Supporting Information

Neher and Shraiman 10.1073/pnas.0812560106

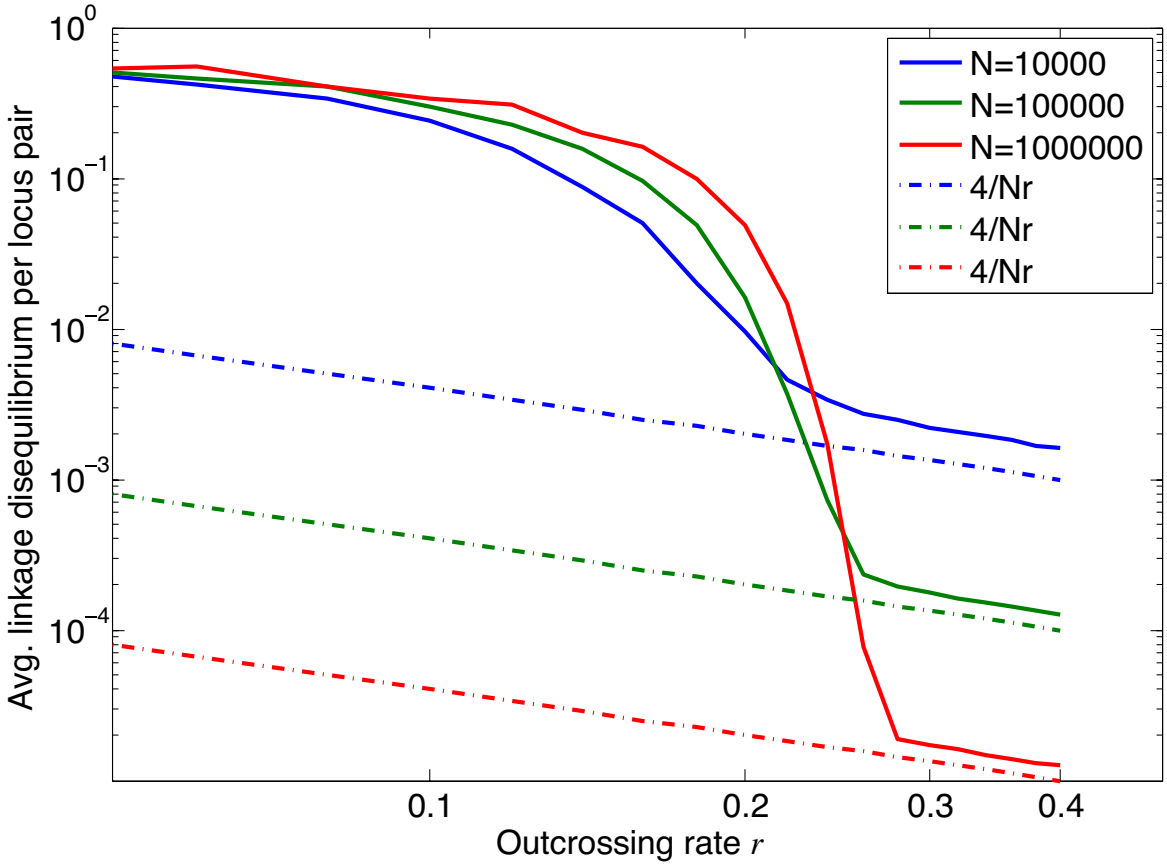

Fig. S1. The population size dependence of linkage disequilibrium. The plot shows the average linkage disequilibrium per locus pair $2/L/(L-1)\,\Sigma_{i<j}\psi_{ij}^2$ in the random epistasis model versus the outcrossing rate r for different population sizes N. Below r_c, LD does not depend strongly on N, whereas LD is proportional to $(Nr)^{-1}$ above r_c (dashed lines). The pairwise epistasis coefficients f_{ij} in the random epistasis model are very small ($f_{ij}^2 \sim V_l 2^{-L}$), such that the stochastic contribution $(Nr)^{-1}$ dominates over the deterministic QLE predicition. Parameters: $L = 100$, $\sigma^2 = 0.005$ and $V_A = 0.1\sigma^2$. LD is measured when the allelic entropy decayed to 70% of its initial value. Data are averaged over 100 realizations for $N = 10^4$, 10^5 and over 10 realizations for $N = 10^6$.

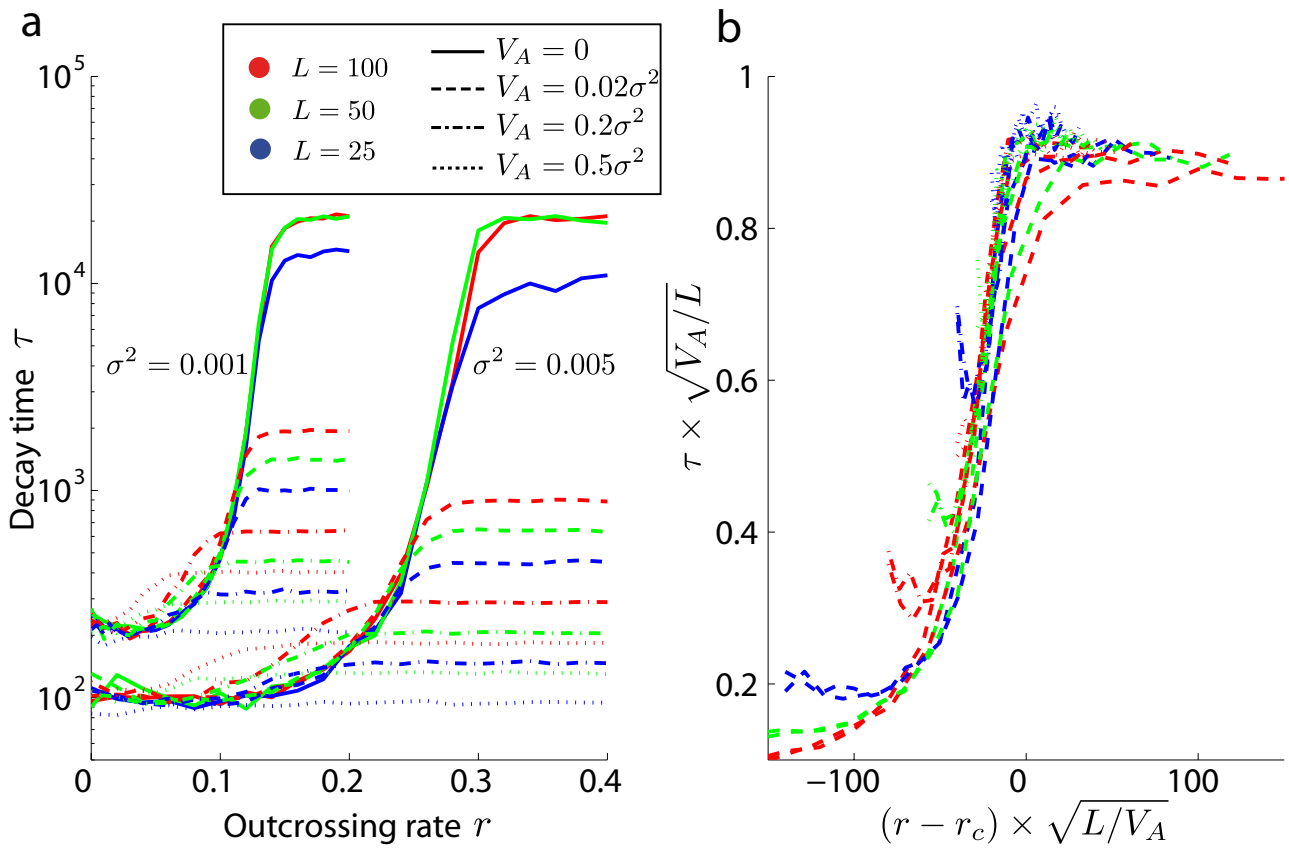

Fig. S2. Scaling collapse for the decay time τ of genetic variation for RE model. (*a*) Raw simulation data on τ, defined as the time after which genetic diversity decreased by 30%. (*b*) Scaling collapse of the data from *a* (except $V_A = 0$). The scaling collapse is achieved by multiplying τ with the typical fitness differential of a single locus $\sqrt{V_A/L}$. After this rescaling, all curves fall on top of each other for large outcrossing rates, which demonstrates that τ is entirely determined by the single locus effects. A subsequent shift of the curves by $r_c = 4\sqrt{V_I}$ and multiplication of the *x* axis by $\sqrt{L/V_A}$ results in an almost complete collapse of the data onto a single master curve. This data collapse demonstrates that the transition becomes abrupt as $L \to \infty$. Furthermore, it confirms the dependence r_c in V_I. The constant of proportionality between r_c and $\sqrt{V_I}$ was determined to achieve the best collapse and depends weakly on the population size, see *SI Appendix*, *The Critical Recombination Rate*. While the data for $V_A = 0$ cannot be included into the scaling collapse, it demonstrates how the finite size effects set the time scale τ in absence of additive fitness contributions for $r > r_c$. On one hand, τ is affected by drift due to random sampling of genotypes, the strength of which is inversely proportional to *N*. Drift is the predominant contribution for $L = 50$, 100, for which τ settles to a value on the order of the population size $N = 100{,}000$, which is independent of σ or *L*. For smaller *L* another finite size effect due to inhomogeneities of the fitness function reduces τ compared to its value for $L = 50{,}100$.

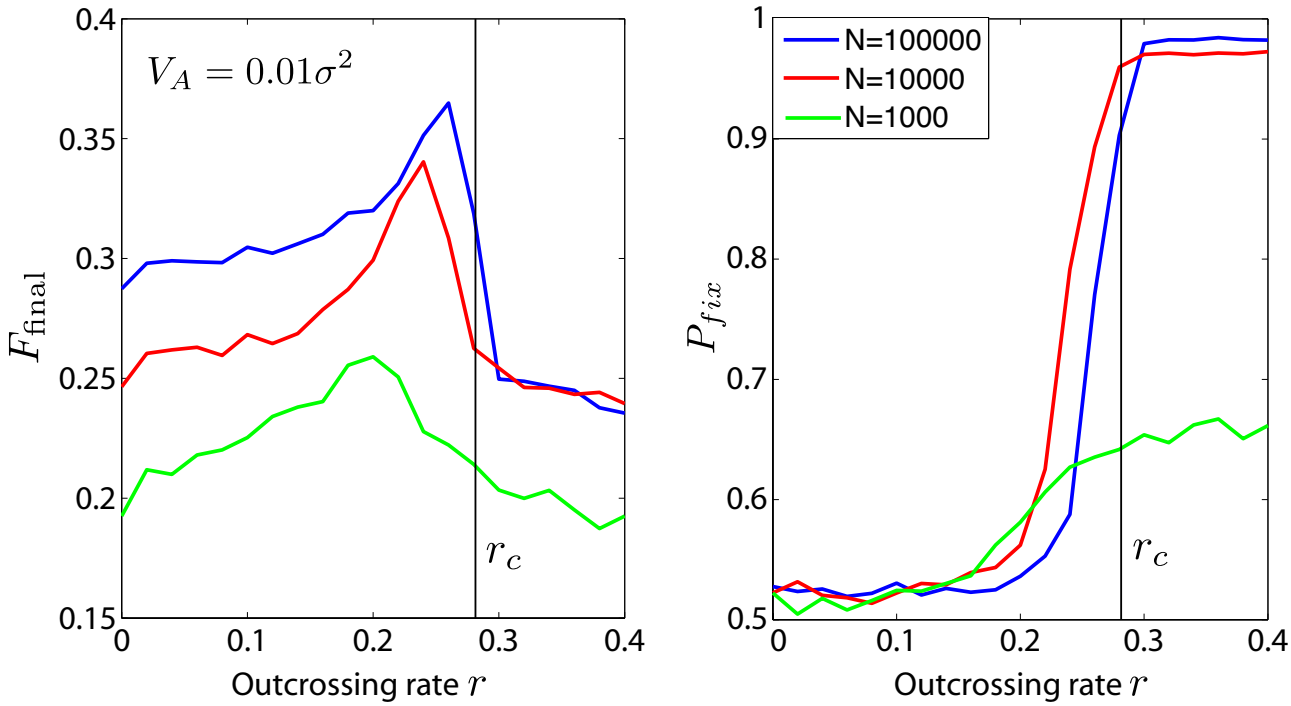

Fig. S3. The fitness of the fixated genotype F_{final} for $V_A = 0.01\sigma^2$ for RE model. (*a*) Some of the data of Fig. 3 and the corresponding curve for the small population size $N = 1{,}000$. The additive effect per locus is $\sqrt{V_A/L} \approx 0.0007$ and hence comparable to N^{-1} for the small population. This results in the markedly smaller fitness for $r > r_c$ compared to larger N. The position of the peak seems to approach r_c from below as N increases, while the drop across r_c becomes steeper. More data are required to decide whether F_{final} is discontinuous at $r = r_c$ in the limit $L \to \infty$, $N \to \infty$. (*b*) The probability P_{fix} that the allele with the favorable single locus effect is fixated. For large N and $r > r_c$, almost all loci fixate the advantageous allele (P_{fix} is close to 1). For $N = 1{,}000$, a large fraction of the loci fixates the disadvantageous allele, in accord with the above reasoning. Well below r_c, P_{fix} deviates only slightly from 0.5 for any N, confirming that selection acts on genotypes rather than alleles. Again, the slope of P_{fix} in the transition zone is increasing with N.

Other Supporting Information Files

SI Appendix