Data Presentation

Module 10

Masato Yamamichi – School of Biological Sciences

Scientific papers in peer-reviewed journals

PROCEEDINGS B

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A shady phytoplankton paradox: when phytoplankton increases under low light

Masato Yamamichi[1], Takehiro Kazama[2], Kotaro Tokita[3], Izumi Katano[4], Hideyuki Doi[5], Takehito Yoshida[1,6], Nelson G. Hairston Jr[7] and Jotaro Urabe[3]

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Research

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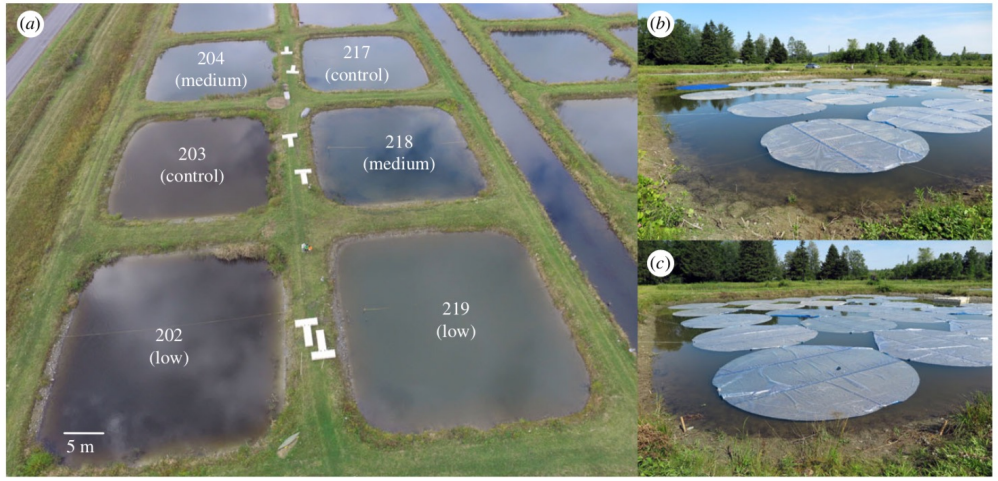

Figure 1. Field site and shading treatments. (*a*) A bird's-eye view of the assigned treatments in CUEPF at the end of the experiment (after floating mats were removed). Control: high-light (0% shading), medium: medium-light (56.5% shading) and low: low-light (75.4% shading). T-shape floating docks (3 m × 1 m each), used for sampling, are visible on dikes between ponds. Floating mats during experiment in (*b*) a medium-light treatment (pond 218) and (*c*) a low-light treatment (pond 219).

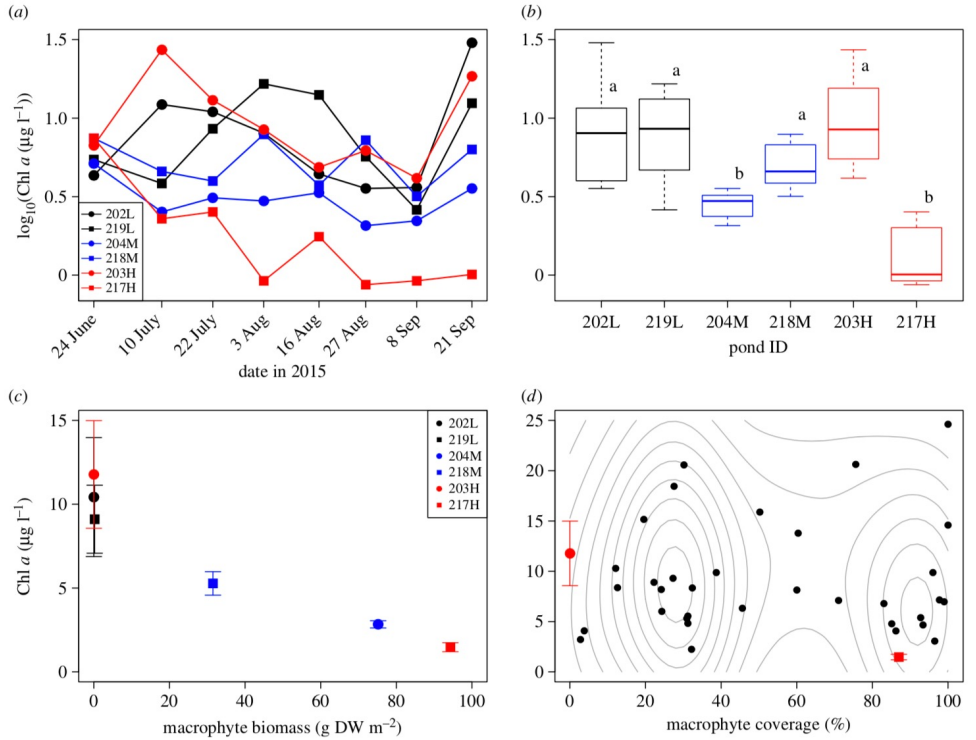

Figure 2. Experimental and observational results. (*a*) Time-series and (*b*) boxplot of log-scaled chlorophyll *a* (Chl *a*) under three light intensities. The bold line in the box indicates the median value. Lower/upper limits of the box are the first/third quartiles, respectively. Lower/upper whisker plots represent the minimum/maximum values within the first/third quartiles minus/plus 1.5× interquartile range, respectively. The different characters on the box indicate significant differences among the ponds using a post-hoc multiple comparison for LMM ($p < 0.002$). (*c*) A correlation between chlorophyll *a* (mean and standard error, s.e.) and submersed macrophyte biomass (Spearman's $\rho = -0.99$; $p = 0.00031$). Red: control, blue: medium-light, black: low-light. Circle: road-side ponds, square: channel-side ponds. (*d*) Macrophyte coverage (%) and chlorophyll *a* concentration in 34 non-shaded ponds in 2016 (we removed a pond with an outlier value of chlorophyll *a*, see text). Black points indicate mean values at two sampling times in 2016. Red points and error bars indicate mean and s.e. values of non-shaded control ponds in 2015. Grey contours indicate a two-dimensional binned kernel density estimation.

Yamamichi et al. (2018) *Proc. R. Soc. B*

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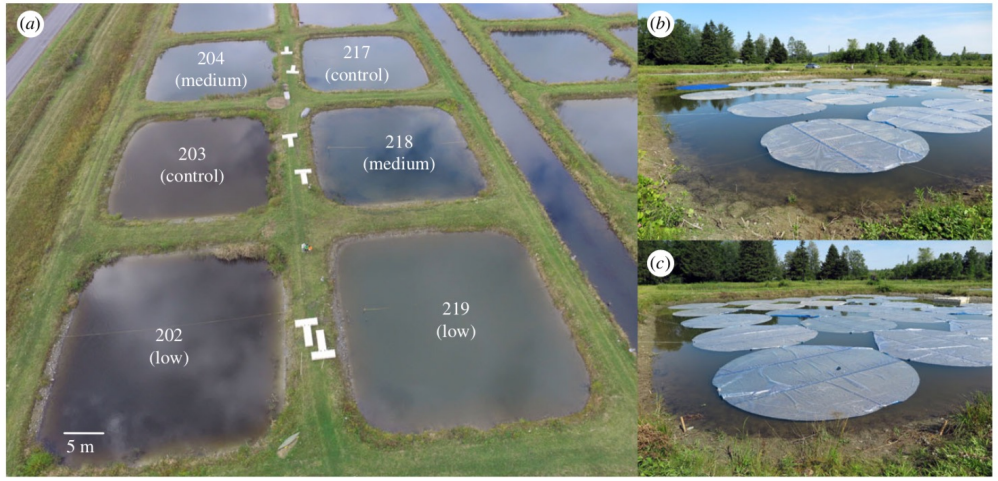

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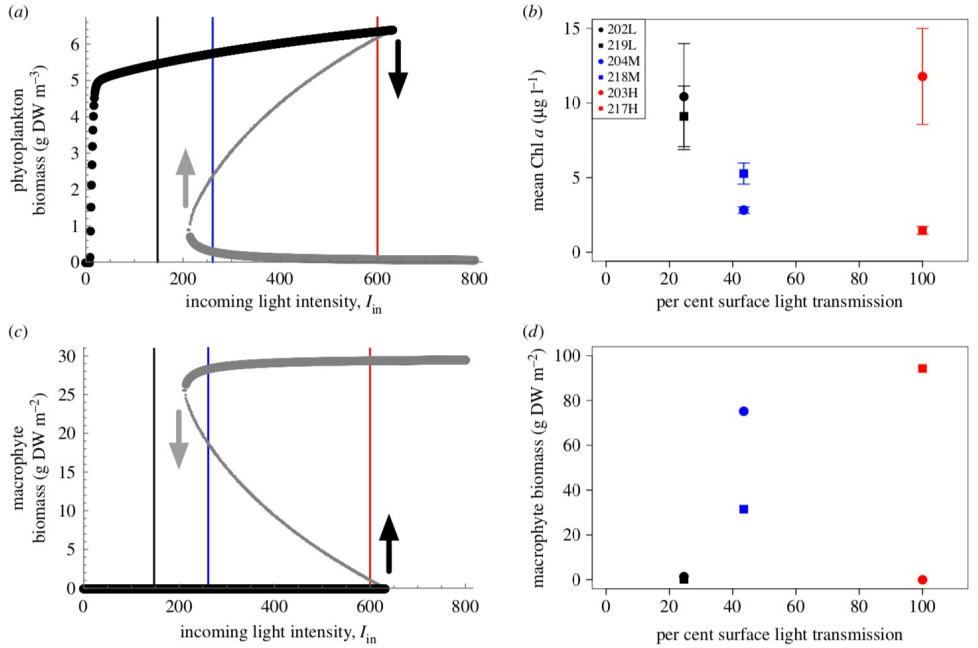
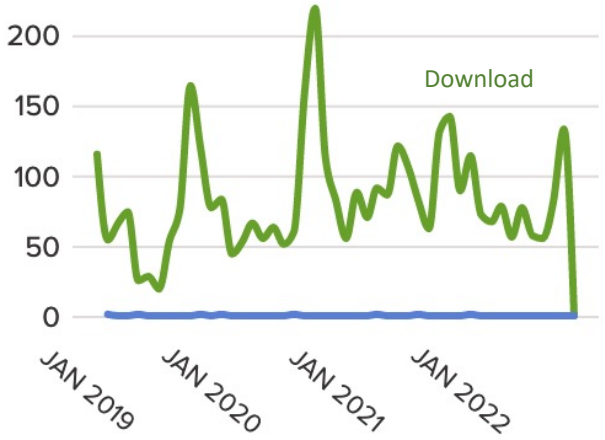


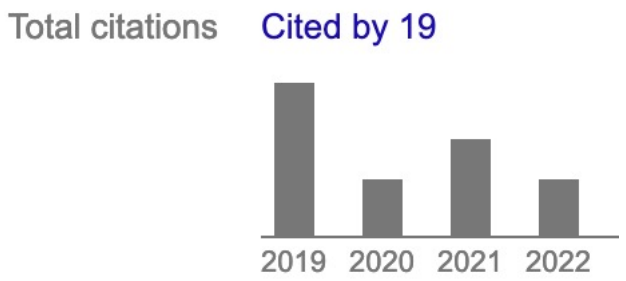
Figure 3. Effects of light on phytoplankton and submersed macrophytes. Bifurcation analyses of (*a*) phytoplankton biomass (averaged over the pelagic and the benthic habitats), $(z_1A_1 + z_2A_2)/(z_1 + z_2)$, and (*c*) macrophyte biomass, z_2S, along incoming light intensity. Large black and grey dots represent stable equilibria and smaller grey dots represent unstable equilibria. Black and grey arrows indicate abrupt shifts due to alternative stable states. Red, blue and black vertical lines indicate light conditions for control, medium-light and low-light ponds, respectively, when the light intensity of control ponds is assumed to be 600 (μmol m^{-2} s^{-1}). The experimental results of (*b*) chlorophyll *a* and (*d*) macrophyte biomass along per cent surface light transmission.

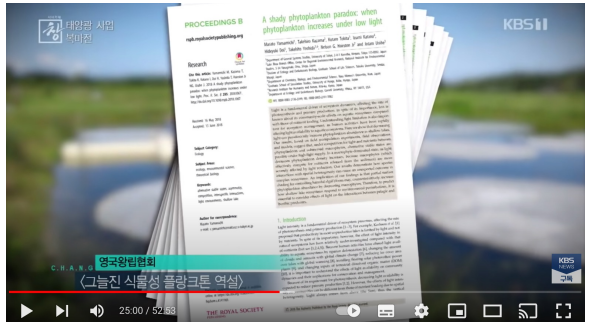
Yamamichi et al. (2018) *Proc. R. Soc. B*

Scientific papers in peer-reviewed journals

- 2014: Grant application
- 2015: Field work
- 2016: Field work
- 2017: Analyzing data & writing a manuscript
- 2018: Submission to the journal, review & revise, publication







KBS

Data presentation

1. In text reporting
2. Tables
3. Figures
4. Figures for presentation

Data presentation

LETTER

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IFITM3 restricts the morbidity and mortality associated with influenza

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The 2009 H1N1 influenza pandemic showed the speed with which a novel respiratory virus can spread and the ability of a generally mild infection to induce severe morbidity and mortality in a subset of the population. Recent *in vitro* studies show that the interferon-inducible transmembrane (IFITM) protein family members potently restrict the replication of multiple pathogenic viruses[1–7]. Both the magnitude and breadth of the IFITM proteins' *in vitro* effects suggest that they are critical for intrinsic resistance to such viruses, including influenza viruses. Using a knockout mouse model[8], we now test this hypothesis directly and find that IFITM3 is essential for defending the host against influenza A virus *in vivo*. Mice lacking *Ifitm3* display fulminant viral pneumonia when challenged with a normally low-pathogenicity influenza virus, mirroring the destruction inflicted by the highly pathogenic 1918 'Spanish' influenza[9,10]. Similar increased viral replication is seen *in vitro*, with protection rescued by the re-introduction of *Ifitm3*. To test the role of IFITM3 in human influenza virus infection, we assessed the *IFITM3* alleles of individuals hospitalized with seasonal or pandemic influenza H1N1/09 viruses. We find that a statistically significant number of hospitalized subjects show enrichment for a minor *IFITM3* allele (SNP rs12252-C) that alters a splice acceptor site, and functional assays show the minor CC genotype IFITM3 has reduced influenza virus restriction *in vitro*. Together these data reveal that the action of a single intrinsic immune effector, IFITM3, profoundly alters the course of influenza virus infection in mouse and humans.

IFITM3 was identified in a functional genomic screen as mediating resistance to influenza A virus, dengue virus and West Nile virus infection *in vitro*[1]. However, the role of the IFITM proteins in antiviral immunity *in vivo* is unknown. Therefore, we infected mice that are homozygous for a disruptive insertion in exon 1 of the *Ifitm3* gene that abolishes its expression[8] (*Ifitm3*$^{-/-}$) with a low-pathogenicity murine-adapted H3N2 influenza A virus (A/X-31). Low-pathogenicity strains of influenza do not normally cause extensive viral replication throughout the lungs, or cause the cytokine dysregulation and death typically seen after infection with highly pathogenic viral strains[9], at the doses used (Fig. 1a). However, low-pathogenicity-infected *Ifitm3*$^{-/-}$ mice became moribund, losing >25% of their original body weight and showing severe signs of clinical illness (rapid breathing, piloerection) 6 days after infection. In comparison, wild-type littermates shed <20% of their original body weight, before fully recovering (Fig. 1a, b). There was little difference in virus replication in the lungs during the first 48 h

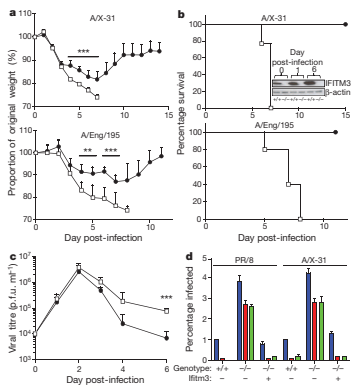

Figure 1 | Influenza A virus replicates to higher levels in *Ifitm3*$^{-/-}$ mice. **a**, **b**, Change in body mass (**a**) and survival (**b**) of wild-type (filled circles) and *Ifitm3*$^{-/-}$ (open squares) mice following intranasal inoculation with A/X-31 and pandemic H1N1/09 Eng/195 influenza ($n > 5$). **b**, Absence of Ifitm3 expression was verified in the *Ifitm3*$^{-/-}$ mice at all time points, but was seen to increase in wild-type mice. **c**, A/X-31 viral load in the lungs of mice ($n > 4$) was calculated over the course of infection by plaque assay. p.f.u., plaque-forming units. *Ifitm3*$^{-/-}$ murine embryonic fibroblasts ($n = 3$ per condition) stably expressing *Ifitm3* (+), or the empty vector (−) were left untreated (blue), or incubated with IFN-α (red) or IFN-γ (green), then challenged with either A/X-31 or PR/8 influenza. **d**, Twelve hours after infection, the cells were assessed for either haemagglutinin expression (PR/8), or nucleoprotein expression (A/X-31) IFITM3 expression was determined to be present (+) or absent (−) by western blotting (Supplementary Fig. 2). Results show means ± s.d. Statistical significance was assessed by Student's *t*-test (**$P < 0.01$; ***$P < 0.001$).

[1]Wellcome Trust Sanger Institute, Wellcome Trust Genome Campus, Hinxton CB10 1SA, UK. [2]Ragon Institute of Massachusetts General Hospital, Massachusetts Institute of Technology, and Harvard University, Charlestown, Massachusetts 02129, USA. [3]Divison of Virology, Department of Pathology, University of Cambridge, Tennis Court Road, Cambridge CB2 1QP, UK. [4]Division of Genetics and Genomics, The Roslin Institute, University of Edinburgh, Roslin EH25 9RG, UK. [5]Department of Critical Care Medicine, University of Edinburgh, Edinburgh EH16 4TJ, UK. [6]Institute of Genetics and Biophysics "A. Buzzati-Traverso", National Research Council (CNR), Naples, Italy. [7]Centre for Respiratory Infection, National Heart and Lung Institute, St Mary's Campus, Imperial College London, W2 1PG, UK. [8]Liverpool School of Tropical Medicine, Pembroke Place, Liverpool L3 5QA, UK. [9]Institute of Translational Medicine, University of Liverpool, Alder Hey Children's Hospital, Liverpool L12 2AP, UK. [10]Gastrointestinal Unit, Massachusetts General Hospital, Boston, Massachusetts 02117, USA. [11]UCL/MRC Centre for Medical Molecular Virology, Department of Infection, University College London, Cleveland Street, London W1T 4JF, UK.
*Lists of participants and their affiliations appear at the end of the paper.

Table 1. Strain number and MIC against Ampicillin (Amp), Streptomycin (Strep), and Norfloxacin (Nor) for ECOR isolates of *E. coli* used in experiments.

ECOR strain no.	Host	Location	MIC Amp (μg/mL)	MIC Strep (μg/mL)	MIC Nor (μg/mL)
1	Human	Iowa	9	67	0.16
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9	Human	Iowa	9	67	0.04
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61	Human	Sweden	9	67	0.08
64	Human	Sweden	9	67	0.32
65	Celebese ape	Washington (zoo)	18	67	0.08
69	Celebese ape	Washington (zoo)	18	67	0.64
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differences in MIC among strains for ampicillin and streptomycin, with slightly larger variation for norfloxacin. The range of this variance is markedly less than that found for persistence and is uncorrelated with the persister fraction for each strain (shown below). This implies that MIC variation does not account for variation in persistence.

TIME-KILL CURVES

Figure 1 shows time-kill curves over 4 h for the 24 ECOR strains treated with (1) ampicillin (24 μg/mL), (2) streptomycin (96 μg/mL), and (3) norfloxacin (0.66 μg/mL). Because three of the ECOR strains (ECOR 3, ECOR 16, ECOR 48) are resistant to streptomycin (Table 1), time-kill experiments using this drug were performed only on the remaining 21 strains (Fig. 1B). Following previous work (e.g. Keren et al. 2004a; Wiuff et al. 2005) we take the fraction of viable cells remaining after 4 h antibiotic exposure to be the putative "persister fraction" for each genotype. We observed highly significant differences in the persister fraction between our 24 focal ECOR strains following ampicillin (One-way analysis of molecular variance [ANOVA] $F_{23} = 5.435$ $P \leq 0.0001$), streptomycin (One-way ANOVA $F_{20} = 9.224$ $P \leq 0.0001$), and norfloxaxin (One-way ANOVA $F_{23} = 13.533$ $P \leq 0.0001$) treatments. There was no association, however, between MIC and persister fractions with any drug (ampicillin: $r^2 = 0.003$, $P = 0.797$; streptomycin: $r^2 = 0.071$, $P = 0.302$; norfloxacin: $r^2 = 0.020$, $P = 0.516$). Furthermore, there were no pairwise correlations between the persister fractions following treatment with any of the antibiotics (ampicillin × streptomycin: $r = -0.158$, $P = 0.507$; ampicillin × norfloxacin: $r = -0.043$, $P = 0.847$; streptomycin × norfloxacin: $r = -0.153$, $P = 0.519$; Fig. 3). This shows that persistence level is both drug and genotype specific.

COMPETITION EXPERIMENTS

To investigate whether there are fitness effects associated with the generation of higher persistence during growth in conditions of fluctuating antibiotic exposure, we carried out competition experiments between three pairs of strains in both ampicillin and streptomycin-fluctuating environments as well in an antibiotic-free environment. For each pair of strains examined, one strain produces a higher persister fraction following treatment with ampicillin but a lower fraction following streptomycin exposure (Fig. 3). From starting cultures of roughly equal frequencies of the two strains, the strain producing the higher persister fraction in ampicillin prevailed during competition with a periodic cycling of this drug in all cases. By contrast, under conditions of fluctuating

Data presentation

1. In text reporting

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1. In text reporting

How can we improve this?

“Mice fed the high-carb diet were significantly heavier than those fed the low-carb diet ($P < 0.05$).”

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ALWAYS report:

1. the test statistic (e.g., if you do an ANOVA, the F ratio)

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2. the size of the experiment (degrees of freedom)

Typically, you would also report the actual *P*-value, unless it is very small.

1. In text reporting

Doing it right:

"Mice fed the high-carb diet were significantly heavier than those fed the low carb diet ($F_{1,98} = 4.25$, $P = 0.042$)."

Data presentation

2. Tables

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differences in MIC among strains for ampicillin and streptomycin, with slightly larger variation for norfloxacin. The range of this variance is markedly less than that found for persistence and is uncorrelated with the persister fraction for each strain (shown below). This implies that MIC variation does not account for variation in persistence.

TIME-KILL CURVES

Figure 1 shows time-kill curves over 4 h for the 24 ECOR strains treated with (1) ampicillin (24 μg/mL), (2) streptomycin (96 μg/mL), and (3) norfloxacin (0.66 μg/mL). Because three of the ECOR strains (ECOR 3, ECOR 16, ECOR 48) are resistant to streptomycin (Table 1), time-kill experiments using this drug were performed only on the remaining 21 strains (Fig. 1B). Following previous work (e.g. Keren et al. 2004a; Wiuff et al. 2005) we take the fraction of viable cells remaining after 4 h antibiotic exposure to be the putative "persister fraction" for each genotype. We observed highly significant differences in the persister fraction between our 24 focal ECOR strains following ampicillin (One-way analysis of molecular variance [ANOVA] $F_{23} = 5.435$ $P \leq 0.0001$), streptomycin (One-way ANOVA $F_{20} = 9.224$ $P \leq 0.0001$), and norfloxaxin (One-way ANOVA $F_{23} = 13.533$ $P \leq 0.0001$) treatments. There was no association, however, between MIC and persister fractions with any drug (ampicillin: $r^2 = 0.003$, $P = 0.797$; streptomycin: $r^2 = 0.071$, $P = 0.302$; norfloxacin: $r^2 = 0.020$, $P = 0.516$). Furthermore, there were no pairwise correlations between the persister fractions following treatment with any of the antibiotics (ampicillin × streptomycin: $r = -0.158$, $P = 0.507$; ampicillin × norfloxacin: $r = -0.043$, $P = 0.847$; streptomycin × norfloxacin: $r = -0.153$, $P = 0.519$; Fig. 3). This shows that persistence level is both drug and genotype specific.

COMPETITION EXPERIMENTS

To investigate whether there are fitness effects associated with the generation of higher persistence during growth in conditions of fluctuating antibiotic exposure, we carried out competition experiments between three pairs of strains in both ampicillin and streptomycin-fluctuating environments as well in an antibiotic-free environment. For each pair of strains examined, one strain produces a higher persister fraction following treatment with ampicillin but a lower fraction following streptomycin exposure (Fig. 3). From starting cultures of roughly equal frequencies of the two strains, the strain producing the higher persister fraction in ampicillin prevailed during competition with a periodic cycling of this drug in all cases. By contrast, under conditions of fluctuating

2. Making Tables

An experiment was designed to test the **effects of copper** on the **fertilisation success** in the free-spawning marine invertebrate, *Galeolaria caespitosa*. Raw data are the number of eggs fertilized per 100. **Note:** The Copper concentrations are in units of milligrams per Litre)

Galeolaria caespitosa

```
> #means
> mean(control$fertilisation_success)
[1] 65.94444
> mean(copper$fertilisation_success)
[1] 56.36111

> # Std errors
>
sd(control$fertilisation_success)/sqrt(nrow(control))
[1] 2.784009
> sd(copper$fertilisation_success)/sqrt(nrow(copper))
[1] 3.667349
```

2. Making Tables

How can we improve this?

Parameter	Control	Copper
Mean	65.94444	56.36111
Standard Error	2.784009	3.6673

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1. Heading, with information about the experiment, and what is shown in the table

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Table 1. The effect of soluble copper on fertilisation success in *Galeolaria caespitosa*. The proportion of fertilised eggs was measured in 36 trials for each copper treatment, and the mean and standard error across trials was calculated.

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Fertilisation success	Copper concentration	
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Mean	65.94444	56.36111
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3. Report units and sample sizes (this might be in the table heading or in the table itself)

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4. Keep the number of decimal places consistent, and make them sensible

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5. Avoid column borders (vertical lines), and only use horizontal borders (lines) for header and the bottom.

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2. Making Tables

Table 1. Strain number and MIC against Ampicillin (Amp), Streptomycin (Strep), and Norfloxacin (Nor) for ECOR isolates of *E. coli* used in experiments.

ECOR strain no.	Host	Location	MIC Amp (μg/mL)	MIC Strep (μg/mL)	MIC Nor (μg/mL)
1	Human	Iowa	9	67	0.16
3	Dog	Massachusetts	18	Resistant	0.08
9	Human	Iowa	9	67	0.04
11	Human	Sweden	18	67	0.02
14	Human	Sweden	9	33	0.08
16	Leopard	Washington (zoo)	18	Resistant	0.08
23	Elephant	Washington (zoo)	18	67	0.04
26	Human	Massachusetts	18	67	0.08
29	Kangaroo rat	Nevada	18	67	0.04
32	Giraffe	Washington (zoo)	18	67	0.04
33	Sheep	California	9	67	0.32
38	Human	Iowa	9	67	0.16
39	Human	Sweden	18	67	0.16
40	Human	Sweden	9	67	0.04
44	Cougar	Washington (zoo)	9	67	0.08
48	Human	Sweden	18	Resistant	0.32
50	Human	Sweden	18	67	0.08
53	Human	Iowa	9	67	0.08
56	Human	Sweden	18	33	0.16
61	Human	Sweden	9	67	0.08
64	Human	Sweden	9	67	0.32
65	Celebese ape	Washington (zoo)	18	67	0.08
69	Celebese ape	Washington (zoo)	18	67	0.64
70	Gorilla	Washington (zoo)	9	67	0.04

5. Avoid column borders (vertical lines), and only use horizontal borders (lines) for header and the bottom.

Sometimes for big tables, approaches like this alternate shading of rows are used to help the reader follow across a row.

2. Making Tables – Principals to Remember

1. Have an informative heading
2. Label columns and rows
3. Include the units of measurement and number of samples
4. Keep the number of decimal places consistent among cells, and consider experimental precision when deciding how many to report
5. Avoid vertical borders, between columns

As long as you state your sample size in the Methods section of a report, it is ok not to report sample size or degrees of freedom in the results section.

True?

False?

Data presentation

3. Figures

LETTER

doi:10.1038/nature10921

IFITM3 restricts the morbidity and mortality associated with influenza

Aaron R. Everitt[1], Simon Clare[1], Thomas Pertel[2], Sinu P. John[2], Rachael S. Wash[1], Sarah E. Smith[1], Christopher R. Chin[2], Eric M. Feeley[2], Jennifer S. Sims[2], David J. Adams[1], Helen M. Wise[3], Leanne Kane[1], David Goulding[1], Paul Digard[3], Verneri Anttila[1], J. Kenneth Baillie[4,5], Tim S. Walsh[5], David A. Hume[4], Aarno Palotie[1], Yali Xue[1], Vincenza Colonna[1,6], Chris Tyler-Smith[1], Jake Dunning[7], Stephen B. Gordon[8], The GenISIS Investigators*, The MOSAIC Investigators*, Rosalind L. Smyth[9], Peter J. Openshaw[7], Gordon Dougan[1], Abraham L. Brass[2,10] & Paul Kellam[1,11]

The 2009 H1N1 influenza pandemic showed the speed with which a novel respiratory virus can spread and the ability of a generally mild infection to induce severe morbidity and mortality in a subset of the population. Recent *in vitro* studies show that the interferon-inducible transmembrane (IFITM) protein family members potently restrict the replication of multiple pathogenic viruses[1–7]. Both the magnitude and breadth of the IFITM proteins' *in vitro* effects suggest that they are critical for intrinsic resistance to such viruses, including influenza viruses. Using a knockout mouse model[8], we now test this hypothesis directly and find that IFITM3 is essential for defending the host against influenza A virus *in vivo*. Mice lacking *Ifitm3* display fulminant viral pneumonia when challenged with a normally low-pathogenicity influenza virus, mirroring the destruction inflicted by the highly pathogenic 1918 'Spanish' influenza[9,10]. Similar increased viral replication is seen *in vitro*, with protection rescued by the re-introduction of *Ifitm3*. To test the role of IFITM3 in human influenza virus infection, we assessed the *IFITM3* alleles of individuals hospitalized with seasonal or pandemic influenza H1N1/09 viruses. We find that a statistically significant number of hospitalized subjects show enrichment for a minor *IFITM3* allele (SNP rs12252-C) that alters a splice acceptor site, and functional assays show the minor CC genotype IFITM3 has reduced influenza virus restriction *in vitro*. Together these data reveal that the action of a single intrinsic immune effector, IFITM3, profoundly alters the course of influenza virus infection in mouse and humans.

IFITM3 was identified in a functional genomic screen as mediating resistance to influenza A virus, dengue virus and West Nile virus infection *in vitro*[1]. However, the role of the IFITM proteins in antiviral immunity *in vivo* is unknown. Therefore, we infected mice that are homozygous for a disruptive insertion in exon 1 of the *Ifitm3* gene that abolishes its expression[8] (*Ifitm3*$^{-/-}$) with a low-pathogenicity murine-adapted H3N2 influenza A virus (A/X-31). Low-pathogenicity strains of influenza do not normally cause extensive viral replication throughout the lungs, or cause the cytokine dysregulation and death typically seen after infection with highly pathogenic viral strains[9], at the doses used (Fig. 1a). However, low-pathogenicity-infected *Ifitm3*$^{-/-}$ mice became moribund, losing >25% of their original body weight and showing severe signs of clinical illness (rapid breathing, piloerection) 6 days after infection. In comparison, wild-type littermates shed <20% of their original body weight, before fully recovering (Fig. 1a, b). There was little difference in virus replication in the lungs during the first 48 h

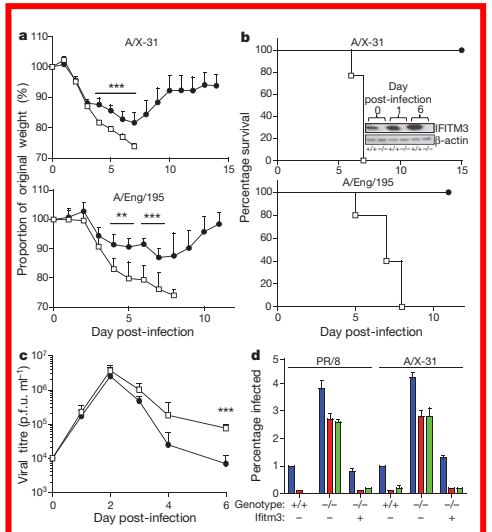
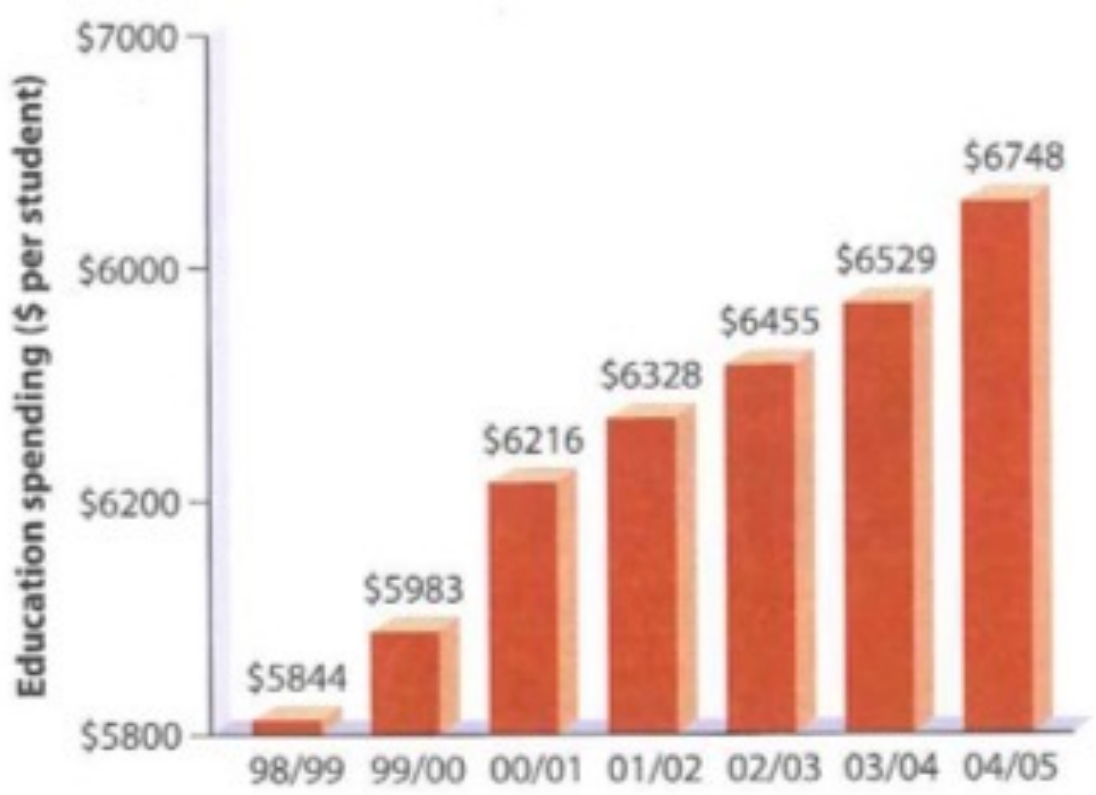


Figure 1 | Influenza A virus replicates to higher levels in *Ifitm3*$^{-/-}$ mice. **a**, **b**, Change in body mass (**a**) and survival (**b**) of wild-type (filled circles) and *Ifitm3*$^{-/-}$ (open squares) mice following intranasal inoculation with A/X-31 and pandemic H1N1/09 Eng/195 influenza ($n > 5$). **b**, Absence of Ifitm3 expression was verified in the *Ifitm3*$^{-/-}$ mice at all time points, but was seen to increase in wild-type mice. **c**, A/X-31 viral load in the lungs of mice ($n > 4$) was calculated over the course of infection by plaque assay. p.f.u., plaque-forming units. *Ifitm3*$^{-/-}$ murine embryonic fibroblasts ($n = 3$ per condition) stably expressing *Ifitm3* (+), or the empty vector (−) were left untreated (blue), or incubated with IFN-α (red) or IFN-γ (green), then challenged with either A/X-31 or PR/8 influenza. **d**, Twelve hours after infection, the cells were assessed for either haemagglutinin expression (PR/8), or nucleoprotein expression (A/X-31) IFITM3 expression was determined to be present (+) or absent (−) by western blotting (Supplementary Fig. 2). Results show means ± s.d. Statistical significance was assessed by Student's *t*-test (**$P < 0.01$; ***$P < 0.001$).

[1]Wellcome Trust Sanger Institute, Wellcome Trust Genome Campus, Hinxton CB10 1SA, UK. [2]Ragon Institute of Massachusetts General Hospital, Massachusetts Institute of Technology, and Harvard University, Charlestown, Massachusetts 02129, USA. [3]Divison of Virology, Department of Pathology, University of Cambridge, Tennis Court Road, Cambridge CB2 1QP, UK. [4]Division of Genetics and Genomics, The Roslin Institute, University of Edinburgh, Roslin EH25 9RG, UK. [5]Department of Critical Care Medicine, University of Edinburgh, Edinburgh EH16 4TJ, UK. [6]Institute of Genetics and Biophysics "A. Buzzati-Traverso", National Research Council (CNR), Naples, Italy. [7]Centre for Respiratory Infection, National Heart and Lung Institute, St Mary's Campus, Imperial College London, W2 1PG, UK. [8]Liverpool School of Tropical Medicine, Pembroke Place, Liverpool L3 5QA, UK. [9]Institute of Translational Medicine, University of Liverpool, Alder Hey Children's Hospital, Liverpool L12 2AP, UK. [10]Gastrointestinal Unit, Massachusetts General Hospital, Boston, Massachusetts 02117, USA. [11]UCL/MRC Centre for Medical Molecular Virology, Department of Infection, University College London, Cleveland Street, London W1T 4JF, UK.
*Lists of participants and their affiliations appear at the end of the paper.

3. Making Graphs

How can we improve this?



Source: Whitlock and Schluter, 2009 p 45

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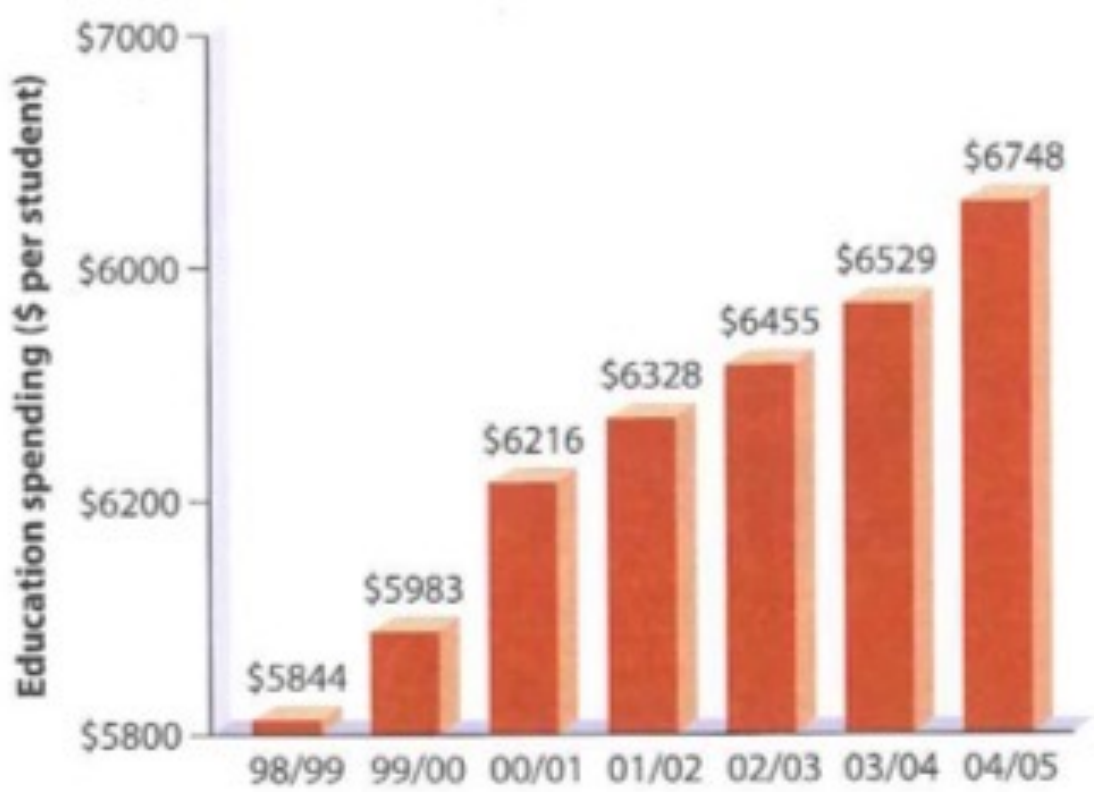

1. Always label both axes, and include units of measurement

Source: Whitlock and Schluter, 2009 p 45

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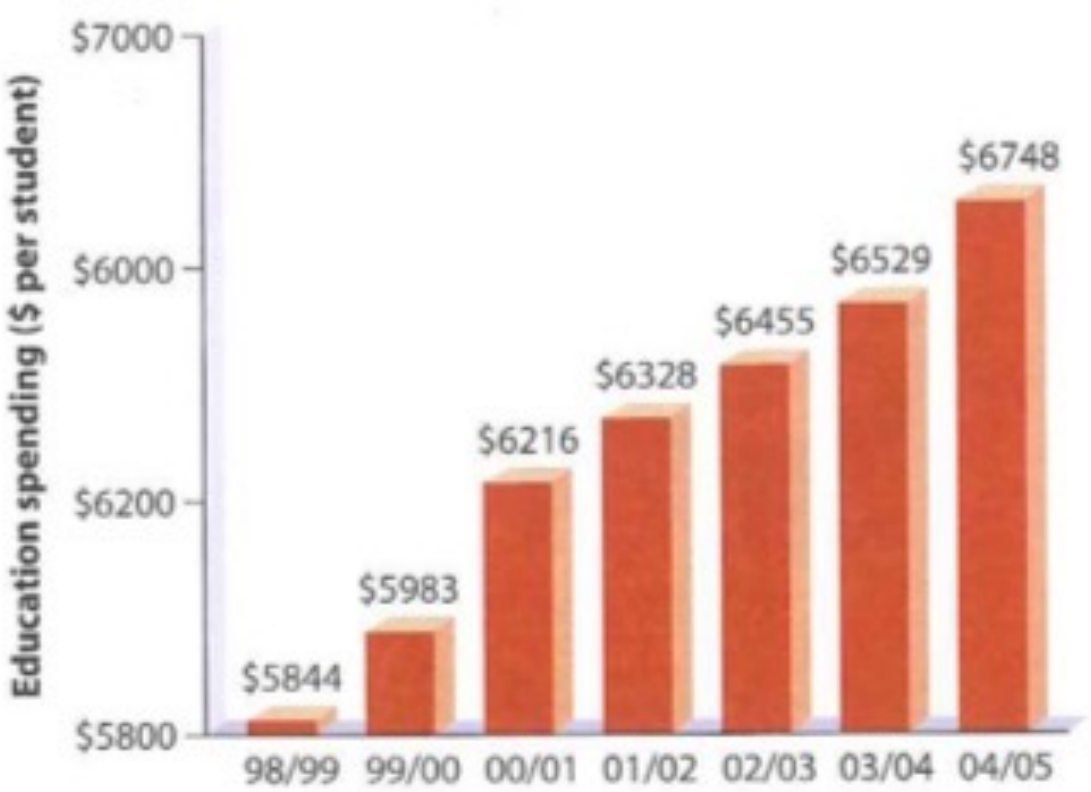

1. Always label both axes, and include units of measurement
2. Choose a meaningful scale

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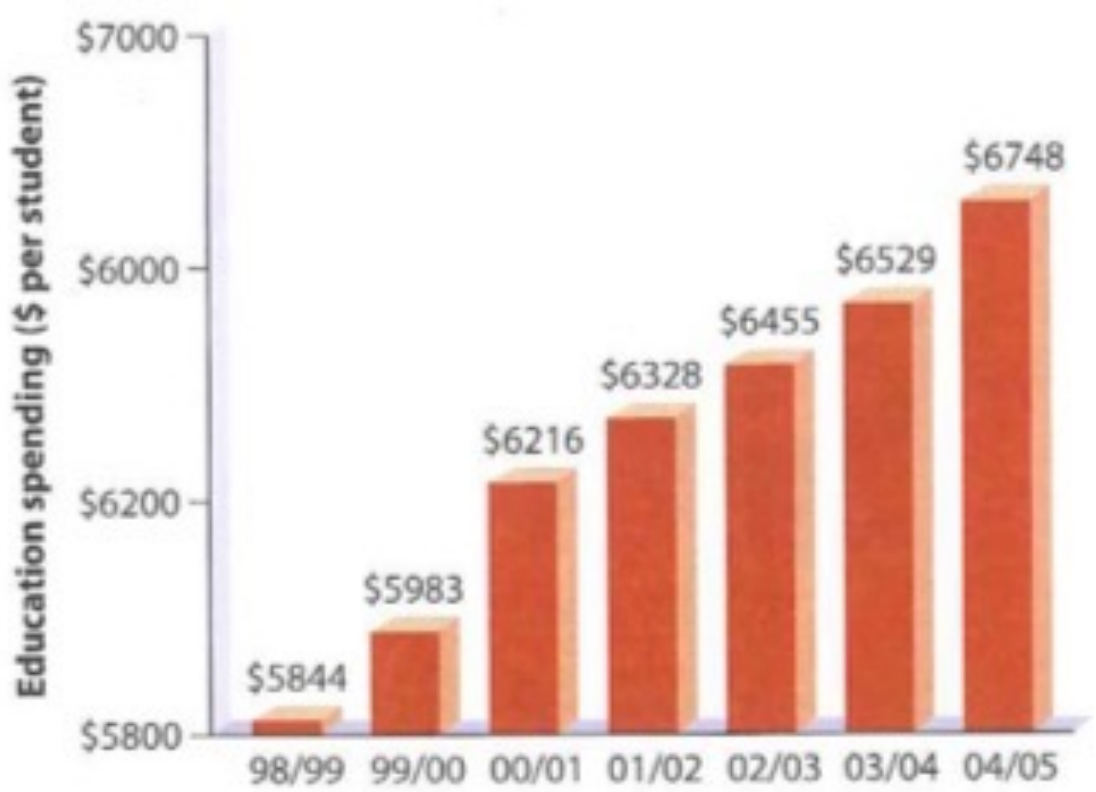

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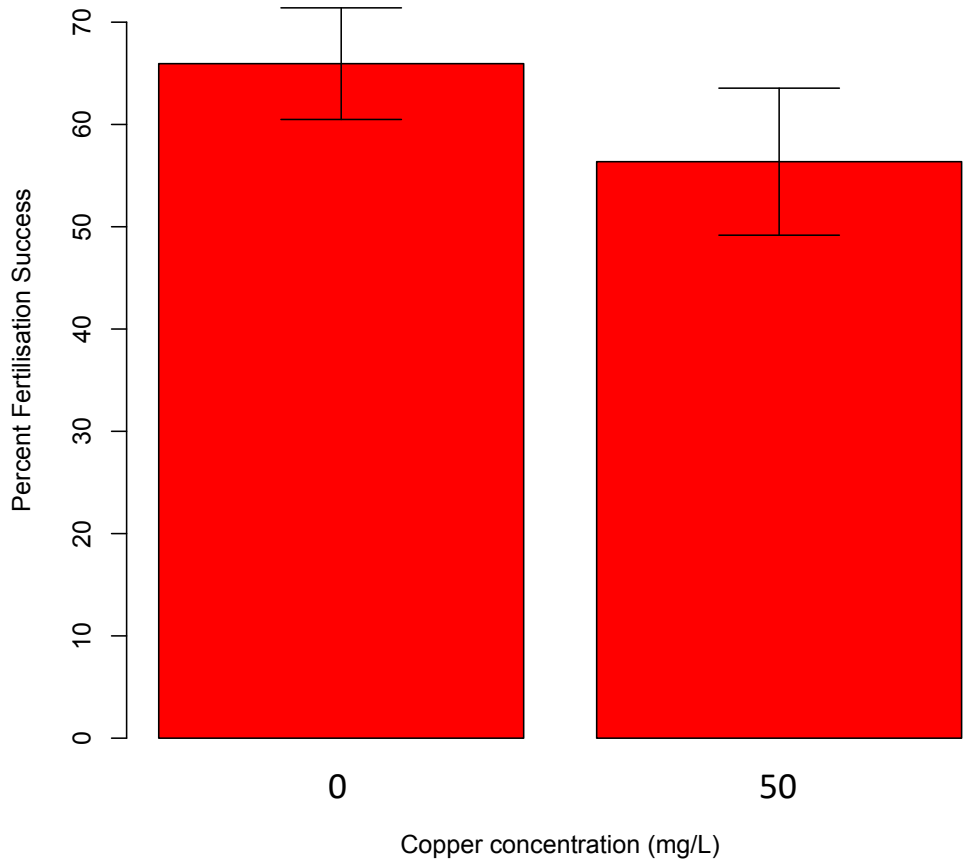
2. Choose a meaningful scale
3. Don't add features that don't convey information – *such as 3D bars to plot 2D data*

Source: Whitlock and Schluter, 2009 p 45

3. Making Graphs

Doing it Better



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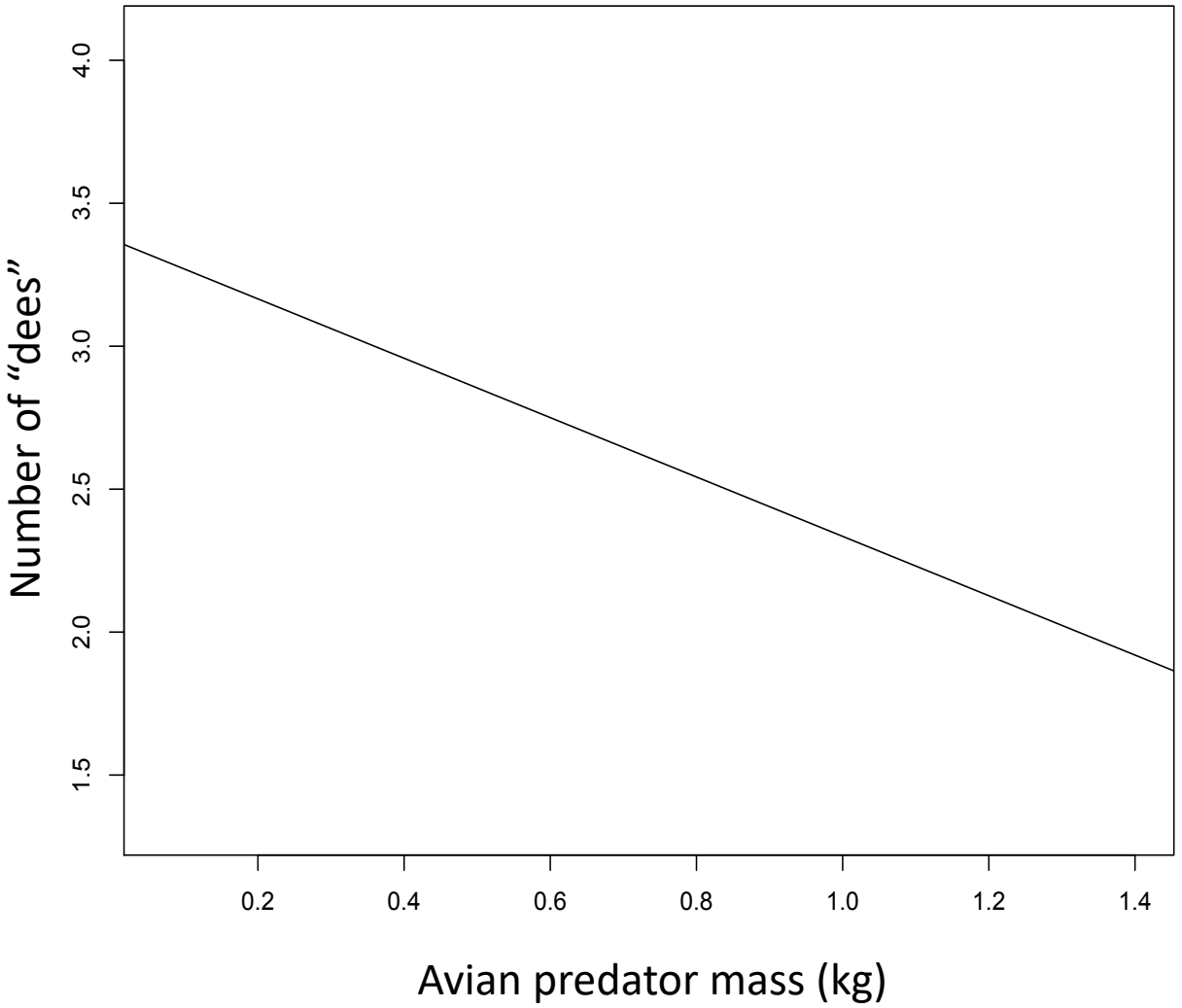

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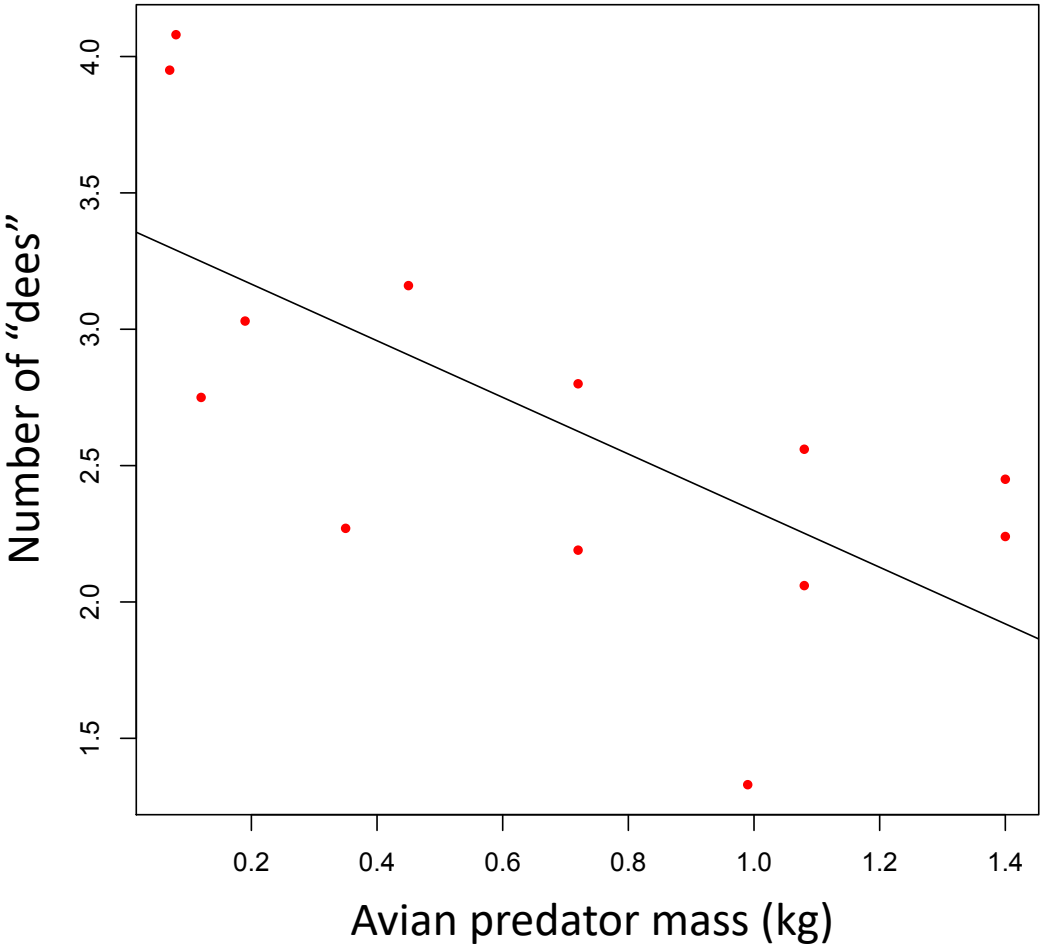
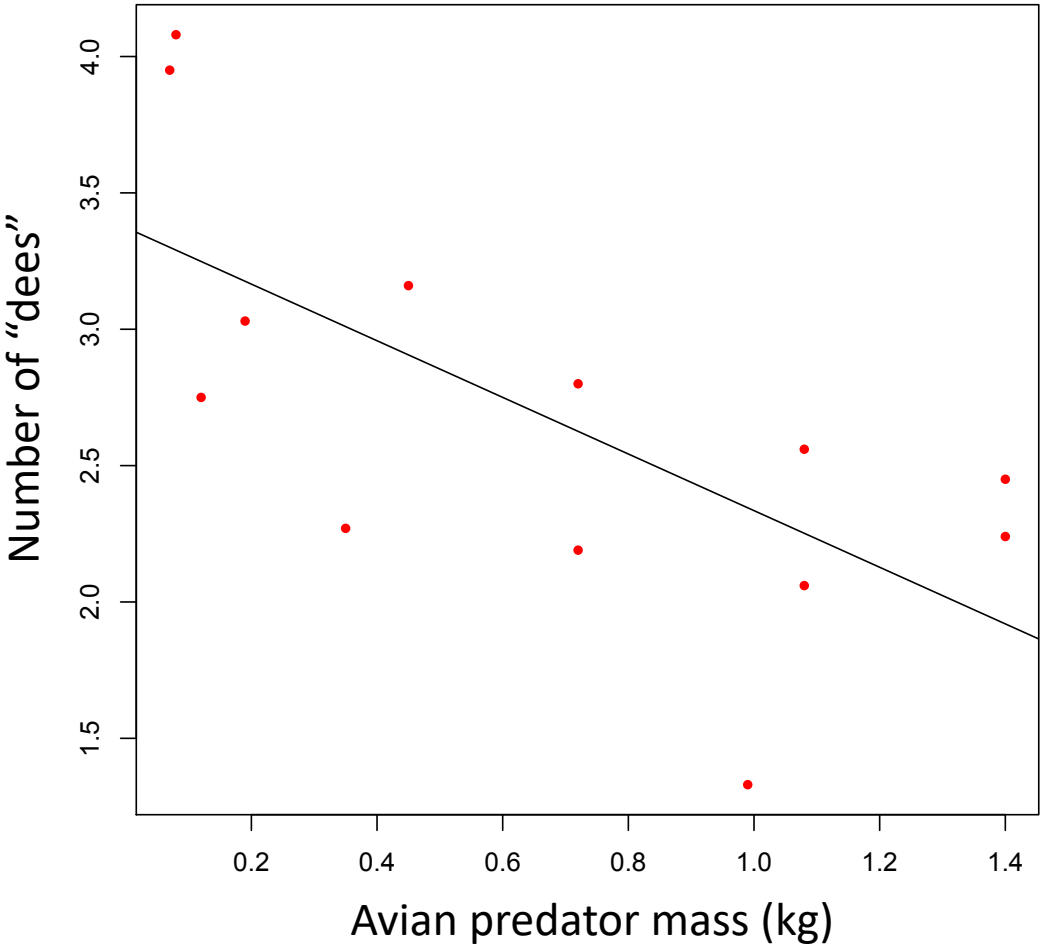


4. Show the data

3. Making Graphs

How can we improve this?



4. Show the data
5. Include a figure legend

3. Making Graphs

How can we improve this?

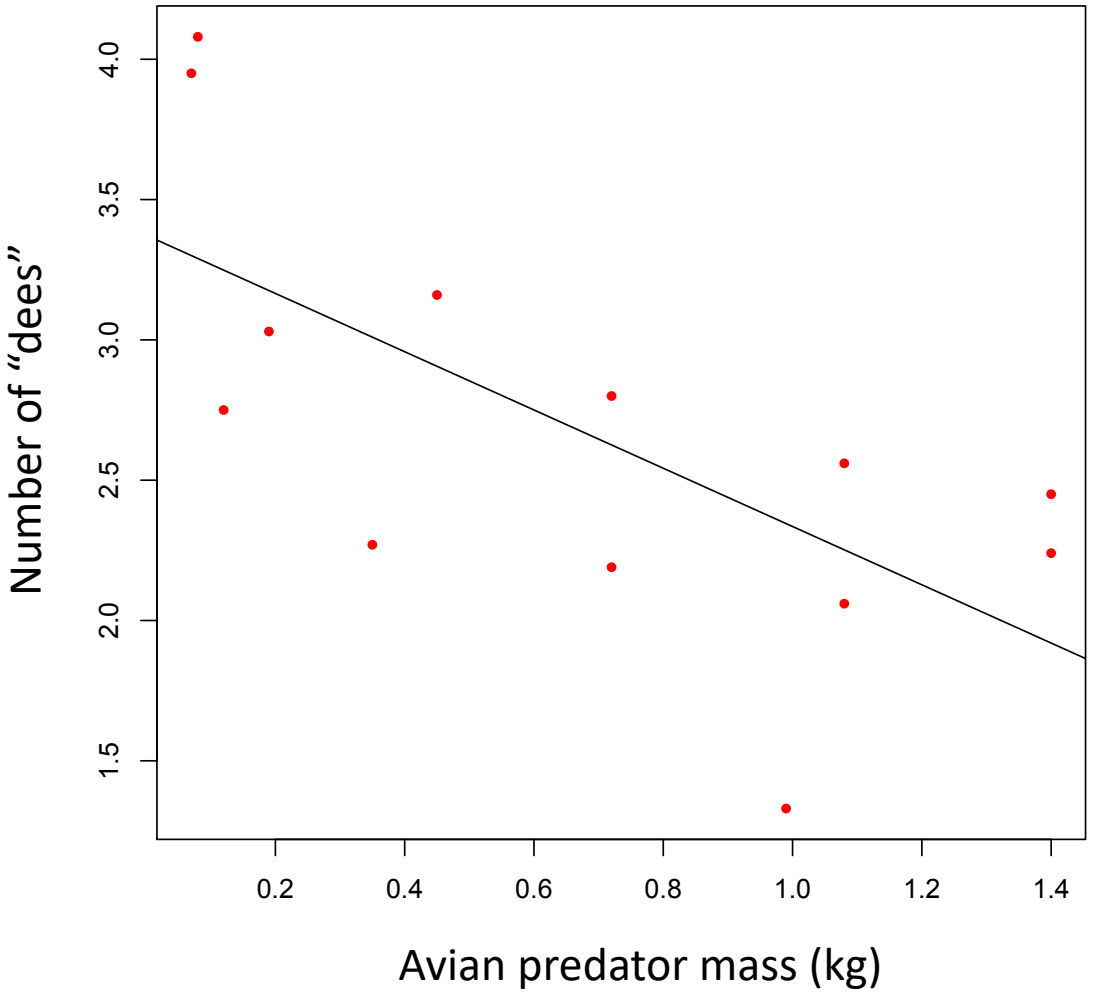


Figure 1. The relationship between the size of avian predators and the number of "dees" in chickadee calls. Calls were recorded during 13 separate observation periods when a predator was present, and "dees" per call counted. The predator was identified to species level, and the mass (in kg) of the predator was determined as a species-average from data collected in a separate study. Data is the "dee" count and predator mass for each of the 13 encounters (shown in red). The least squares mean regression slope is plotted (in black).

3. Making Graphs

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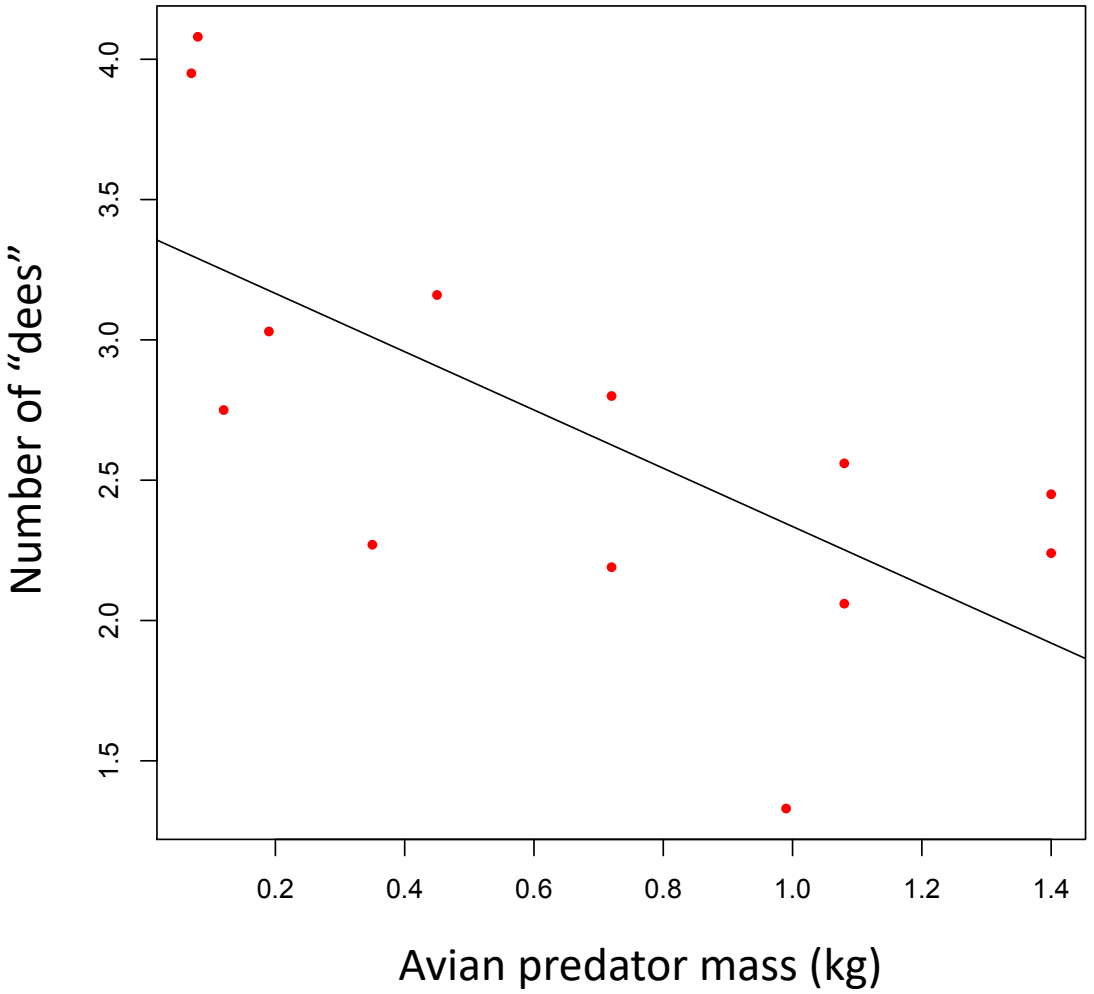

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- Units of measurement

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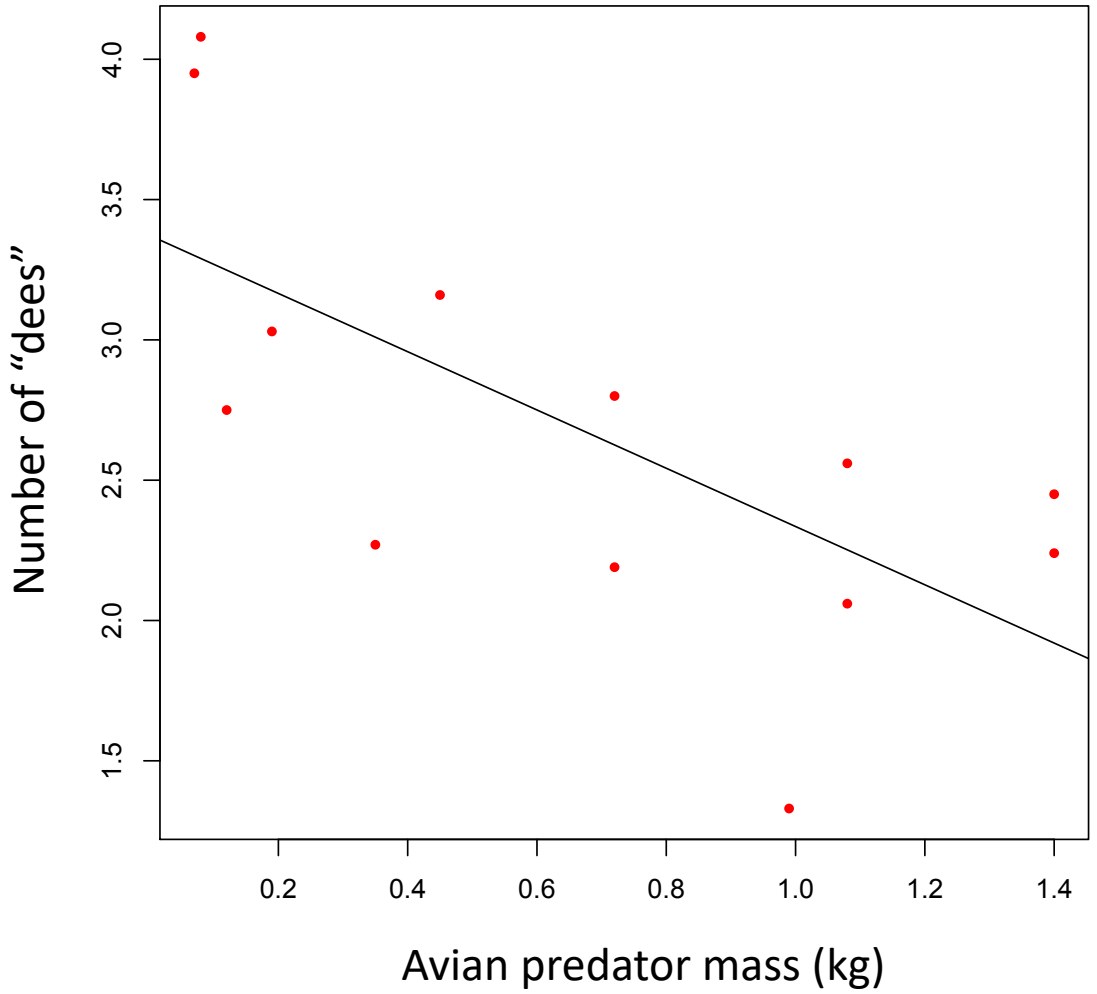

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- Units of measurement
- Sample size

3. Making Graphs

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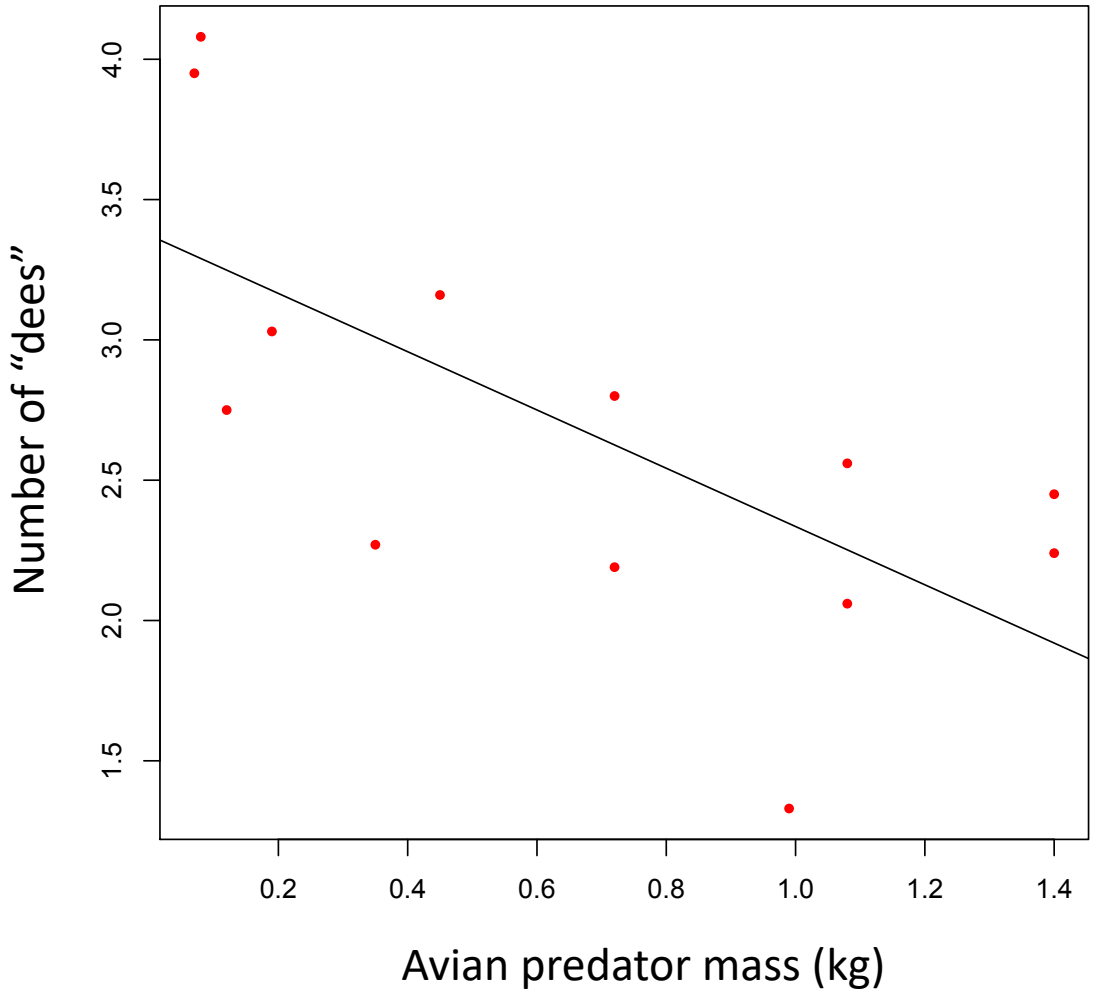

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- Units of measurement
- Sample size
- all elements of the graph

3. Making Graphs

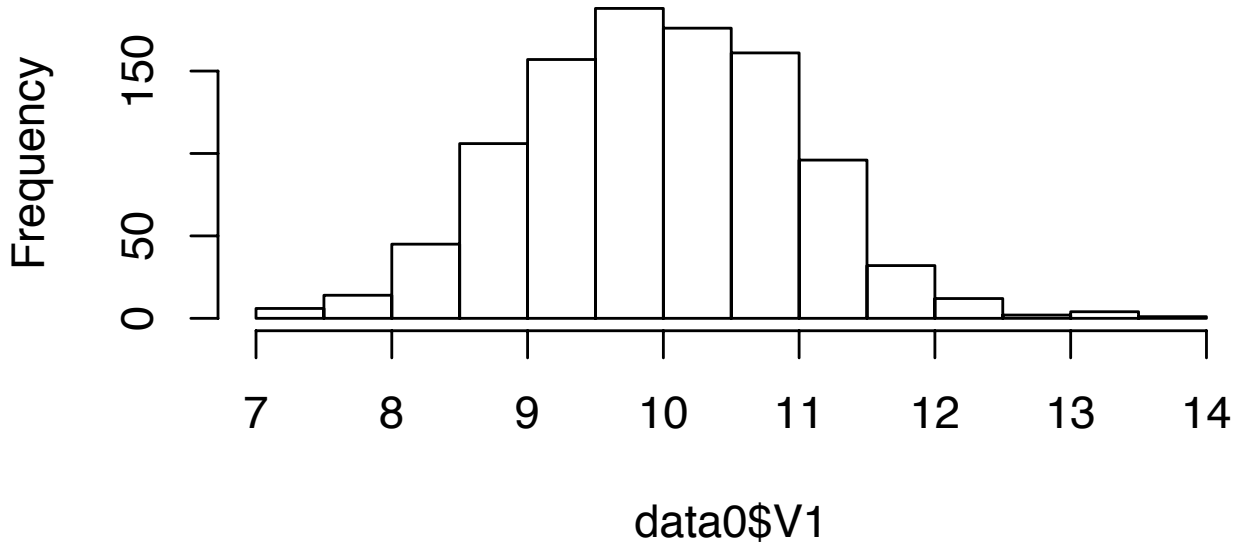
- Choose a meaningful scale
- Label axes clearly and include units
- Maximise “data-to-ink” ratio (i.e. minimise clutter)
    - Don’t use lots of redundant graphics because you think it looks good. Your goal is to convey information, make sure the information doesn’t get lost.
- Show the data
- Include a figure caption
- Match your graphical display to your data analysis

4. Presenting results in a talk

- Follow the same rules for in text, tables and figures!
- Keep it simple
  - One result at a time!

- Make it BIG
  - Could someone in the back row see the points on your graph?
  - Can they read the labels?

How can we improve this graph?



How can we improve this graph?

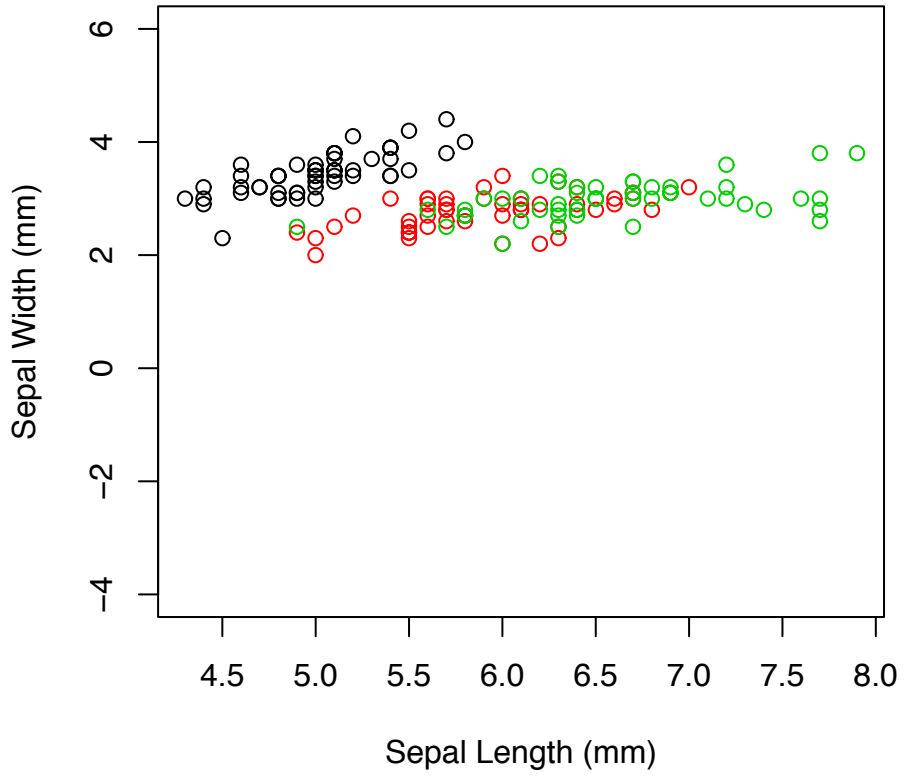


Figure 1. The relationship between sepal width and length.

4. Presenting results in a talk

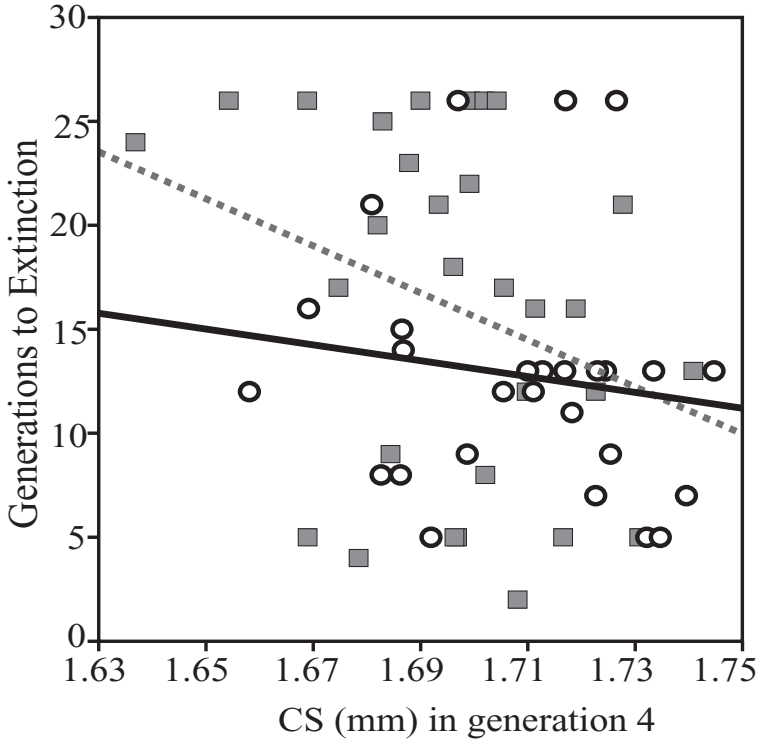
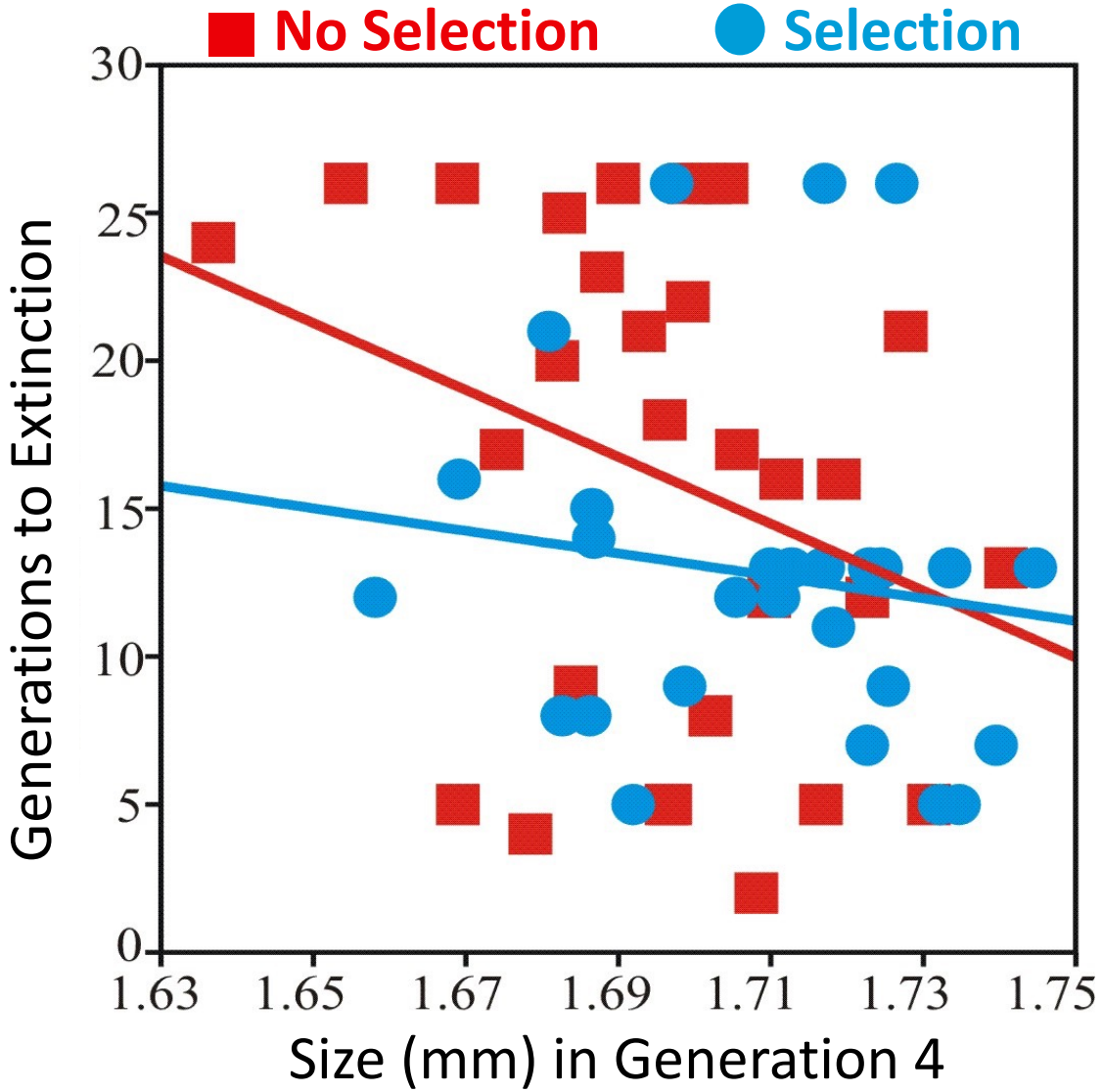
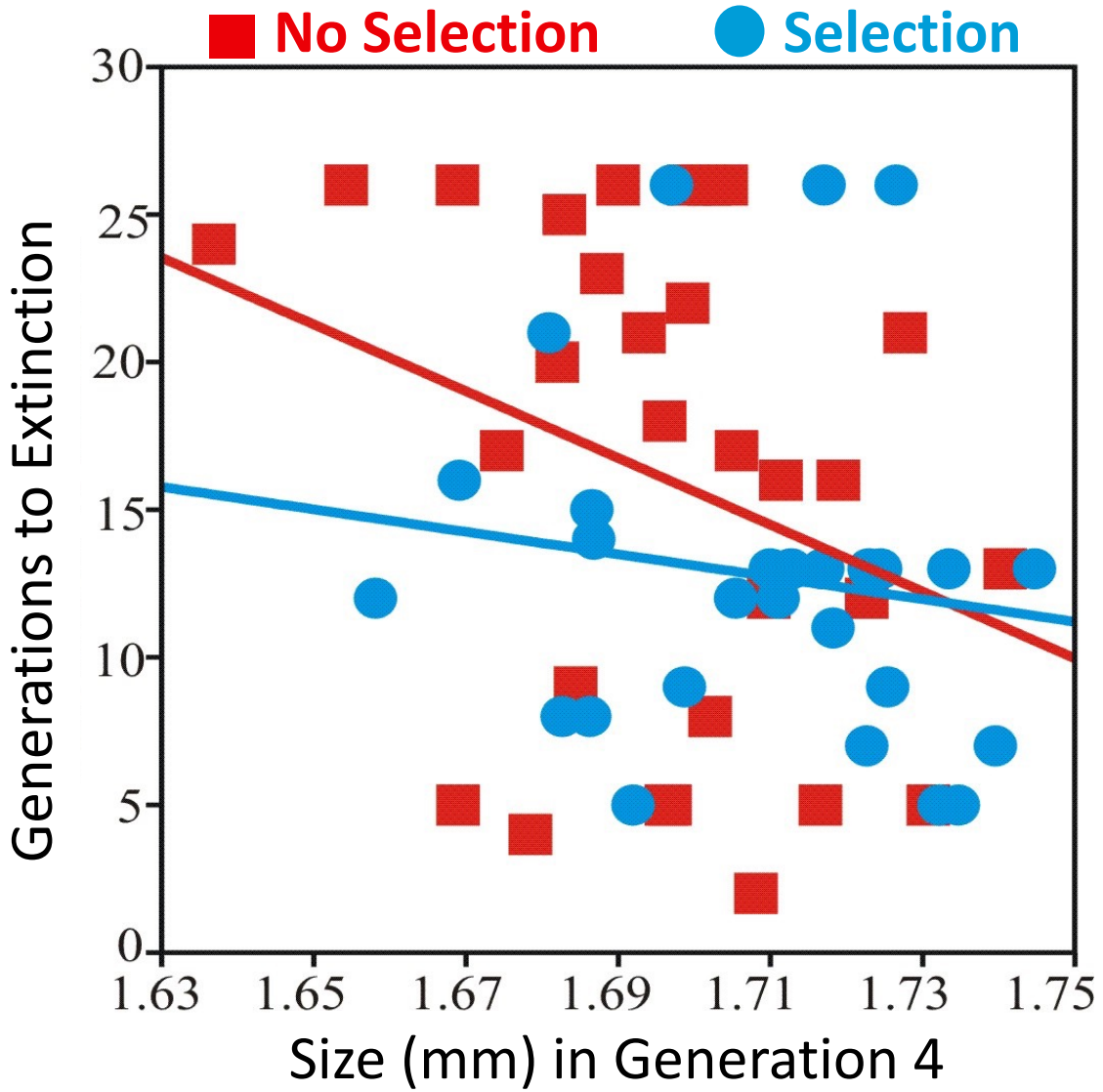


Figure 3. The relationship between the time to extinction (fitness) and mean CS of MA lines that went extinct prior to the conclusion of the experiment for N (shaded squares; dashed line) and S (open circles; solid line) treatments.



4. Presenting results in a talk



$\beta = -0.124, P = 0.0513$

$\beta = -0.047, P = 0.5563$