Supporting Information

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SI Text

Metaanalysis of Published Studies on Allelopathic Interactions Among Plankton. The database was assembled by keyword searches in the ISI Web of Science database covering papers published between 1986 and 2008 and by searching the cited literature in the obtained papers and in recent reviews. The following criteria were used to include publications in the analysis: i) the effect of culture-media extracts, cell-free filtrates or supernatants of a potentially allelopathic species on a target algal species was tested against an appropriate control, ii) the paper included a presentation of the cell biomass (cells volume^{-1}, chlorophyll *a* [Chl *a*], or carbon concentration [C]) of the potentially allelopathic species from which the media extracts, cell-free filtrates or supernatants were prepared, iii) the paper included a presentation of the means, some measure of the variance and the sample sizes for the experimental treatments and for the controls. On these grounds, papers using cultures separated by membranes or filters that cells could not pass were not included as species may compete for nutrients rather than exude allelopathic substances. Furthermore, studies testing the effect of specific compounds present in the potentially allelopathic organism but that were not extracted from the medium (e.g., commercially available ocadaic acid, domoic acid, microcystins or nodularin), or compounds that were extracted from within cells were not included as these studies were not considered ecologically relevant. Studies using bacteria as target species were not included because this interaction was considered defensive rather than competitive. When a study compared several measurements of allelopathic effects using the same control (e.g., when exudates from several potentially allelopathic species were compared with a control consisting of identical medium but without exudates) only one, randomly chosen measurement was included in the database to avoid problems with nonindependence between comparisons. The final database consisted of 21 studies published between 1956 and 2008. The experiments included in the analysis were conducted with a total of 16 potentially allelopathic and 29 target species covering several taxonomic groups.

Response and Explanatory Variables. Allelopathic effects can be evaluated as changes in growth rate or biomass (measured either as fluorescence, cells volume^{-1}, Chl *a*, or C concentration) of the target algal species exposed to exuded compounds from a potentially allelopathic species. Furthermore, some studies investigated mechanisms behind allelopathic effects by measuring biochemical or cellular processes (e.g., photosynthetic efficiency, enzyme activity, membrane permeability or siderophore-bound iron). We only included measures of growth rate or biomass of target species in our analysis, because allelopathic effects may be present although not through the specific mechanism under study. When measurements of cell density were made on the same replicate after different time intervals, only the result from the last time was included. When allelopathic effects of exuded compounds from algal cultures grown under different nutrient or light levels were studied, we only included the control measurements or the measurements that were as close to natural levels as possible.

To test whether there were differences among particular groups of measurements included in the database, the studies were categorized according to the Chl *a* content of the potentially allelopathic species from which the media extracts, cell free filtrates or supernatants were prepared. Studies were categorized into low (<5 μg L^{-1}) or high (≥ 5 μg L^{-1}) Chl *a* content. These classes are typical for coastal waters and many harmful algal blooms (1). We used Chl *a* content, rather than cell concentration, because the potentially allelopathic species included in our database differed widely in cell size and thus in the number of cells per volume at bloom concentration. In studies where cell density was presented, the C content of potentially allelopathic cells was calculated using the formula

$$\log \text{pg } C \text{ cell}^{-1} = \log a + b \log V \quad [1]$$

where V is cell volume (μm^3), $\log a$ is the y intercept and b is the slope in model I least-square regressions of $\log_{10}$-transformed C (pg) and volume data for different species (2). The prolate spheroide volume (PSV) was calculated using the formula

$$PSV = \pi/6\, L\, B^2 \quad [2]$$

where L is the length (μm) and B is the breath (μm) of a cell (3). Chl *a* content was calculated by dividing the C content with 40 (4).

Metaanalysis. Hedge's d was used as a measure of the effect size and was calculated for each individual measurement as the difference between the mean of the experimental treatment and the control divided by their pooled standard deviation and multiplied by a correction term to reduce bias from small sample sizes (5). Consequently, a negative effect size indicates an allelopathic interaction. All analyses were performed using the computer program MetaWin 2.0 (6). Visual data exploration was performed using normal quantile plots (7) to check that the data were normally distributed (data should fall on an approximately straight line and within the 95% confidence limits), and to search for publication bias (the curve is nonlinear or has gaps where data are missing). Rosenthal's fail-safe number was also calculated to test for publication bias in the database (8). This number indicates the number of studies with zero effect that has to be added to the database to change the result from significant to nonsignificant. If this number is sufficiently high ($>5n + 10$, where n is the number of measurements), the results can be considered as robust regarding publication bias (8).

The normal quantile plots for allelopathic interactions showed approximately linear relationships between the standardized effect size and the normal quantiles, indicating that data were normally distributed. No gaps were observed in the linear regression curves, indicating that publication bias is not a problem with the present datasets. Furthermore, Rosenthal's fail-safe number for the total dataset was larger (4,933) than the critical value (485). Therefore, the number of nonsignificant, unpublished studies needed to change the results of the meta-analyses from significant to nonsignificant was sufficiently high to conclude that the observed results can be treated as a reliable estimate of the true effect size.

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