Results

# Methods

### *Effect size*

We calculated the standardized mean difference, assuming heteroscedastic population variances () (Bonett, 2008, 2009), along with its associated sampling error using the *escalc* function in the *metafor* package (vers. 4.6.0) (Viechtbauer, 2010) of R (vers. 4.3.1). In addison, a small sample correction was used to account for possible bias resulting from effect sizes calculated with small sample sizes. We choose to use as our effect size because our data: 1) contained substantial amounts of ratio data (e.g., respiratory control ratios, RCR, relative gene expression) which makes interpretation with alternative effect size measures, such as log response ratio, challenging; 2) percentages and 3) skewed measurement variables. is more robust to these types of measurement variables. We calculated as the mean difference between control and treatment groups divided by the pooled standard deviation. As such, positive effect sizes represent situations where the mean of the experimental group was larger than the control group.

### *Meta-Analysis*

We analysed our data using multilevel meta-analytic (MLMA) and meta-regression (MLMR) models in R using *metafor* (vers. 4.6.0 Viechtbauer, 2010). Meta-analytic mean estimates and meta-regression models were plotted using orchard plots which convey overall meta-analytic means, alongside raw effect sizes and prediction intervals, which describe effect heterogeneity (vers. 2.0, Nakagawa *et al.*, 2021, 2023).

#### Multi-level Meta-analysis (MLMA) Models

Using data subsets for each of our major stressor categories, we first fit multi-level meta-analysis (MLMA) models (i.e., intercept-only models) with , that included study, species, trait type, and phylogeny as random effects to account for non-independence and identify sources of variability. Our MLMA models allowed us to partition variation in among these key sources while accounting for total sampling variance. This allowed us to calculate the proportion of total heterogeneity [i.e., ; *sensu* Nakagawa & Santos (2012); Noble *et al.* (2022)] along with various metrics describing the proportion of variance explained by each random effect level (Nakagawa & Santos, 2012). As a second measure of heterogeneity we also present 95% prediction intervals which describe the expected distribution of effects that are expected from future studies (Nakagawa *et al.*, 2021; Noble *et al.*, 2022).

A phylogeny was derived by first cross-checking taxa names using the Open Tree of Life (OTL) with the *rotl* package in R (vers. 3.1.0) (Michonneau, Brown, & Winter, 2016), and plotted using *ggtree* to visualize the tree (vers. 3.9.0) (Yu *et al.*, 2017). We resolved any taxa names that were outdated or changed. Once names in the dataset and *rotl* database matched we exported taxa names and built a time calibrated phylogeny for the species in our dataset using TimeTree [timetree.org; ]. We used the R packages *ape* (vers. 5.7.1) (Paradis & Schliep, 2019) and *phytools* (vers. 1.9.16) (Revell, 2012) to prune the tree for individual analyses and calculate phylogenetic correlation matrices used in meta-analytic models.

#### Multi-level Meta-Regression (MLMR) Models

After quantifying levels of heterogeneity in each of the different stressor datasets, we fit a series of multi-level meta-regression (MLMR) models to test our key questions. In all models, we included the same random effects as we used in our MLMA models, however, MLMR models differed in the moderators included because each stressor type had unique sources of heterogeneity that we expected would explain effect variance.

# Results

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