by increased risk aversion in higher-condition

individuals due to their greater reproductive expectations (an interpretation consistent with the asset-protection principle applying to the context of nutritional condition and predation-novelty based risk), 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 nutritional state (Ludwig & Rowe, 1990; 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 humans (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 high heterogeneity, our overall effect does not preclude the opposite pattern being applicable in certain systems. Also, our findings focus on nutritional state in contexts often involving direct or indirect predation risk, so state-dependent safety may be more directly

applicable when considering types of state variables that provide a more direct advantage in reducing predation risk (e.g. defensive traits), or in risk-taking contexts where physical condition provides a clearer advantage (e.g. intraspecific contests).

The experimental context of risk-taking behaviour 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

563 variation (Han & Dingemanse, 2015), but there does not appear to be a generalizable
564 direction to this effect. Life-stage effects did show evidence that treatments in juvenile stages
565 had strong and positive effects, while effects in adults were less clear. The effects of life-
566 stage and sex may be interrelated in a way that was not originally anticipated, as the strong
567 effect in unknown sex groups may be related to an overrepresentation of juveniles in that
568 category. Whereas studies where sex was identifiable may have been more likely to involve
569 adult treatments groups, with both sex-specific and adult-specific estimates being smaller.
570 The influence of longevity was ambiguous, but ongoing theoretical support for asset
571 protection to be sensitive to life-history traits (e.g. iteroparous vs. semelparous reproductive
572 strategies; Luttbeg et al., 2020) suggests that a more focused analysis incorporating life-
573 history differences is warranted, particularly in relation to reproductive traits.
574
575 Our exploratory analyses revealed a few key patterns in condition-dependent behavioural
576 responses, and the suitability of our methodology. Modelling studies have suggested there
577 may be non-linearity in state-dependent phenotypic responses in risk-taking behaviour, due
578 to potential factors such as inconstant correlations between condition and reproductive value
579 (Clark, 1994; McNamara & Houston, 1996; Luttbeg & Sih, 2010). While not directly testing
580 this, evidence of a non-linear effect of condition and risk taking was not detected in the
581 analysis of diet manipulation direction. Effects were similar for each group (i.e. reduced vs.
582 standard condition; standard vs. enriched condition, reduced vs. enriched condition),
583 supporting a more constant directional effect of condition on mean risk taking, and
584 suggesting that our methodology of pooling these designs together for analysis was sound.
585 Similarly, the mean effect estimate was positive across all classes of diet treatment analysed
586 (e.g. quality, quantity etc.), such that pooling these experiments was unlikely to influence
587 results. Finally, wild-reared animals did show the largest effect of treatment on mean risk
588 taking (and also a particularly strong negative effect on behavioural variation), suggesting
589 that these animals might be either more sensitive to imposed dietary manipulations or more
590 responsive to predator-based risk due to past experiences in the wild .

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). Variance meta-analyses are expected to be more data hungry, although this is unlikely to be the cause of the overall weak lnCVR effect found in our study given the large dataset used.

Variation in behaviour was sensitive to the experimental context of risk-taking behaviour, with variation in both the strength and direction of context-specific effects. In particular, variance in feeding behaviour within novel environments was far lower in low-condition groups, providing some evidence that being highly motivated to feed in this context is an optimum phenotype for individuals in poor energetic state. In contrast, variation in refuge use in a novel environment was higher in low-condition groups, which may be evidence of the opposite (complementary) pattern where high refuge use is a preferred strategy for high condition individuals. Effects of life stage on behavioural variation are consistent with recent empirical evidence suggesting that developmental diet is related to phenotypic plasticity and personality development (see examples in Royauté & Dochtermann 2017; Kelleher *et al.* 2019). Buchanan, Grindstaff, & Pravosudov (2013) suggested that poor condition during early life stages may reduce an individual's capacity to express behavioural plasticity. This is potentially consistent with our finding of reduced behavioural variation in groups subject to low-condition treatments as juveniles, while the effect in adults heavily overlapped with zero. We also found that treatments that spanned juvenile and adult life stages (often longer term, chronic diet restriction treatments) had a positive effect on behavioural variation. Similarly,

619 the duration of diet treatments had a positive effect on behavioural variation, consistent with
620 the proposition that extremely poor diet conditions can expose cryptic genetic and
621 phenotypic variation (Han & Dingemanse, 2017). Nonetheless, identifying mechanisms from
622 unpartitioned phenotypic variance remains challenging, as the proposed mechanisms for
623 effects on variability in risk-taking behaviour often apply specifically to among- or within-
624 individual levels (Han & Dingemanse, 2015).
625
626 A pertinent question in behavioural ecology is whether phenotypic variation is primarily within
627 or among individuals (Westneat, Wright, & Dingemanse, 2015). Any effects on the variance
628 as estimated in our meta-analysis (and more generally in most meta-analysis using lnCVR)
629 may arise from either source. Individuals might become more variable in their behaviour in
630 response to some treatment (or some environmental effect) as a form of behavioural bet-
631 hedging or reduce accuracy of performance (i.e. within-individual level). Alternatively,
632 individuals might differ in their average responses to changes in conditions if they have
633 intrinsically different reaction norms (i.e. among-individual level). Only repeated
634 measurements per individual would help to separate the two variance components.
635 However, this type of data is usually not available in the literature (Niemelä and Dingemanse
636 2018). Future studies should focus on the relative importance of within- vs. among-individual
637 variance in the variance effects identified in our study.
638
639 Considered together, our publication bias analyses suggest there may be some limited
640 influence on the overall results. Time-lag analysis showed that effect sizes might be
641 decreasing over time, while precision analysis showed a small negative effect, both of which
642 can be signs of publication bias toward a positive effect (Jennions & Møler, 2002; Jennions
643 *et al.*, 2013). Moreover, effect sizes obtained from author correspondence where no data
644 could be extracted from published material showed the lowest and most uncertain effect,
645 suggesting preferential publication of positive effects. Intriguingly, publication bias appears to
646 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) The overall evidence of diet and thus nutritional condition effects on risk-taking behaviour in the literature is clear, as low-condition individuals appear willing to on average take greater risks in ecological contexts relating to predation risk 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).

## IX. References

Andrews, C., Nettle, D., Reichert, S., Bedford, T., Monaghan, P. & Bateson, M. (2018) A marker of biological ageing predicts adult risk preference in European starlings, *Sturnus vulgaris*. *Behavioral Ecology* **29**, 589–597.

Barclay, P., Mishra, S. & Sparks, A.M. (2018) State-dependent risk-taking. *Proceedings of the Royal Society B: Biological Sciences* **285**, 20180180.

Beckmann, C. & Biro, P.A. (2013) On the Validity of a Single (Boldness) Assay in Personality Research. *Ethology* **119**, 937–947.

Bonett, D.G. (2009) Meta-analytic interval estimation for standardized and unstandardized mean differences. *Psychological Methods* **14**, 225–238.

Borcherding, J. & Magnhagen, C. (2008) Food abundance affects both morphology and behaviour of juvenile perch. *Ecology of Freshwater Fish* **17**, 207–218.

Buchanan, K.L., Grindstaff, J.L. & Pravosudov, V.V. (2013) Condition dependence, developmental plasticity, and cognition: implications for ecology and evolution. *Trends in Ecology & Evolution* **28**, 290–296.

Carter, A.J., Marshall, H.H., Heinsohn, R. & Cowlishaw, G. (2012) How not to measure boldness: novel object and antipredator responses are not the same in wild baboons. *Animal Behaviour* **84**, 603–609.

709 Clark, C.W. (1994) Antipredator behavior and the asset-protection principle. *Behavioral*
710 *Ecology* **5**, 159–170.
711 Dall, S.R.X. & Johnstone, R.A. (2002) Managing uncertainty: information and insurance
712 under the risk of starvation. *Philosophical Transactions of the Royal Society of*
713 *London B: Biological Sciences* **357**, 1519–1526.
714 English, S. & Uller, T. (2016) Does early-life diet affect longevity? A meta-analysis across
715 experimental studies. *Biology Letters* **12**, 20160291.
716 Engqvist, L., Cordes, N. & Reinhold, K. (2014) Evolution of risk-taking during conspicuous
717 mating displays. *Evolution* **69**, 395–406.
718 Gosling, S.D. (2008) Personality in Non-human Animals. *Social and Personality Psychology*
719 *Compass* **2**, 985–1001.
720 Grafen, A. (1989) The phylogenetic regression. *Philosophical Transactions of the Royal*
721 *Society of London. Series B, Biological Sciences* **326**, 119–157.
722 Han, C.S. & Dingemanse, N.J. (2015) Effect of diet on the structure of animal personality.
723 *Frontiers in Zoology* **12**, S5.
724 Han, C.S. & Dingemanse, N.J. (2017) You are what you eat: diet shapes body composition,
725 personality and behavioural stability. *BMC Evolutionary Biology* **17**, 8.
726 Hedges, L.V., Gurevitch, J. & Curtis, P.S. (1999) The Meta-Analysis of Response Ratios in
727 Experimental Ecology. *Ecology* **80**, 1150–1156.
728 Hinchliff, C.E., Smith, S.A., Allman, J.F., Burleigh, J.G., Chaudhary, R., Coghill, L.M.,
729 Crandall, K.A., Deng, J., Drew, B.T., Gazis, R., Gude, K., Hibbett, D.S., Katz, L.A.,
730 Laughinghouse, H.D., McTavish, E.J., et al. (2015) Synthesis of phylogeny and
731 taxonomy into a comprehensive tree of life. *Proceedings of the National Academy of*
732 *Sciences* **112**, 12764–12769.
733 Houslay, T.M., Hunt, J., Tinsley, M.C. & Bussière, L.F. (2015) Sex differences in the effects
734 of juvenile and adult diet on age-dependent reproductive effort. *Journal of*
735 *Evolutionary Biology* **28**, 1067–1079.
736 Jennions, M. D., & Møller, A. P. (2002) Relationships fade with time: a meta-analysis of
737 temporal trends in publication in ecology and evolution. Proceedings of the Royal
738 Society of London. Series B: Biological Sciences **269**, 43-48.
739 Kelleher, S.R., Silla, A.J., Niemelä, P.T., Dingemanse, N.J. & Byrne, P.G. (2019) Dietary
740 carotenoids affect the development of individual differences and behavioral plasticity.
741 *Behavioral Ecology* **30**, 1273–1282.
742 Kelly, C.D., Stoehr, A.M., Nunn, C., Smyth, K.N. & Prokop, Z.M. (2018) Sexual dimorphism
743 in immunity across animals: a meta-analysis. *Ecology Letters* **21**, 1885–1894.
744 Krause, E.T. & Caspers, B.A. (2016) Long-term consequences of early nutritional conditions
745 on the behaviour and growth of fire salamanders. *Amphibia-Reptilia* **37**, 69–77.
746 Lajeunesse, M.J. (2011) On the meta-analysis of response ratios for studies with correlated
747 and multi-group designs. *Ecology* **92**, 2049–2055.
748 Lajeunesse, M.J. (2013) Recovering missing or partial data from studies: a survey of
749 conversions and imputations for meta-analysis. *Handbook of meta-analysis in*
750 *ecology and evolution*, 195–206.
751 Ludwig, D. & Rowe, L. (1990) Life-history strategies for energy gain and predator avoidance
752 under time constraints. *The American Naturalist* **135**, 686–707.
753 Luttbeg, B. & Sih, A. (2010) Risk, resources and state-dependent adaptive behavioural
754 syndromes. *Philosophical Transactions of the Royal Society B: Biological Sciences*
755 **365**, 3977–3990.
756 Luttbeg, B., Ferrari, M.C.O., Blumstein, D.T. & Chivers, D.P. (2020) Safety cues can give
757 prey more valuable information than danger cues. *The American Naturalist* **195**, 636–
758 648.
759 McNamara, J.M. & Houston, A.I. (1996) State-dependent life histories. *Nature* **380**, 215–221.
760 Michonneau, F., Brown, J.W. & Winter, D.J. (2016) rotl: an R package to interact with the
761 Open Tree of Life data. *Methods in Ecology and Evolution* **7**, 1476–1481.
762 Moher, D., Liberati, A., Tetzlaff, J., Altman, D.G. & Group, T.P. (2009) Preferred Reporting
763 Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement. *PLOS*

764 *Medicine* **6**, e1000097.
765 Moiron, M., Araya-Ajoy, Y. G., Mathot, K. J., Mouchet, A., & Dingemanse, N. J. (2019)
766 Functional relations between body mass and risk-taking behavior in wild great tits.
767 *Behavioral Ecology* **30**, 617-623.
768 Moiron, M., Laskowski, K.L. & Niemelä, P.T. (2020) Individual differences in behaviour
769 explain variation in survival: a meta-analysis. *Ecology Letters* **23**, 399–408.
770 Moran, N.P., Mossop, K.D., Thompson, R.M. & Wong, B.B.M. (2016) Boldness in extreme
771 environments: temperament divergence in a desert-dwelling fish. *Animal Behaviour*
772 **122**, 125–133.
773 Moschilla, J.A., Tomkins, J.L. & Simmons, L.W. (2018) State-dependent changes in risk-
774 taking behaviour as a result of age and residual reproductive value. *Animal*
775 *Behaviour* **142**, 95–100.
776 Nakagawa, S., Poulin, R., Mengersen, K., Reinhold, K., Engqvist, L., Lagisz, M. & Senior,
777 A.M. (2015) Meta-analysis of variation: ecological and evolutionary applications and
778 beyond. *Methods in Ecology and Evolution* **6**, 143–152.
779 Nakagawa, S. & Santos, E.S.A. (2012) Methodological issues and advances in biological
780 meta-analysis. *Evolutionary Ecology* **26**, 1253–1274.
781 Niemelä, P.T. & Dingemanse, N.J. (2018) Meta-analysis reveals weak associations between
782 intrinsic state and personality. *Proceedings of the Royal Society B: Biological*
783 *Sciences* **285**.
784 Noble, D.W.A. (2019) Functions for aiding meta-analyses in Ecology, Evolution and beyond.
785 *https://github.com/daniel1noble/metaAidR*.
786 Ouzzani, M., Hammady, H., Fedorowicz, Z. & Elmagarmid, A. (2016) Rayyan—a web and
787 mobile app for systematic reviews. *Systematic Reviews* **5**, 210.
788 Paradis, E. & Schliep, K. (2019) ape 5.0: an environment for modern phylogenetics and
789 evolutionary analyses in R. *Bioinformatics (Oxford, England)* **35**, 526–528.
790 Pick, J.L., Khwaja, N., Spence, M.A., Ihle, M. & Nakagawa, S. (2019a) Counter culture:
791 Causes, extent and solutions of systematic bias in the analysis of behavioural counts.
792 *EcoEvoRxiv*, https://ecoevorxiv.org/jq9n6/.
793 Pick, J.L., Nakagawa, S. & Noble, D.W. (2019b) Reproducible, flexible and high-throughput
794 data extraction from primary literature: The metaDigitise r package. *Methods in*
795 *Ecology and Evolution* **10**, 426–431.
796 Réale, D., Reader, S.M., Sol, D., McDougall, P.T. & Dingemanse, N.J. (2007) Integrating
797 animal temperament within ecology and evolution. *Biological Reviews* **82**, 291–318.
798 Royauté, R. & Dochtermann, N.A. (2017) When the mean no longer matters: developmental
799 diet affects behavioral variation but not population averages in the house cricket
800 (*Acheta domesticus*). *Behavioral Ecology* **28**, 337–345.
801 Royauté, R., Garrison, C., Dalos, J., Berdal, M.A. & Dochtermann, N.A. (2019) Current
802 energy state interacts with the developmental environment to influence behavioural
803 plasticity. *Animal Behaviour* **148**, 39–51.
804 Sánchez-Tójar, A., Moran, N.P., O'Dea, R.E., Reinhold, K. & Nakagawa, S. (2019)
805 Illustrating the importance of meta-analysing variances alongside means in ecology
806 and evolution. *EcoEvoRxiv*, https://ecoevorxiv.org/yhfvk/.
807 Sánchez-Tójar, A., Nakagawa, S., Sánchez-Fortun, M., Martin, D.A., Ramani, S., Girndt, A.,
808 Bokony, V., Kempenaers, B., Liker, A., Westneat, D., Burke, T. & Schroeder, J.
809 (2018) Meta-analysis challenges a textbook example of status signalling: evidence
810 for publication bias. *eLife*, e37385.
811 Schielzeth, H. (2010) Simple means to improve the interpretability of regression coefficients.
812 *Methods in Ecology and Evolution* **1**, 103–113.
813 Senior, A.M., Nakagawa, S., Raubenheimer, D., Simpson, S.J. & Noble, D.W.A. (2017)
814 Dietary restriction increases variability in longevity. *Biology Letters* **13**, 20170057.
815 Sih, A., Mathot, K.J., Moirón, M., Montiglio, P.-O., Wolf, M. & Dingemanse, N.J. (2015)
816 Animal personality and state–behaviour feedbacks: a review and guide for
817 empiricists. *Trends in Ecology & Evolution* **30**, 50–60.
818 Temple, S.A. (1987) Do predators always capture substandard individuals disproportionately

819 from prey populations? *Ecology* **68**, 669–674.
820 Toms, C.N., Echevarria, D.J. & Jouandot, D.J. (2010) A methodological review of
821 personality-related studies in Fish: Focus on the shy-bold axis of behavior.
822 *International Journal of Comparative Psychology* **23**, 1–25.
823 Viechtbauer, W. (2010) Conducting meta-analyses in R with the metafor package. *Journal of*
824 *statistical software* **36**, 1–48.
825 Wohlfahrt, B., Mikolajewski, D.J., Joop, G. & Vamosi, S.M. (2007) Ontogenetic changes in
826 the association between antipredator responses and growth variables. *Ecological*
827 *Entomology* **32**, 567–574.
828 Westneat, D.F., Wright, J. & Dingemanse, N.J. (2015) The biology hidden inside residual
829 within-individual phenotypic variation. *Biological Reviews* **90**, 729–743.
830 White, J.R., Meekan, M.G., McCormick, M.I. & Ferrari, M.C.O. (2013) A Comparison of
831 Measures of Boldness and Their Relationships to Survival in Young Fish. *PLoS ONE*
832 **8**.
833 Wilson, D.S., Clark, A.B., Coleman, K. & Dearstyne, T. (1994) Shyness and boldness in
834 humans and other animals. *Trends in Ecology & Evolution* **9**, 442–446.
835 Wolf, M., van Doorn, G.S., Leimar, O. & Weissing, F.J. (2007) Life-history trade-offs favour
836 the evolution of animal personalities. *Nature* **447**, 581.
837 Wolf, M. & Weissing, F.J. (2010) An explanatory framework for adaptive personality
838 differences. *Philosophical Transactions of the Royal Society B: Biological Sciences*
839 **365**, 3959–3968.
840 Zimmer, C., Larriva, M., Boogert, N.J. & Spencer, K.A. (2017) Transgenerational
841 transmission of a stress-coping phenotype programmed by early-life stress in the
842 Japanese quail. *Scientific Reports* **7**, 46125.
843

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# 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 log response ratio (lnRR) and log coefficient of variation ratio (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 log response ratio (lnRR) models, 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 log coefficient of variation ratio (lnCVR) models, 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<br>p < 0.0001 | 38.4 ***<br>p = 0.0002 | 10.22 |
| Hyp. 3. Sex difference in risk taking (*cvr.Full.h3*) | lnCVR | 1235 | *Sex* | 2520.5<br>p < 0.0001 | 5.9<br>p = 0.2066 | 2.44 |
| Hyp. 4. Effects across life stages (*cvr.Full.h4*) | lnCVR | 1153 | *ManipLifeStage* + *RelativeTimeFromTreatment.C* | 2158.2<br>p < 0.0001 | 9.5<br>p = 0.0908 | 16.64 |
| Hyp. 5(1). Life-history effects (*cvr.Full.h5.i*) | lnCVR | 1153 | *MaxLongevity.C,* | 2185.53<br>p < 0.0001 | 1.41<br>p = 0.2348 | 13.81 |
| Hyp. 5(ii). Life-history effects (*cvr.Full.h5.ii*) | lnCVR | 1153 | *lnMaxLongevity.C* | 2187.91<br>p < 0.0001 | 0.34<br>p = 0.5615 | 13.14 |
| Exp a. Effect of manipulation type (*cvr.Full.exp.a*) | lnCVR | 1235 | *ManipType* | 2535.9<br>p < 0.0001 | 3.1<br>p = 0.5406 | 0.00 |
| Exp b. Effect of manipulation direction (*cvr.Full.exp.b*) | lnCVR | 1235 | *ManipDirection* | 2541.4<br>p < 0.0001 | 2.23<br>p = 0.5256 | 0.00 |
| Exp c. Effect of manipulation duration (*cvr.Full.exp.c*) | lnCVR | 1153 | *RelativeManipDuration.C* | 2182.57<br>p < 0.0001 | 4.59 *<br>p = 0.0322 | 16.17 |
| Exp d. Effect of rearing environment (*cvr.Full.exp.d*) | lnCVR | 1235 | *WildLabRear* | 2514.93<br>p < 0.0001 | 4.6<br>p = 0.3312 | 0.86 |

**Table 4:** Parameter estimates for log response ratio (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]** |

**Table 5:** Parameter estimates for log coefficient of variation ratio (lnCVR) hypothesis testing, 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 (*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]** |

## 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 log response ratio (lnRR) and log coefficient of variation ratio (lnCVR) with 95% confidence intervals. The number of effect sizes used in each model is *k*.

**Fig. 2** Category-specific estimates for log response ratio (lnRR) and log coefficient of variation ratio (lnCVR) with 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 log response ratio (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 log response ratio (lnRR) and log coefficient of variation ratio (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) the rearing environment of the experimental subjects. lnRR effects are presented on the left (A, C, D) frames and lnCVR on the right (B, D, F). The areas of the orange 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, respectively.

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**Fig. 1**

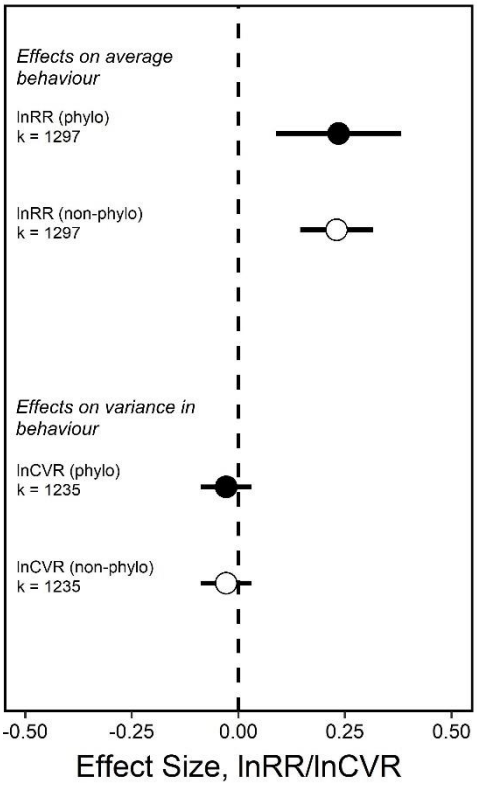


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**Fig 2**

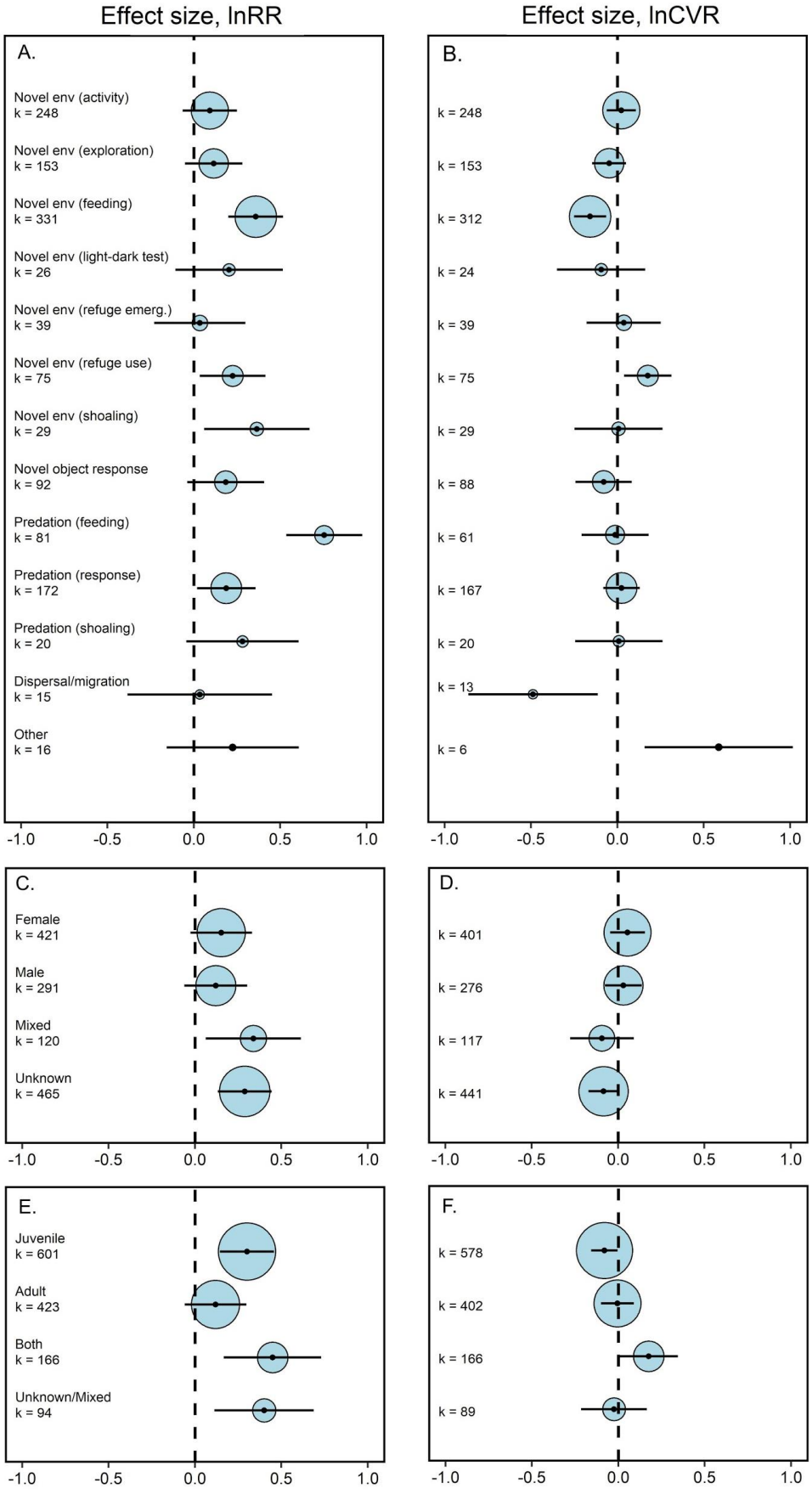


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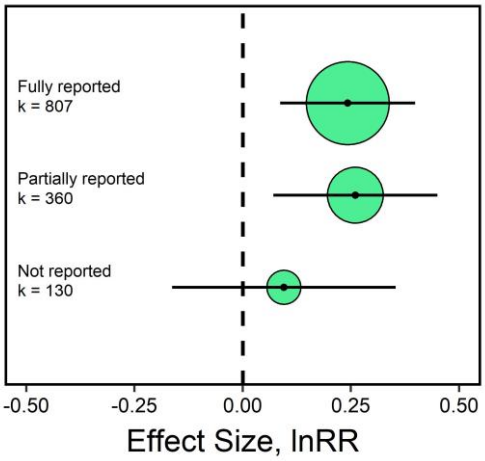
**Fig. 3**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Partially reported
k = 360
Not reported
k = 130
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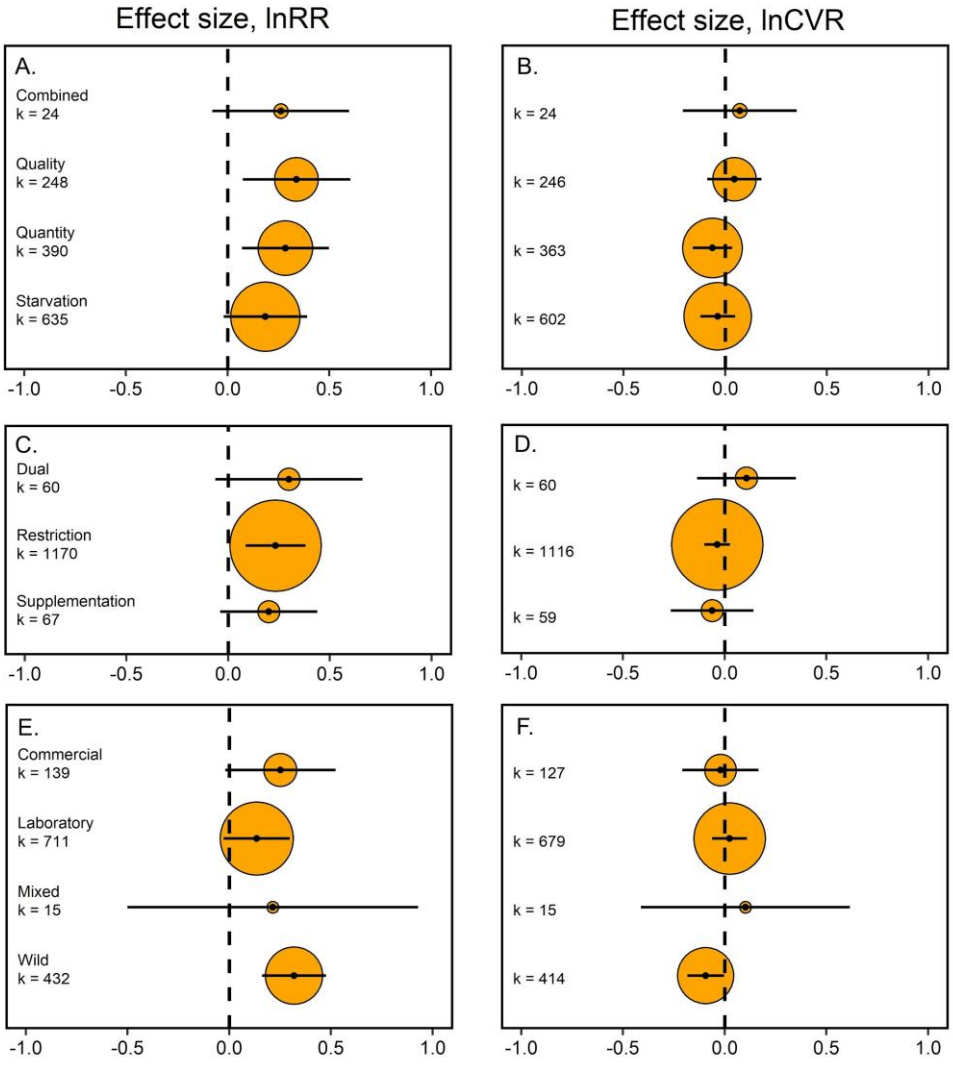
-0.25
0.00
0.25
0.50
Effect Size, lnRR

985 **Fig 4.**

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