**Methods**

**Literature Review**

Studies of the effects of several pollutants on oxidative stress in amphibians were identified via database and search engine searches conducted between the 8th of July and the 25th of November 2021. Specifically, using the search string “(‘Oxidative Stress’) AND (‘Amphibians’) AND (‘Pollution’)”, we performed the search on the Web of Science Core Collection, PubMed, EMBASE (Ovid), EBSCOhost, Scopus, and ECOTOX. We read the title and abstract of studies published between 1998-2021 and assessed whether they contained suitable information for our meta-analysis (details below). 865 studies were identified by the database searches above and seven additional studies were identified subsequently as suitable for our meta-analysis from the reference list of screened studies (Figure S1). After removing duplicates, 361 studies were screened (by reading their title and abstract) and 105 were identified as potentially containing suitable information for the meta-analysis. These 105 studies were fully read to assess whether they had suitable information and met inclusion criteria (see details below). Database searches, study screening and effect size extraction were all performed by one co-author (CM). effect sizes Our final dataset included XXX effect sizes from XXX studies and XXX species.

**Criteria for inclusion**

We were interested in meta-analysing the experimental effects of different pollutants on oxidative stress in amphibians. Therefore, we only included experimental studies that reported: (1) mean oxidative stress values, variation (standard deviation [SD] or standard error) and sample sizes (i.e., number of individuals) for control (i.e., non-exposed to pollutants) and treatment groups (i.e., exposed to pollutants); (2) one of the following biomarkers of oxidative stress Superoxide dismutase, Glutathione peroxidase, Catalase, Glutathione reductase, Glutathione S-transferase (i.e., enzymatic biomarkers), GSH (i.e., non-enzymatic biomarker) or Malondialdehyde (i.e., an indirect indicators of oxidative damage); (3) the development stage (embryos, larvae or post-metamorphic) in which the effect of pollutants had been tested. Additionally, we only included effect sizes from studies that tested one pollutant at a time (i.e., studies not testing the effect of a pollutant in combination with another factor, such as temperature).

**Meta-analytic effect sizes**

To assess the effects of different pollutants on the oxidative stress of amphibian, we computed the log response ratio (lnRR) (Hedges et al., 1999).We calculated lnRR and its associated sampling variance using the R function ‘escalc’ in the ‘metafor’ R package (v3.8-1; (Viechtbauer, 2010)) lnRR was calculated so that positive values meant higher values of a given oxidative stress in the treatment group (i.e., after exposure to a pollutant) than in the control group (i.e., not exposed to a pollutant), and *vice versa* for negative lnRR values. When a given control group was compared to multiple treatment groups, we divided the sample size of the control group by as many comparisons the control group was used for and used this ‘adjusted’ sample size to calculate lnRR and its sampling variance. We carried out sensitivity analysis to validate our main results based on lnRR (see ‘Sensitivity analyses’ below).

**Meta-analysis**

To assess how oxidative stress markers are affected by pollution, we ran a phylogenetic multilevel (intercept-only) meta-analysis and meta-regressions. These models included three random intercept effects: publication identity, phylogeny and species identity (to capture among-species variation not explained by phylogeny). Additionally, an observation identity random term was included to capture variation in effect sizes within studies. For intercept-only models, we estimated total heterogeneity (*I*2total) (Nakagawa & Santos, 2012; Senior et al., 2016) and the amount of variation explained by each random term as implemented in the R function ‘i2\_ ml’ (‘orchaRd’ R package v2.0; (Nakagawa et al., 2021)). For meta-regressions, we report on the proportion of variation explained by each moderator as calculated by the R function ‘r2\_ ml’ (‘orchaRd’ R package v2.0; (Nakagawa et al., 2021)).

To finish this section, I think it would be good to add specific details on each model that we run and show in the results section. Therefore, I will wait to write it until we have the structure of the results written.

**Phylogenies**

To control for phylogenetic history, we extracted phylogenetic trees from Open Tree of Life (Hinchliff et al., 2015; Rees & Cranston, 2017), accessed via the R package ‘rotl’ (v3.0.14; (Michonneau et al., 2016; Redelings et al., 2019)). Tree branch length was calculated following (Grafen & Vickerman, 1989), generating a phylogenetic correlation matrix that was included in all our (phylogenetic) multi-level meta-analytic models. We assessed the phylogenetic importance in our meta-models calculating the proportion of variation in lnRR explained by the phylogeny (*I*2phylogeny; (Cinar et al., 2022)).

**Publication bias**

We tested small-study effects and time-lag effects following Nakagawa et al. ([2022](https://onlinelibrary.wiley.com/doi/full/10.1111/ele.14099" \l "ele14099-bib-0087)). To this end, we ran two additional multilevel meta-analytic models of lnRR. Each of these models included as a single moderator either the square-root of the inverse of the effective sample size or the mean-centred year of study publication (Nakagawa et al., [2022](https://onlinelibrary.wiley.com/doi/full/10.1111/ele.14099" \l "ele14099-bib-0087); Trikalinos & Ioannidis, [2005](https://onlinelibrary.wiley.com/doi/full/10.1111/ele.14099" \l "ele14099-bib-0135)).

We handled the dataset, ran all analyses and produced visualisations using R (v.4.2.1; (R Core Team, 2022)).

Diagram

Description automatically generated

Figure 1: PRISMA Flow Diagram outlining the literature review process (adapted from Moher et al., 2015).