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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1 Abstract

2 Animal behaviour can lead to varying levels of risk, and an individual's physical condition can 3 alter the potential costs and benefits of undertaking risky behaviours. How risk-taking 4 behaviour depends on condition is subject to contrasting hypotheses. The asset protection 5 principle proposes that individuals in better condition should be more risk averse, as they 6 have higher future reproductive potential (i.e. more to lose). The state-dependent safety 7 hypothesis proposes that high-condition individuals that are more likely to survive and 8 maximise the benefits of risky situations may make apparently riskier choices, as their 9 individual risk is in fact lower. We systematically searched for studies that experimentally 10 manipulated animals' nutritional or energetic condition through diet treatments, and 11 subsequently measured risk-taking behaviour in contexts relating to predation, novelty, 12 exploration. Our meta-analysis quantified condition effects on risk-taking behaviour at both 13 the mean and variance level. We preregistered our methods and hypotheses prior to 14 conducting the study. Phylogenetic multilevel meta-analysis revealed that the lower 15 nutritional condition individuals showed on average ca. 26% greater tendency towards risk 16 than high-condition individuals (95% confidence interval: 15% - 38%; n = 126 studies, 1297 17 effect sizes). Meta-regressions revealed several factors influencing the overall effect, such 18 as the experimental context used to measure risk-taking behaviour, and the life-stage when 19 condition was manipulated. Meta-analysis of variance revealed no clear overall effect of 20 condition on behavioural variance (on average ca. 3% decrease in variance in low- vs high-21 condition groups; 95% confidence interval: -8% - 3%; n = 119 studies, 1235 effect sizes), 22 however, the experimental context was an important factor influencing the strength and 23 direction of the variance effect. Our comprehensive systematic review and meta-analysis 24 provide insights into the roles of state-dependency and plasticity in intraspecific behavioural 25 variation. While heterogeneity among effect sizes was high, our results show that poor 26 nutritional state on average increases risk-taking in ecological contexts involving predation, 27 novelty and exploration.

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

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51

52 I. Introduction

53 Animals often must gamble with their lives, with behavioural decisions frequently involving 54 trade-offs between resource acquisition, reproduction and survival. Many of those decisions 55 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), 56 and promise large gains, but also the potential of large losses (Barclay, Mishra, & Sparks, 57 58 2018). The concept of risk may be applied broadly in animal ecology (e.g. participation in 59 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., 60 61 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, 62 63 and thus an important research topic in behavioural ecology. State variables, such as 64 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.

69 Individual condition, considered here as variation in nutritional or energetic state, can lead to 70 differences in morphological, behavioural and cognitive traits among individuals 71 (Borcherding & Magnhagen, 2008; Buchanan, Grindstaff, & Pravosudov, 2013; Han & 72 Dingemanse, 2015), which can subsequently affect risk taking in different ways. Animals in 73 high condition might be risk-averse, as these individuals have a lot to lose in terms of future 74 reproductive potential (the 'asset-protection principle'; Ludwig & Rowe, 1990; Clark, 1994), 75 whereas individuals in low condition have more to gain in terms of improved condition, 76 elevated competitiveness, and starvation avoidance, particularly when an individual is 77 relatively close to their starvation threshold (Dall & Johnstone, 2002; Luttbeg & Sih, 2010; 78 also known as the 'needs-based' explanation, Barclay et al., 2018). Contrastingly, the 'state-79 dependent safety' hypothesis (also known as the 'ability-based' explanation) predicts that 80 individuals may appear to take greater risks where they are better able to survive and 81 maximise the benefits of engaging in risky behaviours, as they individually experience a 82 lower level of risk (Barclay et al., 2018). State-dependent safety might apply if improved 83 condition allows greater investment in physical and/or cognitive capabilities (e.g. increased 84 vigour and/or ability to evade or defend against predation) that reduce the level of risk for the 85 individual (as in Temple, 1987).

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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

93 development in particular can increase investment in traits that improve future survival and 94 fitness, such as cognitive ability (Buchanan et al., 2013). This might allow greater risk taking 95 if those traits provide an advantage in certain risky contexts by altering effective risk levels, if 96 for example high-condition individuals are better protected/less vulnerable than low-condition 97 individuals in the same situation. Theoretical support for any one directional state-effect on 98 risk-taking is mixed, and show that the outcome may depend on environmental conditions, 99 such as overall resource availability or acuteness of the risk factor (Luttbeg & Sih, 2010; 100 Engqvist, Cordes, & Reinhold, 2014). Empirical results are similarly mixed, and thus it 101 remains unknown if there are any generally applicable effects of condition on risk-taking 102 behaviour, or the ecological context in which any one hypothesis applies.

103

104 Regardless of the hypothesis, condition effects on risk taking are often framed as adaptive 105 responses to variation in an individual's future fitness expectations (as in Clark, 1994; Wolf 106 et al., 2007). The key proposition being that decisions to take risks are related to variation in 107 state, where an individual's state includes all intrinsic and extrinsic factors strategically 108 relevant for their fitness (Wolf & Weissing, 2010). State-dependent responses due to 109 nutritional condition may have interactive effects with other state variables, such as life 110 history-differences within- or among-species (McNamara & Houston, 1996). For example, 111 sex is a form of state variation involving differences in reproductive roles, which may alter 112 male and female responses to poor dietary conditions (Han & Dingemanse, 2015). In some 113 cases, males could be more sensitive to condition due to condition-dependent sexual 114 selection, but in other cases, females may be more sensitive to condition since they often 115 bear a disproportionate energetic burden of reproduction (Houslay et al., 2015; English & 116 Uller, 2016). Similarly, interspecific differences in longevity may influence behavioural 117 responses, since long-lived species generally have a larger future reproductive asset and/or 118 more future opportunities to improve their own condition, and thus might be less willing to 119 display risky behaviour (Clark, 1994).

120

121 A subset of ecological contexts where variation in risk-taking behaviour can apply are those 122 involving trade-offs between resource acquisition and (implied or direct) predation risk, which 123 are often used in connection with the concept of 'boldness'. For example, responses to 124 novelty involve inherently high outcome variance, as the potential benefits and dangers of 125 novel situations are unknown to the individual. Furthermore, greater activity or exploration 126 increases the likelihood of both finding new resources or habitat patches, and encountering 127 predators (Réale et al., 2007, Wohlfahrt et al., 2007). Risk taking is therefore often quantified 128 in assays involving the presence of predators directly or via predation cues, which 129 emphasize the risk of mortality (Moschilla, Tomkins, & Simmons, 2018). Furthermore, some 130 studies manipulate the outcome variance of foraging-related behaviour directly (Andrews et 131 al., 2018). Studies of risk-taking behaviour across a variety of contexts have shown different 132 responses, for example between predator and novel object experimental setups (Carter et 133 al., 2012), or between emergence into a novel environment and startle responses 134 (Beckmann & Biro, 2013). As such, we expect condition effects to vary across experimental 135 contexts. For example, state-dependent safety may be more relevant in a predator-response 136 context, if high-condition individuals are less vulnerable to predation. Similarly, the effects of 137 starvation avoidance may be more relevant in experimental contexts where potential food 138 rewards are explicit, where low-condition individuals may show increased risk taking.

139

140 Thus far, most studies have focused on mean behavioural effects of condition (i.e. higher or 141 lower levels of risk taking). There has, however, been growing interest in individual-level 142 variation in recent years (Westneat, Wright, & Dingemanse, 2015), and new tools to meta-143 analyze variances alongside means are revealing that meta-variance effects may be both 144 prevalent and often overlooked (Nakagawa et al., 2015). While a recent meta-analysis of 145 variance has shown diet restriction can increase variation in longevity (Senior et al., 2017), 146 another has shown little evidence of environmental stress (including diet restriction) effects 147 on phenotypic behavioural variance (Sánchez-Tójar et al., 2019). Furthermore, case studies 148 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

We here present a systematic review and meta-analysis of studies that experimentally manipulated individual nutritional or energetic condition through diet quality or quantity treatments, and independently quantified risk-taking behaviours such as exploration, and predation and novelty responses. Specifically, we address six questions, which we preregistered previous to the study (see details below):

Do nutritional 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.

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

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 risktaking 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).

190
6. Does nutritional condition affect the amount of total variation in risk-taking behaviour
191 within high- and low-condition treatment groups? We do not predict an overall clear
192 variance effect between high- and low-condition experimental groups, however, as
193 for hypotheses 1 and 2, we predict variance effects to show high heterogeneity and
194 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.

200

201 II. Methods

202 (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). Nonpreregistered 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).

209

210 (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).

216

217 We screened records to find original experimental studies that manipulated the condition of 218 animals in independent treatment groups through their diet, via both dietary quantity (i.e. 219 partial restriction, complete deprivation or enrichment) or quality treatments (e.g. protein 220 restriction or enrichment), and including both short term and longer term manipulations up to 221 extended periods of weeks-months. Then we screened for studies that then subjected those 222 animals to behavioural observations in contexts relating to risk (e.g. novel environments, 223 novel object, risk-sensitive foraging, predator response) in independent trials (for inclusion 224 and exclusion decision trees see Supporting Information S1). Our aim was to test for 225 adaptive condition-dependent behavioural responses in non-human animals, therefore we 226 excluded studies using species with compromised genetic diversity and/or evolved adaptive 227 responses (e.g. domesticated animals, laboratory breeds, genetically modified organisms; as 228 per Kelly et al., 2018) as well as studies on humans. Studies manipulating the micronutrient 229 content of diets, or subjecting animals to high fat diets were also excluded as the relationship 230 between these diet manipulations and body condition is not clear and considered beyond the

231 scope of this review. Dietary treatments were excluded as 'non-independent': where the 232 behaviour was measured in the presence of high and low food availability, or dietary 233 treatments such as periods of deprivation were applied within the novel environment (i.e. 234 non-independence of treatments from the behavioural assay); where the dietary treatments 235 were coupled with additional non-dietary factors (non-independence of the diet factor within 236 treatments; e.g. temperature); or, the dietary treatments were applied longitudinally (within 237 individuals) rather than cross-sectionally (i.e. non-independence between high and low 238 treatments).

239

240 Both the title and abstract screening of 5453 records (post-deduplication), and the full-text 241 screening of 641 published papers were conducted by two authors (NPM 100%, AST 25% at 242 both stages) to ensure reliability. Title and abstract screening was done using Rayyan 243 (Ouzzani et al., 2016), from which 626 references were included for full-text screening. The 244 title and abstract screening resulted in 67/1377 (4.9%) conflicted decisions between 245 observers, confirming high inter-screener agreement. All conflicted decisions were resolved 246 collectively by both screeners. A few additional references that were not captured by our 247 search but instead identified from different sources were also included for full-text screening 248 ('non-systematic' records, n = 15). Data from five such papers were included in the final 249 analysis, therefore we conducted a sensitivity analysis to test the potential effects of these 250 additional five references by re-running the main effects models without these effect sizes, 251 and results remain very similar (see Supporting Information S3). Full-text screening of 641 252 papers resulted in 5/160 (3.1%) conflicted decisions (i.e. where one screener included a 253 reference, and the other excluded it), that were resolved collectively by both screeners. Fulltext screening identified 147 studies meeting the experimental design criteria for inclusion 254 255 (see https://osf.io/3tphi/ for full-text screening decision database 256 'CD FulltextScreeningDatabase.xlsx', and Supporting Information S1 for the PRISMA 257 diagram and the decision tree summarizing the full-text exclusion reasons).

258

259 Data were extracted as comparisons between the *low-condition* groups (i.e. the treatment 260 group for diet restriction treatments, the control group for diet enrichment treatments) and 261 the high-condition groups (i.e. the control group for diet restriction treatments, and the 262 treatment group for diet enrichment treatments). Extractions were conducted by NPM with 263 data extracted from figures where necessary using the R package 'metaDigitise' v1.0.0 264 (Pick, Nakagawa, & Noble, 2019). Data required to calculate effect sizes were (a) group 265 means and (b) estimates of uncertainty (standard error, confidence intervals) or variability 266 (standard deviation) in combination with sample sizes (N) for the behavioural variables of 267 interest. Full or partial extraction of relevant data was possible from the published material of 268 118 studies (80.2% of all studies included after full-text screening). To recover missing or 269 partially reported data, corresponding authors of 72 studies were contacted via a 270 standardized author correspondence email, such that 395 (29.6%) of 1334 effect sizes in the 271 full final dataset were obtained via author correspondence. Data from 25% of included 272 papers (37 papers) were re-extracted by an independent observer to ensure data reliability. 273 Of 1420 re-extracted values, errors requiring correction were identified in only 6 values 274 (0.4%) affecting only two effect sizes included in the final analyses.

275

276 (3) Effect size calculation

277 We analysed mean effects using the log response ratio of group means ('InRR'; Hedges, Gurevitch, & Curtis, 1999), instead of Cohen's D or Hedge's g, as InRR is less sensitive to 278 279 heteroscedasticity. Variance effects were analyzed using the log coefficient of variation ratio 280 ('InCVR'), as this effect size, unlike log ratio of variances ('InVR'), is less sensitive to 281 potential mean-variance correlations (Nakagawa et al., 2015). Both ratios were calculated 282 using *low condition* over *high condition*, such that a positive effect size represents higher risk 283 taking or larger variance in risk taking in low-condition animals, respectively (effect sizes 284 calculated via R package 'metafor' version v2.1-0, Viechtbauer, 2010). To maintain 285 consistent directionality, effect sizes were reversed for a subset of InRR effect sizes where 286 lower values reflected higher risk behaviours (e.g. 'latency to emerge from a shelter',

'distance from a predator' etc.). Since InCVR 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
(InVR; Nakagawa *et al.*, 2015). Conclusions remained robust (see Supporting Information S4
for details).

293

294 (4) Data analysis - main effects models

295 Two multilevel intercept-only meta-analytic models were run for each effect size, testing for a 296 general effect of condition treatments on risk-taking behaviour at a mean and variance level 297 (using the function 'rma.mv' from the R package 'metafor' v2.1-0, Viechtbauer, 2010). 298 Phylogenetic and non-phylogenetic models were run to investigate whether non-299 independence due to the degree of relatedness between species influenced both the overall 300 effects and their level of uncertainty. Phylogenetic relatedness were estimated based on 301 existing phylogenies and taxonomic information from the Open Tree of Life, and any 302 polytomies were resolved by randomization (Hinchliff et al., 2015; via R package 'rotl' v3.0.7; 303 Michonneau, Brown, & Winter, 2016; for the final phylogenetic tree see Supporting 304 Information S5). Branch lengths were estimated using Grafen's method (Grafen, 1989; via R 305 package 'ape' v5.3; Paradis & Schliep, 2019), and were used to construct a phylogenetic 306 variance-covariance relatedness matrix.

307

In addition to phylogeny, we included other random effects in our models to account for nonindependence 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).

318

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).

326

327 For main effects models, we investigated total, residual and random effect specific relative heterogeneity by calculating 'f' values (Nakagawa & Santos, 2012, via R package 328 329 v0.0.0.9000 'MetaAidR', Noble, 2019), and estimated absolute heterogeneity 'Q'. For 330 moderator models, we calculated the percentage of heterogeneity explained by the inclusion 331 of moderators ' $R^{2}_{marginal}$ ' (i.e. as the estimated percentage decrease in heterogeneity 332 between the moderator model and the non-moderator model), the residual heterogeneity 333 Q_{E} , and moderator specific heterogeneity Q_{M} (via R package 'metafor' v2.1-0, Viechtbauer, 334 2010). Where applicable, estimates are presented with 95% confidence intervals in square 335 brackets (hereafter simply refer to as 'confidence interval').

336

337 (5) Data analysis - hypothesis testing models

338 All hypotheses were tested using phylogenetic multilevel meta-regression models for both

339 InRR and InCVR including random effects as above (for detailed descriptions of all

340 moderators used for hypothesis testing models see Supporting Information S6).

341

342 First, we included a categorical moderator ('RiskContext') to test if effects were context-343 dependent by classifying behavioural variables by both the functional context of the 344 experiment (e.g. assays involving predators or predator cues, novel objects, novel 345 environments etc.; Luttbeg & Sih, 2010) and the specific behavioural measurements (e.g. 346 activity levels, areas explored, willingness to feed and forage, shoaling tendencies etc.; for 347 descriptions of all categories see Supporting Information S6). Second, a categorical 348 moderator ('Sex') tested for differences between male and female experimental groups. 349 Effect sizes were calculated separately for males and females where sufficient data was 350 available, otherwise effect sizes were categorized as mixed (i.e. groups including both 351 sexes), or unknown (i.e. no information about the sex of study subjects). Third, a categorical 352 moderator ('ManipLifeStage') tested for an effect of life-stage at the time of the treatments, 353 with the level of maturity during diet manipulations categorised as juvenile, adult, both (i.e. 354 for treatments spanning both periods), or unknown/mixed. If the paper did not present 355 sufficient information to determine the subject's life-stage, this was inferred from the 356 available information (e.g. age, average length, weight etc.). If life-stage could not be 357 reasonably inferred or if groups may have included both juvenile and adult individuals, these 358 were classed together as mixed/unknown. Since treatments in juveniles may have been 359 imposed a longer time before behavioural testing (e.g. early-life diet treatments with adult 360 behavioural testing) relative to adult diet treatments, life-stage models also included the time 361 between condition treatment(s) and behavioural experiments relative to the species 362 maximum longevity as a continuous moderator ('RelativeTimeFromTreatment.C'). Finally, to 363 assess the role of life-history variation among species, we separately tested for effects of 364 maximum lifespan ('MaxLongevity.C') and the natural logarithm of maximum lifespan 365 ('InMaxLongevity.C') as continuous moderators. Log transformed lifespan was used to better 366 captures the variability in lifespan between species, as estimates for included species were 367 heavily biased towards short lifespans. Lifespan estimates were obtained from online 368 databases (AnAge, genomics.senescence.info; FishBase, fishbase.se, Animal Diversity 369 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).

374

375 (6) Data analysis - publication bias tests

Several meta-regression models were used to assess our InRR dataset for evidence of
publication bias (for all included moderators and descriptions see Supporting Information
S6).

379

380 First, the precision of each effect was included as a moderator, calculated as the square root 381 of the inverse sampling variance ('Precision', a variant of an Egger's regression based on 382 Nakagawa & Santos, 2012), to test for small-study bias. Next, time-lag bias was tested using 383 the year of publication as a continuous moderator ('Year.C'), where a commonly observed 384 trend is a decrease in effect size over time (Jennions & Møler, 2002; Sánchez-Tójar et al., 385 2018). For both the precision and time-lag models, a limited dataset excluding effect sizes 386 obtained through author correspondence was used so that we were specifically testing for 387 effects of publication bias in published material. Finally, using the full dataset, we used a 388 categorical moderator to test whether effect sizes were larger in studies with partial or 389 incomplete reporting of results ('EffectSizesFromPublication', i.e. complete, partial or none; 390 where none refers to studies where all effect sizes had to be obtained via author 391 correspondence). In addition, funnel plots were produced using InRR and precision for a 392 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 393 394 calculated fail-safe N to test the robustness of our results (function 'fsn', R package 'metafor' 395 v2.1-0, Viechtbauer, 2010; see Supporting Information S7). Publication bias tests were not 396 conducted for InCVR, 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 didnot expect publication bias on InCVR.

399

400 (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 InRR and InCVR
(for moderators and descriptions see Supporting Information S6).

404

405 We tested a categorical moderator based on the differing types of diet manipulation 406 included in our analysis ('ManipType'). This included quantity (where the amount of food 407 ration/food access differed between groups), starvation (where one group was entirely 408 deprived of food for an extended period), quality (where the nutritional content of food 409 differed between groups) or combined (where both quality and quantity was manipulated in 410 the same treatment group). Since our main models compared low-versus high-condition 411 treatment groups regardless of whether diets corresponded to restriction or supplementation 412 treatments, we also explored potential effects of this by including a categorical moderator 413 ('ManipDirection'). This categorised treatments as restriction (where low-condition groups 414 were restricted relative to high condition/control groups), supplementation (where high 415 condition groups were enriched relative to low-condition/control groups), and dual (where 416 both the low-condition group was restricted and the high condition group was enriched from 417 standard conditions). To explore how the duration of diet treatments influenced the outcome, 418 a continuous moderator ('RelativeManipDuration.C') was defined as the time that the 419 treatment was applied as a proportion of the maximum lifespan of the species. Finally, the 420 influence of the source of the study subjects was tested using via a categorical moderator 421 ('WildLabRear', wild, laboratory, commercial or mixed).

422

423 III. Results

424 (1) Main effects models

Intercept-only models showed a significant positive effect for InRR, 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: InRR = 0.23 [0.14 – 0.32], phylogenetic method: InRR = 0.23 [0.09 – 0.38]; Table 1, Figure 1). For InCVR, the overall estimate was small, negative and the confidence intervals overlapped zero substantially (InCVR = -0.03 [-0.09 – 0.03]; Table 1, Figure 1). As phylogeny failed to resolve any heterogeneity in InCVR, the estimates from the phylogenetic and non-phylogenetic models were identical.

432

433 (2) Hypothesis testing models

434 The magnitude of the InRR was influenced by the experimental context, with the RiskContext 435 moderator explaining a large amount of heterogeneity among effect sizes ($R^2_{marginal} =$ 436 12.03%; Table 2). Although most context-specific confidence intervals overlapped with zero, 437 all the mean estimates were positive (Table 4). The highest estimates were found for 438 behaviours relating to feeding under predation (InRR = 0.75 [0.53 - 0.97]), feeding in a novel 439 environment (InRR = 0.36 [0.20 - 0.52]), and shoaling in a novel environment (InRR = 0.36 440 [0.06 – 0.67]; Table 4; Fig 2A). The risk context also explained a large amount of 441 heterogeneity in InCVR ($R^2_{marginal} = 10.22\%$; Table 3), and the confidence intervals of some 442 context-specific effects did not overlap with zero, including refuge use in a novel 443 environment (InCVR = 0.18 [0.04 - 0.31]), feeding in a novel environment (InCVR = -0.16 [-444 0.25 - -0.07]), and, dispersal/migration decisions (InCVR = -0.49 [-0.86 - -0.11]; Table 5; Fig 445 2B), showing a reduction in total variance in low-vs. high-condition treatments in those 446 specific risk contexts.

447

448 Sex appeared to have some effect on lnRR (Table 2), but there was no evidence for an 449 effect on lnCVR (Table 3). The lnRR estimates were positive but the confidence intervals 450 slightly overlapped with zero for both females (lnRR = 0.15 [-0.03 - 0.33]) and males (lnRR 451 =0.12 [-0.06 - 0.30]), while effects were strongest for mixed (lnRR = 0.34 [0.06 - 0.61]) and

452 unknown sex groups (InRR = 0.29 [0.14 – 0.44]; Fig 2C). Life-stage also influenced InRR 453 (Table 2), and less clearly also InCVR (although this model showed a particularly high $R^{2}_{marginal}$ = 16.64, Table 3). Life-stage specific estimates for InRR were lowest and 454 455 overlapping zero in adult treatments (InRR = 0.12 [-0.06 – 0.30]), and strongest for 456 treatments that spanned both the juvenile and the adult life stage (InRR = 0.45 [0.17 - 0.73]; 457 Table 4; Fig 2E). Life-stage effects on InCVR showed a negative estimate for juvenile treatments (InCVR = -0.08 [-0.16 - 0.00]), and a positive effect, i.e. an increase in 458 459 behavioural variance in low-condition treatments, when treatments spanned both the juvenile 460 and the adult life stage (InCVR = 0.18 [0.01 - 0.34]; Table 5; Fig 2F). Untransformed 461 maximum lifespan did not appear to influence lnRR (0.00 [-0.08 - 0.09]). However, log-462 transformed lifespan showed a positive InRR effect, with its confidence intervals only slightly 463 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 464 465 appeared to have a clear effect on InCVR, however, these moderators explained a 466 substantial amount of heterogeneity ($R^{2}_{marginal} = 13.81\%$, 13.14% respectively; Table 3, 5). 467

468 (3) Publication bias tests

469 Funnel plots showed some potential evidence of asymmetry (for plots and fail-safe N 470 calculations see Supporting Information S7). The estimated effect of *Precision* on InRR was 471 negative and the confidence intervals slightly overlapped with zero (-0.002 [-0.005 - 0.000]; 472 Table 2, 4), while $R^{2}_{marginal}$ was comparably high (7.81%; Table 2), showing some potential 473 evidence of small-study bias. There was also possible evidence of time-lag bias in published 474 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 475 476 relatively high (8.18%; Table 2). Last, effects calculated from papers where effect sizes could be partially (InRR = 0.26 [0.07 - 0.63]) or completely (InRR = 0.24 [0.09 - 0.40])477 calculated from the publicly available material were relatively large (Fig 3), whereas the 478 479 effect from papers where effect sizes could only be obtained through author correspondence

were small and the confidence intervals overlapped with zero (InRR = 0.10 [-0.16 - 0.35]), however, $R^2_{marginal}$ was zero for this moderator (Table 2). This difference suggests that nonreported results might be biased towards inconclusive (likely statistically non-significant) results.

484

485 (4) Exploratory models

486 There was limited evidence that either the type or direction of diet manipulation influenced 487 InRR with all diet types and directional treatments, respectively, showing positive mean estimates, and no heterogeneity explained by either of those moderators ($R^{2}_{marginal} = 0.00$; 488 489 Table 2, 4; Fig 4A, 4C). The effect of the duration of diet treatments on InRR was almost 490 zero too (Table 2, 4). There a small amount of heterogeneity explained by the rearing 491 environment of the experimental subjects ($R^{2}_{marginal} = 1.44\%$; Table 2, 4), with effect sizes 492 from laboratory reared animals being the smallest (InRR = 0.13 [-0.03 - 0.30]), and effect 493 sizes from wild reared animals being the largest (InRR = 0.32 [0.16 - 0.48]; Fig 4E). 494

Both the type and direction of diet manipulation did not appear to influence InCVR 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 InCVR, with less than 1% of heterogeneity explained by this moderator (Table 3, 5; Fig 4F).

501

502 IV. Discussion

503 Despite our expectations, we found a convincing directional effect on mean risk-taking 504 behaviour, where individuals subject to low condition dietary treatments are more likely to 505 show high-risk behaviour in a range of contexts involving predation and novelty. This 506 condition-dependency may be caused by increased risk aversion in higher-condition

507 individuals due to their greater reproductive expectations (an interpretation consistent with 508 the asset-protection principle applying to the context of nutritional condition and predation-509 novelty based risk), or by increased risk preference in low-condition animals due to their 510 elevated danger of starvation (a starvation avoidance mechanism; Luttbeg & Sih, 2010). 511 These adaptive interpretations contrast with a recent meta-analysis showing that riskier 512 behavioural types had higher survival in the wild (Moiron, Laskowski, & Niemelä, 2020). 513 which may highlight a distinction between behavioural variation due to personality trait 514 differences and due to state-dependent effects. Nonetheless, our result is consistent with the 515 idea of a trade-off between the potential benefits of high outcome-variance behaviours (e.g. 516 accessing resources) and the potential costs (e.g. predation or starvation), which animals 517 balance based on their current or past nutritional state (Ludwig & Rowe, 1990; Clark, 1994; 518 McNamara & Houston, 1996).

519

520 Although our overall effect was relatively strong, there was high heterogeneity in InRR effect 521 sizes with a large proportion (>20%) related to among-species differences. Variation among 522 species, however, was only minimally related to their shared ancestry, with phylogeny only 523 accounting for a very small proportion of heterogeneity (3%). It would be interesting to know 524 if condition-dependence of risk-taking behaviour also applies to humans (Wilson et al., 1994; 525 Gosling, 2008), but the large amount of context-specificity might suggest that the effect 526 might vary between contexts. The high heterogeneity among effect sizes is also evident from 527 the wide prediction intervals estimated, and the substantial heterogeneity among studies and 528 experiments. Since theory predicts that state-dependent effects on risk taking vary in 529 strength and direction with factors such as life history traits (Clark, 1994; McNamara & 530 Houston, 1996) and/or local environmental/ecological conditions (Luttbeg & Sih, 2010), such 531 a pattern of variation among species, studies and experiments was to be expected. Critically, 532 given the high heterogeneity, our overall effect does not preclude the opposite pattern being 533 applicable in certain systems. Also, our findings focus on nutritional state in contexts often 534 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).

538

539 The experimental context of risk-taking behaviour was the most explanatory of InRR 540 moderators, revealing that the effect of condition in certain contexts was clear and 541 particularly strong, such as those involving feeding. This is consistent with studies showing 542 that the choice of experiment used to measure risk taking is important to the outcome, and 543 that different risk-taking behaviours can show divergent patterns of individual-level variation 544 (e.g. Carter et al., 2012). The concept of a 'risky' behaviour can be applied to a broad range 545 of circumstances, as shown by the range of behavioural variables included here, and 'risk-546 taking' can refer to a suite of potentially independent behaviours. A risk context that was 547 particularly strongly affected was shoaling behaviour in a novel environment (and, with less 548 certainty, shoaling when exposed to a predator). Whether decisions to venture from a group 549 can be considered a risk-taking behaviour or boldness trait has been disputed, partly due to 550 overlap with sociability traits (Toms, Echevarria, & Jouandot, 2010), but our findings are 551 consistent with these decisions being related to risk taking as a trade-off between resource 552 acquisition and group safety. Contrastingly, the estimated effect was highly uncertain and 553 close to zero for refuge emergence into a novel environment, a commonly used variable to 554 measure bold-exploratory personalities. Studies have shown refuge emergence to be 555 unrelated to within-species variation in other risk-taking behaviours (e.g. startle responses in 556 Pomacentrus spp., Beckmann & Biro, 2013; or novel object tests in Chlamydogobius 557 eremius, Moran et al., 2016), such that the relationship between refuge emergence and risk 558 taking remains unclear.

559

560 Sex effects on InRR did not show evidence of male-female differences, with both male- and
561 female-specific effects being relatively small and similar to each other. It has been
562 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 .

592 Contrasting with overall mean effects, support for an overall effect of condition on 593 behavioural variation was limited, with only a small, slightly negative and rather uncertain 594 overall InCVR estimate. This contrasts with the expectation that poor condition may increase 595 phenotypic variability (e.g. by exposing cryptic genetic variation), but agrees with a recent 596 meta-analysis showing that environmental stress does not seem to influence variation in 597 behavioural traits across species (Sánchez-Tójar et al., 2019). Heterogeneity was generally 598 lower in InCVR models relative to InRR ones, which is likely because variance effect sizes 599 are generally associated with larger sampling variances (Sánchez-Tójar et al., 2019). 600 Variance meta-analyses are expected to be more data hungry, although this is unlikely to be 601 the cause of the overall weak InCVR effect found in our study given the large dataset used. 602

603 Variation in behaviour was sensitive to the experimental context of risk-taking behaviour, 604 with variation in both the strength and direction of context-specific effects. In particular, 605 variance in feeding behaviour within novel environments was far lower in low-condition 606 groups, providing some evidence that being highly motivated to feed in this context is an 607 optimum phenotype for individuals in poor energetic state. In contrast, variation in refuge use 608 in a novel environment was higher in low-condition groups, which may be evidence of the 609 opposite (complementary) pattern where high refuge use is a preferred strategy for high 610 condition individuals. Effects of life stage on behavioural variation are consistent with recent 611 empirical evidence suggesting that developmental diet is related to phenotypic plasticity and 612 personality development (see examples in Royauté & Dochtermann 2017; Kelleher et al. 613 2019). Buchanan, Grindstaff, & Pravosudov (2013) suggested that poor condition during 614 early life stages may reduce an individual's capacity to express behavioural plasticity. This is 615 potentially consistent with our finding of reduced behavioural variation in groups subject to 616 low-condition treatments as juveniles, while the effect in adults heavily overlapped with zero. 617 We also found that treatments that spanned juvenile and adult life stages (often longer term, 618 chronic diet restriction treatments) had a positive effect on behavioural variation. Similarly,

the duration of diet treatments had a positive effect on behavioural variation, consistent with
the proposition that extremely poor diet conditions can expose cryptic genetic and
phenotypic variation (Han & Dingemanse, 2017). Nonetheless, identifying mechanisms from
unpartitioned phenotypic variance remains challenging, as the proposed mechanisms for
effects on variability in risk-taking behaviour often apply specifically to among- or withinindividual 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 InCVR) 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.

652

653 V. Conclusions

- 654 (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
- 656 greater risks in ecological contexts relating to predation risk and novelty.
- 657 (II) While condition-dependency appears to have broad relevance across the animal
- 658 kingdom, the strength and certainty of this effect may be somewhat overstated due to
- 659 publication bias and large heterogeneity among effect sizes.
- 660 (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.
- 663 (IV) Overall, there appears to be complex and nuanced effects of diet and condition on
- 664 behavioural variance warranting further empirical study. Future research should focus on
- separating among- and within-individual variance effects of individual condition.

666

667 VI. Acknowledgements

- 668 We thank members of the UNSW I-DEEL: Inter-Disciplinary Ecology and Evolution Lab,
- 669 particularly Shinichi Nakagawa, Rose O'Dea and Losia Lagisz for sharing your meta-analytic
- 670 wisdom. Thank you Anna Antonatou and Jonathan Grone for assisting with data extraction.
- 671 We also thank Barney Luttbeg and an anonymous reviewer for their valuable comments on
- our manuscript. This project has received funding from the European Union's Horizon 2020
- 673 research and innovation programme under the Marie Sklodowska-Curie grant agreement No

- 674 836937. The Centre for Ocean Life is a VKR center of excellence supported by the Villum
- 675 foundation. This research was funded by the German Research Foundation (DFG) as part of
- 676 the SFB TRR 212 (NC³) Project numbers 316099922 and 396782608.
- 677

678 VII. Authorship

- 679 NPM: Conceptualization, Investigation, Methodology, Data curation, Formal analysis, Project
- 680 administration, Software, Validation, Visualization, Writing original draft, Writing review &
- editing. AST: Conceptualization, Investigation, Methodology, Data collection, Software,
- 682 Validation, Writing review & editing. HS: Conceptualization, Funding acquisition, Writing -
- 683 review & editing. KR: Conceptualization, Funding acquisition, Supervision, Writing review &
- 684 editing.
- 685

686 VIII. Data Accessibility

- 687 All data and code used (including data processing, preparation, analysis and presentation)
- 688 are available at the Open Science Framework (<u>https://osf.io/3tphi/</u>, doi:
- 689 10.17605/OSF.IO/3TPHJ).
- 690

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Tables

Table 1: Main effects models estimates, with random effect specific heterogeneity estimates

847 (l^2) expressed as percentages, and Q-test for absolute heterogeneity among effect sizes (Q).

848 Square brackets represent 95% confidence intervals. Round brackets represent 95%

849 prediction intervals, i.e. the range in which 95% of future or unknown effects are likely to fall.

850 Positive log response ratio (InRR) and log coefficient of variation ratio (InCVR) effects

851 represent higher either risk taking or variance in risk taking in low-condition animals,

- 852 respectively.

| Effect size | k | Mean effect | f ^e _{Experiment ID} (%) | f ^e _{Study ID} (%) | P ^e _{Species ID} (%) | ۴ _{Phylogeny} (%) | ل ^و _{Effect ID} (%) | f ^e _{Total} (%) | Q |
|----------------------|------|--|--|---|---|-------------------------------|--|--|------------------------|
| InRR (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 |
| InRR (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 |
| InCVR (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 |
| InCVR (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 (<i>rr.Full.h2</i>) | InRR | 1297 | RiskContext | 14657.13 p < 0.0001 | 79.42 *** p < 0.0001 | 12.03 |
| Hyp. 3. Sex difference in risk taking (<i>rr.Full.h3</i>) | InRR | 1297 | Sex | 24006.28 p < 0.0001 | 15.92 ** p = 0.0031 | 0.53 |
| Hyp. 4. Effects across life stages (<i>rr.Full.h4</i>) | InRR | 1214 | ManipLifeStage + RelativeTimeFromTreatment.C | 16753.8 p < 0.0001 | 21.2 *** p = 0.0007 | 0.00 |
| Hyp. 5(i). Life-history effects (<i>rr.Full.h5.i</i>) | InRR | 1214 | MaxLongevity.C | 23933.71 p < 0.0001 | 0.00 p = 0.9651 | 0.00 |
| Hyp. 5(ii). Life-history effects (<i>rr.Full.h5.ii</i>) | InRR | 1214 | InMaxLongevity.C | 22654.52 p < 0.0001 | 3.46 p = 0.0628 | 0.00 |
| Publication bias 1 (<i>rr.Full.pub1</i>) | InRR | 908 | Precision | 13245.28 p < 0.0001 | 2.81 p = 0.0938 | 7.81 |
| Publication bias 2 (<i>rr.Full.pub2</i>) | InRR | 908 | Year.C | 21211.43 p < 0.0001 | 0.97 p = 0.3254 | 8.18 |
| Publication bias 3 (<i>rr.Full.pub1</i>) | InRR | 1297 | EffectSizesFromPublication | 23269.07 p < 0.0001 | 11.43 * p = 0.0096 | 0.00 |
| Exp a. Effect of manipulation type (<i>rr.Full.exp.a</i>) | InRR | 1297 | ManipType | 22616.48 p < 0.0001 | 8.24 p = 0.0833 | 0.00 |
| Exp b. Effect of manipulation direction (<i>rr.Full.exp.b</i>) | InRR | 1297 | ManipDirection | 20399.67 p < 0.0001 | 10.26 * p = 0.0165 | 0.00 |
| Exp c. Effect of manipulation duration (<i>rr.Full.exp.c</i>) | InRR | 1214 | RelativeManipDuration.C | 24024.39 p < 0.0001 | 0.06 p = 0.8007 | 0.00 |
| Exp d. Effect of rearing environment (<i>rr.Full.exp.d</i>) | InRR | 1297 | WildLabRear | 22799.97 p < 0.0001 | 16.57 ** p = 0.0023 | 1.44 |

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Table 3: Hypothesis testing, publication bias and exploratory moderators for log coefficient of variation ratio (InCVR) 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 (<i>cvr.Full.h2</i>) | InCVR | 1235 | RiskContext | 2450.98 p < 0.0001 | 38.4 *** p = 0.0002 | 10.22 |
| Hyp. 3. Sex difference in risk taking (<i>cvr.Full.h3</i>) | InCVR | 1235 | Sex | 2520.5 p < 0.0001 | 5.9 p = 0.2066 | 2.44 |
| Hyp. 4. Effects across life stages (<i>cvr.Full.h4</i>) | InCVR | 1153 | ManipLifeStage + RelativeTimeFromTreatment.C | 2158.2 p < 0.0001 | 9.5 p = 0.0908 | 16.64 |
| Hyp. 5(1). Life-history effects (<i>cvr.Full.h5.i</i>) | InCVR | 1153 | MaxLongevity.C, | 2185.53 p < 0.0001 | 1.41 p = 0.2348 | 13.81 |
| Hyp. 5(ii). Life-history effects (<i>cvr.Full.h5.ii</i>) | InCVR | 1153 | InMaxLongevity.C | 2187.91 p < 0.0001 | 0.34 p = 0.5615 | 13.14 |
| Exp a. Effect of manipulation type (<i>cvr.Full.exp.a</i>) | InCVR | 1235 | ManipType | 2535.9 p < 0.0001 | 3.1 p = 0.5406 | 0.00 |
| Exp b. Effect of manipulation direction (<i>cvr.Full.exp.b</i>) | InCVR | 1235 | ManipDirection | 2541.4 p < 0.0001 | 2.23 p = 0.5256 | 0.00 |
| Exp c. Effect of manipulation duration (<i>cvr.Full.exp.c</i>) | InCVR | 1153 | RelativeManipDuration.C | 2182.57 p < 0.0001 | 4.59 * p = 0.0322 | 16.17 |
| Exp d. Effect of rearing environment (<i>cvr.Full.exp.d</i>) | InCVR | 1235 | WildLabRear | 2514.93 p < 0.0001 | 4.6 p = 0.3312 | 0.86 |

Table 4: Parameter estimates for log response ratio (InRR) 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 | RiskContext | novelenvironment_activity | 248 | 46 | 0.09 [-0.06, 0.25] |
| risk (<i>rr.Full.h2</i>) | | 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 | Sex | female | 421 | 39 | 0.15 [-0.03, 0.33] |
| taking (<i>rr.Full.h3</i>) | | 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 | ManipLifeStage | adult | 423 | 48 | 0.12 [-0.06, 0.30] |
| stages (rr.Full.h4) | | 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 | MaxLongevity.C | intercept | - | - | 0.26 [0.15, 0.36] |
| (rr.Full.h5.i) | 0 | (covariate) | - | - | 0.00 [-0.08, 0.09] |
| Hyp. 5(ii). Life-history effects | InMaxLongevity.C | intercept | - | - | 0.22 [0.02, 0.43] |
| (rr.Full.h5.ii) | | (covariate) | - | - | 0.15 [-0.01, 0.30] |
| Publication bias 1 | Precision | intercept | - | - | 0.28 [0.08, 0.49] |
| (rr.Full.pub1) | | (covariate) | - | - | 0.00 [-0.01, 0.00] |
| Publication bias 2 | Year.C | intercept | - | - | 0.26 [0.07. 0.44] |
| (rr.Full.pub2) | | (covariate) | - | - | -0.05 [-0.14, 0.05] |
| Publication bias 3 | EffectSizesEromPublication | (001411410) 00 | 130 | 13 | 0 10 [-0 16 0 35] |
| (rr. Full. pub 1) | | nartial | 360 | 31 | 0.26 [0.07, 0.45] |
| (apaz .) | | partial | 907 | 27 | 0.20 [0.07, 0.45] |
| Fun a Effect of maximulation | ManinTruna | yes | 007 | 02 | 0.24 [0.09, 0.40] |
| Exp a. Effect of manipulation | Manip i ype | combined | 24 | 4 | 0.27 [-0.08, 0.62] |
| type (n.r un.exp.a) | | quality | 248 | 18 | 0.35 [0.07, 0.63] |
| | | quantity | 390 | 50 | |
| | Marcia Dina atian | Starvation | 035 | 59 | 0.19[-0.04, 0.41] |
| direction (rr Full over b) | ManipDirection | duai | 60 | 1 | |
| unection (<i>n.r-un.exp.b</i>) | | restrict | 67 | 0 | |
| | | supplement | 07 | 9 | 0.20 [-0.04, 0.44] |
| exp c. Effect of manipulation | RelativelylanipDuration.C | Intercept | - | - | |
| | | (covariate) | - | - | -0.01 [-0.07, 0.05] |
| Exp a. Effect of rearing | wiiaLabRear | commercial | 139 | 12 | 0.25 [-0.02, 0.52] |
| environment (<i>m.r-uii.exp.a</i>) | | lab | /11 | 58 | 0.13 [-0.03, 0.3] |
| | | mixed | 15 | 1 | 0.21 [-0.5, 0.93] |
| | | wild | 432 | 5/ | 0.32 [0.16, 0.48] |

Table 5: Parameter estimates for log coefficient of variation ratio (InCVR) hypothesis testing,903and exploratory models, with 95% confidence intervals. k shows the number of effect sizes,904and n_{study} shows the number of studies. Bold estimates correspond to confidence intervals905that do not overlap zero. Note that models with categorical moderators were run as no-

906 intercept models for ease of interpretation.

| Hyp. 2. Context-dependency of risk (cvr.Full.h2) RiskContext novelenvironment_activity 248 46 0.02 [-0.06, 0 novelenvironment_exploration 153 33 -0.05 [-0.15, C novelenvironment_feeding 312 34 -0.16 [-0.25, -1 novelenvironment_refugeemergence 39 7 0.04 [-0.25, -1 novelenvironment_refugeemergence 39 7 0.04 [-0.25, -1 novelenvironment_refugeemergence 75 16 0.18 [0.04, 0. novelenvironment_refugeese 75 16 0.18 [0.04, 0. novelenvironment_shaaling 29 5 0.01 [-0.25, 0. novelenvironment_shaaling 20 4 0.01 [-0.24, 0. predation_response 167 33 0.02 [-0.06, 0. predation_response 167 33 0.02 [-0.80, 0. predation_response 167 33 0.02 [-0.26, 0. dispersalmigration 13 5 -0.49 [-0.86, -0. uhraking (cvr.Full.h3) male 276 37 0.03 [-0.08, 0. mixed <t< th=""><th>Hypothesis (model)</th><th>Moderator(s)</th><th>Level</th><th>k</th><th>nstudy</th><th>Estimate</th></t<> | Hypothesis (model) | Moderator(s) | Level | k | n study | Estimate |
|--|----------------------------------|-----------------------------|----------------------------------|------|----------------|----------------------|
| of risk (cvr.Full.h2) novelenvironment_exploration 153 33 -0.05 [-0.15, C novelenvironment_feeding 312 34 -0.16 [-0.25, C novelenvironment_feeding 312 34 -0.16 [-0.25, C novelenvironment_refugeemergence 39 7 0.04 [-0.18, O novelenvironment_refugeemergence 39 7 0.04 [-0.28, O novelenvironment_refugeemergence 39 5 0.01 [-0.25, O novelenvironment_refugeemergence 39 5 0.01 [-0.24, O novelenvironment_response 161 3 -0.01 [-0.24, O predation_feeding 61 13 -0.01 [-0.24, O predation_feeding 13 -0.01 [-0.24, O difference in risk Sex female 401 38 0.05 [-0.05, O taking (cvr.Full.h3) male 276 37 0.03 [-0.28, O mixed 117 13 -0.09 [-0.28, O -0.08 [-0.17, O taking (cvr.Full.h3) male 276 37 0.03 [-0.28, O unknown | Hyp. 2. Context-dependency | RiskContext | novelenvironment_activity | 248 | 46 | 0.02 [-0.06, 0.11] |
| novelenvironment_feeding 312 34 -0.16 [-0.25, -0.09 [-0.35, C] novelenvironment_lightdarktest 24 5 -0.09 [-0.35, C] novelenvironment_refugemergence 39 7 16 0.18 [0.04, O] novelenvironment_refugemergence 39 5 0.01 [-0.25, O] 0.04 [-0.18, O] novelenvironment_refugemergence 39 5 0.01 [-0.25, O] 0.08 [-0.24, C] novelenvironment_shoaling 29 5 0.01 [-0.24, O] 0.02 [-0.08, O] predation_response 167 33 0.02 [-0.08, O] 0.02 [-0.08, O] predation_response 167 33 0.05 [-0.05, O] 0 dispersalmigration 13 5 -0.49 [-0.86, -G] 0 taking (cvr.Full.h3) male 276 37 0.03 [-0.08, O] mixed 117 13 -0.09 [-0.28, O] 0 unknown 441 56 -0.08 [-0.17, O] 0 stages (cvr.Full.h3) male 276 33 0.09 [-0.28, O] unknown <t< td=""><td>of risk (<i>cvr.Full.h2</i>)</td><td></td><td>novelenvironment_exploration</td><td>153</td><td>33</td><td>-0.05 [-0.15, 0.05]</td></t<> | of risk (<i>cvr.Full.h2</i>) | | novelenvironment_exploration | 153 | 33 | -0.05 [-0.15, 0.05] |
| novelenvironment_lightdarktest 24 5 -0.09 [-0.35, C novelenvironment_refugeemergence 39 7 0.04 [-0.18, 0 novelenvironment_refugeemergence 39 7 0.04 [-0.18, 0 novelenvironment_shoaling 29 5 0.01 [-0.25, 0 novelenvironment_shoaling 29 5 0.01 [-0.24, C predation_response 88 10 -0.08 [-0.24, C predation_shoaling 20 4 0.01 [-0.24, C predation_shoaling 20 4 0.01 [-0.24, C predation_shoaling 20 4 0.01 [-0.24, C dispersalmigration 13 5 -0.49 [-0.86, -C other 6 3 0.59 [0.16, 1. Hyp. 3. Sex difference in risk Sex female 401 38 0.05 [-0.05, 0 taking (cvr.Full.h3) male 276 37 0.03 [-0.08, 0 mixed 117 13 -0.09 [-0.28, 0 -0.08 [-0.17, 0 stages (cvr.Full.h3) male 276 37 0.03 | | | novelenvironment_feeding | 312 | 34 | -0.16 [-0.25, -0.07] |
| novelenvironment_refugeemergence 39 7 0.04 [-0.18, 0 novelenvironment_refugeuse 75 16 0.18 [0.04, 0. novelenvironment_shoaling 29 5 0.01 [-0.25, 0. novelobject_response 88 10 -0.08 [-0.24, 0. predation_response 13 -0.01 [-0.21, 0. predation_response 167 33 0.02 [-0.08, 0. predation_response 167 33 0.02 [-0.80, 0. other 6 3 0.59 [0.16, 1. Hyp. 3. Sex difference in risk taking (cvr.Full.h3) Sex female 401 38 0.05 [-0.05, 0. mixed 117 13 -0.09 [-0.28, 0. -0.08 [-0.17, 0. -0.08 [-0.17, 0. Hyp. 4. Effects across life ManipLifeStage adult 402 45 -0.08 [-0.17, 0. stages (cvr.Full.h4) both 116 7 0.18 [0.01, 0. juvenile 578 63 -0.08 [-0.16, 0. unknown/mixed 89 11 -0.02 [-0.22, 0. -0.02 [-0.21, 0. -0.02 [-0.21, 0. | | | novelenvironment_lightdarktest | 24 | 5 | -0.09 [-0.35, 0.16] |
| novelenvironment_refugeuse novelenvironment_shoaling 75 16 0.18 [0.04, 0. novelenvironment_shoaling 29 5 0.01 [-0.25, 0. novelobject_response 88 10 -0.08 [-0.24, 0. predation_feeding 61 13 -0.01 [-0.21, 0. predation_response 167 33 0.02 [-0.08, 0. predation_shoaling 20 4 0.01 [-0.24, 0. dispersalmigration 13 5 -0.49 [-0.86, -0. other 6 3 0.59 [0.16, 1. Hyp. 3. Sex difference in risk taking (cvr.Full.h3) Sex female 401 38 0.05 [-0.05, 0. mixed 117 13 -0.09 [-0.28, 0. 0.01 [-0.21, 0. 0.01 [-0.21, 0. make 276 37 0.03 [-0.08, 0. 0.01 [-0.21, 0. uhknown 441 56 -0.08 [-0.17, 0. 0.01 [-0.28, 0. uhknown 441 56 -0.08 [-0.17, 0. 0. stages (cvr.Full.h4) bath 116 7 0.18 [0.01, 0. | | | novelenvironment_refugeemergence | e 39 | 7 | 0.04 [-0.18, 0.25] |
| novelenvironment_shoaling 29 5 0.01 [-0.25, 0 novelobject_response 88 10 -0.08 [-0.24, C predation_feeding 61 13 -0.01 [-0.21, C predation_response 167 33 0.02 [-0.08, O predation_shoaling 20 4 0.01 [-0.24, O dispersalmigration 13 5 -0.49 [-0.86, -C other 6 3 0.59 [0.16, 1. Hyp. 3. Sex difference in risk Sex female 401 38 0.05 [-0.08, O mixed 117 13 -0.09 [-0.28, O 0.03 [-0.08, O mixed Hyp. 4. Effects across life ManipLifeStage adult 402 45 0.00 [-0.10, O stages (cvr.Full.h4) 578 63 -0.08 [-0.21, O juvenile 578 63 -0.02 [-0.22, O Hyp. 5(i). Life-history effects MaxLongevity.C intercept - - 0.02 [-0.22, O (cvr.Full.h5.i) (covariate) - - - - 0.03 [-0.09, O | | | novelenvironment_refugeuse | 75 | 16 | 0.18 [0.04, 0.31] |
| novelobject_response predation_feeding 88 10 -0.08 [-0.24, 0] predation_feeding 61 13 -0.01 [-0.21, 0] predation_response 167 33 0.02 [-0.08, 0] predation_shoaling 20 4 0.01 [-0.24, 0] dispersalmigration 13 5 -0.49 [-0.28, 0] dispersalmigration 13 5 -0.08 [-0.17, 0] Hyp. 3. Sex difference in risk Sex female 401 38 0.05 [-0.08, 0] mixed 117 13 -0.09 [-0.28, 0] [-0.17, 0] Hyp. 4. Effects across life ManipLifeStage adult 402 45 0.00 [-0.10, 0] stages (cvr.Full.h4) Bioth 116 7 0.18 <td< td=""><td></td><td></td><td>novelenvironment_shoaling</td><td>29</td><td>5</td><td>0.01 [-0.25, 0.26]</td></td<> | | | novelenvironment_shoaling | 29 | 5 | 0.01 [-0.25, 0.26] |
| predation_feeding predation_response 61 13 -0.01 [-0.21, 0] (0.08, 0] predation_shoaling dispersalmigration 20 4 0.01 [-0.24, 0] dispersalmigration 13 5 -0.49 [-0.86, 0] dispersalmigration 13 5 -0.49 [-0.86, 0] taking (cvr.Full.h3) male 276 37 0.03 [-0.08, 0] mixed 117 13 -0.09 [-0.28, 0] unknown 441 56 -0.08 [-0.17, 0] Hyp. 4. Effects across life ManipLifeStage adult 402 45 0.00 [-0.10, 0] stages (cvr.Full.h4) both 116 7 0.18 [0.01, 0] juvenile 578 63 -0.08 [-0.10, 0] juvenile 578 63 -0.08 [-0.10, 0] [-0.20, 0] -0.03 [-0.09, 0] (cvr.Full.h5.i) (covariate) - - -0.03 [-0.09, 0] (cvr.Full.h5.i) - -0.03 [-0.09, 0] - -0.03 <td></td> <td></td> <td>novelobject_response</td> <td>88</td> <td>10</td> <td>-0.08 [-0.24, 0.08]</td> | | | novelobject_response | 88 | 10 | -0.08 [-0.24, 0.08] |
| predation_response 167 33 0.02 [-0.08, 0 predation_shoaling 20 4 0.01 [-0.24, 0 dispersalmigration 13 5 -0.49 [-0.86, -1 other 6 3 0.59 [0.16, 1. Hyp. 3. Sex difference in risk Sex female 401 38 0.05 [-0.05, 0 taking (cvr.Full.h3) male 276 37 0.03 [-0.08, 0 mixed 117 13 -0.09 [-0.28, 0 unknown 441 56 -0.08 [-0.17, 0 Hyp. 4. Effects across life ManipLifeStage adult 402 45 0.00 [-0.10, 0 stages (cvr.Full.h4) both 116 7 0.18 [0.01, 0 juvenile 578 63 -0.08 [-0.17, 0 Hyp. 5(i). Life-history effects MaxLongevity.C intercept - 0.02 [-0.20, 0 - Hyp. 5(i). Life-history effects MaxLongevity.C intercept - - -0.03 [-0.09, 0 (cvr.Full.h5.i) (covariate) - - - -< | | | predation_feeding | 61 | 13 | -0.01 [-0.21, 0.18] |
| predation_shoaling dispersalmigration 20 4 0.01 [-0.24, 0 dispersalmigration 13 5 -0.49 [-0.86, -1 other 6 3 0.59 [0.16, 1. Hyp. 3. Sex difference in risk taking (cvr.Full.h3) Sex female 401 38 0.05 [-0.05, 0 male 276 37 0.03 [-0.08, 0 0.01 [-0.24, 0 0.03 [-0.08, 0 mixed 117 13 -0.09 [-0.28, 0 0.05 [-0.05, 0 0.08 [-0.17, 0 Hyp. 4. Effects across life ManipLifeStage adult 402 45 0.00 [-0.10, 0 stages (cvr.Full.h4) bath 116 7 0.18 [0.01, 0 0 juvenile 578 63 -0.08 [-0.16, 0 0 0.02 [-0.21, 0 elative TimeFromTreatment.C (covariate) - - 0.02 [-0.20, 0 Hyp. 5(i). Life-history effects MaxLongevity.C intercept - - -0.03 [-0.09, 0 (cvr.Full.h5.i) (covariate) - - - - - - - | | | predation_response | 167 | 33 | 0.02 [-0.08, 0.13] |
| dispersalmigration 13 5 -0.49 [-0.86, -0.60, -0.50] Hyp. 3. Sex difference in risk Sex female 401 38 0.05 [-0.05, 0] taking (cvr.Full.h3) male 276 37 0.03 [-0.08, 0] mixed 117 13 -0.09 [-0.28, 0] unknown 441 56 -0.08 [-0.17, 0] Hyp. 4. Effects across life ManipLifeStage adult 402 45 0.00 [-0.10, 0] stages (cvr.Full.h4) both 116 7 0.18 [0.01, 0] juvenile 578 63 -0.08 [-0.17, 0] Hyp. 5(i). Life-history effects MaxLongevity.C intercept - - 0.02 [-0.02, 0] 0] Hyp. 5(ii). Life-history effects InMaxLongevity.C intercept - - - - 0.03 [-0.09, 0] 0 (cvr.Full.h5.i) (covariate) - - - - - - - - - - 0.03 [-0.09, 0] 0 (cvr.Full.h5.i) (cvr.Full.h5.ii) <t< td=""><td></td><td></td><td>predation_shoaling</td><td>20</td><td>4</td><td>0.01 [-0.24, 0.26]</td></t<> | | | predation_shoaling | 20 | 4 | 0.01 [-0.24, 0.26] |
| other 6 3 0.59 [0.16, 1. Hyp. 3. Sex difference in risk taking (cvr.Full.h3) Sex female 401 38 0.05 [-0.05, 0] male 276 37 0.03 [-0.08, 0] mixed 117 13 -0.09 [-0.28, 0] mixed 117 13 -0.09 [-0.28, 0] unknown 441 56 -0.08 [-0.17, 0] Hyp. 4. Effects across life ManipLifeStage adult 402 45 0.00 [-0.10, 0] stages (cvr.Full.h4) both 116 7 0.18 [0.01, 0. juvenile 578 63 -0.08 [-0.17, 0] unknown/mixed 89 11 -0.02 [-0.21, 0] unknown/mixed 89 11 -0.02 [-0.22, 0] Hyp. 5(i). Life-history effects MaxLongevity.C intercept - - 0.03 [-0.09, 0] (cvr.Full.h5.i) (cvr.Full.h5.ii) (covariate) - - -0.03 [-0.09, 0] - Hyp. 5(ii). Life-history effects InMaxLongevity.C intercept - - - | | | dispersalmigration | 13 | 5 | -0.49 [-0.86, -0.11] |
| Hyp. 3. Sex difference in risk taking (cvr.Full.h3) Sex female 401 38 0.05 [-0.05, 0] male 276 37 0.03 [-0.08, 0] mixed 117 13 -0.09 [-0.28, 0] mixed 117 13 -0.09 [-0.28, 0] unknown 441 56 -0.08 [-0.17, 0] Hyp. 4. Effects across life ManipLifeStage adult 402 45 0.00 [-0.10, 0] stages (cvr.Full.h4) both 116 7 0.18 [0.01, 0. juvenile 578 63 -0.08 [-0.16, 0] unknown/mixed 89 11 -0.02 [-0.21, 0] elativeTimeFromTreatment.C (covariate) - - 0.03 [-0.09, 0] (cvr.Full.h5.i) (covariate) - - -0.03 [-0.09, 0] - Hyp. 5(ii). Life-history effects InMaxLongevity.C intercept - - - - (cvr.Full.h5.i) (covariate) - - - - - - - - - - - <td></td> <td></td> <td>other</td> <td>6</td> <td>3</td> <td>0.59 [0.16, 1.02]</td> | | | other | 6 | 3 | 0.59 [0.16, 1.02] |
| taking (cvr.Full.h3) male 276 37 0.03 [-0.08, 0 mixed 117 13 -0.09 [-0.28, 0 unknown 441 56 -0.08 [-0.17, 0 Hyp. 4. Effects across life ManipLifeStage adult 402 45 0.00 [-0.10, 0 stages (cvr.Full.h4) both 116 7 0.18 [0.01, 0. juvenile 578 63 -0.08 [-0.17, 0 gunknown/mixed 89 11 -0.02 [-0.20, 0 Hyp. 5(i). Life-history effects MaxLongevity.C intercept - - 0.03 [-0.09, 0 (cvr.Full.h5.i) (covariate) - - -0.03 [-0.09, 0 Hyp. 5(ii). Life-history effects InMaxLongevity.C intercept - - -0.03 [-0.09, 0 (cvr.Full.h5.i) (covariate) - - -0.03 [-0.09, 0 - Hyp. 5(ii). Life-history effects InMaxLongevity.C intercept - - - (cvr.Full.h5.ii) (covariate) - - - - - - - - - 0.02 [-0.09, 0 | Hyp. 3. Sex difference in risk | Sex | female | 401 | 38 | 0.05 [-0.05, 0.15] |
| mixed 117 13 -0.09 [-0.28, 0] unknown 441 56 -0.08 [-0.17, 0] Hyp. 4. Effects across life ManipLifeStage adult 402 45 0.00 [-0.10, 0] stages (cvr.Full.h4) both 116 7 0.18 [0.01, 0. juvenile 578 63 -0.08 [-0.17, 0] unknown/mixed 89 11 -0.02 [-0.10, 0] kelativeTimeFromTreatment.C (covariate) - - 0.02 [-0.21, 0] (cvr.Full.h5.i) MaxLongevity.C intercept - - 0.03 [-0.09, 0] Hyp. 5(ii). Life-history effects InMaxLongevity.C intercept - - -0.03 [-0.09, 0] (cvr.Full.h5.i) (covariate) - - -0.03 [-0.09, 0] Hyp. 5(ii). Life-history effects InMaxLongevity.C intercept - - - - - - - - - - 0.02 [-0.09, 0] Hyp. 5(ii). Life-history effects InMaxLongevity.C intercept - - | taking (<i>cvr.Full.h3</i>) | | male | 276 | 37 | 0.03 [-0.08, 0.14] |
| unknown 441 56 -0.08 [-0.17, 0] Hyp. 4. Effects across life ManipLifeStage adult 402 45 0.00 [-0.10, 0] stages (cvr.Full.h4) both 116 7 0.18 [0.01, 0] juvenile 578 63 -0.08 [-0.17, 0] ktop both 116 7 0.18 [0.01, 0] juvenile 578 63 -0.08 [-0.16, 0] unknown/mixed 89 11 -0.02 [-0.21, 0] RelativeTimeFromTreatment.C (covariate) - - 0.02 [-0.02, 0] Hyp. 5(i). Life-history effects MaxLongevity.C intercept - - -0.03 [-0.09, 0] (cvr.Full.h5.i) (covariate) - - -0.03 [-0.09, 0] - Hyp. 5(ii). Life-history effects InMaxLongevity.C intercept - - -0.03 [-0.09, 0] - (cvr.Full.h5.ii) (covariate) - - -0.02 [-0.09, 0] - - -0.02 [-0.09, 0] - Exp a. Effect of manipulation ManipType <td></td> <td></td> <td>mixed</td> <td>117</td> <td>13</td> <td>-0.09 [-0.28, 0.09]</td> | | | mixed | 117 | 13 | -0.09 [-0.28, 0.09] |
| Hyp. 4. Effects across life ManipLifeStage adult 402 45 0.00 [-0.10, 0 stages (cvr.Full.h4) both 116 7 0.18 [0.01, 0. juvenile 578 63 -0.08 [-0.16, 0 unknown/mixed 89 11 -0.02 [-0.21, 0 RelativeTimeFromTreatment.C (covariate) - - 0.02 [-0.02, 0 Hyp. 5(i). Life-history effects MaxLongevity.C intercept - - -0.03 [-0.09, 0 (cvr.Full.h5.i) (covariate) - - -0.03 [-0.09, 0 - Hyp. 5(ii). Life-history effects InMaxLongevity.C intercept - - -0.03 [-0.09, 0 (cvr.Full.h5.i) (covariate) - - -0.03 [-0.09, 0 - (cvr.Full.h5.ii) (covariate) - - -0.02 [-0.09, 0 Exp a. Effect of manipulation ManipType combined 24 4 0.07 [-0.21, 0 upantity 246 18 0.05 [-0.09, 0 - - - - - < | | | unknown | 441 | 56 | -0.08 [-0.17, 0.00] |
| stages (cvr.Full.h4) both 116 7 0.18 [0.01, 0. juvenile 578 63 -0.08 [-0.16, 0] unknown/mixed 89 11 -0.02 [-0.21, 0] RelativeTimeFromTreatment.C (covariate) - - 0.02 [-0.02, 0] Hyp. 5(i). Life-history effects MaxLongevity.C intercept - - -0.03 [-0.09, 0] (cvr.Full.h5.i) (covariate) - - -0.03 [-0.09, 0] Hyp. 5(ii). Life-history effects InMaxLongevity.C intercept - - -0.03 [-0.09, 0] (cvr.Full.h5.i) (covariate) - - -0.03 [-0.09, 0] - - -0.03 [-0.09, 0] - [cvr.Full.h5.ii] (covariate) - - - - -0.02 [-0.09, 0] - Exp a. Effect of manipulation ManipType combined 24 4 0.07 [-0.21, 0] - upper (cvr.Full.exp.a) quality 246 18 0.05 [-0.09, 0] - | Hyp. 4. Effects across life | ManipLifeStage | adult | 402 | 45 | 0.00 [-0.10, 0.09] |
| juvenile 578 63 -0.08 [-0.16, 0] unknown/mixed 89 11 -0.02 [-0.21, 0] RelativeTimeFromTreatment.C (covariate) - - 0.02 [-0.02, 0] Hyp. 5(i). Life-history effects MaxLongevity.C intercept - - -0.03 [-0.09, 0] (cvr.Full.h5.i) (covariate) - - -0.03 [-0.09, 0] Hyp. 5(ii). Life-history effects InMaxLongevity.C intercept - - -0.03 [-0.09, 0] (cvr.Full.h5.i) (covariate) - - -0.03 [-0.09, 0] (cvr.Full.h5.ii) (covariate) - - -0.03 [-0.09, 0] Exp a. Effect of manipulation ManipType combined 24 4 0.07 [-0.21, 0] uppe (cvr.Full.exp.a) quality 246 18 0.05 [-0.09, 0] - | stages (cvr.Full.h4) | | both | 116 | 7 | 0.18 [0.01, 0.34] |
| unknown/mixed 89 11 -0.02 [-0.21, 0] RelativeTimeFromTreatment.C (covariate) - - 0.02 [-0.02, 0] Hyp. 5(i). Life-history effects MaxLongevity.C intercept - - - - - - - 0.02 [-0.02, 0] - Hyp. 5(i). Life-history effects MaxLongevity.C intercept - 0.03 [-0.09, 0] - | | | juvenile | 578 | 63 | -0.08 [-0.16, 0.00] |
| RelativeTimeFromTreatment.C (covariate) - - 0.02 [-0.02, 0] Hyp. 5(i). Life-history effects MaxLongevity.C intercept - - -0.03 [-0.09, 0] (cvr.Full.h5.i) (covariate) - - -0.03 [-0.09, 0] Hyp. 5(ii). Life-history effects InMaxLongevity.C intercept - - -0.03 [-0.09, 0] (cvr.Full.h5.ii) (covariate) - - -0.03 [-0.09, 0] Exp a. Effect of manipulation ManipType combined 24 4 0.07 [-0.21, 0] type (cvr.Full.exp.a) quality 246 18 0.05 [-0.09, 0] 0 | | | unknown/mixed | 89 | 11 | -0.02 [-0.21, 0.16] |
| Hyp. 5(i). Life-history effects (cvr.Full.h5.i) MaxLongevity.C intercept (covariate) - - - - - - - - 0.03 [-0.09, 0] 0.03 [-0.09, 0] 0.03 [-0.09, 0] 0.03 [-0.09, 0] 0.03 [-0.09, 0] 0.03 [-0.09, 0] 0.03 [-0.09, 0] 0.03 [-0.09, 0] 0.03 [-0.09, 0] 0.03 [-0.09, 0] 0.03 [-0.09, 0] 0.03 [-0.09, 0] 0.02 [-0.09, 0] 0.02 [-0.09, 0] 0.02 [-0.09, 0] 0.07 [-0.21, 0] 0.07 [-0.21, 0] 0.05 [-0.09, 0] 0.05 [-0.09, 0] 0.05 [-0.09, 0] 0.05 [-0.09, 0] 0.05 [-0.09, 0] 0.05 [-0.09, 0] 0.07 [-0.21, 0] 0.05 [-0.09, 0] 0.05 [-0.09, 0] 0.07 [-0.21, 0] 0.05 [-0.09, 0] <td></td> <td>RelativeTimeFromTreatment.C</td> <td>(covariate)</td> <td>-</td> <td>-</td> <td>0.02 [-0.02, 0.05]</td> | | RelativeTimeFromTreatment.C | (covariate) | - | - | 0.02 [-0.02, 0.05] |
| (cvr.Full.h5.i) (covariate) - - - - - - - - - - 0.03 [-0.08, 0] - - - - - - 0.03 [-0.08, 0] - - - - - 0.03 [-0.08, 0] - - - - 0.03 [-0.09, 0] - - - - - 0.03 [-0.09, 0] - - - - - - - 0.03 [-0.09, 0] - 0.02 [-0.09, 0] - | Hyp. 5(i). Life-history effects | MaxLongevity.C | intercept | - | - | -0.03 [-0.09, 0.03] |
| Hyp. 5(ii). Life-history effects (cvr.Full.h5.ii) InMaxLongevity.C intercept (covariate) - - - - - - - 0.03 [-0.09, 0] 0.07 [-0.21, 0] Exp a. Effect of manipulation type (cvr.Full.exp.a) ManipType combined 24 4 0.07 [-0.21, 0] quality 246 18 0.05 [-0.09, 0] 0 <td>(cvr.Full.h5.i)</td> <td></td> <td>(covariate)</td> <td>-</td> <td>-</td> <td>-0.03 [-0.08, 0.02]</td> | (cvr.Full.h5.i) | | (covariate) | - | - | -0.03 [-0.08, 0.02] |
| (cvr.Full.h5.ii) (covariate) - - - - - - - 0.02 [-0.09, 0] Exp a. Effect of manipulation type (cvr.Full.exp.a) ManipType combined 24 4 0.07 [-0.21, 0] quality 246 18 0.05 [-0.09, 0] 0 quantity 363 48 -0.07 [-0.16, 0] | Hyp. 5(ii). Life-history effects | InMaxLongevity.C | intercept | - | - | -0.03 [-0.09, 0.03] |
| Exp a. Effect of manipulation ManipType combined 24 4 0.07 [-0.21, 0] type (cvr.Full.exp.a) quality 246 18 0.05 [-0.09, 0] quantity 363 48 -0.07 [-0.16, 0] | (cvr.Full.h5.ii) | | (covariate) | - | - | -0.02 [-0.09, 0.05] |
| type (<i>cvr.Full.exp.a</i>) quality 246 18 0.05 [-0.09, 0 quantity 363 48 -0.07 [-0.16, 0 | Exp a. Effect of manipulation | ManipType | combined | 24 | 4 | 0.07 [-0.21, 0.35] |
| quantity 363 48 -0.07 [-0.16.0 | type (cvr.Full.exp.a) | | 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 | | | starvation | 602 | 54 | -0.04 [-0.12, 0.05] |
| Exp b. Effect of manipulation ManipDirection dual 60 7 0.11 [-0.14, 0 | Exp b. Effect of manipulation | ManipDirection | dual | 60 | 7 | 0.11 [-0.14, 0.35] |
| direction (<i>cvr.Full.exp.b</i>) restrict 1116 106 -0.04 [-0.10, 0 | direction (cvr.Full.exp.b) | | restrict | 1116 | 106 | -0.04 [-0.10, 0.03] |
| supplement 59 8 -0.06 [-0.27, 0 | | | supplement | 59 | 8 | -0.06 [-0.27, 0.14] |
| Exp c. Effect of manipulation RelativeManipDuration.C intercept0.03 [-0.08, 0 | Exp c. Effect of manipulation | RelativeManipDuration.C | intercept | - | - | -0.03 [-0.08, 0.03] |
| duration (<i>cvr.Full.exp.c</i>) (covariate) - 0.05 [0.00, 0. | duration (cvr.Full.exp.c) | | (covariate) | - | - | 0.05 [0.00, 0.10] |
| Exp d. Effect of rearing WildLabRear commercial 127 11 -0.02 -0.21, 0 | Exp d. Effect of rearing | WildLabRear | commercial | 127 | 11 | -0.02 [-0.21, 0.17] |
| environment (<i>cvr.Full.exp.d</i>) [ab 679 54 0.02 [-0.06.0 | environment (cvr.Full.exp.d) | | lab | 679 | 54 | 0.02 [-0.06, 0.11] |
| mixed 15 1 0.10 [-0.41, 0 | · · · · · | | mixed | 15 | 1 | 0.10 [-0.41, 0.62] |
| wild 414 55 -0.09 [-0.18. 0 | | | wild | 414 | 55 | -0.09 [-0.18, 0.00] |

909 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 nonphylogenetic (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*.

915

916 Fig. 2 Category-specific estimates for log response ratio (InRR) and log coefficient of 917 variation ratio (InCVR) with meta-regression models testing the effect of (A, B) the 918 experimental context for risk-taking behaviour; (C,D) sex of study subjects; and (E,F) life-919 stage of study subjects during the diet manipulation treatments. InRR effects are presented 920 on the left (A, C, D) and InCVR on the right (B, D, F). The areas of the blue shaded circles 921 are proportional to the number of effect sizes k used, and bars represent 95% confidence 922 intervals. A positive effect shows higher risk taking or higher variance in risk taking in low-923 condition animals, respectively.

924

925 Fig. 3 Category-specific estimates based on the degree that log response ratio (InRR) 926 effect sizes could be extracted from published material. Fully reported effect sizes are from 927 papers where all effect sizes could be extracted from published material, partially reported 928 effect sizes are from papers where some effect sizes could be extracted but additional effect 929 sizes could be obtained from authors (therefore includes effect sizes from published material 930 and author correspondence), and not reported effect sizes are those that could only be 931 calculated from data obtained through author correspondence. The areas of the green 932 shaded circles are proportional to the number of effect sizes k used, and bars represent 95% 933 confidence intervals. A positive effect shows higher risk taking and higher variance in risk 934 taking in low-condition animals.

935

936 Fig 4 Category-specific estimates for log response ratio (InRR) and log coefficient of 937 variation ratio (InCVR) meta-regression models for effect of (A, B) the type of diet 938 manipulation; (C, D) the direction of the diet manipulation; and (E, F) the rearing 939 environment of the experimental subjects. InRR effects are presented on the left (A, C, D) 940 frames and InCVR on the right (B, D, F). The areas of the orange shaded circles are 941 proportional to the number of effect sizes k used, and bars represent 95% confidence 942 intervals. A positive effect shows higher risk taking and higher variance in risk taking in low-943 condition animals, respectively.











