Supplementary Information

Supplementary Methods S1: Modelling direct and indirect genetic effects

After accounting for the effects of fixed factors and residual autocorrelation, the adjusted mycelium growth rate of a focal genotype *i* growing on the p^{th} plate in stack s was:

$$z_{i_p s} = D_{i_p} + \sum_{k=p-3}^{p-1} f_{j_k} N_{j_k}^{below} + \sum_{k=p+1}^{p+3} f_{j_k} N_{j_k}^{above} + \sum_{k=p-3}^{p-1} f_{j_k} I_{i_p j_k}^{below} + \sum_{k=p+1}^{p+3} f_{j_k} I_{i_p j_k}^{above} + a_s + \varepsilon_{i_p s} \qquad (1),$$

where $z_{i_p s}$ is the growth rate of the focal genotype i and D_{i_p} is its direct genetic effect, j_k is the neighbour genotype growing at position k within the same stack and its main (respectively G x G) indirect genetic effect with the neighbour genotype is $N_{j_k}^{below}$ or $N_{j_k}^{above}$ (respectively $I_{i_p j_k}^{below}$ or $I_{i_p j_k}^{above}$) depending on whether it is placed below or above the focal genotype. f_{j_k} is the intensity of interaction factor (see below), a_s is a random stack effect and $\varepsilon_{i_p s}$ is the residual error term (see Figure S1 below). We did not assume the interactions between two genotypes to be reciprocal. In other words, the effect of a first genotype on the growth rate of a second genotype growing one plate below could differ from the effect of the second genotype on the growth rate of the first genotype (i.e. $I_{i_p j_{p-1}}^{below}$ and $I_{i_{p-1} j_p}^{above}$ were fitted independently).

For model with no genetic covariance between DGEs and main IGEs, we also tested for directionality of IGEs by comparing models where IGEs were similar or different for a given genotype placed above or below the focal plate (for main IGEs: $\boldsymbol{u_n} = \begin{pmatrix} \boldsymbol{u_{n\ above}} \\ \boldsymbol{u_{n\ below}} \end{pmatrix}$ with $V[\boldsymbol{u_{n\ below}}] = \sigma^2_{n\ below}\ I_{n\ below}$ and $V[\boldsymbol{u_{n\ above}}] = \sigma^2_{n\ above}\ I_{n\ above}$ and for G x G IGEs:$\boldsymbol{u_i} = \begin{pmatrix} \boldsymbol{u_{i\ above}} \\ \boldsymbol{u_{i\ below}} \end{pmatrix}$ with $V[\boldsymbol{u_{i\ below}}] = \sigma^2_{i\ below}\ I_{i\ below}$ and $V[\boldsymbol{u_{i\ above}}] = \sigma^2_{I\ above}\ I_{i\ above}$, see Table S2 for the dimensions of the different identity matrices).

To determine the distance over which IGEs occurred, we compared a model where the intensity of IGEs decreased with the inverse of the distance between the plates of the focal

and neighbour genotypes (intensity of interaction factors: $f_{j_{p-1}} = f_{j_{p+1}} = 1$, $f_{j_{p-2}} = f_{j_{p+2}} = \frac{1}{2}$ and $f_{j_{p-3}} = f_{j_{p+3}} = \frac{1}{3}$, Costa e Silva and Kerr, 2013, "distance effect" in Table S2) to a model where IGEs only occur with neighbour genotypes one plate away from the focal genotype (intensity of interaction factors: $f_{j_{p-1}} = f_{j_{p+1}} = 1$ and $f_{j_{p-2}} = f_{j_{p+2}} = f_{j_{p-3}} = f_{j_{p+3}} = 0$, "no distance effect" in Table S2).

To test for differences in IGEs for a given neighbour genotype placed above or below a focal plate (i.e. directionality of IGEs), we compared the model in equation 1 to a model in which a given neighbour genotype had the same IGE when placed above or below the focal genotype (i.e. $N_{j_k}^{below} = N_{j_k}^{above}$ and $I_{i_p j_k}^{below} = I_{i_p j_k}^{above}$).

For datasets 1 and 2, the adjusted growth rate could be respectively modelled as:

$$z_{i_p s} = D_{i_p} + f_{j_{p-1}} N_{j_{p-1}}^{below} + f_{j_{p+1}} N_{j_{p+1}}^{above} + f_{j_{p-1}} I_{i_p j_{p-1}}^{below} + f_{j_{p+1}} I_{i_p j_{p+1}}^{above} + a_s + \varepsilon_{ip} \quad (2)$$

and

$$z_{i_p s} = D_{i_p} + \sum_{k=p-2}^{p-1} f_{j_k} N_{j_k}^{below} + \sum_{k=p+1}^{p+2} f_{j_k} N_{j_k}^{above} + \sum_{k=p-2}^{p-1} f_{j_k} I_{i_p j_k}^{below} + \sum_{k=p+1}^{p+2} f_{j_k} I_{i_p j_k}^{above} + a_s + \varepsilon_{i_p s} \quad (3).$$

See main text and text above for details.

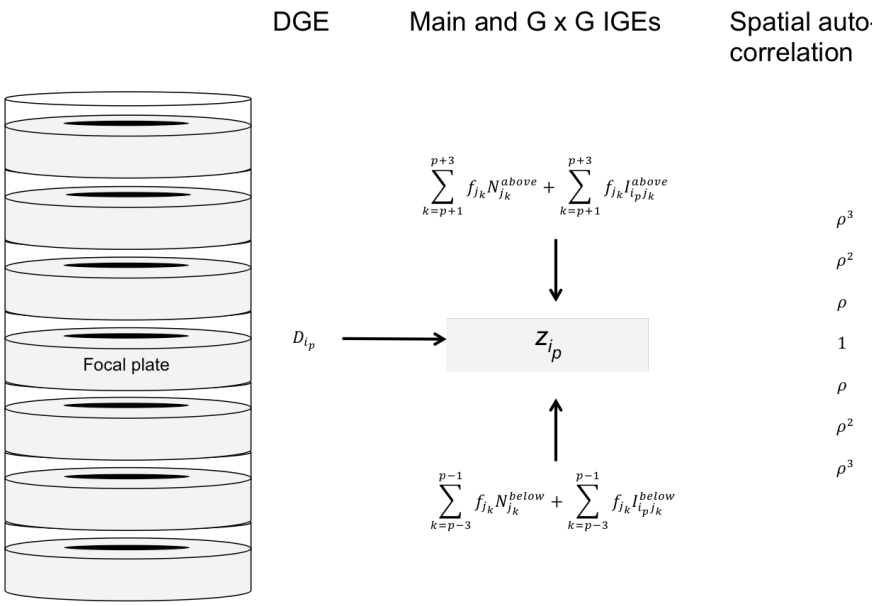

Figure S1 Schematic representation of the different genetic effects fitted (the genetic covariance between D_{i_p} and N_j has been omitted for clarity). D_{i_p} is the DGE of the focal genotype, the different N_j and $I_{i_p j}$ respectively correspond to the main and G x G IGEs of the different neighbour genotypes indexed according to their position relative to the focal genotype at the p^{th} position within the stack. See text above for details.

Supplementary Methods S2: Potential origin of main IGEs

Let's assume that IGEs are proportional to the genotypic growth rate of the neighbour genotype (e.g. the production of a signalling molecule is proportional to growth rate). We can imagine two scenarios that both predict strong main IGEs. In the first scenario, imagine that the growth rate of a focal genotype is altered independently of its own genotypic growth rate value because some genotypes inhibit or stimulate all their neighbours similarly (i.e. there would be universal inhibitor/stimulator genotypes) by depleting a common resource such as oxygen. Under this scenario, we expect to detect a main IGEs and a correlation between DGEs and main IGEs.

In the second scenario, imagine the growth rate of a focal genotype is altered in proportion to the difference between its own genotypic value for growth rate and the genotypic value of its neighbour. Now, a focal strain with an average growth rate would grow

slower when placed with a fast growing neighbour genotype, but would grow faster when placed with a slow growing neighbour genotype. If the IGE is directly proportional to the difference of genotypic values between the focal strain and its neighbour, we have the following expression:

$$\boldsymbol{z_{ijk} = \mu + D_i + \alpha(D_i - D_j) + \varepsilon_{ijk}} \quad (4a),$$

where $\boldsymbol{z_{ijk}}$, is the growth rate of a focal strain of genotype $\boldsymbol{i}$ with genotypic value, $\boldsymbol{D_i}$, placed close to a neighbour strain $\boldsymbol{j}$ with genotypic value D_j. $\boldsymbol{\alpha}$ is the interaction coefficient which relates the observed IGEs to the difference in DGEs between focal and neighbour strains. $\boldsymbol{\varepsilon_{ijk}}$ is the measurement error. Equation 3a can be rewritten as:

$$\boldsymbol{z_{ijk} = \mu + D'_i + D'_j + \varepsilon_{ijk}} \quad (4b),$$

where $\boldsymbol{D'_i}$ is the DGE of focal genotype $\boldsymbol{i}$ (with $\boldsymbol{D'_i = (1 + \alpha)D_i}$) and $\boldsymbol{D'_j}$ represents the main IGE of neighbour genotype $\boldsymbol{j}$ (with $\boldsymbol{D'_j = \alpha D_j}$). In other words, the average growth rate of a focal strain across all neighbours would depend on $\boldsymbol{D_i}$ and $\boldsymbol{\alpha}$, while the effect average effect of a genotype on the growth of its neighbours would depend on $\boldsymbol{D_j}$ and $\boldsymbol{\alpha}$. Although the IGEs biologically represent a G x G IGEs (as the effect of a neighbour genotype depends on the genotype of the focal genotype), it represents a main IGE statistically. Hence, we expect to detect a main IGEs and no G x G IGEs under this scenario.

Supplementary Methods S3: Power analyses

Relative importance of main and G x G IGEs

As the number of levels available to estimate main and G x G IGEs decreased in dataset 3 compared to datasets 1 and 2 (Table S2), we estimated our power of detecting effects, given that they exist, using simulation-based power analyses based on our experimental design (Johnson *et al.*, 2015). We used the estimates from the model with lowest AICc for each variance component. In the first power analysis, we simulated datasets with intensity of interaction factors with neighbour genotypes that were proportional to the distance between

focal and neighbour genotypes (see above). In the second power analysis, we simulated datasets with different $\sigma^2_{n_{p\pm1}}$ (variance of main IGEs due to genotypes one plate way from the focal genotype). The proportion of variance explained by $\sigma^2_{n_{p\pm1}}$ ranged from 0 to 17% ($\sigma_{n_{p\pm1}} =$ 0.001, 0.01, 0.02, 0.025, 0.03, 0.035, 0.04, 0.045, 0.05, 0.055, 0.06, 0.065, 0.075, 0.085, 0.1, 0.125, 0.15, 0.2, respectively). In the third power analysis, we simulated datasets with different $\sigma^2_{n_{p\pm1}}$ (variance of main IGEs due to genotypes one plate way from the focal genotype) and different $\sigma_{dn_{p\pm1}}$ (covariance between the colony diameter of a focal genotype and its inhibition effect as a neighbour one plate away from a focal genotype). The proportion of variance explained by $\sigma^2_{n_{p\pm1}}$ ranged from 0 to 17% ($\sigma_{n_{p\pm1}} =$ 0.01,0.025,0.05,0.06,0.07,0.075,0.08,0.09,0.1,0.125, respectively), while the values of correlation ($\frac{\sigma_{dn_{p\pm1}}}{\sigma^2_d\sigma^2_{n_{p\pm1}}}$) ranged between 0.01 and 0.9 ($\rho_{dn_{p\pm1}} =$ 0.0001,0.005,0.01,0.05,0.1,0.5,0.75,0.9). We simulated independently 1000 datasets for distance-dependent intensity of interaction factor, for each value of $\sigma^2_{n_{p\pm1}}$ and for each combination of $\sigma^2_{n_{p\pm1}}$ and $\sigma_{dn_{p\pm1}}$. We analysed each dataset using either the original model used for the simulations or an alternative model without the tested effect. Power was estimated as the proportion of the 1000 datasets in which the model used for the simulations for amongst the best model (i.e. $\mathrm{AICc(reduced\ model)} - \mathrm{AIC(original\ model)} > -2$. We used an arbitrary threshold of a power of 80% for the analyses (Johnson *et al.*, 2015).

Supplementary Results S1

Relative importance of main and G x G IGEs

For datasets 1, 2 and 3, models including the effects of DGEs and G x G IGEs were strongly supported ($\Delta AIC > 2$ for models without these effects, Tables S3, S4 and S5). In contrast, models including additive IGEs were not supported in the analyses of datasets 1 and 2

($\Delta AIC > 2$ for models with these effects, Tables S3 and S4), and were weakly supported in the analysis of dataset 3 ($\Delta AIC = 1.16$ for a model including directional additive IGEs, Table S5). Models including different vs. the same effects when the neighbour strain was above or below the focal strain (directionality or non-directionally of the G x G IGEs) had different degrees of support depending on the datasets. Models with directional IGEs were weakly supported in dataset 1 ($\Delta AIC = 0.82$ for a model including these directional effects, Table S3), strongly supported in dataset 2 ($\Delta AIC > 2$ for models with non-directional IGEs, Table S4), and were not supported in dataset 3 ($\Delta AIC = 3.98$ for models with directional IGEs, Table S4). G x G IGEs with strains two plates away from the focal strain could only be investigated using datasets 2 and 3. Models including such G xG IGEs were highly supported in dataset 2 ($\Delta AIC = 6.40$ for a model excluding this effect, Table S4), but were weakly supported in dataset 3 ($\Delta AIC = 1.89$ for a model including this effect, Table S5).

Table S1 List of the 41 strains used for the experiment.

Table S2 Number of levels available to estimate DGEs (u_d), main IGEs (u_n, $u_{n\,below}$, $u_{n\,above}$), and G x G IGEs (u_i, $u_{i\,below}$, $u_{i\,above}$) in each of the three datasets analysed.

Table S3A Model selection of focal colony diameter as a function of DGEs, main and G x G IGEs for dataset 3.

Table S3B Incremental Wald test of the G x G IGE model including two fixed effects (agar status focal and agar status neighbour) for dataset 3.

Table S4A Model selection of focal colony diameter as a function of DGEs, main and G x G IGEs for dataset 1.

Table S4B Incremental Wald test of the G x G IGE model including two fixed effects (agar status focal and agar status neighbour) for dataset 1.

Table S5A Model selection of focal colony diameter as a function of DGEs, main and G x G IGEs for dataset 2.

Table S5B Incremental Wald test of the G x G IGE model including two fixed effects (agar status focal and agar status neighbour) for dataset 2.

Table S6 Estimates of the variance components fitted for DGEs, G x G IGEs and spatially correlated environmental effects (dataset 3).

References

Costa e Silva J, Kerr RJ (2013). Accounting for competition in genetic analysis, with particular emphasis on forest genetic trials. *Tree Genet genomes* **9**: 1–17.

Johnson PCD, Barry SJE, Ferguson HM, Müller P (2015). Power analysis for generalized linear mixed models in ecology and evolution. *Methods Ecol Evol* **6**: 133–142.

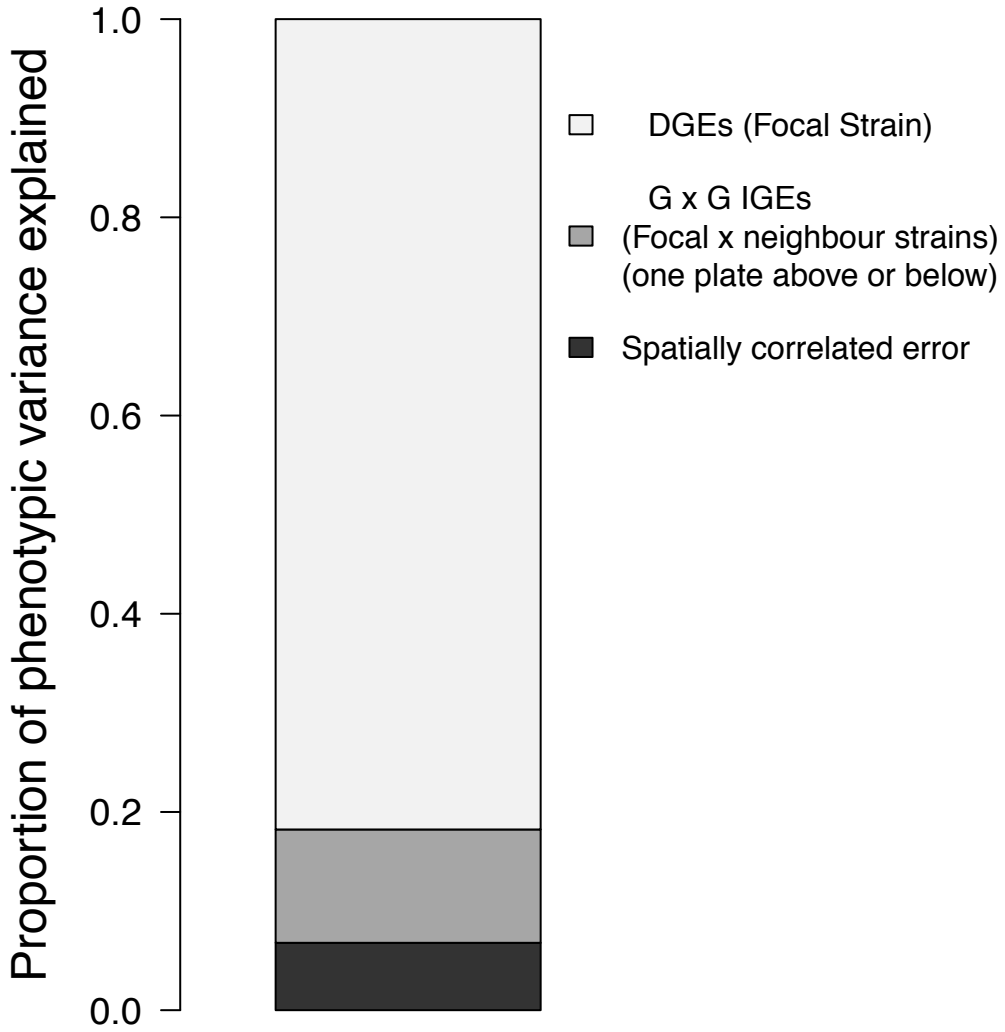

Figure S2 Proportion of variance in growth rate explained by direct genetic effects (DGEs = 81.8%, focal strain), genotype by genotype indirect genetic effects (G x G IGEs between focal and neighbour strains one plate apart = 11.4%) and environmental effects (spatially correlated error = 6.8%). Models including G x G IGEs between focal and neighbour strains more than one plate away or main IGEs had low support (see main text).

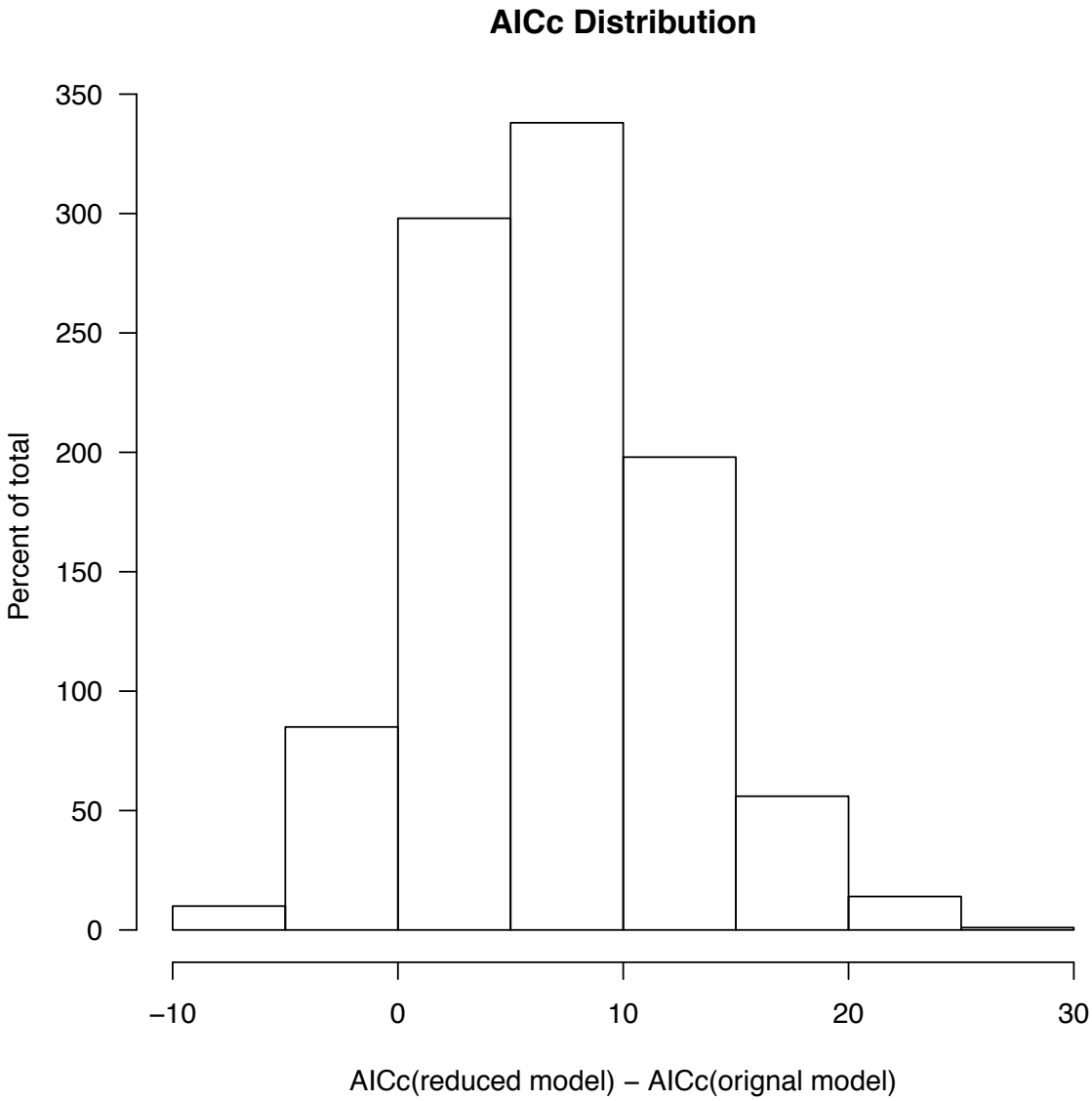

Figure S3 Distribution of AICc differences between the reduced model and the original model used for the simulation. The original model was among the best models in 97% of the simulations (n = 1000 simulated datasets, see Supplementary Methods S2).

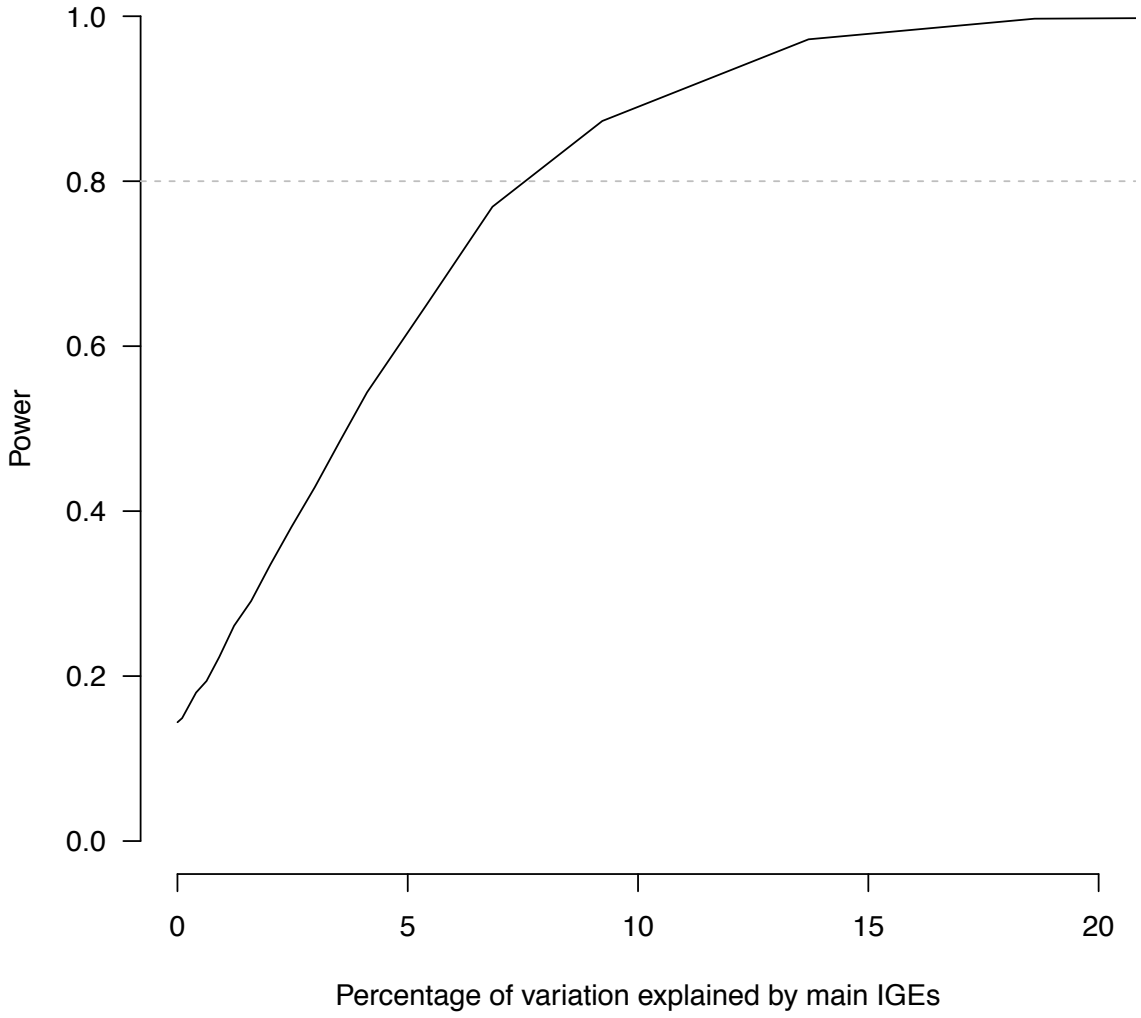

Figure S4 Power as a function of the proportion of the total phenotypic variance explained by the variance in main IGEs. Power was estimated based on 1000 simulated datasets for each value of $\sigma^2_{N_{p\pm1}}$ (see Supplementary Methods S2). The dashed line represents an arbitrary threshold power of 80%.

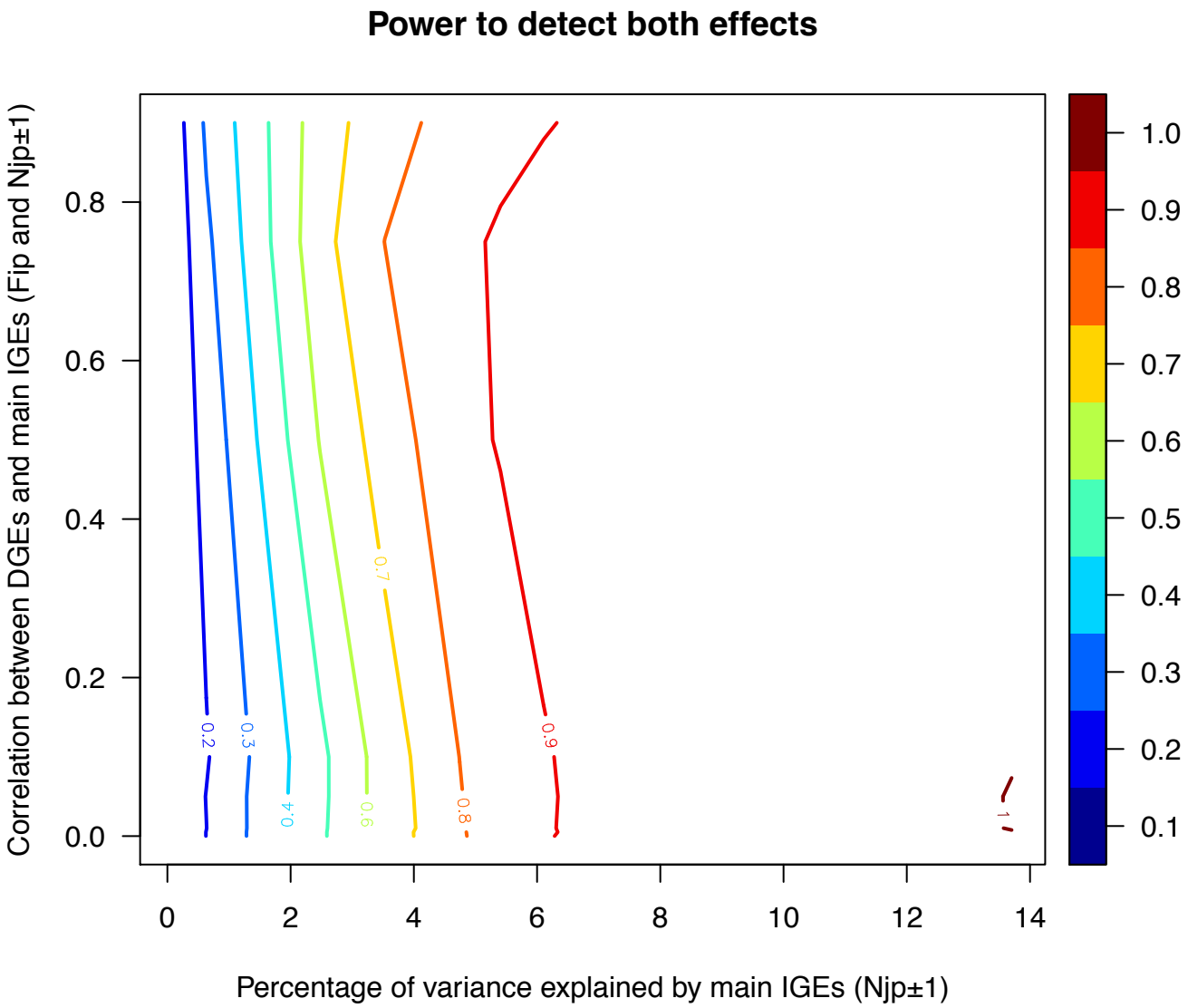

Figure S5 Power as a function of the proportion of the total phenotypic variance explained by the variance in main IGEs and by the genetic correlation between DGEs and these main IGEs. Power was estimated based on 100 simulated datasets for each value of $cor_{F,N_{p\pm1}}$ (see Supplementary Methods S2). The arbitrary threshold power of 80% is represented in orange.

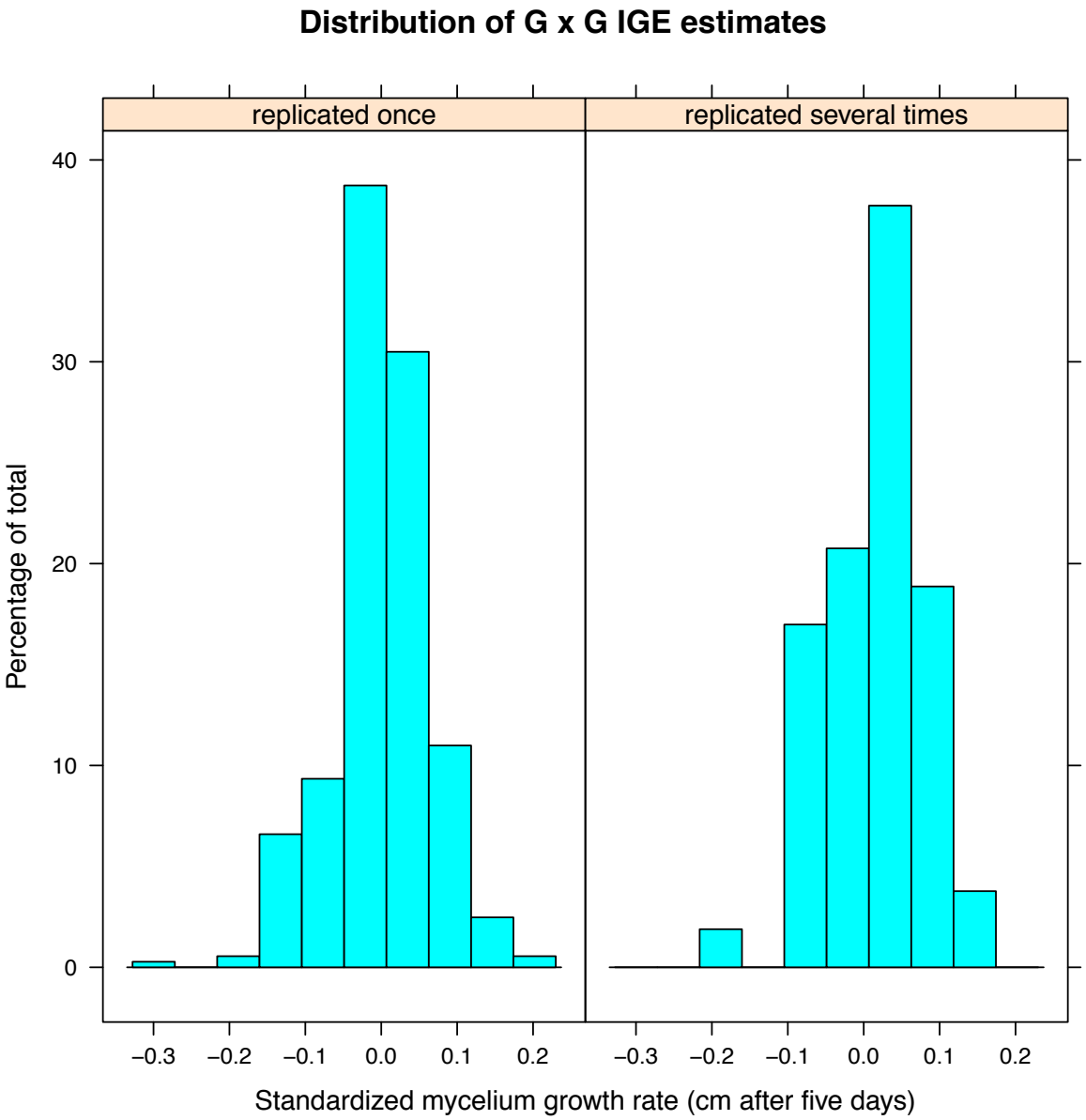

Figure S6 Distribution of G x G IGE estimates for pairs of strains replicated once or several times. The variance between pairs replicated only once does not seem greater than the variance between pair replicated several times.